Second Edition

Advanced Monitoring and Procedures for Small Animal Emergency and Critical Care

Edited By

Jamie M. Burkitt Creedon

Harold Davis



with website

WILEY Blackwell

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Edited by

Jamie M. Burkitt Creedon
School of Veterinary Medicine, University of California, Davis
California, USA

Harold Davis
Retired, University of California
Clinical Educational Veterinary Consultant
California, USA

WILEY Blackwell

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List of Contributors

Damion Asselin RVT, VTS (ECC)
University of Georgia College of Veterinary Medicine, Athens, GA, USA

Amanda Arrowood, BS, CVT/VTS (ECC)
Matthew J. Ryan Veterinary Hospital, University of Pennsylvania, Philadelphia, PA, USA

James O. Barclay LATG
Department of Defense Military Working Dog Veterinary Services, Joint Base San Antonio-Lackland, TX, USA

Linda S. Barter, MVSc, BSc (Vet), PhD, DACVA
University of California, Davis, CA, USA

Karen L. Basher LVT, VTS (Anesthesia and Analgesia)
Anesthesia Department, Cornell University Hospital for Animals, Ithaca, NY, USA

Rachel Bassett, AAS, CVT, VTS (Anesthesia & Analgesia)
Animal Emergency and Referral Center of Minnesota, Oakdale, MN, USA

Søren Boysen, DVM, DACVECC
Faculty of Veterinary Medicine, University of Calgary, Calgary, Alberta, Canada

Benjamin M. Brainard, VMD, DACVA, DACVECC
Department of Small Animal Medicine and Surgery, College of Veterinary Medicine, University of Georgia, Athens, GA, USA

Yekaterina Buriko, DVM, DACVECC
Veterinary Hospital of the University of Pennsylvania, Philadelphia, PA, USA

Jamie M. Burkitt Creedon, DVM, DACVECC
Department of Surgical and Radiological Sciences, School of Veterinary Medicine, University of California, Davis, CA, USA

Andrew G. Burton BVSc (Hons), DACVP
IDEXX Laboratories, North Grafton, MA, USA

Christopher G. Byers, DVM, DACVECC, DACVIM (SAIM), CVJ
Teleconsultant, VetCT, Inc., Omaha, NE, USA

Janice Cain DVM, DACVIM (SAIM)
School of Veterinary Medicine, University of California, Davis, CA, USA

Alessia Cenani DrMedVet, MS, DACVAA
University of California, Davis, CA, USA

Daniel L. Chan, DVM, DACVECC, DECVECC, DACVIM (Nutrition), FHEA, MRCVS
Department of Clinical Science and Services, Royal Veterinary College, University of London, North Mymms, Hertfordshire, United Kingdom

Angela Chapman, BSc(Hons), RVN, DipAVN, VTS(ECC), MMgt
Department of Animal, Plant and Soil Sciences, La Trobe University, Melbourne, Victoria, Australia

Rosalind S. Chow, VMD, DACVECC
Department of Veterinary Clinical Sciences, College of Veterinary Medicine, University of Minnesota, St. Paul, MN, USA

Edward Cooper, VMD, MS, DACVECC
Department of Veterinary Clinical Sciences, Ohio State University, Columbus, OH, USA

Stacey Cooper, RVT, VTS (ECC)
Small Animal Emergency and Critical Care, Veterinary Medical Center, Ohio State University, Columbus, OH, USA

Katherine R. Crosse, MA, VetMB, MANZCVS, DipECVS
School of Veterinary Science, Massey University, Palmerston North, New Zealand

Elizabeth B. Davidow DVM, DACVECC
Timberline Veterinary Emergency and Specialty, Seattle, WA, USA

Autumn Davidson DVM, MS, DACVIM
School of Veterinary Medicine, University of California, Davis, CA 95616, USA

Harold Davis, BA, RVT, VTS (ECC) (Anesth & Analgesia)
Retired, University of California
Clinical Educational Veterinary Consultant
California, USA

Jasmine De Stefano
Woodhull Medical and Mental Health Center, New York, NY, USA

Lindsey Dodd, BSc(Hons), VPAC, VTS(ECC), PgCert in HE, FHEA, RVN
Lumbry Park Veterinary Specialists, Alton, Hampshire, UK

Charlotte E. Donohoe RVT, VTS (ECC), CCRP
Intensive Care Unit, University of Guelph, Guelph, Ontario, Canada

H. Edward Durham Jr., CVT, RVT, LATG, VTS (Cardiology)
Southwest Florida Veterinary Specialists, Bonita Springs, FL, USA

Steven E. Epstein DVM, DACVECC
Department of Surgical and Radiological Sciences, School of Veterinary Medicine, University of California, Davis, CA, USA

Julie Eveland-Baker, RVT, VTS(ECC)
William R. Pritchard Veterinary Medical Teaching Hospital University of California, Davis, CA, USA

Kate Farrell, DVM, DACVECC
Department of Surgical and Radiological Sciences, School of Veterinary Medicine, University of California, Davis, Davis, CA, USA

Sharon Fornes, RVT, VTS (Anesthesia)
VCA Hospitals, California, USA

Rufus W. Frederick LVT
Department of Defense Military Working Dog Veterinary Services, Joint Base San Antonio- Lackland, TX, USA

Sarah Fritz, BA, LVT, RVT

James Mack Fudge, DVM, MPVM, DACVECC
Hill Country Animal League, Boerne, TX, USA

Justina Gerard, MBA, RRT
IngMar Medical, Pittsburgh, PA, USA

Sarah Gray, DVM, DACVECC
Horizon Veterinary Specialists, Ventura, CA, USA

Rebecca J. Greer, DVM, MS, DACVECC
Veterinary Specialty Services, Manchester, MO, USA

Anna Grimes, BS, RVT, VTS (Cardiology)
Wilmington, NC, USA

Julien Guillaumin DVM, DACVECC, DECVECC
Emergency and Critical Care Services, Colorado State University, Fort Collins, CO, USA

Simon P. Hagley, BVSc, DipACVEC, DipECVECC
Vets Now Manchester Referral Hospital, Whitefield, Manchester, United Kingdom

Sarah Haldane, BVSc, MVSc, MANZCVS, DACVECC
Melbourne, Australia

Eric Hilton, BSc, RVT, CVT, VTS(ECC)
Veterinary Medicine Management Group, Philadelphia, PA, USA

Marie K. Holowaychuk, DVM, DACVECC
Reviving Veterinary Medicine, Calgary, Canada

Kate Hopper, BVSc, PhD, DACVECC
Dept of Veterinary Surgical and Radiological Sciences, School of Veterinary Medicine, University of California, Davis, CA, USA

Guillaume L. Hoareau DVM, PhD, DACVECC, DECVECC
Department of Emergency Medicine, University of Utah Health, Salt Lake City, UT, USA

Karl E. Jandrey, DVM, MAS, DACVECC
School of Veterinary Medicine, University of California, Davis, CA, USA

Samantha Jones RVT
Lead Technician, Internal Medicine Service
Veterinary Medical Teaching Hospital
University of California, Davis
Davis, California

Michael S. Kent, DVM, DACVIM (Onc), DACVR (RO), ECVDI (RO-add on)
Department of Surgical and Radiological Sciences, School of Veterinary Medicine, University of California, Davis, CA, USA

Carmen King, LVT, VTS (ECC)
Montreal, Quebec, Canada

Kristine Kofron LVT
Veterinary Teaching Hospital, Colorado State University, Fort Collins, CO, USA

Casey J. Kohen, DVM, DACVECC
MarQueen Pet Emergency and Specialty Group, Roseville, CA, USA

Lucy Kopecny, BVSc (Hons), DACVIM (Small Animal Internal Medicine)
Small Animal Specialist Hospital, North Ryde, New South Wales, Australia

Mary Anna Labato, DVM, ACVIM
Tufts University, Cummings School of Veterinary Medicine, 200 Westboro Road, North Grafton, MA 01536, USA

Michael S. Lagutchik, DVM, MS, DACVECC
Department of Defense Military Working Dog Veterinary Services, Joint Base San Antonio-Lackland Air Force Base, TX, USA

Cathy Langston, DVM, DACVIM
Ohio State University Veterinary Clinical Sciences, Columbus, OH, USA

Jennifer A. Larsen, DVM, MS, PhD
Department of Molecular Biosciences, School of Veterinary Medicine, University of California, Davis, CA, USA

Joyce Lau Jie Ying BS, VTS (ECC)
The Animal Hospital at Murdoch University, Murdoch, WA, Australia

Tami Lind RVT, VTS (ECC)
College of Veterinary Medicine, Purdue University, West Lafayette, IN, USA

Liza Lindeman – van der Mei RVT, VTS (ECC)
Department of Clinical Sciences, Utrecht University, the Netherlands

Alison Liu, DVM
American Society for the Prevention of Cruelty to Animals, New York, NY, USA

Bridget M. Lyons, VMD, DACVECC
Emergency and Critical Care, Cornell University Veterinary Specialists, Stamford, CT, USA

Valerie Madden, DVM, DACVECC
VCA Canada Western Veterinary Specialists and Emergency Centre, Calgary, Alberta, Canada

F. A. (Tony) Mann, DVM, MS, DACVS, DACVECC
Veterinary Health Center, University of Missouri, Columbia, MO, USA

Manuel Martin-Flores MV, DACVAA
Department of Clinical Sciences, College of Veterinary Medicine, Cornell University, Ithaca, NY, USA

Elisa M. Mazzaferro, MS, DVM, PhD, DACVECC
Cornell University Veterinary Specialists, Stamford, CT, USA

Margo Mehl, DVM, DACVS
San Francisco Animal Medical Center, San Francisco, CA, USA

Martin D. Miller
Lower Burrell, PA, USA

Sarah Musulin, DVM, DACVECC
North Carolina State University, Raleigh, NC, USA

Sean Naylor
Hemodialysis and Blood Purification Unit, Veterinary Medical Teaching Hospital, University of California, Davis, CA, USA

Alexandra Nectoux, DVM, MSc, DECVECC
SIAMU, VetAgro Sup Campus vétérinaire de Lyon, Marcy l'Etoile, France

Christopher L. Norkus, DVM, DACVAA, CVPP, DACVECC
Bloomfield, Connecticut, USA

Adesola Odunayo DVM, MS, DACVECC
Department of Small Animal and Clinical Sciences, College of Veterinary Medicine, University of Florida, Gainesville, FL, USA

Jessica L. Oram RVT
Ontario Veterinary College, Health Sciences Centre, Guelph, Ontario, Canada

Laura Osborne, BVSc (Hon I), DACVECC
Western Veterinary Specialist and Emergency Centre, Calgary, Alberta, Canada

Sally C. Perea, DVM, MS, DACVIM (Nutrition)
Royal Canin PHNC, Lewisburg, OH, USA

Jessica Perlini RVT, VTS (Anesth and Analgesia)
Veterinary Teaching Hospital, University of Georgia, Athens, GA, USA

Helen S. Philp, BVMS, DACVECC
Veterinary Medical Teaching Hospital, University of California, Davis, CA, USA

Karen Poeppel, BS, LVT
Schwarzman Animal Medical Center, New York, NY, USA

Jennifer E. Prittie, DVM, DACVIM, DACVECC
Department of Emergency and Critical Care, AMC Schwartzman Animal Medical Center, New York, NY, USA

Jocelyn Pronko BS, CVT, VTS (ECC)
Veterinary Teaching Hospital, Colorado State University for Critical Care Services, Drake Fort Collins, CO, USA

Jane Quandt, DVM, MS, DACVA, DACVECC
University of Georgia College of Veterinary Medicine, Athens, GA, USA

Marc R. Raffe, DVM, MS, DACVAA, DACVECC, eMBA
Veterinary Anesthesia and Critical Care Associates LLC, St. Paul, MN, USA

Louisa J. Rahilly, DVM, DACVECC
Cape Cod Veterinary Specialists, Buzzards Bay and Cape Dennis, MA, USA

Krystle Reagan, DVM, PhD, DACVIM (SAIM)
Veterinary Medical Teaching Hospital, University of California, Davis, CA, USA

Erica L. Reineke, VMD, DACVECC
Advanced Medicine, School of Veterinary Medicine, University of Pennsylvania, Philadelphia, PA, USA

Angel Rivera, CVT, VTS (ECC)
Milwaukee, WI, USA

Nicholas Rivituso, CVT, VTS (ECC)
Steel Center for Career and Technical Education, Jefferson Hills, PA, USA

Joris H. Robben. PhD, DECVIM, CA
Section of Emergency and Intensive Care Medicine, Department of Clinical Sciences, Utrecht University, Utrecht, Netherlands

Elizabeth Rozanski, DVM, DACVECC, DACVIM (SA-IM)
Cummings School of Veterinary Medicine, Tufts University, North Grafton, MA, USA

Elke Rudloff, DVM, DACVECC, cVMA
BluePearl Pet Hospice, Milwaukee, WI, USA

Michael D. Santasieri BS, CVT, LVT, FFCP
Cummings School of Veterinary Medicine, Tufts University, North Grafton, MA, USA

Nicole Saunders
American Animal Hospital, Randolph, NJ, USA

Jessica J. Schavone, BS, LVT, VTS (ECC)
Department of Clinical Sciences, Cummings School of Veterinary Medicine, Tufts University, North Grafton, MA, USA

Kristen Sears, RVT
School of Veterinary Medicine, University of California, Davis, CA, USA

Deborah Silverstein, DVM, DACVECC
Department of Clinical Sciences and Advanced Medicine, University of Pennsylvania, Philadelphia, PA, USA

Sean D. Smarick, VMD, DACVECC
North Huntingdon, PA, USA

Lisa Smart, BVSc (Hons), PhD, DACVECC
Small Animal Specialist Hospital, Tuggerah, NSW, Australia

Andrea M. Steele MSc, RVT, VTS (ECC)
Ontario Veterinary College, Health Sciences Centre, Guelph, Ontario, Canada

Deborah A. Stone, MBA, PhD, CVPM
Ausitn, TX, USA

Lindsey Strang, RVT, VTS (ECC)
Cardiology Service, VCA Western Veterinary Specialist and ER Centre, Calgary, Alberta, Canada

April Summers, DVM, PhD, DACVECC
Blue Pearl Pet Hospital, Maitland, FL, USA

Jane E. Sykes, BVSc(Hons), PhD, MBA, DipACVIM (SAIM)
Department of Medicine and Epidemiology, University of California, Davis, CA, USA

Wan Khoon Avalene Tan, BVSc, DAVECC
Veterinary Teaching Hospital, School of Veterinary Science, Massey University, Palmerston North, New Zealand

Carolyn Tai CVT, VTS (ECC) (SAIM)
Tufts University, Cummings School of Veterinary Medicine, North Grafton, MA, USA

Katherine Vachon, CVT
Cape Cod Veterinary Specialists, Buzzards Bay and Cape Dennis, MA, USA

Chiara Valtolina, DVM, DACVECC
Department of Clinical Science of Small Animals, Utrecht University, the Netherlands

Nicole Van Sant LVT, VTS (ECC)
Cornell University Veterinary Specialists, Stamford, CT, USA

Lori S. Waddell, DVM, DACVECC
Matthew J. Ryan Veterinary Hospital, University of Pennsylvania, Philadelphia, PA, USA
Department of Clinical Sciences and Advanced Medicine, Matthew J. Ryan Veterinary Hospital, University of Pennsylvania, Philadelphia, PA, USA

Courtney Waxman CVT, RVT, VTS (ECC)
Veterinary Nurse Consulting, LLC, Green Cove Springs, FL, USA

Janelle R. Wierenga, DVM, Dipl. ACVECC, MPH, PhD
School of Veterinary Science, Massey University, Palmerston North, New Zealand

Kenichiro Yagi, MS, RVT, VTS (ECC), (SAIM)
Veterinary Emergency Group, White Plains, NY, USA

Preface to the Second Edition

The discipline of small animal emergency and critical care medicine continues to advance and evolve. It has been 11 years since publication of the first edition of our textbook, and we believe that this new edition will help update our community on the monitoring and procedural aspects of care. Our focus continues to be the core, daily hands-on practice of the specialty. This edition features returning and new authors updating previous chapters, and returning and new authors providing additional chapters. We are excited about these additional chapters – among others, they include a comprehensive review of point-of-care ultrasound, an extensive discussion on nursing care of neonates, and the unique and important considerations for handling suspected cruelty cases. We continue to believe the veterinary community benefits from a single reference written by informed, experienced people to improve and expand the standard of care, and we hope this textbook continues to serve that purpose as the first edition did.

Emergency and critical care practice is a team sport that requires cooperation among all its members. Thus, some chapters are authored by a veterinarian, others by a veterinary technician, and some by pairs or groups. The interdependence of all members of the ECC healthcare team requires that veterinary technicians understand why clinicians do what they do, and that veterinarians understand proper ECC nursing care and technical procedures. The collective knowledge and skills of the team fosters a proactive rather than reactive approach to each shift's challenges. The book's contributors come from around the world, from both university and private practice. We aimed to provide the best-referenced, highest-quality textbook that we could. Contributors congenially answered our frequent "Do you have a reference for this?" inquiries and high-quality image requests, so that the reader could have confidence in the recommendations contained herein and see illustrations of how to perform procedures or interpret results. When high-quality references or guidelines were unavailable, these qualified authors made recommendations based on their experience; in such cases, such personal recommendation is noted in the text for transparency.

The textbook is organized roughly by organ system or general topic, but there is considerable overlap in some areas. For instance, some authors of device insertion chapters included a maintenance section, and maintenance of that device may also be covered in another chapter specifically on insertion site maintenance, and so on. Standardized protocols are included for procedures for which they were deemed useful. These protocols are based on best-available evidence and guidelines, and where such citations were unavailable, they are based on author experience. We hope that these protocols will continue to help raise and equalize the standard of care across our profession and serve as the backbone for a protocol book to use in your practice.

We welcome corrections and ideas for future versions of this textbook. Should further editions follow, we are committed to their currency and relevancy, and thus will continue to push for best-practice, evidence- and guideline-based recommendations. We are grateful to the previous contributors, some of whom are now deceased, as their contributions often served as the framework for chapters included here. Finally, we would like to thank each current contributor; they did an amazing job stepping up to the challenges that this unique textbook posed in this unprecedented time in our world and our industry.

Jamie M. Burkitt Creedon
Harold Davis

Acknowledgments

To all the lifelong learners in our profession doing this job out there, thank you for your continual efforts to grow and improve for the sake of our teams and our patients.

To the generous, patient, kind people I am so fortunate to call my friends, you make my days better and I am grateful for you.

And most of all to my family, who remind me every day of what is most important – you are my sunshine and I love you.

"We have two lives, and the second begins when we realize we only have one." – *Confucius*

May you all be on Life Two. *Jamie*

Thank you to all the veterinary health care professionals for all you do to care for our patients, I trust this text will aid you in that endeavor.

To my friends and colleagues, I appreciate your friendship, support, and the fact that you challenge me to be better every day.

To my parents, sister, and extended family this is for you; your love, support and guidance have made this possible and all worthwhile. *Harold*

About the Companion Website

This book is accompanied by a companion website.

www.wiley.com/go/burkitt/monitoring 

This website includes:

- Figures from the book available to download in Microsoft PowerPoint
- Videos
- Protocols from the book

Section One

Fundamental Elements of Emergency and Critical Care Practice

1

Triage

Harold Davis

The word "triage" comes from the French verb trier, meaning to sort. The concept of triage finds its origin in the military, and the goals of triage in human medicine have varied over the years depending upon the situation. After World War II triage came to mean the process of identifying those soldiers most likely to return to battle after medical care. During the Korean and Vietnam conflicts the goals of triage came to mean the greatest good for the greatest number of wounded [1]. In times of disaster, the goals of triage are like those of the military: to concentrate effort and resources on saving the largest number of people possible. Daily human emergency room triage began in the 1960s and has evolved into a method to separate efficiently those patients stable enough to wait for treatment from those who require immediate medical attention. In veterinary medicine we have adopted the goals of our counterparts in the human emergency room. Thus, we prioritize cases by medical urgency when presented with multiple emergencies at the same time.

Triage occurs both by telephone and in the hospital. A client often calls the hospital seeking advice for the care of their pet; the receptionist or veterinary technician must ascertain useful information about the pet in a short period of time. Thus, the receptionist or technician should have the knowledge required to provide appropriate advice. The information obtained during the telephone conversation will also be useful in preparing for patient arrival. On initial presentation to the hospital the veterinary technician is usually first to receive the patient and therefore to perform basic triage. This person must determine whether the patient needs immediate care and, in the case of simultaneous patient arrivals, prioritize treatment based on medical need.

Telephone Triage

In theory, telephone triage requires clinic staff to determine the urgency of a pet's problem and to provide advice based on that determination. However, because the client may not possess the training to give an accurate account of the pet's problem(s), it is generally safest to recommend that the client take the pet to a veterinarian for evaluation. Particularly any patient experiencing breathing difficulty, seizures, inability or unwillingness to rise, or traumatic injury should be seen by a veterinarian without question.

At the beginning of the telephone conversation, staff should establish the animal's signalment (breed, sex, age, and approximate weight) if possible. Questions asked of the owner should be basic and straightforward using lay terminology. Questions should address the patient's level of consciousness (LOC), whether the patient is breathing easily or with difficulty, has abnormal mucous membrane color, experiencing seizures, has obviously broken or exposed bones, or has any pre-existing medical conditions (Box 1.1). Based on the owner's responses, advice can be given on first aid, assuming that the problem can be clearly defined and is simple. See Box 1.2 for a list of problems requiring immediate attention by the veterinary healthcare team.

Information gathered during the phone conversation can aid the veterinary technician in preparation for the arrival of the patient at the hospital. Knowing the animal's breed or approximate weight allows the technician to pre-select appropriate sizes for vascular catheters, fluid bags, and endotracheal tubes.

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Box 1.1 Questions Useful in Telephone Triage, and Suggested Responses

1) Is the animal breathing and conscious?
   A) If neither, institute chest compressions and mouth-to-snout; if yes to either of these, do not.
2) Is the animal having difficulty breathing?
   A) If yes, take immediately to a veterinarian.
3) What color are the mucous membranes (gums)? Do they appear their usual color?
   A) If no, what color is noted?
4) Is the animal actively experiencing a seizure?
   A) If yes, remove from danger of falling, bodies of water, or sharp objects. Take to veterinarian immediately after seizure ends, or if it lasts longer than 1–2 minutes, bring during seizure. Instruct owners to stay clear of the animal's mouth to avoid accidental bite wounds.
5) Has the animal ingested something that may be poisonous within the last two hours?
   A) If yes, take immediately to a veterinarian. In some situations, if the client cannot or will not take the pet immediately to a veterinarian, at-home emesis may be recommended.
6) Is there active bleeding, an obvious fracture, or exposed bone?
   A) Recommend clean towel over the site, pressure if spurting blood. Warn clients to be VERY CAREFUL to avoid being bitten.
7) Does the animal have any ongoing medical problems and or is it on any medications (including over the counter)?
   A) Briefly restate your understanding of the problem(s). If on medications and coming into the hospital, instruct the owner to bring all medications.

Box 1.2 Problems Requiring Immediate Attention by the veterinary healthcare team

- Cardiopulmonary arrest (unconscious and making no regular attempts to breathe)
- Excessive bleeding
- Respiratory distress
- Weakness
- Pale mucous membranes
- Rapid abdominal distension
- Neurological abnormalities
- Inability or persistent straining to urinate
- Protracted vomiting
- Ingestion or topical exposure to toxins
- Burns
- Snake envenomation
- Perforation, wound dehiscence, or open body cavities
- Open fractures
- Prolapsed organs
- Dystocia

Owners should be instructed on safe transport of the animal. Animals that have suffered trauma are often in pain, and owners should be coached on how to approach the pet and place a makeshift muzzle using a necktie, belt, or strips of cloth. If the animal is nonambulatory, owners may be told to place the animal in a box or carrier, or to use a blanket or towel as a stretcher (Figure 1.1). The use of a blanket stretcher makes it easier to get an animal in and out of a car. If the animal is a cat, it should be brought in a cat carrier or box (with holes).

When the caller is not a regular client of the facility, the staff member should obtain the client's phone number early in the conversation in case of disconnection and make the caller aware of the address, location, or easiest directions to the clinic. The client should be informed of the clinic's payment policy.

Finally, the telephone conversation should be documented, giving a complete summary of what transpired. Logs are saved for whatever period is dictated by the regulating body. Telephone logs serve as an extension of the legal medical record.

Hospital Triage

Three major body systems are assessed during the initial triage: respiratory, cardiovascular, and neurological. Triage begins when approaching the patient. Visually assess breathing effort and pattern; presence of blood or other foreign material on or around the patient; and the patient's posture and LOC. Note if there are airway sounds audible without a stethoscope. Note whether the animal responds as you approach. If the animal is conscious, ask the owner about the patient's temperament and take the appropriate precautions regarding physical restraint or muzzling. The veterinary technician cannot rely on the client's statement that an animal "never bites," but if the client states that the patient is aggressive, the patient should be muzzled. Physical restraint and muzzling should be performed with extreme caution in patients with respiratory distress, as such steps can cause acute decompensation and respiratory arrest. If time permits, a brief history should be obtained.



Figure 1.1 (a) Placing a dog in a box for transport. (b) Using a blanket as a stretcher. The animal is placed on a blanket and the edges of the blanket used to lift the patient.

The ABCDEs

A reasonable and systematic approach to triage is the use of the ABCDEs of emergency care, which are: (A) airway, (B) breathing, (C) circulation, (D) dysfunction of the central nervous system, and (E) exposure/examination (Figure 1.2). Patients with respiratory distress or arrest, signs of hypovolemic shock or cardiac arrest, altered LOC, or ongoing seizure activity should be immediately taken to the treatment area for rapid medical attention. Conditions that affect other body systems are generally not life-threatening in and of themselves, but their effects on the three major body systems may be life-threatening. For example, a fractured femur bleeding into a limb can lead to life-threatening hypovolemia.

Airway and Breathing

Expedient respiratory system assessment and rapid correction of abnormalities are critical. First, patency of airway and breathing effort should be assessed. This is done by visualization, auscultation, and palpation. When looking at the animal, an experienced individual can determine whether the animal has increased breathing rate or effort. Some animals with respiratory distress may assume a posture with the head and neck extended and the elbows abducted (held away from the body). Additional concerning signs include absent chest wall motion, exaggerated breathing effort, flaring of the nares, and open mouth breathing in cats. A "paradoxical" breathing pattern can occur when sustained high breathing effort leads to respiratory fatigue or during upper airway obstruction; paradoxical breathing is characterized by opposing movements of the chest and abdominal walls during inspiration and expiration. Cyanosis, a blue or purplish tint to the mucous membranes, usually indicates hypoxemia and warrants immediate medical intervention. The chest wall may be palpated to assess chest wall integrity. Crepitus about the body may indicate subcutaneous emphysema, which can be caused by tracheal tears or chest wall defects.

Assessment questions the triage technician should consider include:

- Is the patient having difficulty breathing?
- Are breath sounds audible?
- Are facial injuries interfering with the airway?
- Has a bite wound disrupted the larynx or trachea?
- Is subcutaneous emphysema present?
- What color are the mucous membranes?
- Does respiratory distress get worse with patient position change?
- Is there evidence of thoracic penetration or an unstable chest wall segment?

Circulation

Many of the signs suggestive of decreased cardiac output are a result of a compensatory sympathetic reflex, which helps maintain arterial blood pressure. Clinical signs suggestive of decreased cardiac output include tachycardia, pale or gray mucous membranes, prolonged capillary refill time, poor pulse quality, cool extremities, and decreased mentation. Decreased cardiac output may be due to hypovolemia from blood or other fluid loss (internally or externally; active or historical), trauma, or cardiac disease.

Circulation is assessed by visualization, palpation, and auscultation if using a stethoscope. The focus of the cardiovascular assessment is the six perfusion parameters (Box 1.3).

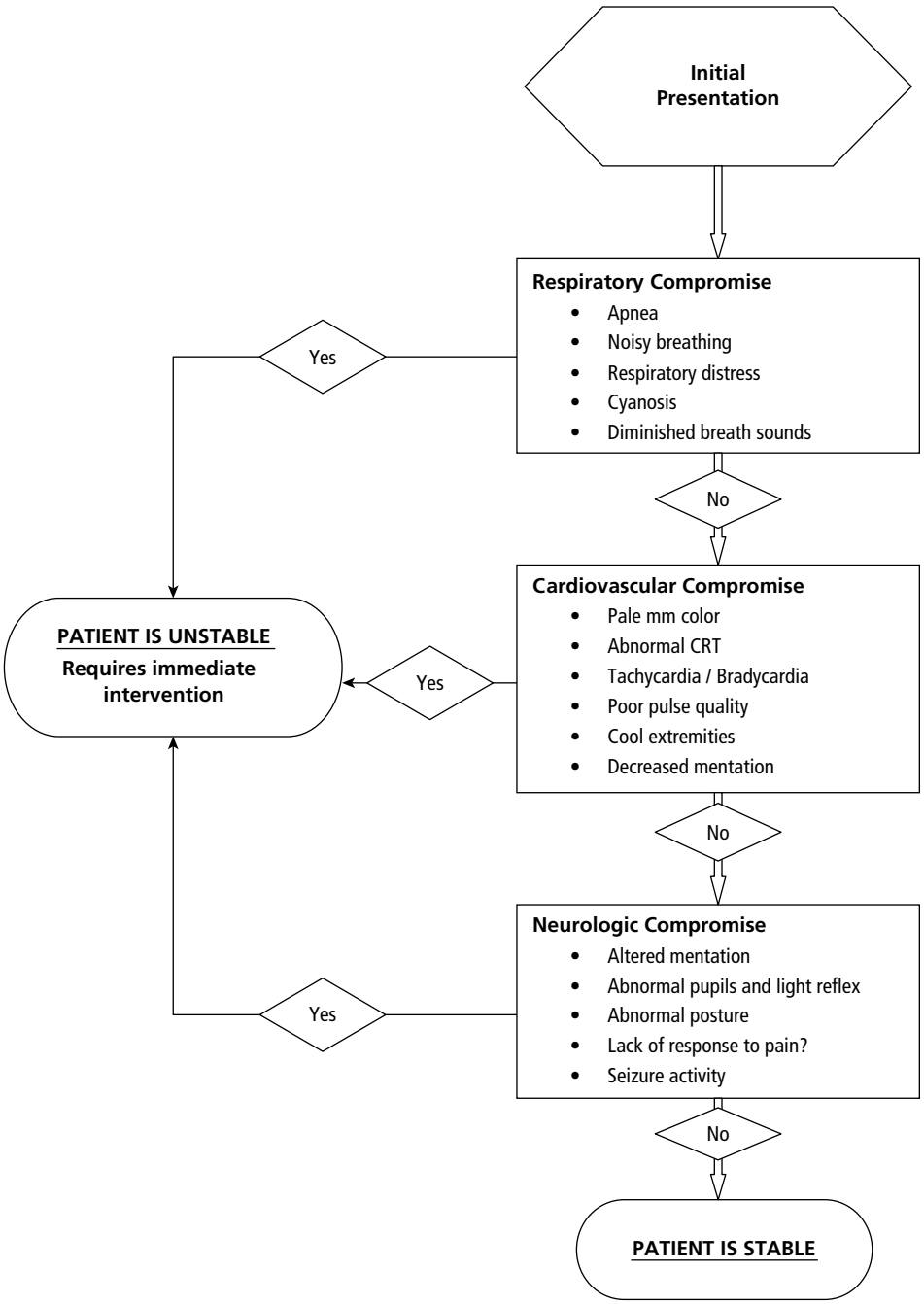


Figure 1.2 Triage algorithm. CRT, capillary refill time; mm, mucous membranes.

Box 1.3 The Six Perfusion Parameters

- Mentation
- Mucous membrane color
- Capillary refill time
- Heart rate
- Pulse quality
- Extremity temperature

Mentation

As previously mentioned, evaluation of mentation starts from afar. The patient's attitude is evaluated without stimulation. A reduced level of mentation is indicated by a loss of interest in the surrounding environment and diminished or absent responses to stimuli such as noise and touch. This can be described as obtundation or depression. As depression implies an assessment of the animal's emotional state, obtundation may be a more appropriate term.

If there is a loss of consciousness, mentation is either stuporous or comatose. Stupor refers to a patient that is unconscious and responsive only to noxious stimuli. Coma refers to a completely unconscious, non-responsive state. Most unconscious animals require intubation to protect their airway.

An altered level of mentation can be the result of primary intracranial disease or significant systemic abnormalities such as hypoperfusion or hypoglycemia. Any abnormality in mentation should be considered serious and a complete triage examination is warranted immediately.

Mucous Membrane Color

After assuring it is safe to do so, evaluate the mucous membranes by examining the color of the gums (Figure 1.3). As an alternative in the fractious animal or patients with pigmented gums, one may examine the conjunctiva, penis, or the vulva. The normal pink color is a result of oxygenated hemoglobin in red blood cells in the capillary bed. Mucous membrane color may vary with circulation-related problems. Pale or white mucous membranes are a consequence of a reduced quantity of red blood cells perfusing the capillary beds of the mucosal tissue. This can be the result of vasoconstriction in compensation to circulatory shock or severe anemia. This abnormality in combination with other evidence of poor perfusion or inadequate tissue oxygenation warrants emergency intervention. Vasodilation increases the flow of blood through the mucous membranes making them a deep pink to red color. Vasodilation may be an appropriate response as seen in a hyperthermic animal following exercise, or it can be pathological as seen in vasodilatory shock. Patients with vasodilatory shock commonly present with concurrent hypovolemia and so on presentation may have pale mucous membranes that turn dark pink or red after adequate fluid resuscitation.



Figure 1.3 Assessing a patient's mucous membrane color.

Cyanotic or blue mucous membranes are an indicator of severe hypoxemia. The absence of cyanosis does not rule out hypoxemia. Icteric or yellow mucous membranes are due to the breakdown of red cells (hemolysis) or hepatobiliary disease. Methemoglobinemia results in brown or chocolate-colored mucous membranes.

Capillary Refill Time

Capillary refill time (CRT), the time it takes for mucous membranes to regain their color following blanching by digital pressure, reflects degree of local blood flow. When perfusion is normal, CRT is one or two seconds. Vasoconstriction reduces the flow of blood through the mucous membranes via arteriolar and precapillary sphincter contraction and it will take longer for the color to return to the tissue after blanching. In severe vasoconstriction the mucous membranes can appear white, and it can be impossible to appreciate any CRT. In patients with vasodilation the CRT can be more rapid than normal as there is less resistance to blood flow and the capillary beds rapidly refill with blood after the digital pressure is removed. A slow CRT is always a concern and suggests poor perfusion. A rapid CRT in conjunction with other perfusion abnormalities can suggest vasodilatory shock.

A study was undertaken to evaluate the relationship between a standardized method of evaluating CRT and various clinical parameters in hospitalized dogs. The authors found that a CRT following blanching for four seconds may provide insight into the hydration status and hemodynamic stability of canine patients [2]. This study may also serve as a basis for establishing a standardized method for evaluating CRT (Box 1.4). It should be noted that a four-second pressing time was used to minimize fluctuations in pressure application time. The gingival mucosa was avoided due to the potential inflammatory changes associated with gingivitis, which may alter CRT. A stopwatch was used to ensure reliability. Avoid immediate (within two minutes) repeating of CRT measurement at the same location as refills may be falsely shortened on subsequent evaluations (presumably due to warming of the site from repeated contact and subsequent vasodilation).

Box 1.4 Standardized Method for Measuring or Assessing Capillary Refill Time

- Use inner lip oral mucosa taking care not to restrict blood flow when everting lip
- Moderate direct pressure is applied for 4 seconds
- Use a stopwatch to determine the time for return of color to the capillary bed
- Avoid immediate (within 2 minutes) measurement of the same site

Heart Rate

Heart rate is a nonspecific parameter. It is usually measured by auscultation of the heart, palpation of the cardiac apex beat, or palpation over an artery. The normal heart rate of a dog varies with body size. In general, large breed adult dogs have resting heart rates of 60–140 beats per minute (bpm), medium adult dogs 70–160 bpm, and small dogs and puppies 100–180 bpm. Normal feline heart rate at a veterinary hospital is usually 160–220 bpm. When arterial blood pressure is threatened either by a drop in stroke volume or because of vasodilation, a baroreceptor-mediated increase in sympathetic tone results in a reflex tachycardia. As tachycardia is a normal response to anxiety, excitement, and exercise, it is a common physical examination finding. The presence of tachycardia in conjunction with other signs of abnormal perfusion (e.g. abnormal mentation, mucous membrane color, CRT) suggests hemodynamic compromise.

Tachycardia is the appropriate and expected response to circulatory shock. The presence of normocardia or bradycardia in canine shock patients (i.e. patients with abnormalities in the other five parameters) is of concern as it suggests decompensated shock and may be associated with greater severity of illness and a poorer prognosis. Feline shock patients often present without tachycardia, and this is not considered to have the same prognostic relevance that it does in dogs.

Auscultable arrhythmias may or may not require immediate medical therapy. Femoral pulse evaluation should be performed simultaneously with auscultation for both time efficiency and recognition of pulse deficits. Arrhythmias without any other signs of poor perfusion are less concerning but these patients should always be prioritized for a secondary evaluation including an electrocardiogram (ECG), as the physical assessment of these abnormalities is limited.

Pulse Quality

Pulse quality is subjectively determined by the digital palpation of the femoral pulse. Obvious abnormalities in pulse quality are concerning, and if present in conjunction with other signs of poor perfusion, the patient should receive immediate medical attention. Unfortunately, patients can have considerable hemodynamic compromise without palpable changes in pulse quality. As a result, the palpation of an adequate femoral pulse cannot be used to indicate a stable patient.

The pulse quality is determined by the difference between diastolic and systolic arterial blood pressure, as well as the duration of the pulse and the size of the vessel. The greater the diastolic–systolic difference, the "stronger" the pulse will feel. For example, a normal arterial blood pressure would be a systolic blood pressure of 120 mmHg, a mean of 85 mmHg, and a diastolic of 70 mmHg. The systolic–diastolic pressure difference (pulse pressure) in this example is 50 mmHg. If there is a fall in blood pressure such that the patient is hypotensive with a systolic pressure of 90 mmHg, mean of 55 mmHg, and diastolic of 40 mmHg, the pulse pressure would still reflect a systolic–diastolic difference of 50 mmHg. The examiner may be challenged to detect any change in pulse quality in such case.

Vasoconstriction tends to diminish palpable pulse quality, leading to the "thready pulse." In contrast, vasodilation increases vessel size and compliance. In addition, vasodilated patients may have an increased stroke volume such that the pulse quality is often appreciated to be normal or exaggerated ("bounding") in these patients.

Studies have looked at the relationship between peripheral pulse palpation and Doppler systolic blood pressures in dogs and cats. In one dog study the authors concluded that absent metatarsal pulses are highly specific in the diagnosis of hypotension. However, dogs with palpable metatarsal pulses can still be hypotensive [3]. In a cat study it was concluded that peripheral pulse quality assessment by emergency room veterinarians correlates with systolic blood pressure. With progressive decreases in blood pressure, metatarsal pulses will disappear and it is only with severe hypotension that femoral pulses are absent [4]. In the case of peripheral pulse abnormities (weak or absent), the index of suspicion for hypotension is high. Conversely, the presence of palpable pulses does not rule out hypotension. Palpation of pulses does not replace the need for an actual blood pressure measurement. Other perfusion parameters should be taken into consideration when assessing peripheral pulses.

Extremity Temperature Compared with Core

The sympathetically mediated vasoconstriction that occurs in response to a fall in cardiac output tends to shunt blood from venous capacitance vessels to the central circulation, preserving blood flow to vital organs at the expense of less vital tissue. This reduction in peripheral circulation will cause a fall in extremity temperature in comparison with core body temperature. If the patient is generally hypothermic, cool extremities that are essentially the same temperature as the rest of the animal does not indicate an abnormality in perfusion. For this reason, extremity temperature (evaluated by manual palpation of the paws and distal limbs) should be interpreted with reference to the measured rectal temperature. Vasodilation is generally associated with warm extremities if the patient is fluid resuscitated.

There has been renewed interest in veterinary medicine to the measurement of toe web and rectal temperature difference in the assessment of perfusion. Its use in the veterinary patient was first described in the late 1970s. Toe web temperature had been shown to decrease far below body

(rectal) temperature during hemorrhagic shock and to return toward body temperature following fluid resuscitation [5]. In the more a recent study looking at rectal–interdigital temperature gradient (RITG) as a diagnostic marker of shock in dogs, RITG was determined by taking a rectal temperature (with a standard, sheathed, battery-operated rectal thermometer). The interdigital temperature was taken with the same thermometer between the third and fourth digit on a pelvic limb. The digits were manually pressed together. Based on the study results, a gradient of 8.5°F may be used as a screen for circulatory shock, and a cutoff of 11.6°F would indicate high suspicion for circulatory shock [6]. The cutoffs used were based on an ambient temperature of 74°F. While the use of RITG should not replace more traditional methods of assessing perfusion, it may serve as an additional tool for the assessment of circulatory shock.

Assessment questions the triage technician should consider include:

- Is there evidence of hemorrhage?
- Is there swelling associated with an extremity fracture?
- Are the mucous membranes pale or injected (deep red)?
- Is the capillary refill prolonged?
- Are the femoral pulses weak and rapid?
- Are the extremities cold/is there increased RITG?

Dysfunction or Disability of the Neurologic System

Dysfunction or disability refers to the neurologic status of the patient. This may be assessed through visualization and palpation. A cursory neurologic examination is performed focusing on the patient's LOC/mentation, pupils (size and response to light), posture, and response to pain (deep tested only if the patient lacks superficial pain perception). Depressed mentation may be a result of poor oxygen delivery or trauma to the brain. Seizure activity may be due to intra- or extracranial causes.

A patient with known or suspected trauma that is recumbent, has an abnormal posture, or is not seen to ambulate or make voluntary movements, should be assumed to have spinal trauma and stabilized on a backboard (Figure 1.4) until proven otherwise.

Assessment questions the triage technician should consider include:

- Is the animal bright, alert, and responsive or obtunded (depressed but rousable), stuporous (roused only with painful stimulation), or comatose?
- Are the pupils dilated, constricted, of equal size, and responsive to light?
- What is the posture of the animal?
- Are there any abnormal breathing patterns?
- Does the animal respond to painful stimuli?
- Is there obvious seizure activity?



Figure 1.4 A patient with suspected head and spinal trauma restrained on a backboard. The cranial end of the board is elevated slightly because of suspected increased intracranial pressure.

Exposure/Examination

In people, skin exposure is an essential aspect of patient evaluation since clothing can hide serious injuries or abnormalities. The same is true for animals, which require fuller examination once airway, breathing, circulation, and neurologic status are evaluated and stabilized. Once an animal is considered safe for movement, the removal of blankets or harnesses, and turning the lateral patient to examine its other side are important. Any source of ongoing harm is removed, such as by bathing the cat with topical permethrin application. Emesis may be induced if the animal recently ingested a toxin.

Finally, a rapid, whole-body examination is performed. The goal is to determine and address any additional problems.

Assessment questions the triage technician should consider include:

- Are there lacerations, wounds, or punctures?
- Is there bruising and is it getting worse?
- Are there palpable fractures?
- Is the abdomen apparently painful or distended?
- Is there evidence of debilitation or other signs of disease?

Body Temperature

Body temperature is not addressed in the standard ABCs and may not be required for the assessment of every patient. Extremes of body temperature are, however, an indication for urgent medical attention and as a result the temperature of at-risk patients should be measured during assessment.

Point-of-Care Ultrasound

Over the past two decades, bedside ultrasonography has become mainstream in the human emergency department [7]. It is an important skill that positively impacts

patient outcomes. Point-of-care ultrasound (POCUS; see Chapter 6) is also beneficial in the veterinary emergency room [8], including during triage [9]. Abdominal point-of-care ultrasound is used in the detection of free fluid in the abdomen (see Chapter 39). There are reports in the veterinary literature that the size of the caudal vena cava can be assessed via ultrasound and aid in diagnosis of hypovolemia in dogs [10]. Thoracic point-of-care ultrasound is used to assess the pleural cavity, looking for free fluid in the pleural and pericardial spaces (see Chapter 17) and for signs consistent with a pneumothorax [11] (see Chapter 27).

In 2007, it was suggested that the training for human emergency and critical care professionals in ultrasound should use the ABCDE and head-to-toe approach. Critical care problems are approached primarily according to the ABCDE or the head-to-toe sequence based on physiological priority. The idea was that introductory ultrasound training should always follow the same pathways and priorities, thus addressing findings in the real order of importance [12]. This POCUS approach or protocol is known as FAST-ABCDE (FAST including airway-breathing-circulation-disabilities/deficits and exposure). FAST-ABCDE is more comprehensive in evaluating patients for life-threating problems (Figure 1.5). The human ABCDE protocol has been adapted for the veterinary patient and studied in dogs [13]; it is called VetFAST ABCDE. The VetFAST ABCDE identifies problems related to airway, breathing, circulation, disability, and exposure (Figure 1.6). The study found that the VetFAST ABCDE protocol in dogs suffering from trauma is feasible, can detect cavitary effusion and pneumothorax with a higher diagnostic accuracy than radiographs, and can detect lesions outside the scope of traditional veterinary ultrasound exams [13].

Airway:
- Airway patency
- Laryngeal/tracheal trauma

Breathing:
- Pulmonary embolism
- Alveolar–interstitial syndrome
- Diaphragmatic lesions

Circulation:
- Intravascular volume estimation
- Cardiac function

Disability:
- Neurological impairment

Exposure:
- Among other injuries in a repeated manner

Figure 1.5 Potential problems [8] identified with the FAST ABCDE protocol.

Airway:
- Tracheal incongruity
- Tracheal collapse

Breathing:
- Pneumothorax
- Lung contusion
- Pleural effusion
- Diaphragmatic hernia
- Alveolar–interstitial syndrome

Circulation:
- Abdominal effusion
- Pericardial effusion
- Cardiac tamponade
- Systolic impairment
- Retroperitoneal effusion
- Caudal vena cava collapse

Disability:
- Presumed increased intracranial pressure

Exposure:
- Worsening or new development of injuries

Figure 1.6 Potential problems [8] identified with the VetFAST ABCDE protocol.

Summary

In some emergencies, minutes count. The triage performed by the veterinary technician should be rapid and efficient. The goal is rapid recognition of and intervention for life-threatening conditions such as hypoxemia and inadequate perfusion. A systematic approach to patient assessment is essential for the best possible patient outcome.

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2

The Small Animal Emergency Room

Martin D. Miller and Sean D. Smarick

Emergency medicine can be defined as "the diagnosis and treatment of unforeseen illness or injury"; however, "emergency medicine is not defined by location, but may be practiced in a variety of settings" according to the American College of Emergency Physicians [1]. In small-animal veterinary medicine, emergency practice takes place on house calls, in primary care clinics, in dedicated "after-hours" free-standing emergency clinics, and in multispecialty referral hospitals. The establishment of a dedicated emergency room (ER), dual-use area, or the ability to make do with some basic equipment must be given careful consideration, as no small-animal clinical veterinary practice is immune to emergent patients: Vaccines can cause anaphylactic reactions, anesthesia-related cardiopulmonary arrests occur, and without warning clients may present a pet with a traumatic injury or critical illness.

The Veterinary Emergency and Critical Care Society (VECCS; www.veccs.org), whose mission includes "To promote the advancement of knowledge and high standards of practice in veterinary emergency medicine and critical patient care," provides guidelines for emergency facilities [2]. In looking to these standards along with applicable state board regulations, a practice should define for its patients, clients, the public, and, for itself, expectations for its emergency services and subsequently, the ER.

The ER, while similar to primary care and other specialty facilities, differs in the urgency and breadth of the patients' conditions that must be addressed. Depending on the degree a practice wishes to diagnose and treat unforeseen illnesses and injuries, varying degrees of adaptations in the physical plant, equipment, inventory, staffing, and hospital systems are needed.

Physical Plant

The ER can range from sharing space in a treatment area within a primary care practice to encompassing a significant area of a large referral hospital occupying in excess of 10 000 square feet and everything in between. The basic needs of an ER and progressive primary care or specialty clinic are similar. The differences between such facilities can be found in the layout and organization of an ER dictated by the level of emergency medicine to be practiced.

ER Design and Flow

When creating a floor plan concept for an ER, a great deal of thought should be given to the specific aspects of the emergency practice. Good "flow" is essential to an efficient clinic. Flow represents the natural movements of patients, clients, doctors, and staff in the daily activity of the practice. Arranging for such movement is almost like choreographing a dance. Thought should be given on how best to move clients from triage through discharge from the hospital and its systems [3].

In larger facilities, the idea of a hub and spoke concept can be applied to the flow of a practice. The hub can be a centralized space such as the treatment area. The spokes from the hub may be clinical areas such as the in-house laboratory, radiology, surgery, patient wards, isolation ward, and pharmacy. In larger facilities, multiple hub and spoke areas may exist, such as one for the intensive care unit (ICU) and one for the emergency service. For more on ICU design, see Chapter 3. Some considerations for the ER are described here.

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Back-up Power

Dedicated ERs should have the ability to operate minimally in the face of a power outage, with emergency back-up lighting and uninterrupted power supply for key equipment, but ideally with a centralized integrated generator or battery back-up system.

Security

Emergency practices have more security concerns because they are open during nonbusiness hours and may be perceived to be rich in cash and narcotics. The perception about controlled drugs is a reality and internal security is equally important. To provide a safe environment for staff and clients, enhanced security measures including well-lit public areas both inside and out, limited-access security doors, video monitoring and recording, alarm systems, and critical incident training and protocols should be considered [4, 5].

Entrance and Lobby

Clear signage such as "Emergency" in red can be used to direct clients to the appropriate entrance and reception area. In shared use facilities, systems to ensure patients presenting to the ER are addressed as emergencies and not confused with other patients waiting in the lobby or presenting for routine care. Large facilities may elect to have a separate ER entrance, similar to human hospitals, which decreases the potential for such confusion. Strategically placed gurneys and portable oxygen systems should be available to readily retrieve nonambulatory or dyspneic patients, respectively. Once presented, a patient should undergo triage (see Chapter 1) following a protocol, to include a predetermined location, whether it be a dedicated triage area, exam room, or the treatment area.

An ER lobby must be large enough to accommodate a simultaneous influx of multiple clients and their pets. Considering an emergency presentation may last hours, amenities may include comfortable seating, a beverage service in the form of a water cooler, coffee dispensers or vending machine, TV and/or wi-fi, and accessible restrooms.

Comfort Rooms

The ER has emotional challenges for clients. Providing clients private areas to visit their pet, receive difficult news, or have a pet euthanized, with a less clinical feel is commonplace. Such rooms include comfortable furniture, extra floor space to accommodate an entire family, noncommercial lighting, and home-like interior appointments. Having an exit from the hospital in close proximity facilitates a private departure for a deceased pet or for emotional owners [6].

Treatment Area

The number and type of treatment tables often are increased in an ER. With no lack of debilitated patients or bodily fluids, lift and "tub tables" are recommended. It is important to account for the space surrounding these tables to accommodate the staff and equipment often needed in addressing emergent patients [7].

With the practice of emergency medicine comes an increase in the number of patient-side infusion pumps, heating devices, medical gas outlets, and monitors. Integrating the equipment near the patient calls for space, power and even connectivity between, above, or below the patient areas. Depending on the existence of an integrated ICU, the number of observable and accessible cages and runs for holding patients will vary but should not be underestimated.

The Isolation Ward

ERs ideally should have (or if dictated by state regulations, may be required to have) an isolation ward distinctly separate from the treatment area and other hospital areas to house patients with communicable diseases. A consultation room may be incorporated into or adjacent to the space. These spaces are not only physically separate from other patient housing areas of the hospital but also have dedicated heating, ventilation, and air conditioning systems. Dedicating cage-side equipment such as heat support and infusion pumps limit the potential for disease transmission to the general hospital population. Stocking the isolation ward with treatment supplies limits foot traffic and increases efficiency when treating patients in the ward [8, 9].

In-House Laboratory

Emergency medicine requires more point-of-care (POC) testing and in-house laboratory equipment often exceeding that of other practices. The laboratory machines require counter space and deserve electrical considerations such as surge suppression, line conditioning, and potentially battery back-up.

Diagnostic Imaging

Diagnostic imaging, and minimally radiology, should be adjacent to the ER. Additional considerations for imaging suites include providing oxygen supplementation, anesthetic gas scavenging, suction, and enough space to perform procedures.

Staff Spaces

In a dedicated ER, efficiency demands a semi-private area for doctors and technicians to call clients, prepare medical records, and consult with each other and be located within

or adjacent to the ER. Some practices additionally include traditional office space distant from the clinical areas to allow for more private and quiet work.

Because emergency practice often involves a larger staff who work long shifts, the design of the staff area should include a place for their personal effects and to store, prepare, and enjoy a meal while on break. Water fountains or coolers should be available so that staff can remain hydrated throughout their shifts. The size of the area and its complement of amenities, such as a refrigerator, microwave, stovetop, seating, and tables are determined by shift staff size and by other purpose(s) of the space (e.g. if the area also serves as a meeting or locker room). Adequate restrooms for the staff, ideally close to the ER, should not be overlooked. The addition of showers may be an appreciated benefit of the staff.

Utility Spaces

The ER is supported by several spaces ranging from storage closets to server rooms to provide for the operations of the ER.

"Janitor's closets" are spaces that include fixtures such as mop sinks to support filling and emptying mop buckets and have room for cleaning supplies and equipment. They should be strategically located to allow for easy access and quick clean-up of any area in or around the ER.

A designated space for a free-standing or walk-in refrigerator or freezer to accommodate deceased pets waiting disposition should ideally be located near the ER and an exit that can allow an owner or service to discreetly remove the deceased from the facility.

If the ER uses towels and bedding, laundry facilities must be adequate to handle such large and often never-ending loads. This almost always goes beyond residential equipment therefore adequate space as well as electrical/plumbing requirements should be considered.

Storage space for the increased amount of equipment and expanded inventory of an ER needs to be considered in the design of the facility.

Advanced emergency practices may start to resemble small hospitals, with central oxygen storage and manifolds, centralized medical or maintenance vacuums, anesthetic gas scavenging systems, and computer servers, which all require space and supporting mechanical systems.

Equipment

Additional equipment must be considered for a dedicated ER because of case load, urgency, and severity of emergent conditions. VECCS provides guidelines for equipment for different levels of emergency facilities [2].

Imaging

Imaging and the rapidity of which it can be acquired plays an important role in many emergency cases. A high-quality radiographic system and a POC ultrasound machine (see Chapters 6, 9, 17, 27, and 39 for point-of-care ultrasound) is the minimum for a dedicated emergency practice. Diagnostic ultrasound and echocardiography, multislice computed tomography and magnetic resonance imaging are commonplace in tertiary care emergency centers.

The In-House Laboratory

According to the VECCS guidelines, an ER should also be able to evaluate packed cell volume, refractometric total solids, complete blood count with manual differential, glucose, lactate, chemistries, electrolytes, blood gases, prothrombin/activated partial thromboplastin time, feline immunodeficiency virus and feline leukemia virus antigen testing, cytology, urinalysis, fecal flotation, blood typing and cross match, and parvoviral antigen testing [2]. In addition to the guidelines, other POC testing may include heartworm, tickborne or other infectious disease tests, co-oximetry, and thromboelastography. The most basic of practices may elect to keep a handheld glucometer, lactate monitor, or other POC analyzers. See Section 7 and Chapters 25 and 26 for additional information on laboratory tests in the ER.

Patient Monitors and Equipment

Patient monitors are needed to screen for physiological abnormalities and to monitor ill patients for disease progression. The monitors recommended include electrocardiography (see Chapters 10 and 11), pulse oximetry (see Chapter 25), capnography (see Chapter 30), temperature, and blood pressure. Blood pressure can be measured by doppler, oscillometric, and direct methods. direct methods are recommended and are found in emergency centers (see Chapters 13 and 14). The monitors may be standalone or multiparameter and fixed or portable. Space, electrical needs, networking, use outside the ER and telemetry are considerations when selecting patient monitors. The most basic of practices could consider refurbished electrocardiogram with or without a defibrillator to meet the standard of care for providing cardiopulmonary resuscitation (CPR; see Chapter 22.) Tonometry is needed to screen for increased intraocular pressure and can be measured with a Schiotz or POC indentation/applanation device.

Fluid and Drug Administration

Fluid infusion pumps, calibrated burettes, and syringe pumps are needed to accurately administer fluids and medications in the ER. Fluid and blood warmers are also found

in dedicated ERs to combat hypothermia, where larger volumes may be administered.

Thermal Support

To address presenting, developing, or iatrogenic hypothermia, circulating warm water, forced warm air, or electrically generated heat devices are warranted in the ER. Each system type has its own advantages based on cost, versatility, reliability, and effectiveness.

Oxygen

Emergent patients often require oxygen supplementation, positive pressure ventilation, or anesthesia; oxygen access is therefore vital in the ER.

Oxygen systems require a source and manifolds, specialized transfer piping or hoses and receptacles. These receptacles have a special fitting (the Diameter Index Safety System) to connect oxygen hosing or a regulator. Quick connects are available in different popular configurations from different manufacturers. Oxygen receptacles can be placed on walls or in ceilings and can be recessed or wall mounted.

A practice not employing oxygen regularly may use as a source of oxygen "E"-size tanks, small or portable oxygen generators, or disposable cans of oxygen, or even a stationary "H" size cylinder(s); large ERs that have multiple and high-flow needs will need centralized systems with H tank banks (as a primary or secondary source), liquid oxygen tanks and systems, and/or oxygen generators.

Patient oxygen administration systems can be simple (i.e. a nasal cannula, e-collar hood or mask), but dedicated ERs often employ specialized cages that are designed to create an atmosphere of higher oxygen concentration.

Patients that do not respond to oxygen administration or are severely hypoventilating require positive pressure ventilation. At the most basic level, this can be accomplished with a bag-valve-mask device, nonrebreathing circuit, or anesthesia machine. An anesthetic ventilator or ideally a critical care ventilator are found in dedicated ERs (see Chapters 24 and 31 for more information).

Anesthesia Waste Gas Scavenging

In a dedicated ER, patients will undergo anesthesia in the radiology and other imaging areas, the treatment area, and the surgical and associated preparation areas. A centralized scavenging system is recommended that can provide connections in strategic locations for anesthesia machines.

Medical Vacuum

Besides providing for suctioning of a body cavity in surgery, medical vacuum provides for airway suctioning and continuous thoracic drainage, all commonly used in emergency medicine. Minimally, a hand-powered device such as the Laerdal V-Vac™ mechanical suction unit (Laerdal Medical Cooperation, Wappingers Falls, NY) can be used for airway suctioning, while dedicated ERs often use a central system similar to the oxygen and waste gas scavenging systems with outlets in strategic areas. Portable, electrically powered suction units can be used to address needs in between.

Inventory

Providing high-quality care for the acutely ill patient requires a full inventory of supplies and drugs. Owing to the breadth and seriousness of disease encountered in emergency practice, an extensive inventory beyond that of a primary care clinic is required for the dedicated ER. Even the most basic of practices should have emergency drugs and supplies on hand to perform CPR, address the preventable causes of death encountered in their practice, such as allergic reactions from vaccines and anesthetic complications, and depending on the scope of their practice and distance from a referral facility, common life-threatening conditions.

Because of the inconsistent nature of emergency practice, usage of inventory items and the inability to obtain such supplies on a moment's notice during off-hours, a robust inventory system is needed. Inventory item usage can be tracked through physical markers signaling an item has been depleted, daily physical inspections, or practice management software that includes inventory control or a supply management system, such as the computer-controlled bank of drawers and cabinets (such as the Omnicell®, Omnicell Corporation Mountain View, CA).

The emergency caseload, resources allocated to inventory and equipment, and geographical location are relevant to the inventory. For instance, an emergency hospital that is near a 24-hour human community hospital may be able to purchase or borrow medications such as antivenin. An ER without such resources will need a full complement of medications. VECCS provides extensive guidelines for supplies and equipment for an ER; however, a checklist of recommended emergency drugs and supplies for practices without a dedicated ER are shown in Box 2.1. See also Figure 2.1 and 2.2 for an "ER in a box" that can provide the drugs, supplies and equipment to address emergencies in practices without an ER, such as a mobile practice, a vaccination clinic, or other type of practice, without the overlapping resources to address basic emergencies.

Box 2.1 Basic Emergency Inventory Considerations

Supplies and equipment:

- Endotracheal tubes (various sizes)
- Laryngoscope and blades (various sizes)
- Bulb syringe/suctions tips and device
- Bag-valve-mask
- Polypropylene catheters
- Oxygen source
- Oxygen tubing and connectors, mask
- Soft Tubes (various Fr sizes)
- 1-inch tape
- Disinfectant and alcohol (pads)
- Intravenous catheters (24–14 gauge)
- Injection caps
- Three-way stopcocks
- Extension tubing
- Fluid administration set
- Syringes 1 ml to 60 luer tip
- Needles (20 gauge × 1 inch)
- Illinois bone marrow needle
- Christmas tree/other adapters
- Sterile procedure pack:
  - Scalpel handle and blades (#11 or 12)
  - Kelley forceps
  - Tissue forceps
  - Needle drivers
  - Mayo scissors
  - Tracheal hook
- Suture, monofilament, cutting and taper needle, 0 to 3-0
- Bandaging: hemostatic/lap sponges, roll gauze, elastic tape or wrap, roll cotton
- Stethoscope
- Electrocardiogram and defibrillator, electrode gel
- End tidal CO_2
- Glucometer and strips

Drugs (injectable unless specified):

- Epinephrine
- Sodium bicarbonate
- Atropine
- Lidocaine
- Lactated Ringer's solution and/or 0.9% saline 1-liter × 2
- Diphenhydramine
- Dexamethasone sodium phosphate
- Dextrose
- Narcotic
- Ketamine
- Midazolam
- Furosemide
- Albuterol inhaler and spacer
- Calcium
- Apomorphine
- Activated charcoal in suspension
- Ticarcillin + clavulanic acid
- Famotidine
- Ondansetron
- Propranolol
- Diltiazem
- Proparacaine ophthalmic
- Fluorescein dye strips
- Eye wash
- Artificial tear (ointment)



Figure 2.1 "ER in a box." Adapted toolbox to store emergency supplies where an emergency room is not available.

Medical Supplies

Basic supplies to support the ABCs (airway, breathing, and circulation) should be stocked in every practice, while additional supplies may be found in more advanced emergency practices.

Airway Management

A laryngoscope with multiple appropriate blade sizes, endotracheal tubes, and a bulb syringe for suctioning are basic emergency airway supplies that all practices should have. A source of medical vacuum as discussed above and catheters or suction tips should be considered. Lidocaine sprayed from an atomizer or directly instilled to a cat's larynx will help prevent laryngospasm. A polypropylene urinary catheter or bougie can act as a guide for the



Figure 2.2 Contents of the author's "ER in a box." Top tray: needle and syringes, emergency drugs, sedatives and analgesics, eye wash, stethoscope, disinfectant wipes. In main compartment: 1-liter bag Lactated Ringer's solution, administration set, disposable colorimetric CO_2 detector, bag-valve-mask. Endotracheal tubes, oxygen cannula, portable oxygen, manual suction device, saline prefilled syringes, intraosseous needle, intravenous catheters, laryngoscope and blades, bulb suction syringe, bandaging supplies and scissors. Not pictured: suture, scalpel, tracheal hook, forceps, ophthalmic supplies, injection caps, gloves (exam and sterile) additional bandaging supplies, three-way stopcock, butterfly needles, extension set, and oxygen regulator.

endotracheal tube during difficult tracheal intubation (see Chapter 28).

Ideally, supplies to perform a tracheostomy or cricothyroidotomy should be available in all practices; such supplies include self-retaining retractors or tracheal hook, scalpels, sutures, needle drivers, and forceps. Commercially available kits and devices are available via emergency medical services suppliers and provide (in trained hands) alternatives to the traditional surgical approaches. Many tracheostomy tubes are sold commercially but a shortened endotracheal tube can be used if a tracheostomy tube is not available (see Chapter 29).

Breathing

Rescue breathing can be delivered with mouth-to-snout or endotracheal tube, but because such practice compromises caregiver safety, it is recommended only when no other option exists. Cost-effective bag-valve devices are available through veterinary distributors. Practices that use general anesthesia usually have an anesthesia machine and circuits that can be used to provide positive pressure ventilation.

Many emergency patients need oxygen supplementation even when they do not require endotracheal intubation. A practice should therefore have other equipment to deliver oxygen. Such items could include rigid plastic masks, hoods, which are commercially available or can be made from a plastic wrap-covered Elizabethan collar or a plastic bag, and nasal cannulas, which can be made from red rubber catheters or human bilateral nose prongs. Plastic wrap over the front of a cage or around a cat carrier with some area left for a vent can also be effective (see Chapter 24, for more information).

Oxygenation and ventilation can be compromised by pleural space filling defects and supplies to address should be in the emergency area or kit. Butterfly needles, peripheral intravenous (IV) catheters, dedicated over-the-needle, Seldinger, or trocar thoracostomy tubes compared with other general use tubes should be considered, together with extension tubing, three-way stopcocks, syringes, tube connectors, and Heimlich valves (see Chapter 34 for more details).

If blood gases are measured in the practice, supplies (e.g. syringes, needles, and heparin), or specialized pre-heparinized vented syringes are needed (as discussed in Chapter 26).

Circulation

Maintaining effective circulating volume is crucial for the emergency patient, and every practice should have the ability to stop external hemorrhage and gain vascular access or a bag-valve-mask with an oxygen tube and reservoir. External bleeding can usually be addressed with direct pressure and bandaging; such supplies should be readily available to arrest severe hemorrhage. Besides IV catheters ranging from 24- to 14-gauge for peripheral placement, intraosseous cannulation, an often-overlooked vascular access technique, can be accomplished with hypodermic needles, spinal needles, or purpose-designed or mechanically placed intraosseous needles. A pressure infusion bag can assist in delivering large volumes of fluids in a short amount of time See Chapter 7 for more specific information.

The goal of maintaining effective circulating volume is to deliver oxygen to the tissues. When anemia is severe, an oxygen-carrying fluid must be provided. Disposables required for transfusion to include equipment for blood typing and crossmatch, blood administration sets, inline filters, and collection supplies such as anticoagulant and blood collection bags may be warranted if no blood

products or emergency center is nearby See Section 9 for more information.

Advanced emergency practices may include long-term single or multilumen central IV catheters to administer multiple infusions simultaneously and obtain repeated venous blood samples, all of which provide a continuum from emergent to critical care.

Additional Supplies

The dedicated ER should have specific supplies available to cannulate any patient orifice or cavity, whereas soft generic catheters are cost-effective versatile substitutes for, for example, urinary catheters, thoracostomy tubes, oxygen cannulas. The type-specific tubes may offer the advantages of less tissue reactivity, integrity of asepsis, and the ease of their intended use. With each tube comes the need for specific collectors (e.g. urinary catheter bags, chest tube drainage systems), connector(s), and adapters, so appropriate pieces should not be overlooked. See Section 4 for further information.

Pharmacy

As with equipment, every practice should be able to minimally address cardiopulmonary arrest and shock and effect referral whereas dedicated ERs need to be able to address a wide range of conditions.

Shock and Cardiopulmonary Arrest

Current CPR guidelines call for a vasopressor (namely, epinephrine and justifiably vasopressin), atropine, and sodium bicarbonate as a buffer and for treatment of hyperkalemia. See Chapter 20 for more information.

Shock, defined as inadequate cellular energy production, is usually the result of hypovolemia, hypoglycemia, hypoxemia, sepsis, or cardiac failure, and is the primary acute condition that every practice should be prepared to address.

Oxygen, not often thought of as a drug, was discussed under supplies and can be a life-saving treatment for shock.

The most common cause of shock is hypovolemia, and restoration of effective circulating volume with crystalloids and blood products may be needed. There is no universally ideal solution for resuscitation, so the more emergencies a practice sees, the more bags and types of fluids and blood products are warranted. Minimally, isotonic saline (0.9% NaCl) or a buffered, balanced electrolyte solution such as lactated Ringer's solution should be stocked. If blood products are not stocked, donors and blood collection supplies may be considerations if distant from a referral facility.

Every practice should be prepared to address anaphylactic or serious allergic reactions with epinephrine, diphenhydramine with or without a histamine type 2 receptor (H_2) antagonist and corticosteroids.

Diuretics, antiarrhythmics, afterload reducers, and inotropes may be needed to address cardiac emergencies. For disturbances in electrical rhythm that may compromise cardiac output, atropine and antiarrhythmic drugs from the sodium channel blocking, beta and calcium channel blocker classes are recommended.

Bronchodilators and corticosteroids are warranted in the patient compromised from allergic bronchitis and epinephrine can be used if severe and no other drugs are available.

Patients in septic shock, by definition, are not responsive to IV fluids alone and require vasopressors and possibly positive inotropes. Many inotropes and vasoactive medications require constant rate infusions. Infusion or syringe pumps to deliver these medications along with appropriate diluents (e.g. dextrose 5% in water) are required for their administration. Additionally, recent sepsis guidelines in people recommend starting antibiotics early [10].

Anti-Infectives

Antibiotics are used to treat, and in very specific situations to prevent, bacterial infections. Parenteral antibiotics effective against the organisms encountered in the emergent setting requires stocking antibiotics to address Gram-positive and Gram-negative, aerobic, and anaerobic organisms. Beta-lactams, fluoroquinolone, and aminoglycoside antibiotics are basic requirements, and metronidazole, macrolides, and tetracyclines play important roles. Antiprotozoal and anthelmintic medications round out the anti-infective inventory, with consideration given to the organisms and parasites encountered in the practice. Basic practices may consider stocking a dose or two of a second- or third-generation cephalosporin, extended spectrum penicillin with a beta lactamase inhibitor, and/or fluoroquinolone injectable that can be administered prior to referral. When used appropriately, early administration of antibiotics can affect outcome in conditions such as sepsis and wound management.

Analgesics, Sedation, and Anesthesia

With the standard of care including analgesia, the essential emergency pharmacy should have drugs for parenteral administration that interfere with the transduction, transmission, modulation, and perception of pain. Local anesthetics, alpha-2 agonists, N-methyl-D-aspartate (NMDA) antagonists, nonsteroidal anti-inflammatory drugs, and opioids are classes of drugs to stock for analgesia. An

analgesic that can also check the box for sedation and be considered cardiac sparing should be considered for every practice to "relieve suffering" during referral or prior to euthanasia.

Patients with an acute presentation usually raise cardiopulmonary concerns beyond those of a healthy patient; the drugs stocked for sedation and anesthesia in emergency practice should reflect this. Balanced anesthetic techniques with cardiovascular sparing drugs are most appropriate, and such drugs include opioids, benzodiazepines, and NMDA antagonists. Propofol, alpha-2 agonists, and promazines still play important roles in the emergent setting, despite their more profound cardiovascular effects. Section 6 provides more in-depth information.

Intoxications

Intoxications may vary by geographical location but universal considerations where timing is crucial (i.e. gut decontamination) justifies stocking emetics and activated charcoal. Dedicated ERs should have specific antidotes in stock, such as ethyl alcohol for ethylene glycol, vitamin K_1 (phytonadione) for anticoagulant rodenticide intoxication, lipid solutions for fat-soluble intoxications.

Gastrointestinal Medications

A patient presenting with signs referable to the gastrointestinal tract is one of the most common presenting emergencies. As such an ER should consider carrying parenteral anti-emetics from several classes and proton pump inhibitors and/or H_2 antagonists.

Endocrine Emergencies

While most endocrine emergencies can initially be treated with supportive care if the patient is going to be immediately referred (i.e. IV fluids), dedicated ERs should be prepared to treat hypoadrenocortism, diabetic ketoacidosis, and aberrations in calcium homeostasis. For Addison's disease, injectable dexamethasone is an important drug, as it will not interfere with adrenocorticotropic hormone stimulation testing due to the lack of mineralocorticoid activity in dexamethasone. Hydrocortisone or prednisone will be required for subsequent doses if a mineralocorticoid (such as deoxycorticosterone pivalate or fludrocortisone) is not available. Diabetic ketoacidosis requires intramuscular or IV administration of insulin.

A high-percentage dextrose solution should be stocked to address hypoglycemia from insulin overdose or secreting tumors and complications from sepsis, neoplasia, and intoxications. Hypercalcemia can be treated with IV fluids, a loop diuretic, an alkalinizing agent, and, in certain instances, a corticosteroid. Hypocalcemia requires parenteral calcium supplementation.

Urogenital Emergencies

Urogenital emergencies amenable to pharmacologic intervention include dystocia and complications from hyperkalemia. Hyperkalemia's effect on the heart may be antagonized by a slow IV push of calcium, while insulin and alkalinizing agents shift the potassium into the intracellular space. Oxytocin, along with dextrose and calcium supplementation, is used to treat uterine inertia.

Ophthalmologic Emergencies

Ophthalmologic examinations require a topical anesthetic for the cornea, fluorescein stain, and sterile, buffered saline eye rinse. Artificial tears and ointment are needed to prevent exposure keratitis in patients with decreased tear production or blinking, and in those under sedation or anesthesia, even for a short time. Every practice should consider stocking these basic supplies. Glaucoma in the acute setting requires lowering the intraocular pressure with a topical and/or systemic carbonic anhydrase inhibitor, an osmotic diuretic, and/or prostaglandin analogues and depending on available referral and pharmacy options, the ER should consider having these medications on hand. Inflammatory diseases use a topical steroid product in the absence of corneal ulceration, and most bacterial infections can be addressed with a few choices in antibiotic ointments or drops. A dilating agent such as topical atropine offers relief from ciliary spasm and prevents synechia formation in corneal lesions.

Neurological Emergencies

Medications to control seizures (e.g. a benzodiazepine) and to address hypoglycemia should be found in every practice, while dedicated ERs may include drugs to lower intracranial pressure and loading doses of long-term anticonvulsants.

Staffing the Emergency Practice

Depending on caseload variations of the season, day of the week, and time of the day, the scope of the practice, and the population serviced, staffing of a dedicated emergency practice can range from a single veterinarian and assistant to multiple veterinarians supported by a plethora of support staff.

Typical veterinary staff (certified veterinary technicians/veterinary nurses, assistants, kennel aides, and

receptionists) have roles in an emergency practice as they would in primary care. It is ideal to have a high ratio of technicians to veterinarians with the support staff in dedicated positions. This can be accomplished with consistent and high caseloads; however, the caseload in an emergency practice can vary tremendously and both veterinarians and support staff may be called upon to assume multiple or nontraditional roles.

The specialized skills and knowledge of the emergent practice staff will often find even certified veterinary technicians and employees with years of experience in other practices needing additional training. In-house training programs are crucial at the basic level. The Academy of Veterinary Emergency and Critical Care Technicians and Nurses offers an avenue to become a veterinary technician specialist and gain recognition in the field by pursuing advanced training.

The nature of an emergency practice means that unique staffing considerations exist. Scheduling for a practice operating off-hours or continuously for 24 hours a day has challenges of shift transitions and continuing education, performance review, and staff meetings. The highly emotional environment of an emergency practice may also lead to compassion fatigue; Section 11 addresses these issues.

Hospital Management and Systems

As discussed, facility, equipment, supplies, pharmacies, and staffing of a dedicated ER are often larger and more complex than those for primary care practice. The off-hours and possibly continuous operating nature of the emergency practice makes communication among co-workers and between management and staff difficult, yet it is crucial for the successful delivery of emergency care. The hospital systems in an emergency practice must address these challenges to keep operating smoothly.

Medical Records

Medical records contain the history and chronology of the medical care given to the patient. Their contents may be regulated by the state veterinary board and the VECCS recommends that the problem-oriented medical record as outlined by the American Veterinary Medical Association be followed and be kept at the emergency facility.

While referral practices usually have robust record systems to ensure that everyone (internal staff, client, and referring veterinarian) follows the same procedures, every emergency warrants a detailed record, even in the throw of chaos, to document the care provided and to be beyond reproach if that care is ever called into question (see Chapter 5).

Practice Management Software

A significant number of a patents medical record can be managed by current veterinary practice software. Veterinary practice software is commonplace and addresses not only medical records but also client and patient databases, inventory control, invoicing, and accounting. Software designed for referral practices can also include treatment orders, white boards, and referring veterinarian databases and portals, which may be advantageous for dedicated ERs.

Other Information Management

VECCS requires select references and continuing professional education for staff to be a certified emergency facility, but every practice should have resources including textbooks, online group memberships, subscriptions, or telemedicine options, poison control center information, professional memberships (e.g. VECCS, RECOVER), and associated resources to be able to meet the standard of emergency care for their type of practice.

Management

Management of a dedicated ER practice is similar to the other practices; however, the chaotic nature of patient presentations and a continuously operating hospital bring additional challenges. Managing these challenges requires more anticipation, implementation, and monitoring beyond that of a practice operating during more traditional hours. Chapter 4 provides invaluable insight into addressing some of these challenges.

ER management is the focus of large corporate entities, specific veterinary management groups or veterinary study groups, and organizations such as VECCS. Also, there is no lack of opinions on online forums in which to engage. However, it is equally important for the non-emergency hospital to define, maintain, and even practice their emergency policies and procedures to effect successful resuscitation and referral.

Summary

Every clinical practice should have a minimal set of tools to treat common emergencies that are likely to be experienced in their practice. While an ER shares many features with other types of veterinary facilities, the design, equipment, inventory, staffing, and hospital systems become more specialized to provide care for patients with unforeseen illness and injuries.

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3

Intensive Care Unit Design

Joris H. Robben and Lindsey Dodd

The primary objective of an intensive care facility is to provide a high level of continuous patient care. A well-designed intensive care unit (ICU) is necessary for the creation of a safe, efficient, and, not the least, pleasant environment for patients, personnel, owners, and other visitors. Safety relates to several aspects, such as environmental services, ergonomics, and mental wellbeing. However, infection control and prevention will have an ever-increasing influence on the design.

In veterinary medicine, no consensus or detailed standards exist regarding what should constitute an ICU for companion animals. Currently, there is a wide variety of ICU types in various clinical settings. Considering this variety, a description of current practices or effective design options can never be complete. Nevertheless, as in human intensive care medicine, veterinary medicine should strive for unification and basic, general guidelines for ICU design to ensure a predefined and, preferably, centrally guided, high quality of intensive patient care. The topics that are addressed here are intended to constitute a basis for such future guidelines.

In smaller practices with a variable need for intensive care it may be most economically viable to combine the ICU facility with the emergency and recovery rooms [1]. Although these facilities are closely related, they should be considered separate units with different operational functions. Physical separation is particularly important for the ICU as it helps to prevent unnecessary commotion and traffic, which aids with infection control (one of the primary aims of ICU design in human medicine along with improving patient comfort). This discussion focuses on intensive care medicine only, and the ICU will be considered as an independent, functional unit.

A well-designed ICU should not only have a relevant floor plan, interior design, and equipment, but also take into account staffing, the intended operations within it, and should have standardized operating procedures described in protocols and guidelines. For a successful design, all these aspects must be considered and put into context with one another. However, for reasons of constraint and in accordance with the intention of this text, this chapter does not discuss the important aspect of staffing. The aspect of operations is discussed if relevant for the design.

The Design Process

Development

ICU design has a significant impact on daily patient care and staff wellness. The extensive investment of time and effort in the long and complicated design process will be rewarded when one experiences its positive impact on the day-to-day operations. A stepwise approach helps to organize the development from initial concept to completed structure (Box 3.1). The planning and design process should include research that leads to evidence-based recommendations for materials in compliance with local legislation directives. A major step in the development process is the description of the ICU program goals and objectives. These can be best defined by a planning team that includes ICU veterinarians and technicians, pet owners, administrators, and design professionals.

The intensive care facility and all its contents should support, and be a logical extension of, the manner in which the ICU is operated. Therefore, the design should creatively reflect the vision and spirit of ICU staff and pet owners. It is important to allow everyone involved in the development process to comment, as design aspects must be judged from different perspectives. A close interaction

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Box 3.1 Steps in Planning and Designing a Veterinary Intensive Care Unit

1) Development of the vision and goals for the project.
2) Education on design planning and processes for a changing organizational culture.
3) Review of articles on delivery of patient care, team-building, (evidence-based) design, facility planning, and other relevant aspects of clinical practice.
4) Visits to new and renovated units.
5) Vendor fairs.
6) Legislation and local directives relating to the build.
7) Development planning.
8) Space planning, including methods to visualize three-dimensional space if available.
9) Operations planning, including traffic patterns, functional locations, and relationship to ancillary services.
10) Interior planning.
11) Surface material selection.
12) Review of blueprints, specifications, other documents, and mock-ups.
13) Preparation and planning for change in practice for staff and pet owners in the new unit.
14) Building and construction.
15) Post-construction verification and remediation.
16) Post-occupancy evaluation.

Source: Based on White et al. [2].

between external experts in hospital construction and an internal interdisciplinary team is mandatory to reach a professional design that agrees with user requirements.

Location in the Hospital

The ICU must be situated within the hospital in relation to other facilities such as emergency services, anesthesia induction and recovery rooms, intermediate or general wards, and ancillary services. Close proximity to the surgical theaters, recovery and emergency rooms reduces the distance to move critically ill patients and enables the easy transfer of staff, equipment sharing, and the common use of ancillary functions. Furthermore, the sharing of engineering services can more easily be accomplished during the building process. The ICU should be located with consideration for other services that receive ICU patients or personnel traffic. Such services include the diagnostic imaging facilities, pathology, and the clinical laboratories if there is limited or no point-of-care testing available in the ICU. In the absence of imaging technicians and laboratory staff, ICU personnel should be able to use these facilities promptly and with ease.

Facility Configuration

An ICU can be divided into three major areas: the staff work area, patient area, and ancillary services. The last is either part of the facility or located elsewhere in the hospital and shared with other services. As with the location of the ICU within the hospital, the topographic positioning of areas within the ICU should be considered and defined early on during the design process. The value of the relationships between different rooms, areas, and functions can be *prioritized* by a system of grading. It helps in this conceptual phase to think of the ICU as a group of concentric circles with the patient modules at the center. (For the purpose of this discussion, each individual patient care area, i.e. cage and direct surroundings, is considered a *patient module*.) By positioning the different rooms, areas, and functions within the smaller and larger concentric circles surrounding the patient modules, relationships can be discussed and adjusted accordingly.

The *character* of the relationship between areas, rooms, and functionalities must also be described, and is based on the need for visual or auditory contact with patients, and the patterns of physical movement of patients, people, and materials (being either clean or dirty, small or large) through the space.

The configuration of the facility has to be drawn within the limitations of the building and based on the priorities for ICU positioning within the hospital, the priorities for the supporting areas and functionalities within the ICU, the need for shared engineering services, and the design considerations from other services.

ICU Size

Several aspects must be taken into consideration when deciding on ICU size (Table 3.1). A facility that can accommodate 6–12 patients with a mean occupancy of 60–70% is often considered a practical and viable companion animal ICU size. Smaller units may not benefit from economy of scale, and may receive an insufficient caseload to maintain skills and expertise of personnel. Larger units may reduce the overview of activities by the staff and present problems with clinical management [3, 4]. Larger units should only be considered when the facility admits patients to the ICU that need less intensive care, such as when a facility combines intensive care with

Table 3.1 Considerations to determine the size of the intensive care facility.

Aspect	Considerations
Historical information	Size of previous ICU facility
	History of patient refusals at prior size
Regional influence	Role in the regional veterinary community
Hospital/practice	Size
Size and type of other specialties and facilities	Presence, size, and level of the emergency service
	Presence, size, and level of other in-hospital specialties
	Number of surgical theaters, surgical case load
Organizational restrictions	Space available
	Staffing available
	Type of patients (admittance policy)
	Level of care
	Number of elective (planned) admissions
	Financial restrictions

medium care functions. Larger institutions could consider the design of subunits that manage patient care independently, such as a separate cat and dog patient module area to provide enhanced and optimal care for both species [5].

The Ground Plan

Staff Work Areas

Technicians' Station

A central technicians' station can improve organization and efficiency of patient care and improve comfort, convenience, and wellbeing for technical staff [6]. Such a station should be an area of sufficient size to accommodate all necessary work activities. The station should be situated such that it offers a clear, unobstructed view of the patient area. Sliding glass doors and glass partitions offer optimal sight and at the same time function to separate the technicians' station and the patient area acoustically (both ways) (Figure 3.1a). If an isolation ward is part of the ICU, visual and audible contact should be available from the station (Figure 3.1b).

The station should contain an area or desk with computer terminals for patient record maintenance and Internet access. This setup facilitates more detailed record compilation, completion of requisitions, and into and out of hospital telephone communications. The station could also accommodate storage facilities for protocols, patient chart material, request forms, and other medical stationery. Depending on the space available a medical book collection, printers, document scanner, fax, photocopier, and a table to perform patient rounds can be made part of the area. Anything that makes work efficient and the working experience attractive should be considered for this area.

If the technicians' station cannot provide enough room for all these facilities and functions, a separate medical or administrative office should be available in close proximity to the patient area.



Figure 3.1 (a) The entire patient area can easily be surveyed from the technicians' station through large glass windows. (b) With the use of cameras in the isolation ward and wall-mounted monitors in the technicians' station, the isolation ward can be monitored from within the station. Auditory surveillance is possible through the installation of microphones and speakers.

Staff Break Room

Work in an ICU can be physically and mentally demanding, and staff should have the opportunity to break away from the clinical environment when necessary and as part of scheduled breaks to aid their wellness. Staff break areas should provide private, comfortable, and relaxing environments with external windows, comfortable seating and distractions such as magazines, radio, and television. Kitchen facilities such as a sink, microwave, food refrigerator, and dishwasher may be integrated or directly adjacent. Lockers for safekeeping of personal belongings can be installed. The room should be equipped with communication systems such as telephones, computers, intercom, and emergency call buttons.

Conference or Multipurpose Room

This room facilitates staff meetings, teaching sessions, and other group meetings. For this purpose, it should contain proper seating, projection facilities, a white board, and illuminated viewing box. It can also be used as a library by equipping it with journals, reference books, internet-connected computer terminals and printers. Both the staff break room and the conference room should be in close proximity to the ICU so that staff are not dispersed during their absence, but can certainly be shared with other services.

Patient Area

The patient area should be compact and offer close, unobstructed visual (and audible) contact with all patients. A link between poor visualization of patients by nursing staff and physicians and mortality in critically ill patients has been demonstrated in a human ICU [7]. The number of staff in a veterinary ICU is often limited, and one staff member should be able to observe multiple patients at once. This factor should play a major role in the positioning of the different patient modules. Furthermore, the requirement for a relatively small unit must be balanced with the need for increasing numbers of cage-side monitors and therapeutic equipment, the importance of infection prevention and control (IPC) measures, and the need for a pleasant, comforting and ergonomically designed environment for patients, staff, and owners.

The patient area should allow unobstructed movement of staff, owners, visitors, patients, and equipment. Therefore, doors, aisles, and corridors should be designed to accommodate the transport of stretchers, patient carts, and large equipment within the unit and into and out of the facility.

Besides the presence of patient modules and a treatment area for specialized procedures, the patient area is often used for storage of disposables, smaller (surgical) equipment, and larger medical equipment for monitoring and treatment. However, the storage of such disposables and equipment in the patient area should be minimized, as it contributes to the impression of chaos and limited space which can have a detrimental effect on patient care, efficacy and staff wellbeing.

Patient Modules

Patient modules consist of the patient's cage and its direct surroundings, the "cage-side". The modules should be arranged and designed in such a fashion that observation of *all* patients by direct or indirect (e.g. by video monitor) visualization is possible from many locations in the ICU [3]. Such an arrangement permits the observation of patient status during both routine and emergency circumstances.

Patient modules should be designed to support all necessary healthcare functions. Intensive care staff spend a considerable time monitoring, treating, and nursing patients. Therefore, patients should be easily accessible, with special attention to ergonomics and infection control measures. The more complex the care required, the more cage-side space needs to be available to position the supporting and monitoring equipment. However, this should not impede access and nursing care to the patient.

These considerations force us to rethink the design of the patient area with special attention to the concept of the patient module. Different frequencies and intensities of care may warrant different types of patient modules to cater to different types of patients, their needs and nursing requirements. The admittance policy should be considered when deciding on the type of patient modules to use. A well-balanced mix of different patient module types or an adaptable cage concept to accommodate moderate, advanced, and intensive one-on-one patient care options probably serves the overall needs best. This variety allows for more economic use of the limited space available.

"Cage-side" Utilities

One of the most valuable resources to have at the cage-side is space. Open space maximizes access for caregivers, reduces cross-contamination between patients, and provides space for owners who are visiting [8]. A visiting room for owners offers more privacy and comfort but many patients require continued care, and this will not always be a suitable option.

Every patient module should have its own lighting. This enables the staff to care for one animal during quiet, nightly hours without having to use the main light that illuminates the whole patient area. Preferably, every cage is equipped with one or more devices for warming the patient, such as floor heating or a heating lamp.

Light switches, electrical power sockets, and other outlets must be organized to ensure safety, easy access, flexibility, hygiene, and maintenance. Depending on the type of cage, outlets can be mounted on the wall above or on both sides of the enclosure. Alternately, they may be brought from the wall or ceiling in a more free-standing arrangement to a boom or trunk over the cage, via a "stalactite" structure from the ceiling or via a "stalagmite" structure from the floor. The number and location of the power sockets depend on the amount of electrical equipment, but a minimum of eight per patient module seems reasonable for most patient modules, especially with the routine use of infusion pumps (Figure 3.2). The current standard in human medicine is as many as 16 power sockets per bed [9].

The number of outlets for vacuum, oxygen, and compressed air may vary with the type of patient module, but every cage should have easy access to at least one of each. Patient modules designed for mechanical ventilation need more service outlets and may need an extra outlet for scavenging of anesthetic gases.

Equipment may be supported on wall-mounted rails, shelves, the top of the cage (when health and safety is carefully considered), or on mobile service stands ("drip poles"). When equipment is too heavy, too bulky, or is shared between patient modules, it can be mounted on a trolley. A combination of these arrangements may be suitable. Bedside monitoring equipment should be located to permit easy access and viewing, and should not interfere with the visualization of, or access to, the patient. The status of each patient should be readily observed at a glance. This goal can also be achieved by a central monitoring station feed by telemetry or an internal camera system that permits the observation of more than one patient simultaneously, preferably from within the technicians' station.



Figure 3.2 With the ever-increasing use of infusion pumps and other medical equipment, the need for power outlets increases. Eight per patient module seems sufficient in most instances, which can be reduced with new inventions like docking stations servicing multiple infusion pumps on one power outlet.

Storage units (cupboards, baskets, shelves) could be placed in more advanced patient modules to provide easy access to specific drugs, disposable materials for specific patient care procedures, such as mouth care and emergency resuscitation equipment.

The patient's daily record and flow sheet can be located in the patient module or at the central technicians' station. Their position should be easily accessible to the nursing and medical staff (Figure 3.3). Complete paperless bedside recording as is now custom in human medicine may be introduced in veterinary medicine in the near future.

Cage Designs

As care and treatment of patients is the core duty of an ICU and is primarily performed in and around patients, the design of the cages is of the utmost importance. Many different "cage" designs can be found in veterinary ICUs today (Figure 3.4).

A kennel or run can be described as a small, fenced space with a door at the front (Figure 3.4a); such a space is often used to enclose larger dogs. When the patient is recumbent, care and treatment must be performed on the floor of the kennel, often with the patient positioned on padded bedding or a mattress. Runs make it difficult to position equipment, and often drip poles are used in or in front of the run. From the perspective of infection control, practicality, and working conditions for the staff this is less than ideal for the treatment for most types of ICU patient.

"Stacked" cages can be made of stainless steel, fiberglass, or plastic and can be stacked in different arrangements (Figure 3.4b). The cages have a metal wire or plastic front with a door and all other sides closed. This setup of cages appears most widely used in companion animal ICUs. Such a conformation uses limited space, which is often important in an ICU. Although the construction is more elevated than a run, the close contact between patients is not ideal for maintaining hygiene and reducing cross-contamination. Cages that are positioned in the second or third row from the bottom offer an ergonomic advantage. Patient visibility is not optimal, as one must stand directly in front of the cage to be able to see clearly inside. Equipment, wires, and infusion lines can only reach the patient from the front. With several patients housed directly next to and on top of each other, this arrangement



Figure 3.3 (a) A small writing tableau attached to the cage makes it easy to keep the patient's flow sheet current. The tableau can be folded away when not in use to increase space for maneuvering carts and equipment. (b) An alcohol dispenser is placed conveniently alongside some tableaus so staff can disinfect hands and forearms between patients.

often makes the whole situation chaotic and difficult to oversee. Additionally, banks of cages such as these make procedures that require two or more staff members difficult (or impossible) to perform with the animal in the cage. Adding equipment and lines to the front of the cage, this setup makes patient care challenging.

Ideally, oxygen cages control not only the oxygen concentration in the closed environment of the cage, but also temperature, humidity, and carbon dioxide levels. Many ICUs are fitted with one or two oxygen cages, as they are an easy way to administer oxygen with minimal patient stress. Some ICUs have made the oxygen cage their standard cage, mainly because they are convenient. The oxygen cage makes it difficult to have frequent, direct physical contact with the patient without sacrificing oxygen supplementation. Furthermore, current oxygen cage designs sometimes fail to maintain the environment at preset values. Although the smooth inner surface of many designs makes cleaning of the cage easy, the disinfection of the cages' internal temperature and humidity control, and carbon dioxide extraction systems can be cumbersome. Some oxygen cage designs provide more space for the patient and service openings on the cage's side; such designs make visual contact with and approach to the patient easier than with stacked cages. Auditory contact is often hampered by the closed environment and the noise of the air conditioning.

Wall-mounted or free-standing singular cages are designed such that the bottom of the cage is at table height (Figure 3.4c). The design as shown in Figure 3.4c originates from combining the characteristics of a metabolic cage with those of a fume cabinet. Three or all sides of the cages are made of glass windows or bars that give ideal visualization of the patient. These cages can be opened from two sides, which makes it possible for two or more people to handle the patient at the same time. Equipment and lines can easily be organized around the cage. Of course, such a cage takes up more space, but it is ideal for dealing with ICU patients that need elaborate intensive care. These cages are primarily intended for smaller patients because of their size and patients often need to be lifted into and out of the cage.

The "playpen" type cage is an excellent alternative for large dogs (Figure 3.4d). Its bottom is conveniently lifted about 40 cm from the floor: easy for the patient to step into and raised enough to contribute to hygiene. The whole construction is made of stainless steel, and the doors and bottom can easily be taken out for proper cleaning and disinfection. The bottom is lined with a watertight cushion. The playpen height is more comfortable for staff than a floor-level run. Equipment and lines can be easily organized around the cage.

"Mechanical ventilation" Station

An ICU that has the resources to perform long-term mechanical ventilation should have one to three stations specifically designated for this task (Figure 3.5). Rather than being restrained by a cage, patients are immobilized by anesthesia or neuromuscular disease. Ventilated patients need intensive care with frequent contact with caregivers and connection to many monitoring devices and other equipment such as infusion pumps. An open area



Figure 3.4 Different cage designs that are used currently in companion animal ICUs. (a) Kennel/run (Cummings School of Veterinary Medicine at Tufts University); (b) stacked cages (Cummings School of Veterinary Medicine at Tufts University); (c1, c2) wall-mounted singular type; (d1, d2) "playpen" type.



Figure 3.5 Mechanical ventilation station.

with a table for the patient in the center serves these needs best. The ventilator and most supporting equipment can be arranged at the head of the table.

It is ideal to be able to change the height and tilt of the patient table. Patients that are immobile over a long period need special attention to prevent pressure lesions. The table surface should be padded and large enough for the patient to lie comfortably without its legs extending beyond the edge of the tabletop. For most hydraulic tables this means the top must be extended beyond the standard size.

Procedure Area

A procedure area is often included in the patient area because many minor interventions such as catheter placement and wound management are performed in the ICU (Figure 3.6), and a different room would separate staff from the patients for too long. The treatment area is organized around one or two procedure tables. A hydraulic tilt-top table offers advantages for some medical interventions. A wet table enables patient bathing and wound care; however, close attention to hygiene is imperative when considering such a construction in the ICU.

High-intensity lights should be mounted above every table. The table should be easily accessible by multiple staff from different directions. On one or two sides, the procedural table can be surrounded by countertops on which to arrange and prepare disposables and equipment. A hands-free handwashing station should be available in this area. Positioning of rails or shelves for support of monitoring equipment above the countertop permits easy access and viewing. Light switches, electrical power, oxygen, compressed air, vacuum, and gas evacuation outlets must be easy to access. They can be mounted on the wall above the counters. The number and location of the power sockets depend on the number of electrical appliances present on the shelves or counter. A free-standing, ceiling-, or floor-mounted utility column directly adjacent to the procedural table improves access and flexibility; the column can also contain controls for the lighting. The use of mobile devices such as poles, carts, and trolleys should be limited, as they often hinder patient and staff mobility. However, there should be room to place the crash cart, mechanical ventilator equipment, or other larger equipment that may be required near the procedural table(s). Some storage facilities such as cupboards, drawers, refrigerator, freezer, and incubator for fluids can be positioned under the counters or above them. As the treatment area is often centrally located in the patient area and used for cardiopulmonary resuscitation (CPR) efforts, this is a suitable place to park a fully equipped crash cart.



Figure 3.6 (a) The procedure area contains all the necessary equipment to perform small surgical interventions. (b) When the ceiling is not available for a service unit, a mobile arm mounted on the adjacent wall can bring all necessary wires and tubes to the table, which prevents people from tripping over them.

Isolation Ward

The isolation ward is ideally maintained as a separate facility near to the ICU. The entrance to the isolation ward should consist of a separation corridor (Figure 3.7a). This area should make it possible to prepare to enter the ward with appropriate protection, and to leave it with minimal risk of breaching isolation. The corridor should be divided into two separate areas, potentially separated with a shower. The corridor should enable the change from regular hospital clothing to personal protective equipment (PPE)/barrier nursing attire (e.g. gowning, hair cap, footwear, and gloves), and should include a hands-free handwashing station. When a completely separate facility is not feasible, a closed room adjacent to the ICU with a large glass window for easy observation of patients can be effective. Sometimes the only practical and cost-effective alternative is to isolate patients in part of the main ICU patient area; however, this puts the general ICU population at higher risk. Besides dedicated personnel, design considerations such as temporary physical barriers should be considered.

The isolation ward should be designed to function independently as much as possible without needing to introduce disposables or equipment (Figure 3.7b). Ideally, the isolation area is a smaller version of the main patient area with storage facilities, refrigerator, a procedure area, and patient area. As this room is to isolate patients, only a limited number of cages (two to four) are necessary. Very rarely do pets have to be isolated as a group and probably not many facilities can provide for that in a cost-effective manner. Any disposables or contaminated equipment should leave the isolation area in sealed containers for further processing.

Ventilation systems for isolation rooms should be independent of other systems in the hospital with 100% exhaust to the outdoors. Ideally, the system should have the capacity to create negative or positive air pressure (relative to the open area). If the room is designed to control airborne infections, all walls, ceilings, and floors, including doors and windows, should be sealed tightly.

Separate isolation facilities should have electronic means of communication. Remote patient monitoring capability via cameras and microphones with a central viewing location in the ICU is necessary (Figure 3.1b).

Outside Runs

ICU patients often have limited mobility. However, for a successful recovery the incentive of a small outside area close to the ICU can be important. The use of patches of (artificial if natural is not available) grass invites patients to relieve themselves more easily, but hygiene is more difficult to maintain compared with a concrete surface. As

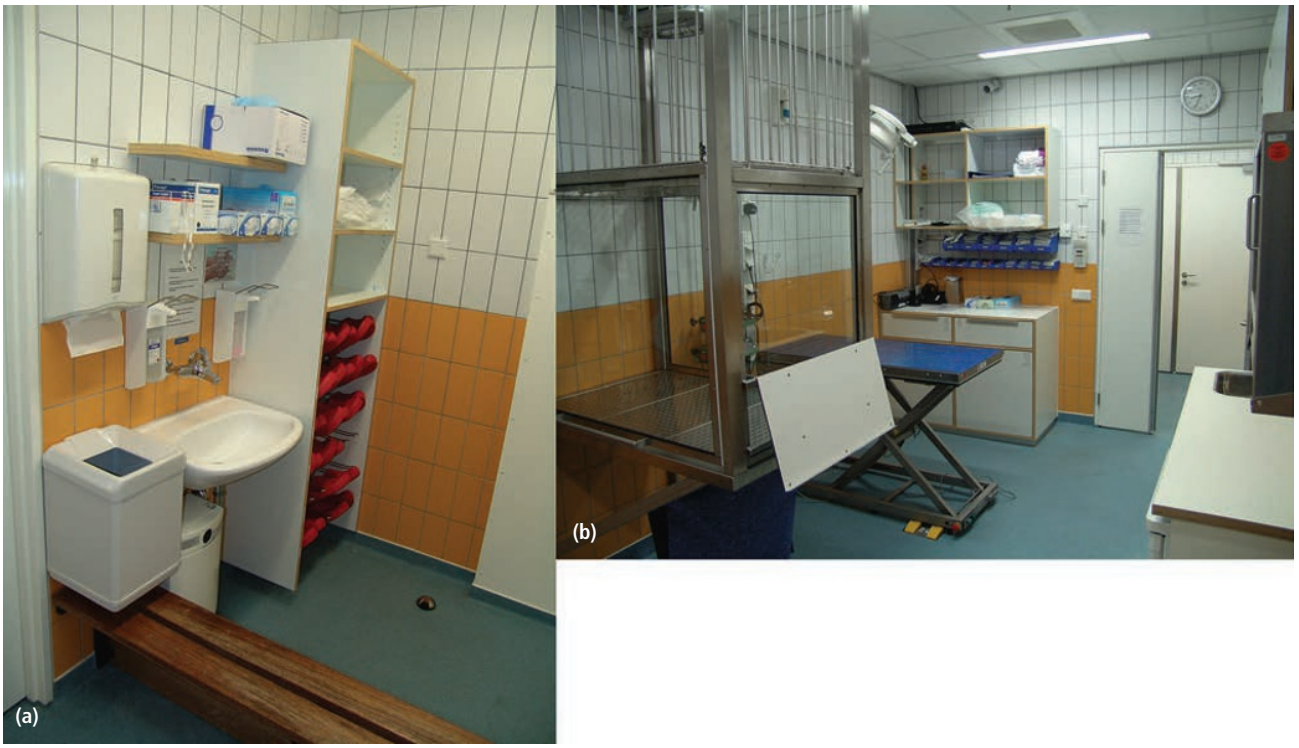


Figure 3.7 (a) The isolation ward can be accessed via a separation corridor, which enables staff to change clothing and wash and disinfect hands. (b) As much as possible, the isolation ward should function as a stand-alone space. A small treatment area is thus part of the isolation ward.

there is constant risk of cross-contamination, this outdoor area is dedicated to ICU patients alone and not shared with other hospitalized animals, and (as much as possible) these areas should be cleaned and disinfected after every patient visit.

Ancillary Rooms

Laboratory Facilities

The ICU should have easy access to a laboratory facility which supplies emergency-directed 24-hour clinical laboratory services. When such services cannot be provided by the central hospital laboratory, a satellite laboratory within the ICU must serve this function. It is advantageous to have the laboratory facility close to the patient area so that technicians are never far away in case of an emergency in the ICU.

At a minimum, it is ideal to have 24-hour capability to measure packed cell volume, total protein, albumin, electrolytes, glucose, blood urea nitrogen, creatinine, coagulation parameters, blood gas and lactate, urine specific gravity, and to have a microscope to evaluate blood smears and cytology [10]. The laboratory bench should offer enough space and be equipped with enough power outlets for all the necessary equipment. Network connections may be necessary to link laboratory equipment with the hospital network and download laboratory results directly into the patient's medical record.

Fume hoods may be advantageous or even legally required if stains for cytological preparations are used. A sink and water tap can sometimes be fitted with a device to produce laboratory quality deionized water. There should be enough room to store instruction manuals and administrative records, and to stock laboratory disposables and reagents, in a refrigerator or freezer if necessary. Also, a refrigerator, freezer, and heating incubator dedicated to the temporary storage of biological specimens must be readily available. Connections and space for a computer terminal and a communication system may be considered. Local regulations may demand an emergency shower or eye wash installation and first aid box.

Pharmacy

Guided by local considerations and organization, a satellite pharmacy can be part of the ICU. A separate room is warranted if the ICU is serviced by its own pharmacy. The air-conditioned pharmacy should have room for storage of medications including a lockable cabinet for controlled substances, a refrigerator for pharmaceuticals, storage for intravenous (IV) fluids, and a refrigerator and a freezer for the storage for blood products. A small counter with storage cabinets, drawers, a sink, and water tap must be provided to prepare medications. A glassed wall can be used to permit visualization of patients and ICU activities while preparing medications if this area is enclosed.

Medical Equipment Storage

Unused drip stands, trolleys, gurneys, pediatric incubators, portable suctioning devices, ultrasound machine, mechanical ventilator, and so on cannot stand in the patient area, as they contribute to a cluttered, disorganized appearance of the ICU. This equipment is best stored in a separate room with easy access for retrieval (Figure 3.8). Appropriate, grounded electrical outlets should be provided within the storage area in sufficient numbers to permit recharging of battery-operated items. Lockable storage should be provided for small but valuable items and fiberoptic equipment.

Consumable Supply Storage

Any ICU depends heavily on a multitude of disposables and recyclable equipment, like sterile minor surgery packages. Depending on the size of the practice or hospital, a three–stage storage management system may be desirable. The central supply of the hospital/clinic constitutes the first storage area. The second storage area consists of a utility room adjacent to the ICU. Storage can be organized on open scaffolding, on shelves, in cupboards, and in drawers. A desk with a computer and sufficient room for administrative papers allows for convenient management of inventory. The third and final storage space is situated



Figure 3.8 A separate room for storage of medical equipment helps to keep the patient area organized and uncluttered. The walls of the room can be fitted with shelves, rails and power sockets. Part of the wall should be kept clear to accommodate free-standing items. Shelving should be shallow for easy location of desired equipment.

in the patient area itself for items frequently used. Attempts to stock everything in the ICU itself causes overcrowding, as needs always grow over time.

Soiled Bedding and Waste Disposal Storage

There should be a dedicated area for storing soiled bedding, waste materials, and material for recycling until collection. This material should not be kept in the main ICU patient care area. The size and nature of this space will depend on the arrangements for collection. If the practice or hospital has a central facility, this can also be used.

Facilities Outside the ICU Complex

What should be part of the ICU complex and what should be supplied in the rest of the hospital or clinic is somewhat arbitrary. In addition to the numerous previously mentioned requirements, other functions and rooms that must be considered are a kitchen for preparation of patient food, a staff room for any onsite overnight watch, a medical office including a library, individual office space, a storage area for patient records, a receptionist's office and area, a waiting area, an owner visiting room, a patient groom room, and a linen room. Furthermore, ICU staff require ready access to facilities for changing with showers, toilets, and lockers. Both the changing room and lockers must be individually lockable.

Interior Design

Environment

Lighting

Ambient lighting should be available throughout the ICU, and especially around the cages and in the treatment areas. Disruptions in circadian rhythm may have a negative impact on critical illness [11, 12]. It therefore seems prudent to have adjustable lighting in the patient area, from both natural and electrical sources. The lights should dim without flicker. If windows or skylights are present, shading devices should be in place and easily controlled and cleaned. It may also benefit the unit to have tinted glass panes in the exterior windows. During the night, the main lights in the facility should be dimmed or turned off completely.

The patient modules and treatment areas should have spotlighting that can be controlled independently to prevent increased lighting for other patients, especially during periods of rest or at night (Figure 3.9). Mobile spotlighting and lighting at low level under the cages or tables to illuminate drains and underwater seals (i.e. for thoracostomy drainage systems) may be added. Treatment areas should have separate procedure lighting with adjustable intensity, field size, and direction, to properly evaluate patients or perform procedures. Independent lighting of support



Figure 3.9 (a) Every patient module should have its own light. (b) Each module is lit by bright LED lights that can be dimmed stepwise and do not emit unwanted heat.

areas, such as the handwashing stations, medication preparation area, and charting areas, can be beneficial.

Heating, Ventilation and Air Conditioning

The air-conditioning system to the ICU patient area should provide a thermoneutral temperature of 60–80°F (16–27°C) and a relative humidity of about 30–60%, while avoiding condensation on wall and window surfaces. Control of humidity and condensation can be a challenge, as regular cleaning of cages and floors requires abundant amounts of water. System capacity and room air change frequencies should take this into account. Ventilated air delivered to the ICU should be filtered and the ventilation pattern should inhibit particulate matter from moving freely in the space. The air-conditioning system should be accessible such that regular monitoring and maintenance services may easily be performed.

Acoustic Design

Reduction of both incoming noise and internally generated noise should be considered [13]. Background noise produced by equipment and mechanical systems such as air conditioning should be minimal and absorbed as much as possible. Floor coverings that absorb sound should be used, keeping the need for infection control, maintenance, and movement of equipment under consideration. Walls and ceilings should be constructed of materials with high sound absorption capabilities. Especially in smaller rooms such as the isolation ward, special attention should be paid to unacceptable noise levels as a result of barking.

Ambience

Staff Everybody remembers the classic gloomy, white appearance of hospitals. However, when patients are monitored, treated, and cared for around the clock, special attention should be paid to alleviate this strenuous working environment and aid wellness. The ambient atmosphere can have an enormous and positive effect on potentially stressed staff, owners, and sick animals away from their familiar homes. Words like soothing, relaxed, bright, calm, organized, pleasant, and familiar should apply. Not only visual but also auditory input, such as from audio equipment, helps to create this pleasant environment for both staff and patients. White or gray should not be the dominating color in the ICU. Natural daylight with an outside view is essential for both patients and staff. Visual distractions such as pictures and other features such as plants should not be reserved for the owner visiting room but should be strategically placed throughout the ICU to enhance a positive atmosphere.

Consideration should be given to provide a space easily accessible to all staff that will provide an opportunity for exposure to higher-intensity light levels for at least 15 minutes per shift to ameliorate the effects of working at night and "seasonal affective disorder" [14, 15]. The presence of natural daylight is also considered beneficial to staff and owners.

Patient There is a growing awareness that the design of the ICU environment should support the wellbeing of patients. It needs as much attention as anything else in the design process. Disruptions of the circadian rhythm and the normal routine of the patient while hospitalized may even have a negative impact on the wellbeing of patients and their recovery [16]. Besides the impact of light, noise and temperature, the impact of odors, especially in veterinary patients with a different sense of smell from humans, should not be underestimated [17]. Design measures to limit the effect of odors should be carefully considered.

In the ICU, cats and dogs are still often housed in the same patient area, with higher priority given to other intensive care principles than patient comfort and stress reduction. However, there is growing awareness and evidence that both patient groups, but especially cats, need a species-specific approach also in the hospital [5]. Although an appropriate balance and solution has not been reached yet, whenever possible, cats should be housed in a separate area to reduce visual, auditory but also odor stressors produced by dogs (see Recommended Reading).

Owner Owners are becoming more aware of their pet's medical status and want transparency in regard to it. It enables them to cope with the anxiety caused by the hospitalization of their companion and enables them to make informed decisions about their pet's care. However, visiting opportunities often need to be restricted with limited staffing and time. Video streaming of patient images over the Internet could be an additional means to address the need of the owner to be more involved and stay connected to their pet while hospitalized [18]. Images can be produced by webcams that are directed at the ICU cage and are linked to the internet. Via a dedicated website, owners have access with the use of a password protected logon. They can view the video images produced by the webcam if it has been turned on by the staff.

Utilities

Electrical Power, Network, and Communication Cables

Electrical power, network, and communication cables should be routed via easily accessible conduits that are wall-mounted or located above the ceiling to make additional cabling relatively simple. The power supply must be single-phase with a single common safety ground. Supply lines must not cause interference with monitoring or computer equipment. Standard multiplex electrical outlets may not be suitable, since some outlets may not be accessible when oversized equipment plugs are in use.

It is critical that the ICU staff have immediate access to the main electrical panel if power must be interrupted or restored in case of an electrical emergency. A set of lights in the ICU and several power outlets supported by a backup generator should be available in case of a power outage.

Water Supply and Plumbing

Sinks with hot and cold running water supplies must be located throughout the ICU. Depending on local regulations, heating boilers that reduce the risk of bacterial growth in the plumbing system must be installed directly adjacent to each water supply. Sinks, water taps, and their direct surroundings are notorious for harboring bacteria that can cause nosocomial infections. It is thus important to have separate stations for handwashing (see the section on personal hygiene), cleaning of equipment, and disposal of (contaminated) bodily fluids. The installation of showerheads can help with cleaning both equipment and the sink itself. The sinks should be large and deep enough to ensure that the direct surroundings are not easily soiled. Screen barriers around the sink can help to prevent contamination of the direct surrounding (Figure 3.12b). Separate water fountains for drinking should be supplied.

Gas Supply

The ICU patient areas should be fitted with connections for central oxygen, compressed air, and vacuum. Gas scavenge systems should be implemented at the design stage if sedation with use of nitrous oxide or volatile anesthetic gases in the ICU is planned.

Service of the systems can be made easier by leading the pipes through wall- or ceiling-mounted, water-resistant conduits. The choice of service location (e.g. wall, stalactite, stalagmite) will have a major impact on the service arrangements and on operational use. The outlets must consist of keyed plugs to prevent accidental interchanging. For detailed requirements for pressures and flows, human guidelines can be applied with specific local guidelines taken into account (see Recommended Reading).

Floors, Walls, and Ceilings

There are many things to consider regarding the surfaces in the patient area. Floor surfaces should be easy to clean and should minimize the growth of microorganisms. They should be highly durable and dense to withstand frequent cleaning and heavy traffic. They should keep their new appearance and glossiness, have acceptable acoustical properties, and give enough grip for staff and patients to walk upon safely, even when wet. In addition, the floors should be aesthetically pleasing to the eye. To choose an appropriate floor for the patient areas is certainly a challenge. It helps in the design phase to pay special attention to floors when visiting other facilities, with a focus on durability.

The walls should be easy to clean, noise-reducing, and durable with extra protection provided at points where contact with movable equipment is likely to occur. Some walls may need to be reinforced if wall-mounted cages, rails, or shelves are used.

Ceilings should also be easy to clean, noise-absorbing, and invulnerable to dust accumulation, both on the ceiling itself and on ceiling-mounted light fixtures. The ceiling structure in the patient module or procedure area should be strong enough to carry the weight of any suspended equipment.

Furnishings

In the patient area the use of cabinets with doors and drawers is preferred over shelves, as closed cabinets contribute to a visually uncomplicated environment. Designs specific for use in medical facilities are ideal, as they have standardized sizes and take into account unique requirements of the medical field.

Chairs and free-standing furnishings such as cabinets and carts in the patient area and ancillary rooms should be easily cleanable with minimal seams (Figure 3.10). Countertops, in particular, should have few or no seams. Furnishings should be of durable construction.

Disposal of Waste

Separate receptacles for biohazardous and non-biohazardous waste, according to local regulation, should be available throughout the ICU (Figure 3.11; see



Figure 3.10 Furniture that is used in the patient area and ancillary rooms should be easily cleanable, with the fewest possible seams.



Figure 3.11 Separate disposal bins should be available for biohazardous and non-biohazardous waste throughout the ICU according to local legislation. The use of a foot control limits the contact of hands with the waste bin.

Chapters 62 and 65 for more details). Sharps disposal containers should be present on or near every counter to facilitate quick and easy disposal.

Medical Equipment

A comprehensive list of required equipment is beyond the scope of this chapter; Table 3.2 gives an abbreviated outline of ICU equipment possibilities. Many pieces of equipment are discussed throughout this book, so the discussion here has been limited to a general overview with some specific ICU-related considerations.

Laboratory Equipment

Equipment in an ICU laboratory must provide results quickly. Furthermore, as nonspecialized personnel often need to make use of these machines, they should also be easy to use, maintain, and calibrate.

Table 3.2 General overview of location and type of equipment in the intensive care unit.

General use	Location	Type	Apparatus/function
Diagnostic/monitoring	Laboratory	Sample preparation and storage	Blood tube centrifuge
			Microtube centrifuge
			Cytospin centrifuge
			Freezer
			Refrigerator
			Heating incubator
			Ice-making machine
		Measurement	Microscope
			Hematology
			Coagulation
			Biochemistry
			Blood gas machine
	Patient module and procedure area	Monitoring	Electrocardiograph
			Ultrasound machine
			Blood pressure monitors (invasive and non-invasive)
			Pulse oximeter
			Capnograph
			Continuous body thermometers
			Scale
			Etc.
Therapeutic	Patient module/medical equipment storage	Continuous infusion	Syringe pump
			Volume fluid pump
		Mechanical ventilation	Mechanical ventilator
			Humidifier
			(oxygen/air blender)

Table 3.2 (Continued)

General use	Location	Type	Apparatus/function
		Renal replacement therapy	Hemodialysis
			Continuous renal replacement therapy
			Plasmapheresis
		Other	Mobile suction unit
	Procedure area	Cardiopulmonary resuscitation	Crash cart
			Defibrillator
Other	Patient area	Patient care	Central supply cart
	ICU	Temperature control	Refrigerator
			Freezer
		Disposal	Non-biohazardous waste receptacle
			Biohazardous waste receptacle
			Sharp safe
	Procedure area		Heating incubator
	Soiled utility/holding room	Disposal	Receptacle for soiled fleeces and blankets
			Receptacle for surgical instruments

Monitoring Equipment

While in human ICUs the bedside monitoring setup is complete and committed to one bed only, this is seldom financially feasible in veterinary medicine. To make more economic use of the expensive equipment, extensive monitoring is either limited to those cages that are intended for the most critically ill patients; monitoring devices are positioned in such a way they can service multiple cages; or the equipment is mobile and can be moved between patient modules as necessary. In some situations, every cage can be set up with monitoring modules that deliver a basic set of monitoring parameters (Table 3.2). The setup chosen is based on the design of the ICU, the type of patients admitted to the ICU, staff preferences, and financial constraints. The introduction of a modular setup for multiparameter monitoring devices is a very attractive development.

Of special interest are telemetry monitors, which offer several advantages. Telemetry is mostly used for electrocardiography, but larger setups can be used for multiple parameters. When used for one or two parameters, the transmitter device is often small enough to tape to the patient. This allows freer movement and minimizes the patient getting caught in wires and leads. Also, all collected data can be sent to a central monitor in the technicians' station, which supports continuous monitoring of the parameter(s).

Crash Cart

In settings such as the emergency room and ICU where cardiopulmonary arrest has to be anticipated, it is important to have a complete set of equipment and supplies for CPR available and ready for use at all times. As an arrest can occur anywhere in the ICU, a mobile cart or trolley is preferred over a fixed CPR station [19]. However, the arrest cart should be placed in a designated, clearly indicated area to retrieve it without delay in case of an emergency.

Advanced Supportive Therapies

Patients receiving mechanical ventilation, renal replacement therapy and plasmapheresis constitute the "high end" of patient care in the companion animal ICU. A mechanical ventilator for the ICU should have an easy-to-use interface, give a wide array of options for different ventilation settings, and should be able to ventilate a wide range of patient sizes. Mechanical ventilators designed for use in anesthesia generally do not suffice. Equipment that specifically monitors respiratory parameters related to tidal volume, gas flow, and airway pressures should be part of the ventilation unit. The ventilator should be equipped with a humidifier and heating unit, as a heat and moisture exchanger device does not work in all patients. Though they are built into modern critical care ventilators, some older units may need an ancillary oxygen and air blender.

The options for renal replacement therapy in veterinary medicine are increasing. For more information the reader is referred to the literature [20].

Central Supply Cart

Although the patient area may have enough room to stock all necessities for direct patient care, it is often convenient to have one or two central supply carts that contain the commonly used equipment and disposables (Figure 3.10). A limited range of frequently used drugs such as potassium chloride, sterile water, or sodium chloride 0.9% to use as diluent for patients' medications or flushing fluid lines, eye lubrication, and xylocaine jelly can be stocked in this cart. The cart can be moved to anywhere in the patient area as needed. If the top of the cart is kept free, it offers an extra area to place, prepare, and organize equipment and disposables prior to an intervention.

Special Design Considerations

The discussion so far has addressed the physical components ("hardware") of the companion animal ICU. However, the organization or "software" is also an essential aspect of ICU design [21]. Intensive care includes many different staff, and intensive care medicine requires and can only be successful with a team approach. The team consists of different groups such as managers, veterinarians, technicians, and animal care assistants, and cleaning staff. Staffing of the ICU regarding number, qualifications, individual job descriptions, and appropriate training is the cornerstone of the ICU organization and the service it can provide its patients and clients. As in any larger organization, cooperation and effective communication between the different groups determines the quality of the care provided and ultimately patient outcome. In recent years, several directives have been published by critical care organizations in human medicine to define optimal staffing and operational requirements [4, 9, 22]. Such directives have identified IPC and written communications as important management aspects that contribute to the performance of a facility.

Infection Prevention and Control

The frequency and impact of hospital-acquired or nosocomial infections and infections by multidrug-resistant pathogens in veterinary ICUs is a growing concern. Improvement and refinement of our antimicrobial strategies no longer suffice, and additional measures that prevent these infections from developing in the first place are becoming a major focus. The spread of pathogens can occur by direct and indirect contact with people, other patients, equipment, and the environment. The risk of infection may be relevant not only to patients but also to the caretakers, as some of these microbes have the potential to be zoonotic. The focus here is on the measures that can be taken from the point of design to prevent the establishment and dissemination of infections in the ICU.

Infection and prevention control measures dominate the ICU design in human medicine [4, 9, 22] (see also Recommended Reading). The concept for the necessity of implementing effective measures that may limit the number of nosocomial, and particularly multi-resistant, infections into veterinary hospital policies appears to lag behind [23]. Therefore, the next challenge will be to implement the increasing knowledge and information on IPC into the design process, as design aspects can significantly affect discipline and behavior of staff. For example, the absence of proper facilities can frustrate and inhibit the intention to follow hand hygiene protocols.

However, adjustments in the ICU design will not be effective by itself as an abundance of sinks will not necessarily ensure that handwashing behavior improves. The lack of compliancy of personnel in the ICU is a great concern and a challenge for anyone who is responsible for developing hygiene measures in the ICU [24]. The establishment of an IPC team can be very beneficial, not only to prepare policies and to initiate interventions to reduce the spread of pathogens but, more importantly, to educate and continually encourage staff to follow these policies in their daily duties [25]. Packages of measures known as "care bundles" have been demonstrated to be an effective preventive method [4].

Personal Hygiene

Staff and visitors should be made aware that they may act as a reservoir for nosocomial microbes and should understand their roles in the possible introduction of these pathogens to the ICU. They can also play a role in the spread of nosocomial infections *within* the ICU. The first measure to reduce the introduction and spread of nosocomial infections is to reduce any unnecessary contact. It is important to reduce unnecessary traffic in and out of the ICU, but also between patients. Moving from one patient to another should trigger hand hygiene measures and a change in barrier equipment (gloves, gown/apron etc.). The team members' personal equipment, such as thermometers and stethoscopes, should be cleaned and disinfected between patients. Thermometers could have a thermometer cover applied and changed between

patients. Also, visitors should be made aware of their role in spreading infections using posters and informational brochures. The ICU team should indicate and demonstrate to owners what, how and when to wear the required PPE/barrier nursing attire to handle their pet within the ICU.

There is an essential awareness that nosocomial infections originate and are spread primarily by those parts of the body that make the most contact with the patient: the lower arms and hands of personnel. Extensive instructions have been described for proper hand and lower-arm sanitation [24, 26, 27]. The ICU should be designed to facilitate any attempt to maintain a high level of hygiene. Handwashing and disinfection facilities should be strategically placed outside the entrance and within the ICU (Figure 3.12). The stations should be used only for handwashing and not to clean dishes or other equipment or to dispose of organic materials such as bodily fluids. The stations should have hot and cold running water that can be turned on and off by hands-free controls, and the basins should be large enough for surgical handwashing. To dry one's hands, disposable towels or hot air dryers can be used. An antimicrobial hand-rub (often alcohol-based) dispenser should be present at every handwashing facility. The setup should be complemented with clear instructions on when and how to wash and disinfect hands and lower arms correctly. Additional antimicrobial hand-rub dispensers should be present at strategic points throughout the ICU (Figure 3.3b). The dispensers are a visual cue for cleanliness and when used appropriately, can be quickly used before and after handling an animal and before touching pens, keyboards, and so on [25, 28].

Barrier nursing attires such as examination gloves and gowns are most effective in the event of contact with soiled materials and during the disposal of bodily fluids. Face and eye protection should be worn if aerosols are likely to be generated [25, 28]. The primary role of barrier nursing attire is personal protection and to reduce contact and soiling of one's own hands and clothes. A convenient and easily accessible stock of protective barrier materials and bin to dispose of them in the patient area supports staff to use, and more importantly, regularly exchange them.

Patient-Related Considerations

Infected or colonized animals may act as reservoirs for further transmission to humans or other patients; this is an important reason to limit interpatient contact and respect a certain distance between cages, which also can facilitate the addition of temporary barriers. In the presence of a known multidrug-resistant microbial infection or an extremely immunocompromised animal, pre-emptive isolation measures may be warranted and should be available within the facility.

But the main focus should be the awareness that an infection with all its detrimental consequences can only develop *after* contamination. The risks of contamination and the way to reduce them are well known in relation to intravascular catheters, urologic catheters, and mechanical ventilation [27, 29]. Every ICU should have protocols that address the proper application and standard care of these devices or



Figure 3.12 (a) Initial handwashing station in pharmacy (b) Adjustment of station with the introduction of glass partitions preventing water spillage from the handwashing station on the counter on which medications are prepared. The adjustment was instigated by a *Serratia marcescens* epidemic in a Dutch human ICU caused by a contamination of infusion fluids as a consequence of similar circumstances as shown in (a).

procedures, with emphasis on the prevention of contamination. Protocols may recommend the location where the intervention should take place (cage, ICU treatment area, surgical theater); the surgical preparation of the procedure site, hand hygiene and the use of PPE/barrier nursing attire; the use of sterile equipment and single-use disposable equipment; and the proper disposal of contaminated material.

Environmental Considerations

Proper cleaning with removal of all (biological) dirt must precede disinfection efforts, as many disinfectants are inactivated prematurely if in contact with biological material. It is important to screen the cleaning protocols and address the effectiveness of the disinfectants to eradicate pathogens. In this respect, surfaces that are in regular contact with personnel and patients are important to address. These surfaces can be difficult to clean, such as computer keyboards, monitoring equipment, infusion pumps, telephones, doorknobs, patient cages, and the handles of cabinets and drawers. During the design process the choice of materials should receive explicit consideration in this respect, for example, by the introduction of fully submersible keyboards that can easily be cleaned and disinfected.

The main contact area for a patient is its cage. Cages and their bedding should be cleaned regularly, at least once a day and immediately after soiling. Disinfection should be performed after discharge or whenever sensible, such as during a long patient stay. All these measures have proven less effective if not combined with adequate numbers of staff and suitable space and facilities [30].

Decontamination measures are not the only strategy that may be applied. The movement of people and patients makes long-lasting decontamination of the floor an impossible task. Therefore, the floor should always be considered "contaminated," and any contact with it should be followed by stringent cleaning and disinfection of anything that has been in contact with the floor. This strategy relates to persons, patients, equipment, disposables, and other materials. If it is not possible to clean and disinfect, the object (such as a blanket or a syringe that is accidently dropped) that has been in contact with the floor should be discarded. The floor should not be considered a convenient "table", and nothing should be stored on it without additional precautions.

Routine Culturing of Patients and the Environment

An infection control team or infection control experts should be consulted to advise on the use and sense of sampling. Routine culturing may have some merit to establish potential risk sites in the ICU. Results should be used to change surfaces or equipment, and guide and improve cleaning and disinfection procedures.

While much can be done within the ICU itself, multidrug-resistant infections in the ICU often reflect difficulties elsewhere in the hospital and in veterinary health service generally, in terms of the control and prevention of healthcare-associated infection [30].

Written Communications

Patient Record Keeping

Proper record keeping is an important part of patient management and serves a specific purpose. Primarily, it ensures and improves communication among ICU personnel regarding the patient. But it is also important for retrospective evaluation of the care given to the patient, both for internal quality control, research, and external scrutiny.

Medical Record The medical record (Chapter 5) is mainly kept by the veterinary staff and is often held in digital form. The record describes the complete period of the patient's hospitalization until discharge. From any period of hospitalization or visit the record should contain at least information on the patient's signalment, history, physical examination, problem list, rule-outs, additional diagnostic and therapeutic plans and interventions, and progress notes, including (presumptive) diagnosis, treatment, and prognosis [31].

Daily ICU Orders It is the responsibility of the overseeing veterinarian (closed ICU) or the case clinician (open ICU) to write the orders that describe diagnostic, therapeutic, monitoring, and nursing care instructions for the next 24 hours for each patient (Figure 3.13). These orders can be written directly in the patient treatment or "flow" sheet, but given the complexity of ICU patient orders and the frequency with which critically ill patients' orders change in a day, a separate order sheet is more useful. It is the task of the technician to be able to read, interpret, and evaluate the orders, and to clarify with the clinician what is not understood.

A dedicated sheet with patient-specific information related to the event of CPR, such as owner's resuscitation wishes, drug dosages, and owner and primary veterinarian contact information, may be part of the daily ICU orders.

Flow Sheet The essence of critical care is continuous patient monitoring, which is beneficial only if the data is collected and recorded in a useful manner. A patient flow or hospitalization sheet is the traditional method for recording all information relevant to an ICU patient for a 24-hour period. Maintaining the flow sheet is primarily the responsibility of the nursing staff caring for the patient

VETERINARY MEDICAL TEACHING HOSPITAL
UNIVERSITY OF CALIFORNIA, DAVIS

CLINICIAN: ANONYMOUS
PHONE # 555-7480 PGR:
STUDENT: JANE
PHONE # : PGR:

SAICU - CLINICIAN ORDERS - ENTRIES MUST BE SIGNED AND UPDATED TWICE DAILY

DATE 1/22/09 TIME 8:00 AM / PM

FLUIDS /MEDS / TREATMENTS	DOSAGE	ROUTE	START TIME
NaCl 0.9% qs pp @ 100 ml/hr			
FENTANYL 0.1-0.4 µg/kg/min (KEEP AS LOW AS POSSIBLE)			
CEPHAZOLIN 25 mg/kg IV SLOW q 6 hrs			
PHENOBARBITAL 4 mg/kg IV q 12 hrs			
DEXAMETHASONE SP 0.1 mg/kg IV @ 10 AM			
FAMOTIDINE 0.5 mg/kg IV OR SQ SID			
SEIZURE WATCH — IF SEIZURES, GIVE MIDAZOLAM 0.25 mg/kg IV BOLUS AND START CRI @ 0.25 mg/kg/hr + CALL DR A			
KEEP HEAD ELEVATED 30°			

Monitor:
Observation q 4 hr (mentation; breathing rate; breathing effort)
☑ Vital signs q 12 hr (observations plus: heart rate; auscult heart & lungs; mucous membr color & CRT)
Temp q 12 hr ECG q ___ hr Doppler BP q ___ hr Dinamap BP q ___ hr Direct BP q ___ hr CVP q ___ hr
Pulse Oxim q ___ hr Urine output q ___ hr ___ q ___ hr ___ q ___ hr ___ q ___ hr

Laboratory:
☑ ICU panel 1 q 8 hr Art/Ven (oximetry, blood gases; electrolytes; glucose; lactate)
☐ ICU panel 2 q ___ hr (panel 1 plus osmolality; colloid osmotic pressure)
☑ PCV/TS q 24 hr ☐ Lactate q ___ hr ☐ Vet-test BUN q ___ hr ☐ Urine Sp Gr q ___ hr
☐ ACT q ___ hr ☐ Glucom q ___ hr ☐ Vet-test Creat q ___ hr ☐ Urine dipstick q ___ hr
☐ COP q ___ hr ☐ ABL glucose q ___ hr ☐ ABL electrolytes q ___ hr ☐ Urine electrolytes q ___ hr
☐ Osmolality q ___ hr ☐ ABL blood gas q ___ hr ☐ ___ q ___ hr ☐ ___ q ___ hr

General nursing care:
Standard care: Weigh q 24 hrs; indwelling catheter care as per protocol; heparin lock unused catheters
☐ Recumbent care: Standard care + resposition and physical therapy q 4 hrs
☑ Comatose patient care: Recumbent care + oral & eye care as per protocol
☐ ___ q ___ hr ☐ ___ q ___ hr ☐ ___ q ___ hr

Nutrition:
☑ NPO ☐ NPO except oral meds ☐ No food, water ad lib ☐ No food, offer ___ ml water q ___ hr
☐ Water ad lib; feed ___ (amount of food) of ___ (type of food) q ___ hr ☐ TPN: ___ (formula) as per protocol; goal: ___ (daily Kcals)

Contingencies (change/cancel any default contingenies?)
ANY ACUTE CHANGE IN MENTAL STATE OR NEUROSTATHS OR RESPIRATORY PATTERN, PLEASE CALL DR A.

CPR status: ☐ Full CPR ☐ Closed-chest CPR ☐ No CPR Signature: Anonymous

Form #38SRevised 12/23/04

Figure 3.13 Daily order sheet with written instructions for the patient's care.

(Figure 3.14a). The flow sheet is used to document the results of "bedside" diagnostic, therapeutic, monitoring, and the associated nursing care instructions, and to document that the orders have been carried out with the associated time of completion [32]. Flow sheets are helpful in registering subtle trends, and as such, their design should include tables and graphs (Figure 3.14b). The flow sheet can contain both subjective and objective assessments. However, subjective assessments and observations are best communicated verbally with the attending veterinarian and others involved in the patient's care and then recorded on the record.

Protocols

In a complicated environment such as an ICU, having consistent protocols and standard operating procedures (SOP) to describe best practices and techniques is essential. The development of such protocols should include the close cooperation between all hospital groups, both within and outside the ICU, that are involved in patient care or the organization of the ICU. Protocols and SOPs are necessary for several reasons:

- To improve, support, and reduce unnecessary repetition of oral communications and instructions among the team.
- To standardize patient care, organization, hygiene, and other strategies among team members.
- To disseminate the best available knowledge so that it may be implemented if appropriate (evidence-based veterinary medicine). Protocols should contain references when available.
- To record and describe in clear detail any actions that need to follow legal instructions or legislation; for example, the use of controlled substances in the ICU.
- To scrutinize, audit and review the strategies that are described within the protocols.

Every protocol or SOP should contain the following information: title, classification or subject group, names of author(s), date of compilation, names of author(s) who made changes, dates of amendments, date of review, and the persons to whom the protocol or SOP is of interest. For administrative purposes it may be helpful to introduce a coding system.

Protocols and SOPs are often written by different people, with the ICU staff often performing the initial writing. It is a challenge to keep them as effective as possible by keeping them concise, informative, and complete without becoming too elaborate. Proper referencing can help the interested reader to find more background information if necessary. The ICU protocol and SOP book is constantly under construction and never finished as a result of rigorous scrutiny and improvements, adaptation to the latest information, and adjustments to changes in the ICU organization. Books published in recent years can be very helpful in setting up an ICU procedure book [33]. Many protocols and SOPs are available throughout this textbook to serve as a starting point.

Bulletin Boards

There is always a need for brief communications among the staff. Bulletin boards are most often used to communicate in this fashion and have the additional advantage of being available to everyone with one quick glance.

A strategically placed patient board (Figure 3.15) contains the numbers of the patient modules, the patients' names that occupy them, their problems and/or diagnosis, their location if not in its cage, their therapies, the current plans, and the name of the staff member who bears primary responsibility. Additional patient identification numbers and a telephone number or other contact information of owner and staff can be helpful. This is of benefit during busy times to keep overview of all patient-related activities.

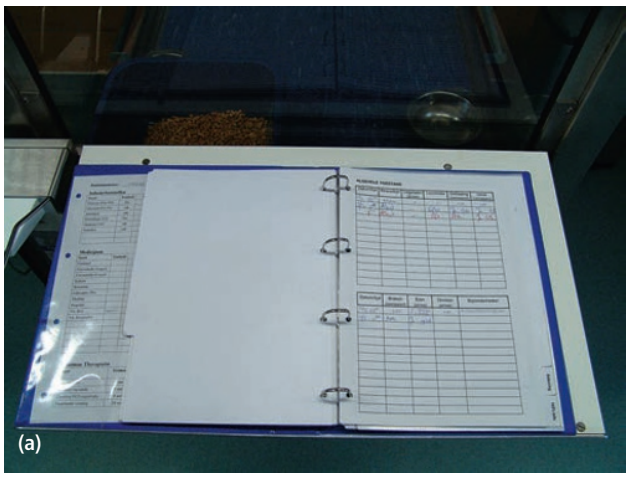

(a)

Figure 3.14 (a) At the ICU at the Department of Clinical Sciences of the Utrecht University, the flow sheet consists of a folder with divider sheets for different aspects such as patient particulars, problem list, monitoring, fluid therapy, and medications. (b1, b2) Tables and graphs are important means to present the collected information in an orderly fashion that facilitates the interpretation of data.

(b1)

ICU LABORATORY

TIME	1752	1820	2202	0208	0606	0810
PCV (%)	37			46	45	
TP (Gm/dl)	4.7			5.6	5.5	
Plasma color	CLR			CLR	CLR	
Plasma transparency	CLR			CLR	CLR	
Arterial/Venous	A	A	A	A	A	A
Body temperature (C/F)	99.5	98.4	98.5	99.2	99.5	99.5
Inspired oxygen (%)	21	74m	21	30	21	25
Hemoglobin (Gm/dl)	12.1	12.2	13.0	14.6	14.0	13.7
Oxyhemoglobin (%)	91.6	98.8	91.9	97.4	99.5	96.0
Methemoglobin (%)	0.1	0.2	0.0	0.0	0.0	0.2
Carboxyhemoglobin (%)	0.4	0.3	0.6	0.7	0.6	0.6
Oxygen content (ml/dl)	15.3	16.7	16.5	19.6	17.8	18.1
Potassium (mEq/L)	3.9	4.0	3.9	3.8	4.1	4.1
Sodium (mEq/L)	148	147	146	148	150	149
Calcium(ionized) (mM/L)	1.34	1.39	1.38	1.41	1.40	1.41
Chloride (mEq/L)	120	118	117	120	124	121
Glucose (mg/dl)	130	125	150	140	139	137
Lactate (mM/L)	0.9	0.6	0.5	0.3	0.2	0.2
pH tc (units)	7.207	7.199	7.225	7.243	7.229	7.233
PCO_{2tc} (mmHg)	54.1	56.7	54.4	49.5	52.3	50.5
PO_{2tc} (mmHg)	86.1	192	83.6	123	85.2	109
A-a pO2 (mmHg)						
SBE c (mEq/L)	-6.1	-5.6	-4.8	-5.6	-5.4	-5.8
HCO_{3c} (mEq/L)	20.5	21.3	21.7	20.5	20.9	20.4
COP (mm Hg)						
Osmolality (mOsm/Kg)						
Creatinine (Vet-test)						
BUN (Vet-test)						
Azostick						
Glucometer						
Activ Clotting Time (sec)						

Urinalysis Time	1800	2200	0200	0600
Collection method	UCS	UCS	UCS	UCS
Color	YELL	YELL	YELL	YELL
Transparency	CLR	CLR	CLR	CLR
Specific Gravity	1.016	1.013	1.010	1.014
Osmolality (mOsm/Kg)				
Sodium (mEq/L)				
Potassium (mEq/L)				
Chloride (mEq/L)				
Protein	TRACE			
pH	7			
Blood	+++			
Ketones	NEG			
Bilirubin	1			
Glucose	NEG			

Figure 3.14 (Continued)

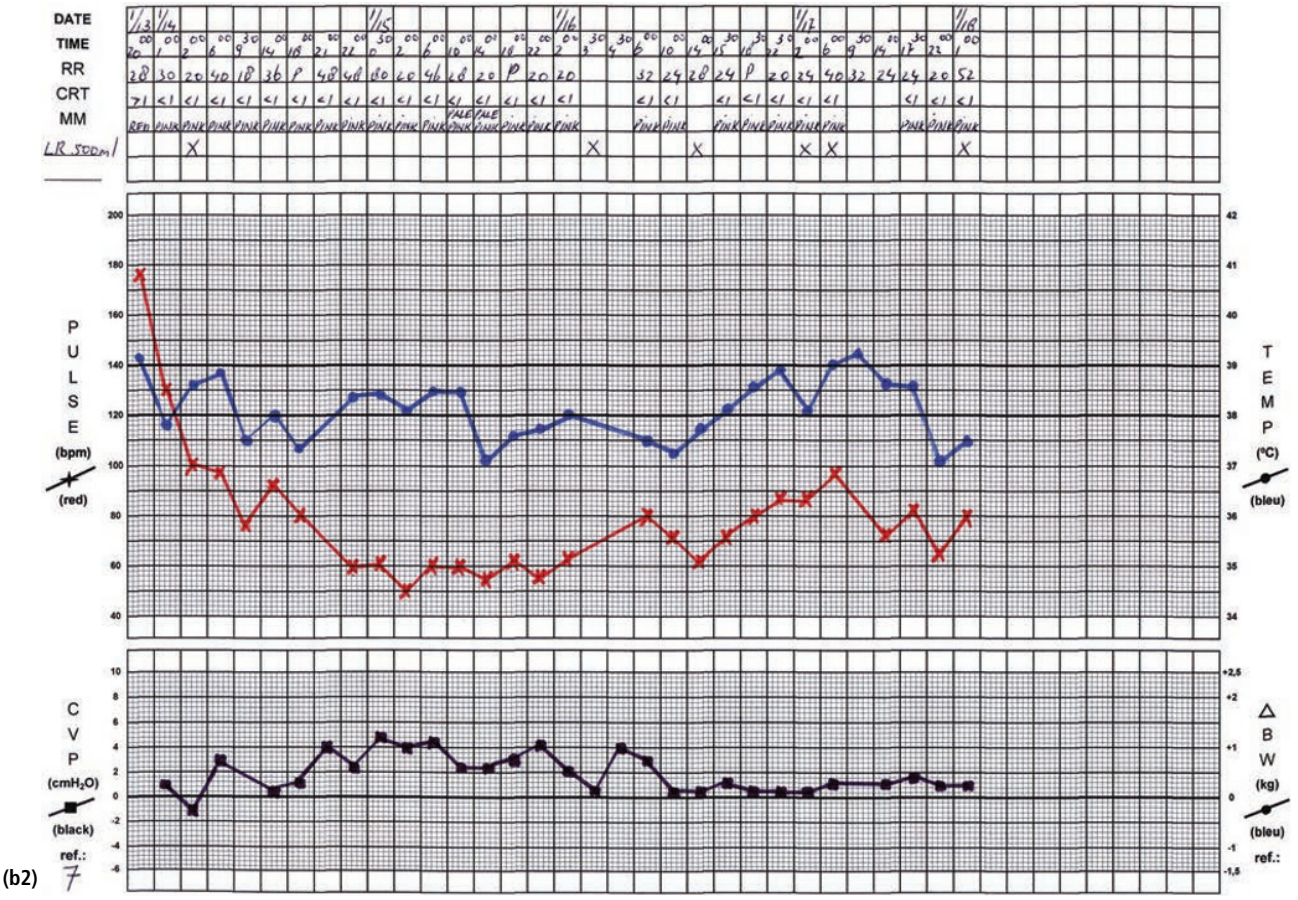


Figure 3.14 (Continued)



Figure 3.15 A strategically placed patient board containing accurate, current information gives anyone who requires it a quick and complete overview of ICU patient status.

When kept up to date bulletin boards can be used to facilitate quick and important communication among ICU staff or between ICU staff and other hospital staff, such as the pharmacy or those responsible for ordering and stocking. The transferred information needs to be kept current so that the information is kept relevant to the time and day. It is especially helpful in maintaining communication among staff on different shifts. With the use of different bulletin boards, different groups or topics within the ICU organization can be targeted on each board.

Safety and Security Measures

Depending on the hospital situation, it may be necessary to have areas of the ICU that can lock from the inside to secure the safety of personnel and patients, particularly after regular office hours. Areas or storage units that contain valuable equipment should be locked. However, ICU staff should have use of all essential equipment and areas; therefore, a key cabinet available to all personnel can be located centrally in the ICU to allow such access.

The safety of patients, personnel, and visitors can be related to fire hazards, other hazards caused by failure or malfunction of services or equipment, and chemical or biohazards. Local fire safety regulations determine certain aspects of ICU design, and the consultation of fire safety officials should be part of the design process. At least two independent escape routes should be available, both

accessible for personnel, mobile patients, and those on gurneys. Most local regulations will require installation of a sprinkler system.

Low-pressure warning systems for the gas services must be visible and audible in the ICU. Shut-off valves or switches for the ICU should be located adjacent to the unit where their operations can be controlled by the staff.

The main electrical panel should preferably be located with easy access to ICU personnel. Each outlet cluster within an ICU should be serviced by its own circuit breaker in the main panel. The electrical panel should be connected to an emergency power source that will quickly resupply power in the event of power interruption. If capacity of the backup power source is limited, special attention should be paid during the design process to identify which operations need backup. Emergency power source sockets should be distinguishable, for example, by color coding.

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Recommended Reading

The International Society of Feline Medicine has described standards for cat-friendly hospitalization facilities:

- Cat Friendly Clinic: https://catfriendlyclinic.org/vets-nurses/hospitalisation-facilities

The following websites provide insight to the approach in human medicine to define and design intensive care facilities. Although far from the reality of companion animal intensive care medicine, the information certainly gives a lot to consider when designing a companion animal ICU, and stimulates "out-of-the-box" thinking necessary to explore design options to the maximum.

- Learn ICU (Society of Critical Care Medicine): www.sccm.org/LearnICU/Home

  Online educational materials, many available only to SCCM members.
- Guidelines for Design and Construction of Health Care Facilities (Facility Guidelines Institute): https://fgiguidelines.org/guidelines/2018-fgi-guidelines
- ESICM guidelines and recommendations (European Society of Intensive Care Medicine): https://www.esicm.org/resources/guidelines-consensus-statements
- Intensive Care Society guidelines: www.ics.ac.uk/Society/Guidance/Guidance
- Faculty of Intensive Care Medicine guidelines: https://www.ficm.ac.uk/standards-safety-guidelines
- College of Intensive Care Medicine of Australia and New Zealand: www.cicm.org.au/Resources/Professional-Documents#Statements

4

Developing and Using Checklists in Practice

Elizabeth B. Davidow and Carmen King

> *"Just ticking the boxes is not the ultimate goal here. Embracing a culture of teamwork and discipline is."*
>
> Atul Gawande, *The Checklist Manifesto* [1]

Introduction

In 2005, research published in the *Lancet* [2] showed that providing handwashing instructions, soap and a six-point checklist of when to wash could dramatically decrease the incidence of common diseases in children in Pakistan. Households that were provided the soap, instructions and six-point list of when to wash had 53% less diarrhea, 50% less pneumonia, and 34% less impetigo than control households. It did not matter if the soap was antibacterial or not. Many households already had soap prior to the study and much of the difference was attributed to the clear six-point checklist of *when* to wash.

A year later, Peter Pronovost published an article in the *New England Journal of Medicine* [3], which showed that use of a simple five-point checklist, when administered by nurses in 103 intensive care units across Michigan, could dramatically decrease the incidence of central line infections. The checklist is simple:

1) Wash your hands.
2) Clean skin with 2% chlorhexidine.
3) All involved wear sterile gloves.
4) No femoral insertion.
5) Ask daily about removal.

The checklist was developed by distilling pages of recommendations from the Centers for Disease Control and Prevention into the five items with the most evidence and the least barrier to use. This created a list short enough that it could easily be read, checked off, and followed with every catheter placement. The finding that a checklist could drop central line infections to near zero challenges the assumption that some complications are unavoidable. If a checklist could avoid central line infections, what other complications could it avoid?

The results of these two studies spurred interest in further uses for checklists in medicine. In 2009, the World Health Organization's (WHO) Safe Surgery Saves Lives program published its surgery checklist study [4]. This study demonstrated that the implementation of a simple checklist before, during and after surgery in eight hospitals in eight different countries decreased both complications and mortality. This checklist added no cost and minimal time but on average, decreased postoperative complications by 36%.

A similar surgery study [5] has now been done in veterinary medicine and has confirmed the human medical findings. In a prospective clinical trial, 300 dogs and cats undergoing surgery were followed to document a baseline incidence of complications and mortality. A surgery checklist was then implemented and used with the following 220 surgeries. The research team found a statistically significant reduction in the incidence of all complications after the implementation of the checklist.

Checklists and algorithms have now been widely adopted in many areas of medicine. However, experience over the last decade has shown that checklists are only successful in saving lives when designed appropriately and implemented carefully.

Why Checklists?

Humans have limitations in their attention span and in their memory. Studies have shown that humans decline in both accuracy and speed when problems have increasing complexity and more variables [6].

Advanced Monitoring and Procedures for Small Animal Emergency and Critical Care, Second Edition. Edited by Jamie M. Burkitt Creedon and Harold Davis.
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Companion website: www.wiley.com/go/burkitt/monitoring

Checklists are used in many industries to assist with these limitations. They are not detailed "how to" instructions but serve as precise directed reminders of crucial steps in a process. The goal is not to teach professionals how to do their job. Instead, they serve as reminders of steps that might be forgotten, even by experienced professionals when they are tired or forced to multitask. They also can be used to bring items front of mind in situations that are less common. In addition, when designed to be read out loud as a group, they promote teamwork within a work environment. Checklists can help a team agree to and provide consistent and reliable care by making sure certain tasks happen with every patient, every time. Finally, if updated regularly, they can help move new evidence into practice faster [7].

Types of Checklists

There are several different types of checklists that can be used in medical settings. They can be roughly divided into three categories: do–confirm, read–do, and dynamic.

In do–confirm checklists, a set of tasks are done from memory but then a list is consulted and checked to confirm that all items have been completed. Another name for do–confirm is static parallel. These types of checklists are useful for tasks such as checking set up of an anesthetic machine prior to a procedure.

In a read–do checklist, each item is read, the task is done and then the item is checked off. These checklists are also known as static sequential. In a static sequential checklist with verification, one person reads the task while a second verifies. This is the type of checklist used for a jugular line placement. In a static sequential checklist with verification and confirmation, there may be multiple people who verify and confirm their specific tasks. This type of checklist is used in surgery.

Dynamic checklists are flowcharts that can be used to guide through a complex process with decision points. These checklists can be created as diagnostic and treatment pathways for specific diseases or medical situations. A dynamic checklist has been used to improve time to antibiotic administration in dogs with septic peritonitis [8].

Design Considerations

Recommended steps in checklist design and implementation are listed in Box 4.1. The first step is the decision that a checklist is needed. It is important to be clear on the specific goal of the checklist so that it is designed with its mission in mind. Are you trying to prevent a specific complication? Avoid missing an important treatment step? Move recent literature into practice? No matter how short, checklists do add some time to a work process and thus they need to be very targeted to the objective.

Box 4.1 Steps in Checklist Design and Implementation

- Decide that a checklist is needed:
  - Define need and desired outcome
- Review the existing literature
- Convene a multidisciplinary group to design:
  - Who
  - When
  - Where
  - How it fits in with other procedures
  - Format (paper, electronic)
  - Content
- Rapidly test and revise prototypes to determine initial version
- Train and implement the initial version:
  - Clear *why* and detailed *how*
  - Anticipate concerns and answer
  - Train in actual situations
  - Debrief and revise if needed
- Continually evaluate and revise
- Build accountability
- Celebrate successes and courageous moments

A review of the relevant medical literature can be helpful to determine which steps have the most evidence behind them in helping to achieve the stated goal. In addition, a literature review may uncover previously developed and tested checklists that can be modified for use.

Checklists will only be used if they are work site specific and fit in a sensible manner with the flow of the day [9]. The most successful checklists are developed by the teams that will use them, not by administrative staff. The team should involve all relevant parties. Surgery checklists are best developed with both the veterinarians performing surgery and the veterinary nurses and veterinary anesthetists involved in these procedures.

The multidisciplinary group should decide who is the best person to run the checklist. If a do–confirm checklist is appropriate, the person doing the checklist is the same person doing the task. In a read–do checklist, the reader has the power to stop a procedure until the step is completed. Both the jugular central line placement study and surgery checklist study showed the highest compliance and success when nurses were set up to run the checklist [3,4].

The multidisciplinary group will also need to decide when the checklist is performed, how it fits into the flow of the procedure, whether the checklist is on paper or electronic, and what items make the list. Ideally, the most critical items are listed near the top. The listed items should be

Box 4.2 Important Elements in Successful Checklist Designs

- Sans serif font
- Lots of white space
- Minimal color
- Precise, simple language
- Most critical items at the beginning
- As short as possible
- Fit on one page

as short as possible and should fit on a single page. Language should be precise and simple.

Studies in the aviation industry have shown that both the content and the appearance matter in creating a successful checklist. Even the font used can influence its usability, with sans serif fonts such as Arial and Helvetica being recommended over serif fonts such as Times New Roman, which can be harder to read quickly [1, 9]. Other important considerations for design are listed in Box 4.2.

Testing

Once an initial prototype is developed, it should be tested in practice by the multidisciplinary group. Most checklists will need a rapid cycle of quick tests and revisions to get a version that can then be tested by a larger group. In the process of testing, it is common to find that the language may not be as precise as thought, that the timing of items needs to be altered, that the list is too long, or that the placement of the checklist needs to be adjusted to fit into the flow of the day.

After a prototype is developed, it should be tested with a small group that was not on the development team. Further modifications will likely be needed. The incorporation of suggestions by this additional group will provide multiple staff who are now vested in the success of the checklist.

After this testing process, the preliminary checklist is now ready to be implemented in the hospital.

Implementation

Studies of implementation of the surgery checklist across hospital systems show that decreases in complications and mortality are directly related to the strength of the implementation process [10–12]. When the WHO surgical safety checklist was implemented in hospitals across Britain, the full expected drop in complications was not seen. A detailed study found that the full checklist was only finished in 62% of cases and in 66% of those cases, not all items were read aloud [11] Resistance from senior clinicians was found to be a large barrier to implementation [12]. During implementation, an effort should be made to anticipate and counteract likely concerns [9]. A study of surgical checklist use in several Washington state hospitals demonstrated that successful implementation included a strong story of *why* the checklist is needed and a detailed explanation of exactly *how* to use the checklist [10].

An important step in implementation is challenging the myth that more experienced doctors do not need checklists. A lesson from aviation can be used. In flight school, pilots are taught that, even when experienced, that their memory and judgment are unreliable and that lives depend on their recognition of this fact [1].

In the author's hospital, the first step in building a *why* for the surgery checklist, was holding a journal club on the book, *The Checklist Manifesto* [1]. Because the book is quite compelling, the *why* was clear. The impetus for a checklist could also come out of a medical error, a missed process step, or a post-surgical complication. In general, people are more compelled to make a change from emotional stories than from facts or studies [1].

A common skipped step in the WHO surgical safety checklist is taking the time for each person to introduce themselves and state their role. While this step does not appear to be related to patient safety, it is actually crucial to build effective teamwork in the case of the unexpected. The most likely surgical complications to result in death are those that are rare such as thromboembolism and ventilator malfunction. Successful response depends on fast teamwork. People work much better as a team if everyone knows each other's name [1].

Once a compelling *why* is described and the staff believes in the process, they then need to be shown exactly *how* to proceed. In-person training using the members of the development team is likely to be most effective. Reference materials for implementation should be developed. Figures 4.1 and 4.2 show a sample surgery checklist and the associated training descriptions for each item on the first part of the checklist.

It can be difficult to incorporate a new process into a daily workflow. Thus, when a new checklist is implemented, reminders and visual clues should be used to encourage habit building. This may include reminders in a weekly staff memo, prominent colored signs around the hospital or stickers to highlight a new checklist on a form (Figure 4.3).

Modification

Although the checklist has likely gone through testing prior to implementation, it will likely need to be modified over time. These modifications may happen due to unexpected issues found during implementation. However, modifications may

Prior to Anesthesia		Before Skin Incision		Before Leaving the OR	
Owner Permission/Deposit	❑	Case Review for team	❑	All Procedures Done	❑
CPR Code	❑	Site Confirmed & Prepped	❑	Gauze count	❑
Med Hx (NSAIDS & steroids & ABX)	❑	Antibiotic prophylaxis	❑	Biopsy/culture/images (ID & label)	❑
Met ✓ reviewed by DVM N/A ❑	❑	Sterility checked	❑	Implant Review (soft/ortho)	❑
Preop BW reviewed by DVM	❑	Patient issues addressed	❑	Post-op Imaging	❑
Blood Trans N/A ❑	❑	Warmed flush available	❑	Extubation Orders	❑
Type/X Match/Supply	❑	Addtl induction drugs avail	❑	PCV/TS, bloodwork	❑
Rx Allergies	❑	Eye Lube	❑	Pain addressed	❑
Appropriate suture available	❑	Gauze Count	❑	Fluids	❑
Special equip needs	❑			Antibiotics	❑
Cautery Setup	❑			Incision Care (Tegaderm/Bandage/Ice)	❑
NPO time	❑			Nail Trim	❑
Verify side of body	❑			Diet	❑
Owner Called Prior to Sx?	❑			Sharps	❑
				Check/Mark Baralyme Prep ❑ OR	❑
Initials_____		Initials_____		Initials_____	

Figure 4.1 Example of a surgery checklist.

Prior to anesthesia

Owner permission – either a signed authorization form or a verbal authorization provided by the admitting doctor

Deposit – check for a sufficient deposit for the surgical procedure. Look at the numbers. A deposit collected for ER admit and hospitalization is not sufficient for surgery and follow up care.

CPR code – a CPR code is required.

Med Hx confirmed – Know what recent meds your patient has taken and how those affect the drug protocol provided by the doctor. For instance, a patient that has received steroids should probably not receive an NSAID.

Preop BW reviewed by DVM – self explanatory

Blood trans – Is it possible that your patient will need a blood transfusion. Be prepared in advance with type and crossmatch. Be sure there is adequate blood supply available for your patient.

RX allergies – are there any known prescription allergies? Check the chart and/or ask your doctor.

Appropriate suture available – not just any suture will do for every procedure. Ask the surgeon. A lar par requires specific suture. Make sure you have the basics on hand – 0 PDS, 2-0 PDS, 3-0 monocryl, 3-0 ethilon and then ask if the surgeon will need a specific suture for the procedure.

Special equip needs – make sure you can locate and set up anything special the doctor may need. Ligasure? Mixters? Delicate metz? Ortho equipment? Not sure what these things are? Ask your doctor. Find them before you need them in surgery.

NPO time – double check the last time your patient ate. Inform the doctor if it was recent. Take precautions when intubating and extubating.

Figure 4.2 Descriptors of elements listed on the surgery checklist (Figure 4.1).

also be needed in response to other flow changes in the hospital. Modifications may also be needed to reflect new evidence or changes in standard of care practice. Willingness to change the checklist to reflect changing conditions will further encourage a culture of feedback and engagement.

Ongoing Accountability

Even with a successful implementation, the team must be kept accountable over time. It is important to add new hospital checklists, with their *why* and *how*, into training manuals for new staff. New staff may not understand the importance of checklists if stories of their development and use are not included in training and orientation.

In the authors' hospitals, a chart audit of surgery checklist completion was completed on several occasions and then published to the hospital to demonstrate what was happening and what was possible. While one department thought the checklist took too much time, another department had no trouble reaching close to 100% completion. Publication of the results and competition between departments encouraged better completion prior to the next audit.

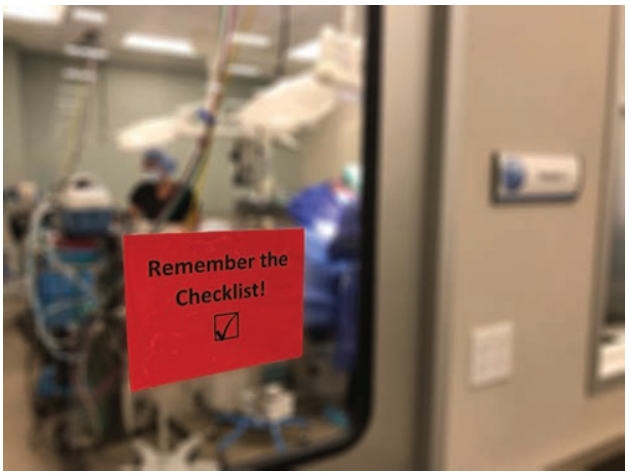


Figure 4.3 Brightly colored signs regarding the use of checklist can serve as a reminder or visual cue when implementing the new process.

Celebrate Successes

The continuing successful use of checklists depends on continuing to demonstrate *why* they are needed. In the authors' hospital, an autoclave malfunctioned. Following the surgical checklist item, "check pack sterility," it was noted that although the outside pack indicator tape had changed, the interior sterility indicator showed lack of adequate heat penetration. Because of the checklist, the unsterile pack was not used and the autoclave was serviced and repaired in a timely fashion. The "catch" by the team was celebrated, and also allowed us to figure out the problem and fix it before any pet was affected.

The Virginia Mason hospital system celebrates safety success by giving a monthly "good catch" award that is announced to the entire organization. This award is given to a staff member who provides an alert of a safety concern that helps to avoid ongoing problems. This recognition is an ongoing way to tell stories of safety success and to encourage staff to actively participate in continuous improvement [13].

Checklists Help Build a Reliable Culture of Safety

Checklists save lives, in part, by helping to remember easily forgotten steps in a process. The goal is thus not to just tick boxes [8]. The ultimate goal is to build a highly reliable hospital that can handle complex cases with minimal to no mistakes. Standardization helps add reliability into a complex environment [13]. When checklists empower nurses, it allows them to insist that those higher in the hierarchy adhere to safety procedures [7]. The use of multidisciplinary teams to develop and refine new checklists creates a culture of quality continuous improvement [8]. The idea that checklists need to be continuously improved, adds additional safety for addressing emergency problems [14]. Successful implementation and use can also change the culture of a hospital to one that emphasizes patient safety and a team approach to care.

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5

Medical Charting

Karl E. Jandrey and Sharon Fornes

Veterinary medical records serve several purposes. Medical records are a document of information that provides what has occurred to the patient while they have been in the care of the veterinary health care team in the hospital. There are four main reasons to maintain accurate and up-to-date medical records.

1) **A medical record is a legal document of patient care.** As a legal document, a complete medical record can be used in court as a representation of the treatment that was planned and completed on an animal. If the record is incomplete or is in error, the courts may rule in favor of the client even if no negligence can be proven from the record [1]. The components of a complete medical record will be discussed later in the chapter.
2) **The medical record makes the path of patient care obvious to all readers.** A primary function of the medical record is to document the path of patient care and the thought process behind it. To this extent, a complete medical record should detail all patient data and the assessment of those data. One study found only 64.4% of the observed and discussed problems during a consultation were actually recorded in the electronic medical record (EMR) [2]. This patient-centered information leads to the unique and particular diagnostic and therapeutic path. For example, the reader of a medical record should be able to easily identify whether a patient's data are within normal reference intervals, whether a trend is improving over time, which procedures were performed on the patient, and the results of a particular intervention. A properly executed and comprehensive medical record will facilitate the development of future diagnostic or therapeutic plans for the continuing treatment of the animal.
3) **The medical record allows for the documentation of all communication between veterinary staff and clients.** Whether it documents communication between the animal's owner and the staff at the practice or within the practice itself, the medical record is an essential tool to maximize continuity of care. By facilitating effective communication, medical records ensure that all doctors and associated hospital staff members involved in the patient's care are aware of the treatment plan. If documented correctly, a medical record can thereby allow for consistent and accurate standardization of patient care [3].
4) **The medical record allows for documentation for research, disease surveillance, and publication** [3–5]. Along with the legal implications of medical records, the importance of a complete and comprehensive medical record is underscored by its use in clinical research. Medical records provide data from which case reports or research papers may be written. Missing medical record information can grossly diminish the impact of a research publication by reducing the amount of usable data, which then may function to reduce the sample size. To avoid this issue, the medical record should present its information in a clear and concise format that allows research personnel to obtain the information quickly.

Medical Record Documentation

Because a medical record is frequently referenced, an accurate and clearly written record is of the utmost importance. Taking care to appropriately document data can greatly facilitate the clarity and accuracy of a medical record. There are seven components to proper medical record documentation.

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1) **Documentation of authorization for patient care.** Authorization for patient care is necessary before treatment of the animal can commence. It is of great importance to ensure that appropriate forms have been signed and that witnessed oral consent is documented [3]. These consent forms for each visit/procedure can be placed in the plans and progress notes of the complete medical record. The consent form also establishes a relationship between the veterinarian, the client and the patient.
2) **Timely documentation of information in the record.** This functions to better ensure accurate recollection of the data. It is important to time- and date stamp any entry if possible. For example, if the animal is unstable, information that was obtained in the initial excitement of an emergency presentation may be recalled with less clarity hours later. Some computer software systems will not allow alterations after a set period. Changing the information after this period may render the medical record inaccurate as it may be viewed as tampering of the information, should the record be called into court. Any history and pertinent information should be recorded in the appropriate place in the chart as soon as it becomes possible. Alternatively, an assistant can document information as it unfolds if their participation is not essential to the emergency interventions ongoing for the patient.
3) **Clear indication of the person who performed a task or treatment.** This is usually accomplished by documentation of the person's initials on the record or treatment sheet. The purpose to initial the records will allow for questions to be directed to the appropriate parties. If there is are many team members inputting information into the medical record, a list of full names of employees along with the initials should accompany the record to add verity to the information in the entries. A date and time should also be entered to verify time of treatment or communication. This can be done automatically with an EMR.
4) **Legible handwriting.** Legibility of the record is essential to prevent misinterpretation. If one cannot write legibly, one should consider typed or computerized medical records.
5) **Appropriate notation of corrections to the record.** Care should be taken if a correction in a medical record is required. To overwrite, scratch out, erase, black out with a marker, or use correction fluid or tape are inappropriate methods for correction. The appropriate method of correction is to initial and draw a single line through the entry that was created in error. The correction should then be written and initialed near the entry that was replaced. The use of any method other than the accepted convention could be considered as tampering with the record and may be used against the veterinary healthcare team in a court of law [1, 6].
6) **Use of proper writing implements.** Permanent ink should be used to make entries in the handwritten medical record. There is controversy about the appropriate color of ink to use. Local regulation and clinic preferences (standard operating procedures) may have some variation. The following are arguments for the exclusive use of black pen in a medical record: [7] better reproduction on a photocopier, better contrast on white paper, and the tendency to be more permanent. However, an advantage of blue ink over black ink is to provide contrast to the black ink of preprinted forms. With blue ink, new entries on preprinted forms may be more easily distinguished.
7) **Use of acronyms and abbreviations.** Acronyms and abbreviations used in the clinic should be standardized. Confusion may arise, for example, in determining whether "mm" refers to millimeters or mucous membranes. The Academy of Veterinary Technicians in Anesthesia and Analgesia (http://www.avtaa-vts.org) has a published list of acceptable abbreviations. The development of a list of acceptable abbreviations for each individual hospital could also be helpful to avoid miscommunication between staff members. Beware, however, that records are often shared between facilities. Other facilities may not understand a particular hospital's abbreviation standards unless provided with a key. Success after implementation of a novel EMR with adoption of a controlled vocabulary permitted standardized filing, as well as retrieval of information [8].

Medical Record Organization

A standardized medical record organization is important for many reasons. During a patient's stay in a veterinary hospital, much information is collected and assessed daily. If these data are consistently written in the same format, information retrieval is timely and accurate. This may help in legal cases, to evaluate therapeutic goals, and to use the medical record for clinical research. Data are most useful and clinical efficiency is maximized when information is placed in a consistent location.

An organized medical record is most commonly formatted in a chronological order. Reverse chronological order (last visit on top and the first visit on the bottom) is a common method for assembly of the medical record [9].

Medical records can also be organized by section (e.g. financial, authorization forms, treatment sheets, pharmacy, laboratory, plans, and progress notes). The creation of sections within a patient record facilitates quicker reference in a large comprehensive medical record.

Medical Record Format

There are two common formats used for the documentation of medical records: the conventional method and the problem-oriented medical record (POMR). The conventional method documents information as it is obtained. It may be less time-consuming than the POMR and tends to be used in general practice. The POMR records patient data according to the patient's problem. POMRs are often used in academic and specialty practices to clearly document and transmit the logical forward-thinking approach that is required for patients with complicated disease processes. POMRs are also used as effective teaching tools because they allow the reader to readily uncover the thought process of the writer. However, although POMR records are very organized and detailed, a disadvantage is that they may be more time-consuming to produce [6].

A source-oriented veterinary medical record information is organized by subject areas rather than by the problem(s) of the patient's visit. Information may be accessed in each separate area of the record. Clinical findings and result may be separated by tabs or dividers to separate the information. Laboratory findings may be separated from plan notes and treatment sheets from the same visit of the patient. It may be difficult to find information about one visit, because the information is found in many different locations. It is helpful however to find all the radiological findings in one location, so outcomes of the various visits can be compared [3].

Components of a Complete Medical Record

A complete medical record should include a thorough and accurate daily description of all data obtained for a particular patient, the assessment of these data, and a discussion of the resultant plan (which is composed of diagnostic, therapeutic, and client education components). The complete medical record should include nine components:

1) Client information
2) Patient information
3) Presenting complaint
4) History (from both the client as well as other prior medical records)
5) Physical examination
6) Problem lists
7) Progress notes
8) Communication log
9) Comments

Client Information

This area of the medical record is devoted to the client where all pertinent information about the animal owner(s) is kept. All contact information (physical and mailing addresses, electronic contact, landline and mobile telephone numbers, and special notations) about the animal owners should be placed here. These listed people are the legal guardians of the animal and permit access to this information. Those not listed in this are unable to access patient information under privacy and confidentiality agreements. Although these are not commonplace in veterinary medicine, patient confidentiality must be respected by the hospital and all members of its staff. Release of patient information to a third party must be approved by the client.

Patient Information

The patient data is recorded here and includes the signalment (age, breed, sex, birth date). Pedigree or individual medical information (allergies, behavioral, blood type/previous transfusions) can be placed here.

Presenting Complaint

The presenting complaint is recorded by the reception or medical records staff when the appointment is made. This is the information in the words of the client that transmits the reason for which they seek veterinary medical care.

History

A comprehensive history is obtained from the client on the initial visit and is updated periodically as the animals' health status changes. A history needs to be taken every time an animal presents for a new illness (known as the history of the presenting illness). Past pertinent history can be helpful in determining onset of the problem or relationship to the history of the presenting illness. Past pertinent history may include recording previous treatment prior to the current hospital visits or previous veterinary care from other veterinary hospitals.

Physical Examination

A physical examination may be completed one or multiple times daily and should be documented at least once daily throughout the animal's hospitalization. All the body systems should be examined and documented properly to ensure and prove that they were examined. Salient normal and abnormal findings need to be documented. Abnormal findings should be well documented and can then be elevated to a level of a problem in the problem list, as indicated by the problem-oriented approach of the clinician.

Problem Lists

Problem lists enumerate the conditions being managed during a hospitalization period. In a problem list, "problems" can be created, resolved, combined with other problems and renamed, or inactivated at any time. This provides the veterinarian with a means of obtaining an overview of all problems that the animal may have had and whether they were addressed or resolved, without the requirement to examine the entire record [6].

A master problem list (Figure 5.1) is often created and placed on the first page of the patient record. The master problem list is a summary of all the problems for which the patient has been examined. This includes the date the problem was identified, as well as when it was resolved (if applicable). It functions as a quick glance into the patient's medical history and can thereby facilitate a focused search, much like a table of contents.

Progress Notes

Progress notes are the daily subjective, objective, assessment, plan (SOAP) of the patient. Each day for each problem, an entry is created that contains the data relevant to that problem. *Subjective* and *objective* data are placed in this section and should include the information gained from diagnostics and therapeutic interventions, as well as the new physical examination or any physiologic measurements. A patient's response to treatments is also recorded in this section. A differentiation is made in human medicine between the information that comes from the patient (subjective) and that measured in analysis (objective). In veterinary medicine, the information given by the client may be treated as subjective. However, historical data from a client can be measured and clearly objective; therefore, "data" is a more proper term. The SOAP would therefore be referred to as the data, assessment, and plan (DAP). An *assessment* follows the data and should include information pertinent to the patient's prognosis. The purpose of the assessment is to refine and document the new thoughts of the clinician. Based on the assessment of all the previous data, a new *plan* is created. This plan must discuss at least one of three distinct areas of focus: diagnostic plans, therapeutic plans, and/or client education plans.

Communication Log

The communication log is the section of the POMR that contains the information about any and all contact between members of the hospital staff with the client and/or referring veterinarian(s). This includes detailed phone calls, emails, text messages, or client visits. Proper documentation also includes reference to client education, date, time, names, and content of the communication. In a referral hospital, the names, contact information, and addresses of the primary care provider and, if applicable, primary veterinarian should also be on the record.

The information found in the communication log is often equally as important as the daily SOAP. As this information is not likely to be organized in any other area of the POMR, the communication log should be carefully and comprehensively documented. EMRs may be finalized and locked with a time and date stamp. However, communications logs should not be locked. Added information via telephone communication may arrive without attachment to a hospital visit and, therefore, no daily POMR. This message may be added into the record in chronological order when the record is not locked. Information from delayed diagnostic tests can be recorded back to the visit to which they pertain. This communication is essential to provide continuity for the patient's medical and surgical treatment data since it may alter the assessment of the patient problem.

Comments

The comments section is the part of a complete medical record designed for other miscellaneous details. Often an additional page or pages are available if there was insufficient room in the space provided on a medical record or form. In some computerized medical record systems, this is the only other editable section to which information can be added once the medical record is finalized and locked.

Other Additions to the Patient Record

Examples of additions to the patient record when applicable include: medications (particularly drug sensitivity), pertinent patient information such as aggressive/caution, special needs, mentation, appetite, food intake, visual analog pain scales, body conditioning score, and lab results. Other additions may include warning labels placed on the outside of the medical record or in the header of the computerized medical record. Alternatively, these may be addenda to the plans and progress notes for hospitalized patients that are included in the description of patient observations.

Clinician Order Sheets and Treatment/ Flow Sheets

In addition to details about patient data, future treatment and diagnostic plans, and client education, a medical record should include clinician order sheets and treatment/flow sheets. These function to help ensure quality and continuity of care for the veterinary patient. They are part of the progress notes since pertinent data from these are abstracted into the computerized medical record. In paper medical records, these sheets are inserted chronologically to accompany the

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NUMBER	PROBLEM	DATE ENTERED	DATE RESOLVED

D2760 (12/90) Form #48-R **MASTER PROBLEM LIST**

Figure 5.1 Master problem list is a summary of all the problems for which the patient has been examined. *Source:* Veterinary Medical Teaching Hospital / The Regents of University of California.

daily DAP. The following section focuses on the information found in order sheets and flow sheets, and highlights appropriate methods to notate findings and interpret information.

Clinician Orders

Many hospitals combine the clinician's orders with patient observation or flow sheets. Order sheets can be simple for wards patients that are stable (Figure 5.2 for general ward patients and Figure 5.3 for intermediate care ward patients), or they can be elaborate for intensive care unit (ICU) patients (Figure 5.4). In all cases, the appropriate treatment, diagnostic, and monitoring plans should be legibly written for clear documentation.

Treatment Sheets

Treatment sheets are the part of the medical record that contain recorded data collected throughout the animal's hospitalization. Treatment sheets (Figure 5.5 for patients on the general ward and Figure 5.6 for intermediate care ward patients) can be as simple as recording observations and the treatments provided to an animal. The preferred format is to write in the medical order using a clear format; for example: "lactated Ringer's solution qs 20 mEq/l at 120 ml/h IV" (as needed, 20 milliequivalents per liter at 120 ml/hour intravenously) or "famotidine 10 mg IV q12h" (10 mg intravenously every 12 hours).

Daily Patient Flow Sheets

Patient daily flow sheets may be complicated, multipage treatment sheets that give detailed information in areas of subsections of the document outlining treatments, monitoring, and observations (Figure 5.6 for intermediate care ward patients and Figure 5.7 for ICU patients). Although the format of these flow sheets is often tailored to the purpose and individual clinic, completed treatment sheets are considered part of the patient's medical record and must contain basic information.

Patient Identification

First, the patient's basic identification must be found on each page of all forms on the flow sheet as well as every piece of the medical record. This enables a page that becomes detached from the record to be easily returned to the record. The date should also be included on each page, and a time may be appropriate for certain entries.

Patient Weight

The patient flow sheet should include the patient's weight at presentation as well as daily updates. This is important to determine effective pharmaceutical treatment and the assessment of need for other treatments. A patient's weight may also be used to calculate the charges for care provided. Recording of weight may be delayed until certain initial interventions required for more life-threatening conditions are completed.

Nutritional Considerations

What is to be fed and the frequency to offer food is essential. The volume in cups/cans or weight in grams should be noted for both the amount offered as well as the amount ingested. Special dietary needs and feeding instructions should be clear, especially if using various enteral feeding tubes or parenteral nutrition. In addition, nutritional considerations should be notated on the record. The patient that has no oral administration of food or medication should be labeled "NPO" (*non* per os). This is important for animals that are going to be anesthetized because preanesthetic protocol may require the removal of food from the animal at a certain time. Therefore, this information should be easily distinguished on a record so that the animal can undergo anesthesia at the intended time. If a hospitalized animal is to be fed or given water, the amount, type of food, and appetite or water consumption should be notated on the record. Some methods to indicate appetite are a number scale (0–5), where other methods use plus or minus (±) symbols to indicate whether an animal did or did not eat/drink. Notation of the patient's nutritional considerations is important on two levels. First, the more nutritional information included in the record, the more fully will the staff understand the individual patient's eating preferences. Second, because some owners may bring the patient's own food or favorite treats to the hospital to encourage appetite, providing a record of nutrition will allow the hospital staff to tally and keep watch on the patient's ingested calorie content. Ideally, the exact calorie content ingested should be documented.

Laboratory Measurements

The data obtained from patient-side laboratory evaluation should be placed in the appropriate section of the medical record and flow sheet. This notation in the record may be the only area it is recorded since some point-of-care machines do not have hard-copy printouts of these data, nor are these data automatically integrated into the EMR.

Patient Treatments and Observations

Medications

The "rights" of pharmacotherapy include right drug, right patient, right time interval, and right route. Accurate

Patient General Ward Orders

Patient Sticker Here

Date:_____

Code Status: _____ Weight (kg):_____

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Cage/run#			
Faculty:		Phone:	
		Alt #:	
House Officer:		Phone:	
		Alt #:	
Student:		Phone:	
		Pager:	
Working Diagnosis:			

Non-shaded Area is the *Service Responsibility* | Shaded Area is the *SAPCS Responsibility*

Treatments	*FREQ*	1200	1400	1600	2000	2400	0200	0400	0800
Temperature, Heart Rate, Respiratory Rate	Q12hr				8				8
Flush unused IV catheter ports	Q6hr		14		8		2		8
IV catheter care (8am or 8pm)	Q24hr								
If IV catheter out: ☐ replace ☐ don't replace									

Pain Management Orders

Clinician Signature		1200	1300	1400	1500	1600	1700	1800	1900	2000	2100	2200	2300	2400	0100	0200	0300	0400	0500	0600	0700	0800	0900	1000	1100

Student Signature: _____ Clinician Signature:_____

Controlled drugs require a separate clinician signature next to the controlled drug order.

Figure 5.2 Example of a patient general ward orders form. *Source:* Veterinary Medical Teaching Hospital / University of California, Davis.

UC DAVIS VETERINARY MEDICINE

Standard Contingencies

Unless orders state otherwise, SAPCS technicians will contact the clinician when any of the following occur:

If temp is >102.5 or <99 F: call clinician
IV catheters:
IV catheter care q 24 hours
All unused catheters/ports flushed q 6 hours
Urinary catheters:
U-cath care q 8 hours, except male cats which are done q 24 hours
If leaking or not patent: ☐ call clinician ☐ remove and do not replace
Incisions, bandages, Fentanyl patches: checked no less than q 6 hours
Elizabethan Collars:
If off, will be replaced
Will be placed on patients as needed
Seizures: Diazepam 0.5 mg/kg IV will be given, then Dr. will be called
Vomit/Regurgitation ☐ call doctor ☐ do not call doctor

Contingencies (these will override standard contingencies)

Doctor is aware of/don't call if:

Figure 5.2 (Continued)

Patient Intermediate Care Ward Orders

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Patient Sticker Here

Date:_____

Code Status:_____ Weight (kg):_____

Cage/run#			
Faculty:		Phone:	
		Alt #:	
House Officer:		Phone:	
		Alt #:	
Student:		Phone:	
		Pager:	
Working Diagnosis:			

Non-shaded Area is the *Service Responsibility* | Shaded Area is the *SAPCS Responsibility*

Treatments		1200	1300	1400	1500	1600	1700	1800	1900	2000	2100	2200	2300	2400	0100	0200	0300	0400	0500	0600	0700	0800	0900	0100	1100
Temperature, Heart Rate, Respiratory Rate	Q12hr									20												8			
Flush unused IV catheter ports	Q6hr			14						20						2						8			
IV catheter care (8am or 8pm)	Q24hr																								
If IV catheter out: ☐ replace ☐ don't replace																									

Pain Management Orders

Clinician Signature		1200	1300	1400	1500	1600	1700	1800	1900	2000	2100	2200	2300	2400	0100	0200	0300	0400	0500	0600	0700	0800	0900	1000	1100

Student Signature:_____ Clinician Signature:_____

Controlled drugs require a separate clinician signature next to the controlled drug order.

Figure 5.3 Example of a patient intermediate care ward orders form. *Source:* Veterinary Medical Teaching Hospital / University of California, Davis.

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Standard Contingencies

Unless orders state otherwise, SAPCS technicians will contact the clinician when any of the following occur:

If temp is >102.5 or <99 F: call clinician
IV catheters:
IV catheter care q 24 hours
All unused catheters/ports flushed q 6 hours
Urinary catheters:
U-cath care q 8 hours, except male cats which are done q 24 hours
If leaking or not patent: ☐ call clinician ☐ remove and do not replace
Incisions, bandages, Fentanyl patches: checked no less than q 6 hours
Elizabethan Collars:
If off, will be replaced
Will be placed on patients as needed
Seizures: Diazepam 0.5 mg/kg IV will be given, then Dr. will be called
Vomit/Regurgitation ☐ call doctor ☐ do not call doctor

Contingencies (these will override standard contingencies)

Doctor is aware of/don't call if:

Figure 5.3 (Continued)

Patient Sticker

UNIVERSITY OF CALIFORNIA, DAVIS - VMTH
SMALL ANIMAL INTENSIVE CARE UNIT

CLINICIAN_____
Ph1# _____Ph2#_____
STUDENT _____
Ph1# _____Ph2# _____

Primary Complaint _____
DATE _____ **TIME** _____ **Patient Weight** _____ **kg**
****ORDERS ARE VALID UNTIL 12 NOON****

FLUIDS / MEDICATIONS / TREATMENTS

_____
_____
_____
_____
_____
_____
_____
_____
_____
_____
_____
_____

Monitoring
Observation q___hr (mentation, respiratory rate, respiratory effort)
Vital signs q___hr (observation & heart rate, auscult heart & lungs, mucous memb color & CRT)
Temp q____hr ECG q____hr Doppler BP q____hr Cardell BP q____hr Direct BP q____hr
Pulse Ox q____hr Urine Output q___hr Weigh q____hr _____ q ____hr _____q____hr

Laboratory
ICU panel q____hr PCV/TP q____hr Glucose/Lactate ABL q____hr ACT q____hr
ICU PTT q____hr ICU PT q____hr ICU Chemistry q____hr Urine dipstick q____hr
COP q____hr Osmolality q____hr Urine SG q_____hr _____ q_____ hr
Clinical Path lab work (6-9am) _____

General nursing care

Standard care (all patients) ☐ Recumbent care ☐ Comatose patient care

Nutrition
☐ NPO ☐ NPO except oral meds ☐ No food, water ad lib ☐ No food, offer _____ml water q____hr
☐ Water ad lib: feed _____ of _____ q____hr ☐ TPN: _____ per protocol; goal: _____
(amount) (type of food) (formula) (kcal/day)

Contingencies (change/cancel any default contingencies)

_____
_____
_____
_____

Full CPR Closed-chest CPR No CPR **Signature:** _____

Figure 5.4 Example of an order sheet for intensive care unit patients. *Source:* Veterinary Medical Teaching Hospital / University of California, Davis.

Standard Default Contingencies for ICU Patients

Unless instructed otherwise, the ICU technicians will contact the primary clinician when any of the following criteria have been met. In addition, ICU technicians will call when an important change in the patient or any parameter is noted, even if it does not meet a contingency.

General

Temperature < 99°F (37.5°C) or >104°F (40°C)

Urine output

- If catheterized: < 1 ml/kg/hr for any 4 hour period
- If not catheterized: Has not urinated in last 12 hours and bladder palpates empty

Laboratory

PCV < 20%	pH <7.2 or > 7.6	Potassium <2.5 or > 5.5
Hb < 6.5 g/dL	$PaCO_2$ < 20 or > 60 mmHg	Sodium <135 or > 160 mEq/L
COP < 12 mmHg	$PvCO_2$ <25 or > 65 mmHg	or any change > 10 mEq/L/12 hrs
Lactate > 3 mmol/L	PaO_2 < 60 mmHg	Glucose < 70 mg/dL
iCa < 0.80 mmo/L	HCO_3 < 14 mmol/L	Osmolality < 280 or > 320

Cardiovascular

HR dog < 60 or > 200	ABP mean < 60 or > 140	Significant or worsening
Cat < 90 or > 240	systolic < 80 or > 200	ventricular arrhythmia of which
Persistent change > 50%	CVP < 0 or > 10 cmH_2O	clinician is unaware

Pulmonary

Respiratory rate < 8 or > 60	SpO_2 < 90%	Significant change in
or persistent change > 50%		respiratory effort

Neurological

Seizures: 1) Administer diazepam (0.5 mg/kg IV) unless other contingencies; 2) Call clinician
Mentation – any obvious deterioration or abnormal excitation
Changing pupil size to anisocoria, miosis or mydriasis

Patient Care

Standard care: Weigh q24 hrs, indwelling catheter care per protocol; heparin lock unused catheters
Recumbent care: Standard care, reposition & physical therapy q4hrs
Comatose patient care: Recumbent care + oral & eye care per protocol

6/2017

Figure 5.4 (Continued)

Cage # _____

GENERAL WARD OBSERVATION RECORD

Feces
N normal
A abnormal
0 none
D diarrhea

Patient sticker

Date	Time	Feces	Urine	Vomit/ Regurgitation	Observations	Initials

Pain/Sedation Medication:

Date	Time	Drug and Dose	Route Given (IM/SQ/PO/IV)	Initials

Figure 5.5 Example of a general ward observation record.

Controlled Drug Record					
Drug	Mg	Route	CRI/Bolus	Time	Name

Controlled Drug Record					
Drug	Mg	Route	CRI/Bolus	Time	Name

CPR: ☐ FULL CPR ☐ CC/CPR ☐ D.N.R.

Date:

Time:

Ward/Cage:

Weight:

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S.A. ICW OBSERVATION SHEET

Patient Sticker Here

Time	Observations	Initials

Time	Shift Observations	Initials

12/15

Figure 5.6 Example of an intermediate care ward observation sheet.

	1200	1300	1400	1500	1600	1700	1800	1900	2000	2100	2200	2300	2400	0100	0200	0300	0400	0500	0600	0700	0800	0900	1000	1100	
BLOOD PRESSURE																									
HEART RATE																									
RESPIRATORY RATE																									
BREATHING EFFORT																									
BODY POSITION L/R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	
BLOOD GLUCOSE																									
INSULIN TYPE:																									
INSULIN TYPE:																									
FEMORAL PULSE QUALITY / DORSAL PEDAL PULSE QUALITY																									
SPO2																									
HEART SOUNDS																									
BREATH SOUNDS	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	L R	
BREATHING EFFORT																									
MUCOUS MEMBRANE COLOR																									
CAPILLARY REFILL TIME (seconds)																									
MENTATION																									
PCV																									
TP																									
TEMPERATURE																									

	1200	1300	1400	1500	1600	1700	1800	1900	2000	2100	2200	2300	2400	0100	0200	0300	0400	0500	0600	0700	0800	0900	1000	1100	
Water Amount Drank																									
Food Type: Amount Eaten																									
Food Type: Amount Eaten																									
FLUIDS:																									
b:																									
c:																									
d:																									
e:																									
Urine ☐ Voided ☐ Catheter																									
Defecation/diarrhea																									
Emesis/regurgitation																									
Other																									
Other																									
Fluids																									

OUTPUTS

Pulse Quality: B=bounding; G=good, F=fair, P=poor; U=undetectable

Heart sounds: N=normal; M=murmur; G=gallop; MF=muffled

Breath sounds: C=clear; Cr=crackle; W=wheezes; I=increased; D=decreased; RUA=referred upper airway

Breathing effort: N=normal; I=increased; D=decreased

Mucous membrane color: Pi=pink; Pa=pale; R=red; C=cyan; I=icteric; W=white

Mentation: A=alert; O=obtunded; C=comotose; An=anesthetized; AnR=anesthesia recovery; As=asleep

Figure 5.6 (Continued)

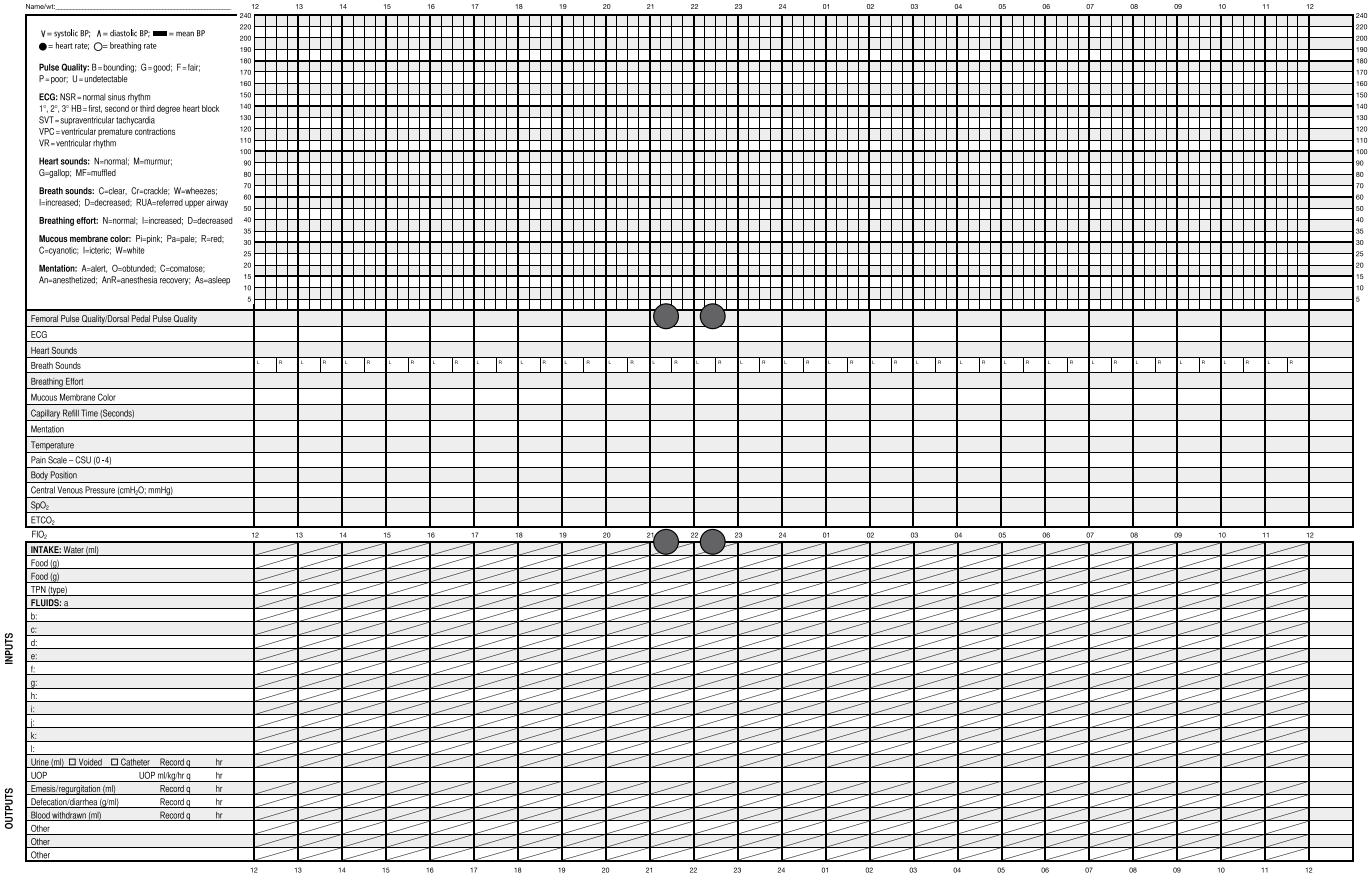


Figure 5.7 Example of an intensive care ward observation sheet. *Source:* Veterinary Medical Teaching Hospital / University of California, Davis.

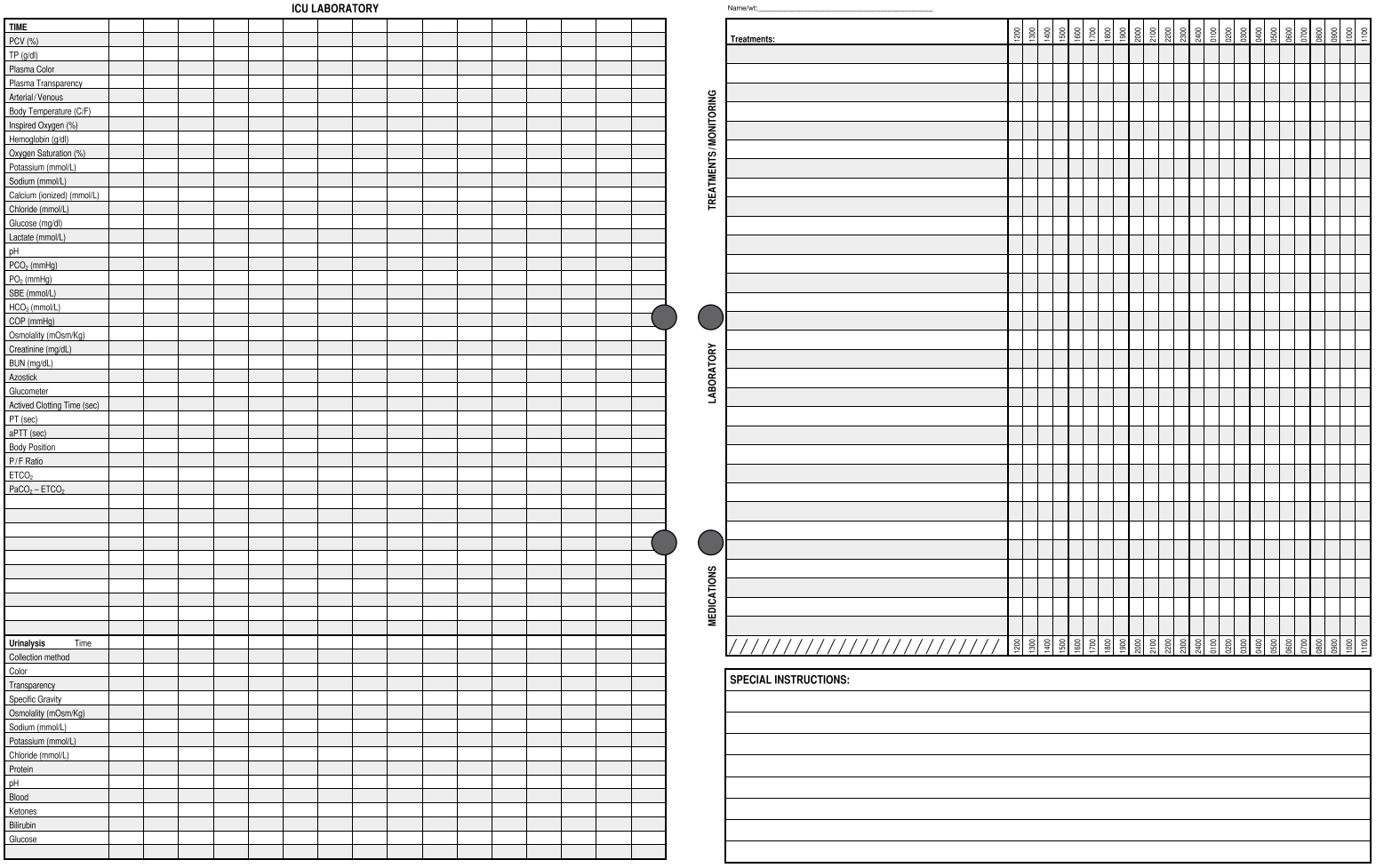


Figure 5.7 (Continued)

Name/wt:.....

OBSERVATIONS

Controlled Drug Record

Drug	mg	Route	CRI/Bolus	Time	Name

OBSERVATIONS

Figure 5.7 (Continued)

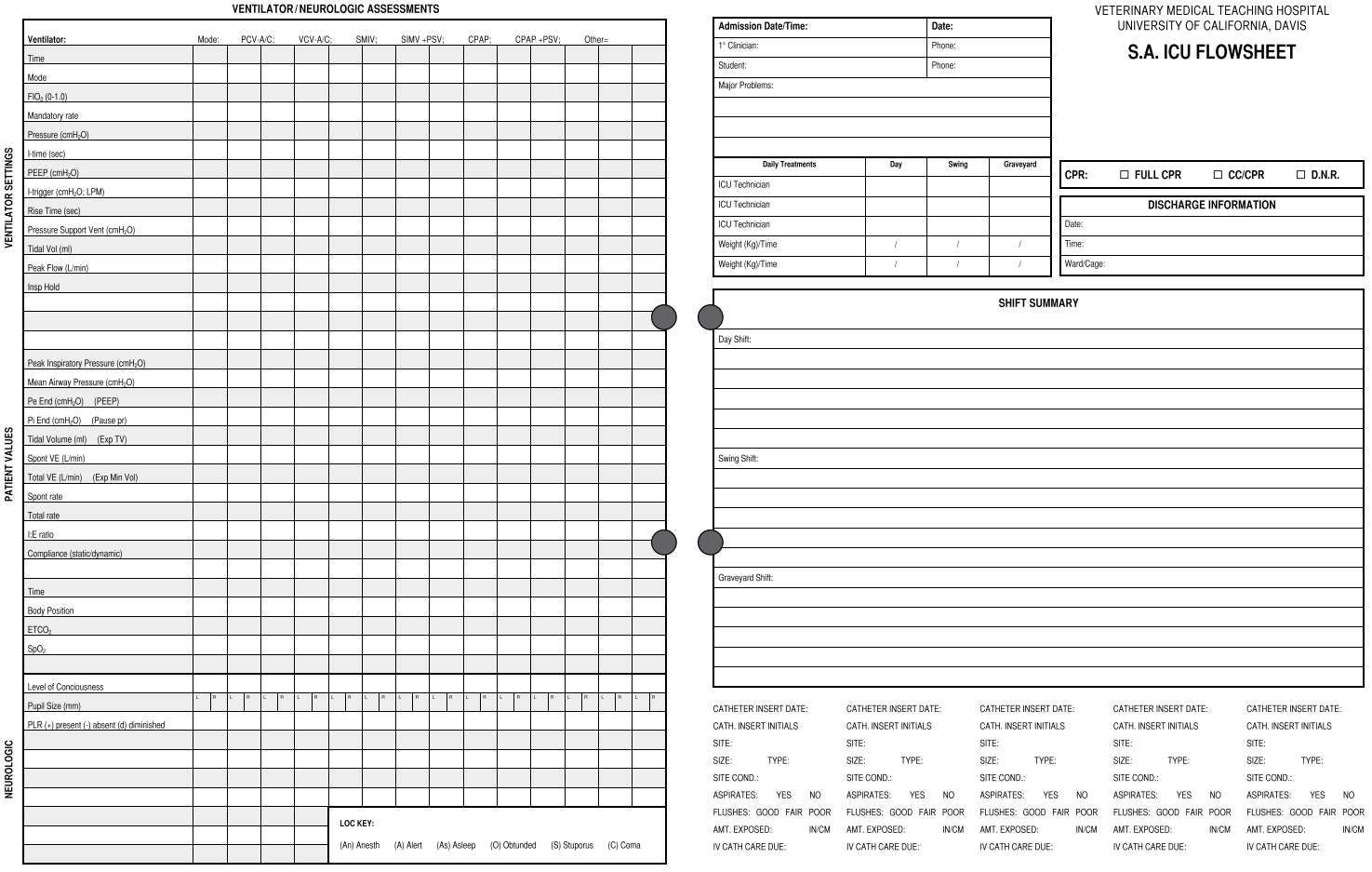


Figure 5.7 (Continued)

notation of pharmacologic information into the flow sheet plays a major role in ensuring the correct method of administration of a drug. Medications and treatment regimens should be recorded in the sheet exactly as the veterinarian prescribed [10]. Standardized orders require all medications to be written in the exact amount of drug in milligrams administered. It is preferred to write the total dosage in milligrams (mg) with the appropriate time interval and not just a dose per body weight (mg/kg). Drug volumes should not be used due to the varying concentrations of preparations between manufacturers. It is expected that the veterinarian who prescribes the medication will write the order clearly (e.g. ampicillin 250 mg IV q8h). An order in total dose such as "250 mg" is much clearer than "22 mg/kg" because drugs dosed in milligram/kg (mg/kg) are also subject to computational error. Before being written in the medical record or delivered to the patient, any clarifications should be addressed to the clinician who wrote the order. In some hospitals, the time for the treatment to be completed is indicated on the treatment sheet by an open circle. When the treatment is completed, the time at which it was delivered is written in the circle. Alternatively, some hospitals prefer the treatment order to be written as a number on the hour at which treatment should be delivered. When the treatment is completed, the number is then circled indicating completion. A hospital standard for consistency in format of the orders should be followed.

As part of the medication section, special legal considerations and maintenance of a controlled drugs log is essential. All controlled drugs administered to patients must be noted in both the patient record and in the controlled drug log. Some facilities have a drug log created at the time of dispensation by the use of automated dispensing equipment (with or without a witness). This log will be proof required during audits by the Drug Enforcement Agency or the state veterinary board. This should include patient information as well as the amount and route of the drug administered. The starting volume and remainder in a multi-use vial are also recorded. The names or initials of the dispensing and witnessing veterinary professional should be included on the patient flow sheet.

Fluids

Accurate fluid orders include many specific parameters. The type of fluid, dose and concentration, rate (per unit time), additive solutions or medications, and total hourly/daily tallies should be clearly indicated to ensure adherence. The complete measurement of fluid input can be compared with all net fluid output over time to direct adjustments in fluid treatment orders. Whether the fluids were administered via intravenous (IV) or subcutaneous bolus with or without the use of a fluid pump should be noted. Daily catheter evaluations should also be noted. How often the catheter was checked (every 24 hours at a minimum or as indicated) and by whom is also part of the fluid orders. Any information regarding the catheter replacement (date, personnel, anatomic site, catheter size and length) is also helpful for optimal patient care as well as troubleshooting in the event of a catheter mishap. Annotate the removal of the old catheter and the site of placement of the new catheter (including gauge, length, amount exposed, vessel quality, and whether it aspirates or can be flushed easily). Other information to include on the record regarding a new catheter placement includes the date of placement, the initials of the person who placed the catheter, site (i.e. left rear limb, lateral saphenous vein, or right jugular vein), and catheter size (e.g. 22-gauge 1½ inch over-the-needle catheter or 5 Fr, 10-cm guidewire, double lumen catheter). Notations of the patient's hydration status as measured by skin turgor, tear film, mucous membranes, and/or ocular position will help to gauge efficacy of therapy.

Body Systems Evaluations

The veterinarian uses the information found in the flow sheets to assess the response to treatment, to plan the next daily treatment, diagnostic, and monitoring plans, as well as to predict recovery of the animal. The body systems used for evaluation include: cardiovascular, respiratory, neurologic, and urinary. These body systems have parameters the veterinary technician can evaluate and notate in the medical record. Typically, data from individual body systems are organized in the medical record in proximity to one another. This arrangement facilitates evaluation by the caregivers to organize constellations of data into a more global perspective of the patient's status.

Cardiovascular System

Initial or serial vital measurements (e.g. temperature [temp], heart rate [hr], pulse rate [pr], respiratory rate [rr]) must be included in the patient flow sheet. These will help to assess whether a particular treatment is successful and sufficient. For example, a flow sheet should include the following pieces of information necessary for the understanding of the patients' perfusion: heart rate/pulse rate, pulse quality, mentation, extremity temperature, mucous membrane color, and capillary refill time. Using these parameters, poor distal perfusion may be assessed in shock, where the rectal-extremity (interdigital) temperature difference may be large (9°F; >4°C) due to peripheral vasoconstriction. Similarly, pale mucous membranes with a slow capillary refill time, tachycardia, weak femoral pulses,

and decreased mentation are all signs of poor perfusion. Normal capillary refill time (CRT) should be less than 2 seconds approximately. Conversely, an extremely rapid CRT accompanied by bright pink or red mucous membranes may indicate vasodilation [11,12]. Electrocardiogram interpretations or rhythm strips should be part of this portion of the medical record.

Respiratory System

Important parameters to annotate in the section devoted to the pulmonary system are respiratory rate, effort (apparent ease, origin of effort; e.g. thorax vs. abdomen), associated sound (type of sound, origin, volume change in reference to phase of respiration), or irregularities in respiratory pattern.

Nervous System

Upon initial presentation or triage of the animal, observations of the patient's level of consciousness and response to the surroundings is essential to the examining veterinarian [7]. The mentation of an animal may range from alert to obtunded to stuporus to comatose. In the daily flow sheet, the writer must mention the level of consciousness and any behavioral changes in the patient. Changes in level of mentation are important markers or improvement or decline in health status. Behavior may also give an indication that there may be some neurologic changes in the animal. The animal may circle, head-press, or become aggressive or withdrawn. Modifications of these mental states should be interpreted in light of the treatments given as well as in postoperative states after anesthesia or pain control has been administered [9, 10].

Urinary System

Some animals have preference for the substrate on which to eliminate or respond to special commands taught by the owners. These unique data should be annotated in the area related to the urinary system. Any urinary catheter, as is the case with an IV catheter, should have information regarding the date of placement (and by whom), catheter type and frequency of care, and any problems encountered (e.g. positional flow). The amount of urine production (hourly/daily) is important to note in milliliters whether obtained as an estimates from voided urination or specific amounts measured from the urinary collection systems. Collected urine samples may be weighed and subtracted from the weight of hospital bedding to estimate the urine output (UOP) as closely as possible. Normal UOP is 1–2 ml/kg/hour. Volumes more or less than this need to be addressed by the clinician once discovered. An Elizabethan collar may also be required to prevent premature removal of the catheter by the patient. This should also be notated on the record to ensure that nursing personnel keep the collar on the patient until the urinary catheter is removed.

Computerized or Electronic Medical Record

A large number of veterinary practices now utilize computers and electronic software for documentation of veterinary medical records. There are variety of software and products available for medical record keeping. The benefit of these systems, if designed properly, is the ease of retrieving information from the record and the sharing of information electronically. With the adoption of standard medical terminology and structured reporting, exchange of information across hospitals and institutions would be simplified [13]. Multiple users may enter information in the record at the same time. Misplacement of the physical copy of the patient file is eliminated. Another potential benefit of an EMR is to link information resources directly to the record for references to enhance evidence-based practice and support clinical decision-making [14].

Besides cost, considerations for choosing the appropriate software for the hospital might be how easily it will be to integrate the current paper system to a paperless or paper-light record. How much training is needed to implement the new software into the practice? How will it be to train new employees to use the system? How will records be shared in the hospital and also with other hospitals? Will it contain all data needed in the record (i.e. diagnostic results, inventory control, or client communications)? Ease of invoicing, billing and scheduling needs for the hospital must be considered. Is there availability of alerts systems to check for errors in prescriptions and contraindicated or adverse treatments of the patient? Once the software is mastered, will it actually increase the efficiency of the entry of the information into the record? [15, 16] Additional under-used benefits of the EMR, such as improved population health, identification of emerging disease, outweigh the perceived risks for technological problems, time constraints, and cost [17].

Patient Privacy

Although there are strict regulations and laws in place to protect the identification of human patients in both verbal and research communications, veterinary medicine does not have a global policy for client/patient privacy [17]. Veterinary caregivers should be sensitive to client/patient privacy especially with the advent and growth of social

networking sites. Written, photographic, and verbal confidentiality should be maintained for clients and patients. Client consent forms for the use of patient images and data are used to avoid inappropriate use against the clients' wishes. There may be local or regional confidentiality agreements. Be aware of the laws regarding the patient and client confidentiality. Obtain a client release [6] for anything that you may need to disclose to a third party.

Conclusions

The most accepted charting methods are those that are found to be user-friendly. The choice of charting method lies within the judgment of each clinic. For example, the use of a 24-hour clock may be preferred to a 12-hour clock. Despite the fact that a 24-hour clock is best used to avoid any confusion in a hospital where 24-hour service is provided, most people are not comfortable with this method. Internal standardization within a practice enables clear and precise communication among the veterinary healthcare team.

Storage of records may vary by area. State veterinary medical boards have mandated the minimum length of time that records must be maintained. When records are purged, security must be maintained due to the confidential information therein. Shredding of documents is an acceptable and preferred method of securely purging medical records. Many companies provide this service when a large number of medical records are culled.

Standards and guidelines of veterinary medical record keeping can be found at the local, state, and national veterinary associations. The following are some suggested associations where the salient details can be found: American Veterinary Medical Association (www.avma.org), American Animal Hospital Association (www.aahanet.org), state veterinary medical boards (e.g. for California, go to www.vmb.ca.gov), and the Veterinary Emergency and Critical Care Society (www.veccs.org).

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6

Point-of-Care Ultrasound for Emergency and Critical Care

Søren Boysen and Valerie Madden

Introduction

With technological advances leading to smaller, more portable ultrasound machines with better image quality, faster start up times, and more intuitive user interfaces, ultrasound is being employed more frequently and with greater efficacy by non-specialist veterinary clinicians. Although ultrasound has been used by non-specialist clinicians for years, veterinary point-of-care ultrasound (VPOCUS) has only recently evolved as a specific arm of diagnostic imaging, tracing its origins back to the focused assessment with sonography for trauma exam published by Boysen et al. in 2004 [1]. As such, VPOCUS is now defined as focused real-time ultrasonography brought to the patient and performed by the attending clinician in conjunction with the clinical examination to answer specific questions (often binary) or to guide interventions [2–5]. Although clinicians from diverse backgrounds have become adept at using VPOCUS, it continues to be most prevalent in veterinary emergency and critical care (ECC), where it is one of many point-of-care (POC) diagnostics designed to expedite triage and time to diagnosis, with the ultimate goal of decreasing morbidity and mortality. It is a diagnostic modality that provides clinically significant information that is otherwise unattainable on physical examination alone, and is therefore complementary to triage, physical examination, and other point-of-care (POC) diagnostic and clinical tests or findings; it does not replace them.

VPOCUS is not meant to replace comprehensive sonographic examinations, which are traditionally consultative in nature being performed and interpreted by radiologists, cardiologists, or other board-certified specialists. Consultative sonography is intended to comprehensively evaluate extensive anatomy and physiology, often being performed with numerous open differential diagnoses in mind. VPOCUS applies specific goal-directed questions to well-defined clinical scenarios or problems with the objective of expediting patient care (e.g. presence of free fluid yes/no, dilated left atrium yes/no, visible B-lines at the lung surface yes/no). VPOCUS is designed to interpret a limited number of conditions and therefore follows a different standard of practice compared with consultative sonography (Table 6.1). For example, an emergency clinician may initially perform VPOCUS of the abdomen in a cardiovascularly unstable older Golden Retriever that presents for collapse, pale mucous membranes, weak pulses, an abdominal fluid wave, and a low hematocrit with a high pretest probability of hemoabdomen based on history and physical examination. The initial focused objective of the scan is to identify the presence or absence of free abdominal fluid, which if present can be aspirated and evaluated, subsequently guiding further diagnostic tests. In contrast, a radiologist-performed, consultative ultrasound of the dog's abdomen (when stable) would describe the entire anatomy of the abdomen, including a systematic, detailed description of the solid and hollow viscus organs. By keeping VPOCUS focused to specific goal-directed questions, the chance of errors is decreased, the most important clinically relevant questions are prioritized, and user confidence is increased.

The ability of VPOCUS to interpret clinical findings in real time has led to its widespread use with the applications continuing to expand rapidly as the capacity for research increases. However, it must be kept in mind that despite human studies validating POCUS as a safe, expedient, and cost-effective clinical adjunct that improves patient care, research in veterinary medicine is currently limited.

Depending on the question being asked, VPOCUS involves a series of focused ultrasonographic examinations restricted to certain organs or body regions to interpret specific underlying conditions in patients with defined clinical symptoms (e.g. dyspnea, hypotension,

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Table 6.1 Comparison of formal compared with point-of-care ultrasound.

Formal ultrasound	Point-of-care ultrasound
Consultative assessing all organs, anatomy, and structures	Focused to key structures to answer specific (often binary) clinical questions
Requires years of training	Requires minimal experience
Often takes > 30–60 minutes	Performed in < 5–10 minutes
Usually performed by specialists: cardiologists, radiologists	Often performed by non-specialists: emergency room doctors, general practitioners
Patients often stable	Patients often unstable
Patient taken to the machine	Machine taken to the patient
Placed in lateral or dorsal recumbency	Rarely if ever dorsal; lateral, sternal, standing preferred
Typically clipped	Rarely clipped
Gel preferred as the coupling agent	Uses alcohol ± gel as the coupling agent

acute abdomen). In general, ECC VPOCUS can be divided into three major components to help facilitate learning: (i) the abdomen; (ii) the pleural space and lungs; and (iii) the cardiovascular system. Interventional ultrasound-guided procedures are applicable within all three components. Additionally VPOCUS is applied at four different time points: (i) triage VPOCUS is applied as a tool to rapidly identify the most immediately life-threatening and critical conditions; (ii) serial VPOCUS is applied to monitor progression or resolution of any pathology, and response to therapy; (iii) systemic VPOCUS is aimed at detecting asymptomatic conditions, new developments, and/or to ensure sonographically detectable problems have not arisen prior to undertaking procedures, anesthesia, or discharge; (iv) therapeutic VPOCUS is used to reduce complications of interventions where applicable (Figure 6.1) [2]. The branches of VPOCUS often overlap but may be assessed individually or concurrently depending on the triage examination and initial clinical findings. For example, if a dog presents with respiratory distress and clinical signs suggestive of hypovolemic shock after having been hit by a car, all three components of VPOCUS are rapidly assessed to gain a systemic understanding of the potential underlying causes. On the other hand, if a dog presents with sudden onset of respiratory distress following an episode of vomiting, in the absence of abdominal discomfort or cardiovascular instability, the lungs are

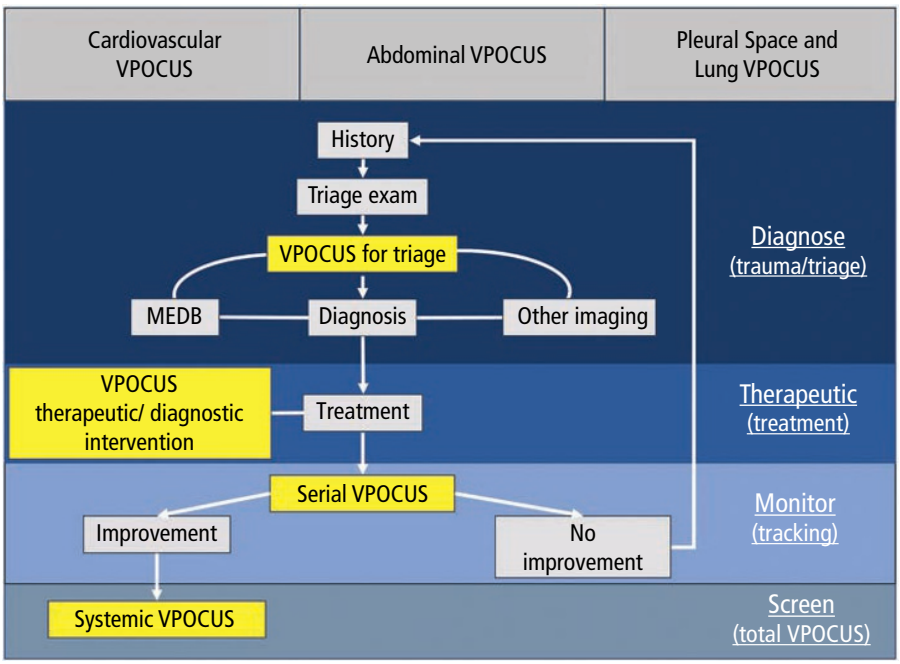


Figure 6.1 Veterinary point-of-care ultrasound (VPOCUS) in the emergency and critical care (ECC) settings currently involves three major components: (i) abdominal (ii) pleural space/lung and (iii) cardiovascular, which are assessed in a holistic manner. VPOCUS is also applied at four key integrated time points: (i) At the time of presentation, concurrent with history, triage, and other clinical findings, VPOCUS facilitates making a diagnosis or guiding further workup by thoroughly interpreting specific clinical scenarios. In this manner, VPOCUS is applied as a triage tool to identify rapidly the most immediately life-threatening and critical conditions. (ii) Serial VPOCUS is applied to monitor progression or resolution of any pathology, and response to therapy. (iii) Systemic VPOCUS is aimed at detecting asymptomatic conditions, new developments and/or to ensure sonographically detectable problems have not arisen prior to inducing anesthesia, performing procedures, or patient discharge. (iv) Finally, therapeutic VPOCUS is used to reduce complications of interventions where applicable. MEDB, minimum emergency database. *Source:* Adapted from Soni and Lucas (2015) [6].

assessed first, followed by the other VPOCUS as dictated by a systemic clinical assessment of the patient. Newer and other specialty-specific applications of VPOCUS (e.g. nerve blocks, optic nerve sheath diameter assessment) will likely play a future role in the ECC setting but are not covered here.

Machine Settings, Transducers, and Materials for ECC VPOCUS

- Bring the ultrasound machine to the patient! Do not discontinue stabilization efforts or compromise patient safety to perform ECC VPOCUS.
- A designated ECC portable ultrasound unit, independent of specialist console units (e.g. cardiology, radiology), is recommended for busy emergency clinics to ensure ultrasound is available at the time of patient presentation and can be transported to the patient as needed.
- A 5–8 mHz frequency microconvex/curvilinear transducer can be used for all VPOCUS applications. A linear array probe is helpful for ultrasound-guided procedures such as ultrasound-guided intravenous (IV) catheter placement, and a phased array probe can be used for echocardiography, but neither is essential.
- Although ultrasound consoles have many functions, the key machine functions for VPOCUS include frequency, gain, depth, and focal position.
- Frequency is adjusted to highlight structures of interest, typically 5–7.5 mHz (see individual sections). As for any sonographic examination, higher frequency settings are indicated for more superficial structures, while lower frequency settings are required to evaluate deeper structures.
- The received ultrasound signal can be modified by adjusting the gain. Decreasing the gain yields a darker (blacker) image with a loss of detail, while increasing the gain yields a brighter (whiter) image. Gain is adjusted to user preference and the structures of interest, depending on the binary question to be answered (see individual sections).
- Depth is adjusted to visualize the region of interest. Begin with a somewhat higher depth setting to obtain a "big picture" assessment to find the structure of interest, and then gradually decrease the depth to visualize the desired structures in more detail.
- Focal position is adjusted to the area of interest.
- Isopropyl alcohol is often used as the coupling agent; part the fur so the skin is visible before applying alcohol. Ultrasound gel can also be used alone or in combination with alcohol.
- Alcohol can create artifact on subsequent radiographs. Alcohol should be replaced with gel if electrocautery or defibrillation is a possibility due to the risk of fire.
- Sedation is rarely required for VPOCUS since it is noninvasive and requires minimal restraint. In some situations, sedation may decrease anxiety and work of breathing, which may improve image acquisition in certain settings, particularly for the lungs and heart.
- If image resolution is questionable, clipping of fur with addition of ultrasound gel may improve image quality.

Transducer Movements

Acquiring good sonographic images of the desired object requires both broad and fine adjustments to the transducer, in multiple planes and directions. Even the tiniest movements of the transducer (millimeters or 1–2 degrees) can have a significant impact on determining whether or not the desired object can be correctly imaged: applying a single transducer movement at a time is often necessary to avoid simultaneously manipulating multiple imaging planes and making it difficult to obtain the best image. There are five key transducer manipulations that must be understood to perform VPOCUS: fanning, rocking, sweeping, sliding, and rotating:

1) Fanning: the transducer head remains stationary while the tail of the transducer is moved side to side relative to the transducer's widest axis, similar to the movement of a handheld paper fan.
2) Rocking: the transducer head remains stationary while the tail of the transducer is moved side to side relative to the transducer's shortest axis, similar to the motion made when cutting something with the side of a fork.
3) Sweeping: the entire transducer is moved across the area of interest (the point of contact between the probe and patient is changed) in the short axis direction, without changing the angle of the probe relative to the target structure.
4) Sliding: the entire transducer is moved across the area of interest (the point of contact between the probe and patient is changed) in the long axis direction, without changing the angle of the probe relative to the target structure.
5) Rotating: the transducer head is rotated in a clockwise or counterclockwise direction while maintaining the same point of contact and probe angle relative to the target organ.

With fanning, rocking, and rotating transducer manipulations, the contact point between the transducer and the patient remains the same, and the transducer is manipulated around the point of contact. With sliding and sweeping, the transducer head is moved away from the initial point of contact between the transducer and the body surface of the patient. See Figure 6.2 for a visual representation of these movements.

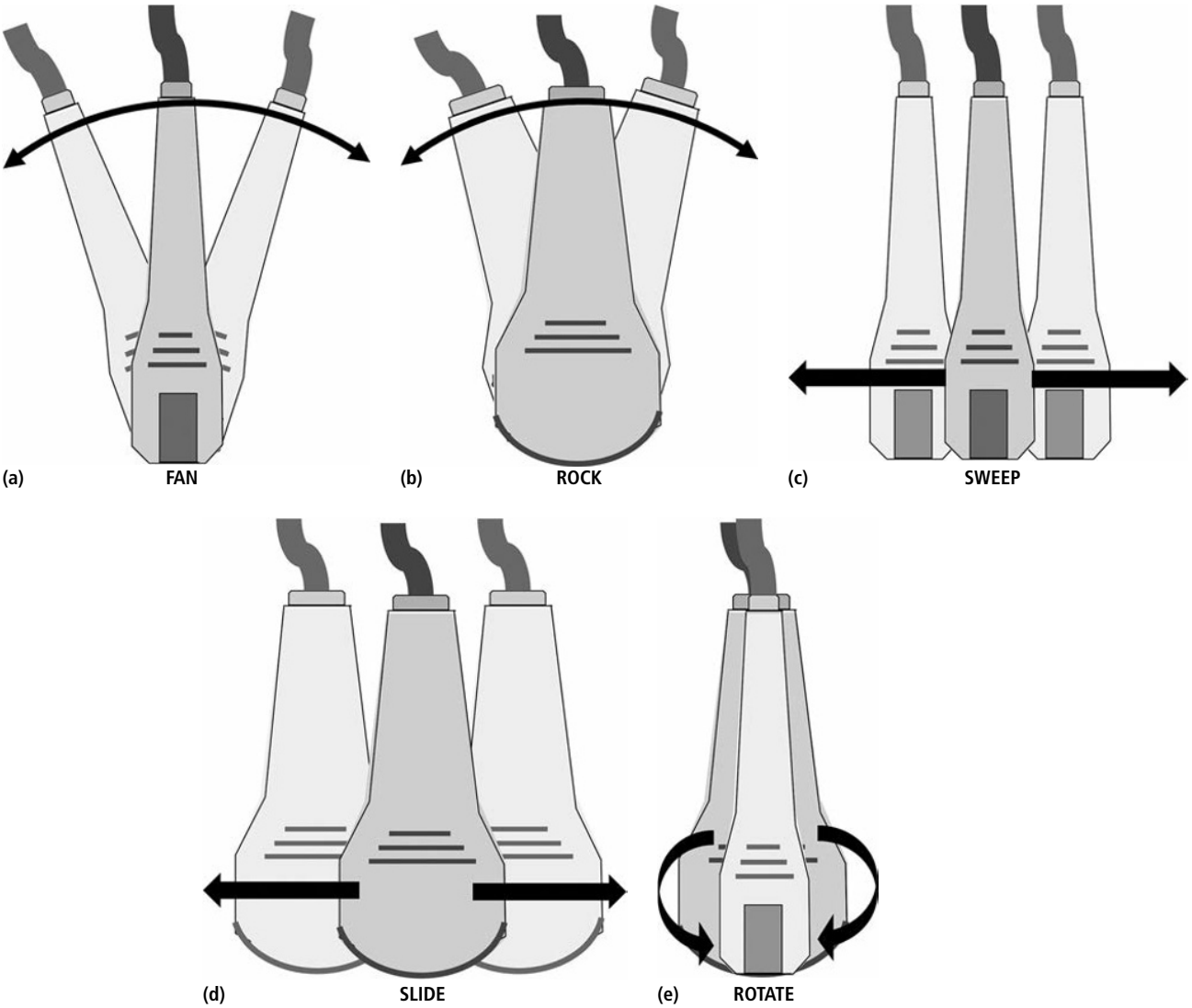


Figure 6.2 (a) Fanning: the transducer head remains stationary while the tail of the transducer is moved side to side relative to the transducer's widest axis. (b) Rocking: the transducer head remains stationary while the tail of the transducer is moved side to side relative to the transducer's shortest axis. (c) Sweeping: the entire transducer is moved across the area of interest (the point of contact between the probe and patient changes) in the short axis direction, without changing the angle of the probe relative to the target structure. (d) Sliding: the entire transducer is moved across the area of interest (the point of contact between the probe and patient changes) in the long axis direction, without changing the angle of the probe relative to the target structure. (e) Rotating: the transducer head is rotated in a clockwise or counterclockwise direction while maintaining the same point of contact and probe angle relative to the target structure.

Indications

There is no limit to the indications for VPOCUS, with new applications being developed as research grows; however, the applications will vary depending on the clinical scenario encountered and the sonographer's comfort and skill level:

- Any patient as part of the triage examination.
- Trauma patients.
- Unstable patients.
- Respiratory distress.
- Patients not recovering as expected from surgery.
- Routine daily assessment of hospitalized patients.
- To assess for peritoneal effusion of any cause.
- Suspicion of pneumoperitoneum.
- Concern for adequate urine production.
- Suspicion of gastric retention or gastrointestinal ileus.
- Suspected pneumothorax.
- Suspected pleural effusion.
- Suspected alveolar or interstitial lung disease.
- Patients with suspected congestive heart failure.
- Assessment of hypovolemia or volume overload.

Serial VPOCUS

Serial VPOCUS is recommended to: (i) monitor progression/resolution of patients with positive VPOCUS findings (e.g. resolution or progression of cavitary fluid volumes, left atrial-to-aortic ratio in response to fluid therapy, or resolution/development of B-lines); and (ii) to detect new developments in patients over time, particularly those that become unstable in the absence of an identifiable cause, and/or have received significant quantities of IV fluids or other therapeutic interventions (Figure 6.1). The frequency of serial VPOCUS examinations depends on the patient and may vary from several minutes to hours. It should be repeated as often as required to identify the reason a patient is unstable if no cause is evident on ancillary diagnostic tests, or to determine why a patient changes from stable to unstable. If the patient is stable and the goal is to simply follow resolution or progression of underlying pathology, VPOCUS can be repeated every two to four hours.

Limitations

In most cases, VPOCUS is better at ruling in pathology than ruling out pathology; a negative VPOCUS result does not exclude pathology. In people, POCUS has low sensitivity for penetrating abdominal injury and retroperitoneal injury, which is also likely to be true in veterinary patients. However, it is still worth evaluating patients with penetrating injury and suspected retroperitoneal injury since a positive result may dictate further diagnostics and therapy. Initial hypovolemia or severe dehydration may limit detection of effusion, making it important to use serial VPOCUS during and following adequate resuscitation. There is a learning curve to VPOCUS and sonographers should know their limitations; some skills are easier to acquire than others. Finally, although VPOCUS aims to answer POC binary questions, the clinical interpretation of VPOCUS is highly dependent on concurrent clinical findings such as signalment, history, physical examination, as well as other binary VPOCUS questions (e.g. a cat presenting for collapse with the VPOCUS finding of a thick left ventricular wall and a small left atrium would be suggestive of pseudohypertrophy secondary to hypovolemia, while a thick left ventricular wall associated with an enlarged left atrium and increased B-lines would be suggestive of hypertrophic cardiomyopathy). As mentioned above, VPOCUS is part of the holistic approach to patient care.

Conclusions

VPOCUS is focused, real-time ultrasonography brought to the patient, performed by the attending clinician in conjunction with the clinical examination, to answer specific questions (often binary) or to guide interventions. It is best applied as a problem-based assessment to enable the clinician to gather key pieces of information in real time to help narrow or determine a diagnosis, streamline care, guide ongoing management, and reduce cognitive errors. The dynamic, real-time findings are correlated directly with the patient's presenting signs, and scans can be repeated in a serial fashion. POCUS is a skill that is easily applied by appropriately trained clinicians, particularly when using a binary approach. Although evidence-based research for the role of POCUS in veterinary medicine is lacking, preliminary results suggest it can be used as a rapid and reliable diagnostic tool.

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Section Two

Cardiovascular Procedures and Monitoring

7

Catheterization of the Venous Compartment

Andrea M. Steele and Jessica L. Oram

Intravascular catheter placement is the most common method used to gain access to the vasculature to allow for fluid therapy, medication administration, serial blood sampling, and hemodynamic monitoring. Anatomical selection and precise placement of an intravascular catheter are essential whether performing a single intravenous (IV) injection or providing long-term vascular access in an ill or injured patient. Understanding the care of intravascular catheters is key to avoiding adverse events.

Selection of Catheter Insertion Site and Type

Many factors will influence the type of IV catheter used and the site chosen for its placement (summarized in Boxes 7.1 and 7.2). The length and diameter of the catheter selected must be appropriate for its intended use. Peripheral IV catheters are ideal in emergent patients as they are simple to place and allow for rapid fluid resuscitation. Central venous catheters may be preferable for long-term patients, for serial blood sampling, central venous pressure monitoring and for additional therapies such as parenteral nutrition (PN).

Choosing the right catheter size is often left to the individual performing the task and it is rare to follow a protocol in the veterinary field. In contrast, in human medicine, clear guidelines have been established for catheter size based on the type of therapy being initiated. The recommendation is to avoid catheter to vessel ratios greater than 45% (recently increased from 33%). For veterinary patients, there is often a desire to obtain the largest catheter possible, often fully occluding, or even stretching the vessel, in an effort to push fluids as fast as possible. This is often unnecessary, and frequently delays therapy due to multiple attempts at catheterization.

Table 7.1 summarizes the flow rates of one particular catheter brand (each brand will have slight individual variances), showing that even the smallest 24-gauge catheters can deliver a significant volume of fluid in one hour by gravity drip. This rate far exceeds the "shock rate" most commonly used in veterinary patients using realistic sizes and suggests that successful fluid resuscitation can occur with smaller catheter sizes than previously thought. The use of a fluid pump or pressure bag may further increase this rate. As a guideline, cats 24–22 G, small dogs less than 10 kg 24–22 G, dogs 10–30 kg 22–20 G and dogs over 30 kg 20–18 G would more than meet the needs of maximal fluid rates, and also be easier to place when a patient is in cardiovascular collapse. In the author's experience, obtaining a smaller catheter in the emergency phase is better than delaying therapy attempting to obtain the largest possible catheter.

In a patient in cardiovascular collapse, the peripheral veins may not be accessible, even with the smallest catheter. In this case, consider the jugular veins, as they will often still be visible. A traditional over-the-needle catheter (using a longer catheter for extra stability) can usually be placed easily and sutured in place until the patient is stabilized and the catheter can be replaced in a peripheral vein.

Emergently placed IV catheters should always be replaced as soon as possible, as there was likely inadequate skin preparation (including minimum contact time and hand hygiene), and increased manipulation of the skin or vessel, which could increase risk of contamination.

There is a vast market of IV catheters (Figure 7.1). Catheters are generally categorized as over-the-needle, through-the-needle, or winged ("butterfly"), and as either single or multilumen catheters.

Butterfly catheters are not meant for extended use and are best for blood collections or short injections. These are nothing more than a steel needle with wings and a built-in extension set. Butterfly catheters should not be affixed to

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Box 7.1 Catheter Insertion Site Location Considerations

- Patient size and species
- Venous accessibility
- Presence of physical barriers (wounds, fractures, neoplasia, neurologic insufficiencies)
- Restraint requirements
- Temperament of patient
- Hemodynamic stability
- Coagulation abnormalities
- Vessel size
- Skill of the person placing the catheter

Box 7.2 Catheter Selection Considerations

- Length of hospitalization
- Frequency of blood sampling
- Coagulation abnormalities
- Nutritional delivery
- Hemodynamic monitoring plan

Table 7.1 Gravity flow through various size and length catheters.

Catheter gauge (G)	Length		Gravity flow	
	(inches)	(mm)	(ml/minute)	(ml/hour)
24	0.75	19	20	1200
22	1.00	25	37	2220
20	1.00	25	63	3780
20	1.88	47	54	3240
18	1.16	29	95	5700
18	1.88	47	87	5220
16	1.16	29	193	11580
16	1.88	47	185	11100

Source: Becton Dickinson Data: Insyte Autoguard® Shielded Catheters.

the patient, and must be removed immediately upon completion of the procedure.

The structural material of the catheter can influence rigidity, ease of placement, and the potential for thrombosis or phlebitis of the vessel. Common materials for IV catheters include polytetrafluoroethylene, polypropylene, polyurethane, and silicone. Different brands of catheters may be more flexible or have thicker-or-thinner walls, may be easier to affix than others (some catheters have wings on the hub which can make taping or suturing easier). Each type has its advantages and disadvantages, and generally speaking, users become comfortable with certain catheters over time, and rarely like to change brands/types. Ideally, each hospital will have a brand/type of catheter that the majority of users are comfortable with, have good success

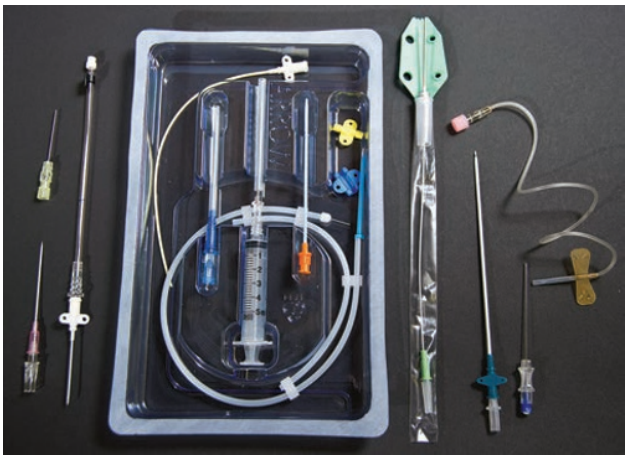


Figure 7.1 There are many different venous catheters available. Catheter selection should be based on patient need and length of hospital stay. Source: Insyte Autoguard® Shielded Catheters, BD.

in placing, have been tested in their patient population, and will train new personnel on their use, rather than providing a selection of catheter brands.

Also available are specialty catheters impregnated with radiopaque metal salts for radiographic confirmation of correct anatomical placement, or to locate accidentally freed catheter fragments. In addition, IV catheters coated with antimicrobial substances marketed to decrease the incidence of infection are now commercially available, although there is little evidence in veterinary medicine to support the claims of reduced infection.

Placement of any type of catheter should have a universal protocol in the hospital for placement. This protocol should encompass the selection of vessel and catheter type and size, hand hygiene and skin preparation, placement of the catheter, affixing the catheter to the patient, and finally, care and maintenance of the catheter. Suggested placement protocols for each type of catheter discussed in this chapter are presented below.

Care bundles for various procedures have been in use in human medicine for many years, and provide a framework for the placement and maintenance of many devices, with the main goal of preventing device related hospital associated infection and complications, and critically evaluating the need of the device on a daily basis. The care bundle is essentially a checklist of "best practices," and takes into account preparation of supplies and patient, hand hygiene, wearing of caps/masks/sterile gloves and maintaining a sterile field, and ensuring all steps are followed. The care bundle is then used every 12–24 hours to ensure that the device is being maintained and closely monitored and the veterinary team is critically evaluating whether the device is still necessary. Care bundles can be created for any device, such as IV catheter, central line, urinary catheter, or arterial catheters, to name a few. An example of a care bundle created by the primary author is shown in Figure 7.2.

Patient Label

CENTRAL LINE BUNDLE

Clinician's Orders:

Requested Catheter Type: _____

Requested Catheter Site: Jugular ○ (Preferred) Saphenous ○

Procedure: Stable Patient ○ Emergent ○

Sedation Orders: _____

Concerns: _____

Clinician Signature: _____

Insertion Checklist

Date Placed: _____ **New Catheter ○ Replacement ○**

Performed by: _____

Catheter Brand/Size/Lot #	
Location	R ○ L ○ Jugular ○ Saphenous ○

# of attempts: _____ **R Side** _____ **L side** _____ **New Catheter ○ or Introducer ○ each attempt?**

Cutdown: Yes ○ No ○ Performed by: _____

Procedure Checklist

Safety Practice	**Yes**
Assess location, clip area, apply Maxilene ointment & cover with plastic dressing	○
Assemble all supplies	○
Position patient and perform surgical skin prep of area, allow 5-minute contact time	○
Aseptically open catheter kit & prepare supplies	○
Wear a cap and mask	○
Hand Hygiene	○
Don sterile gloves	○
Use a sterile drape to cover field	○
Maintain a sterile field	○
Did the restrainer wear a cap and mask?	○
Apply sterile dressing to insertion site	○
Apply bandage to neck/limb to secure	○
Date & initial catheter	○

Form Completed by: _____ Signature: _____

SEE BACK FOR CATHETER CARE Checklist

Figure 7.2 Central venous catheter care bundle example.

CENTRAL LINE BUNDLE

Catheter Care Checklist **Date Placed:** _____

Day #	Bandage Changed*	Catheter Assessed (Q12 hrs) and Comments	Catheter Care Initials	Need Assessment (q 24 hrs)
1	am ◯	am ◯		◯ Continue ◯ Remove
	pm ◯	pm ◯		Clinician's Initials _____
2	am ◯	am ◯		◯ Continue ◯ Remove
	pm ◯	pm ◯		Clinician's Initials _____
3	am ◯	am ◯		◯ Continue ◯ Remove
	pm ◯	pm ◯		Clinician's Initials _____
4	am ◯	am ◯		◯ Continue ◯ Remove
	pm ◯	pm ◯		Clinician's Initials _____
5	am ◯	am ◯		◯ Continue ◯ Remove
	pm ◯	pm ◯		Clinician's Initials _____
6	am ◯	am ◯		◯ Continue ◯ Remove
	pm ◯	pm ◯		Clinician's Initials _____
7	am ◯	am ◯		◯ Continue ◯ Remove
	pm ◯	pm ◯		Clinician's Initials _____
8	am ◯	am ◯		◯ Continue ◯ Remove
	pm ◯	pm ◯		Clinician's Initials _____
9	am ◯	am ◯		◯ Continue ◯ Remove
	pm ◯	pm ◯		Clinician's Initials _____
10	am ◯	am ◯		◯ Continue ◯ Remove
	pm ◯	pm ◯		Clinician's Initials _____

***Bandage change q 12 or 24 hrs: highlight preference (Minimum q 24 hrs)**

Figure 7.2 (Continued)

Peripheral Catheter Placement

Peripheral venous catheters are the most commonly used tools for obtaining vascular access in the small animal patient. Ease and speed of insertion, low cost and versatility of access in different anatomical locations make peripheral catheters advantageous over other catheter types. Most peripheral catheter types are categorized as over-the-needle, meaning that the catheter is fitted outside or over a steel needle (Figure 7.3). The steel needle extends slightly beyond the catheter tip to facilitate venous entry. Once venipuncture is accomplished, the catheter is slid off the needle into the targeted vessel.

Anatomical locations for placement of peripheral catheters include the cephalic, accessory cephalic, or even the jugular vein during stabilization. Other easily accessible sites for placement include the lateral saphenous vein in the dog or medial saphenous vein in the cat. Less common placement sites include the dorsal common digital vein, auricular vein, or lingual vein. Other common uses for the over-the-needle catheter type include pericardiocentesis, abdominocentesis, and thoracocentesis.

Peripheral catheters are available in a variety of gauges and lengths, typically ranging from 24 G to 12 G and from 0.75 inches (1.9 cm) to 12 inches (30 cm) in length. Longer catheters used for pericardiocentesis or abdominocentesis are typically fenestrated for optimum fluid collection [1].

Overall, ease of insertion and low costs associated with placement and maintenance make the peripheral IV catheter ideal for initial venous access in the emergency patient. Peripheral over-the-needle catheterization is the most common venous access technique in veterinary medicine.

The technique is described in Protocol 7.1, however some common pitfalls that can be avoided are described below. Peripheral catheters should not be placed on, or close to, a moving joint (e.g. elbow) to avoid positional (flow) complications. The cephalic vein is the most common insertion site in dogs and cats and offers the radius and ulna as ideal long bones to stabilize the catheter. Initiating catheter insertion too high or using too long of a catheter should be



Figure 7.3 The most common peripheral catheter is the over-the-needle type, meaning that the catheter is fitted outside or over a steel needle. Such catheter types are very versatile and can be placed in many different anatomical locations.

avoided, as this can cause the catheter tip to terminate in the elbow; or alternatively, too low a placement will require hub stabilization over the carpus. Other insertion sites described above can require more creative stabilization.

The authors do not recommend the *routine* use of a relief incision (not to be confused with the mini-cutdown described below) to gain access to the vessel, as it causes trauma to the insertion site and increases mobility of the final catheter. This is performed by using a similar, or slightly larger size needle then the intended catheter, piercing the skin alongside the vein with the tip, and rotating so the cutting edge of the bevel is facing up. The edge of the needle is then pulled up and through the skin, resulting in a small 1–2 mm incision. This technique is used by many as a default for all insertions, however a proper insertion angle, and using high-quality catheters can usually solve insertion issues. Placing gentle traction on the skin and using a 30-degree insertion angle to allow the bevel to cut through the skin facilitates a smooth, atraumatic piercing of the skin. A flat angle is a common mistake, requiring additional force to pierce the skin resulting in burring of the catheter and trauma to the skin.

After placement of the peripheral catheter, it is important that the catheter not move within the vein, as damage to the

Protocol 7.1 Peripheral Catheter Placement

Items Required

- Clippers
- Hand sanitizer or handwashing station
- Surgical scrub
- 1-inch porous medical tape
- T-port or infusion cap
- Saline flush
- Bandage material
- Appropriate work surface and at least one assistant

Procedure

1) Select the best placement site, taking into consideration patient comfort, ease of accessibility, and anticipated catheter dwell time. If chemical restraint is necessary, allow adequate time for optimum results. Consider the use of a topical anesthetic.
2) Collect necessary supplies.
3) Closely clip a generous area (2–2.5 inch – approximately 5 cm) around the intended insertion site and remove any loose fur. There is no need to clip circumferentially around the limb, but feathers should be trimmed to avoid hair contamination of the insertion site and make tape removal more comfortable.
4) Perform hand hygiene.
5) Aseptically prepare the area using hospital protocol.
6) Perform hand hygiene again after prepping.
7) Restrain patient; avoid excessive stress to patient. Restrainer should occlude the vessel proximally to ensure maximum visualization.
8) Unfold a sterile 3 × 3-inch gauze and place at the bottom of the prepped area (Figure 7.4). Use the gauze to apply gentle traction on the prepped area and minimize vessel roll. The gauze also prevents the catheter from contacting hair at the bottom of the prepped area.
9) Visualize targeted vessel, if palpation is necessary, do so away from the anticipated insertion site; point of entry should be directly on top of or beside the intended vessel.
10) Introduce catheter into the skin with bevel side up at an angle of 30 degrees, the optimal angle to cut through the skin. Once through the skin, reduce the angle of the catheter to enter the superficial vessel.
11) After a "flash" of blood is visualized in the stylet hub, advance the catheter and stylet an additional 1–2 mm, confirming blood is still flowing into stylet and thus remains in the vessel.



Figure 7.4 Demonstrating the use of an open piece of gauze which keeps the catheter from contacting hair at the bottom of the prepared area. The gauze can be used to place traction on the skin to facilitate placement. Note the 30-degree angle used to enter the skin. Once through the skin layer, flatten the catheter to enter the vessel.

12) Slide the catheter off the needle and into the vessel. During catheter advancement, ensure the stylet remains stationary, and the catheter is advanced off the stylet.
13) Remove the stylet fully and attach a sterile infusion cap to the catheter hub. If restrainer uses digital pressure to staunch blood flow during this transition, ask them to occlude at the catheter tip, and avoid touching near the insertion site.
14) If blood is required from the patient, remove from the catheter at this point, using gentle negative pressure. This may not be possible in small or cardiovascularly collapsed patients.
15) Flush catheter with saline to ensure and maintain patency. Replace injection cap with a preflushed T-port if preferred.
16) Anchor catheter with adhesive tape. Avoid tape touching the insertion site, placing a sterile adhesive bandage ("plaster bandage") over the insertion site prior to final taping (Figure 7.5).
17) Apply a light, protective bandage, again per hospital protocol, and anchor infusion set to the outside of the bandage.

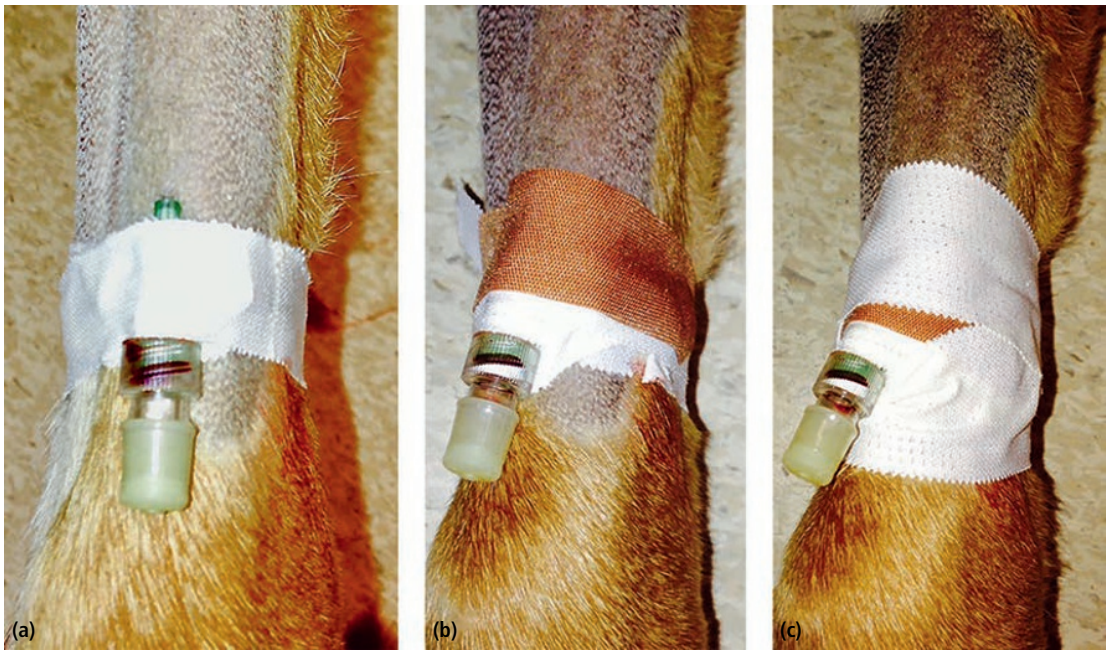


Figure 7.5 (a) Initial tape pieces secured only to the hub of the catheter, and do not contact insertion site. (b) A sterile adhesive bandage is placed over the insertion site, with sterile pad contacting the insertion site. (c) Final tape is placed over top of the bandage to secure.

vessel may occur and increase the risk of thrombus formation and phlebitis. There are numerous techniques for taping catheters in place, and this should be a hospital-wide protocol that all technicians and nurses utilize. Avoid techniques that use a single piece of tape as these are inherently unstable. The technique used must prevent movement of the catheter and should secure the catheter at the same angle as placed, avoiding kinks or bends. The use of a sterile adhesive bandage over the insertion site can act as a sterile buffer between the tape (which is frequently contaminated) and the insertion site and may reduce infection risk. If the patient is to be administered IV fluids, anchoring a small portion of the tubing line directly to the bandage will help reduce catheter migration and movement.

Protocol 7.2 Mini-Cutdown

Items Required

- Clippers
- Surgical scrub
- 20- or 18-gauge hypodermic needle
- Peripheral catheter
- Saline flush
- T-port
- Catheter cap
- 1-inch medical tape
- Bandage material

Procedure

1) Select best placement site, taking into consideration patient comfort, ease of accessibility, and anticipated catheter dwell time. If chemical restraint is necessary, allow adequate time for optimum results.
2) Collect necessary supplies.
3) Closely clip a generous area (2–2.5 inches, approximately 5 cm) over the intended insertion site and remove any loose fur.
4) Perform hand hygiene.
5) Aseptically prepare the area using hospital protocol.
6) Perform hand hygiene and don sterile gloves.
7) Restrain patient; avoid excessive stress to patient. Restrainer should occlude the vessel manually and proximally to ensure maximum visualization.
8) Visualize target vessel.
9) Use edge of bevel of 22–18 G hypodermic needle as a "scalpel" to cut the skin in a distal to proximal direction adjacent and directly parallel to the targeted vessel.
10) Dissect around vessel further as needed, using needle as a mini scalpel with bevel directed parallel to vein.
11) Visualize vein and insert catheter.
12) Secure catheter as usual and place a sterile dressing. Suture or staple incision closed if necessary.

Peripheral Cutdown Techniques

In emergency situations, if traditional peripheral catheterization attempts are unsuccessful and the patient is in peril, venous catheterization may require a cutdown procedure (e.g. exposing the vein surgically). Cutdowns allow quick vessel visualization and ensure catheterization on the first attempt. In addition, cutdowns can allow placement of a larger catheter than what can typically be placed percutaneously. Aseptic technique should be followed except in dire situations. The veterinarian performing the cutdown procedure should wear sterile gloves.

Mini-Cutdown

The mini-cutdown technique is performed to gain access to either the cephalic or lateral saphenous vessel that may be collapsed or difficult to enter (Protocol 7.2). While this procedure does not isolate the vessel like a surgical cutdown described below, it does provide better visualization, which can increase catheter insertion success.

Using a sterile 18–22 G hypodermic needle, hold the needle with the bevel up and use the needle tip to cut the skin directly parallel to the targeted vessel (Figure 7.6). Infusion of subcutaneous lidocaine prior to the mini-cutdown is rarely needed. Once the incision has been made, the skin defect can be moved directly over the vessel. Tearing of the skin will release skin tension without damaging the underlying vessel, allowing for better visualization necessary for rapid catheter placement. A small incision can also be made adjacent to the vein with a number 11 scalpel blade, using caution to avoid incising the vessel and subcutaneous tissue. Blunt dissection with mosquito forceps may be required to visualize the vein; minimal dissection is usually required for the placement of an over-the-needle or through-the-needle catheter.



Figure 7.6 A sterile 18–22-gauge hypodermic needle can be used to create a mini "cutdown" when venous visualization is difficult.

Surgical Cutdown

A surgical cutdown is sometimes necessary to obtain vascular access in patients with hypovolemia or if central venous access is needed for hemodynamic monitoring, parental nutrition or hemodialysis (Protocol 7.3). Surgical cutdowns can be used to access the external jugular, cephalic or lateral

Protocol 7.3 Surgical Venous Cutdown

Items Required

- Clippers
- Surgical scrub
- Sterile gloves
- Sterile mosquito forceps
- Sterile thumb forceps
- Sterile sharp-sharp scissors
- Sterile needle driver
- Sterile catheter
- Sterile syringes filled with saline
- Sterile injection port and T-port
- Sterile absorbable monofilament suture
- Sterile scalpel blades (#10, #11, #15)
- Sterile 4 × 4-inch gauze squares
- Small eye drape
- Bandage material
- Optional: catheter introducer, towel clamps

Procedure

1) Select best placement site, taking into consideration patient comfort, ease of accessibility, and anticipated catheter dwell time. If chemical restraint is necessary, allow adequate time for optimum results.
2) Collect necessary supplies.
3) Closely clip a generous area (2–2.5 inches – approximately 5 cm) around the intended insertion site and remove any loose fur.
4) Aseptically prepare the area using hospital protocol.
5) Perform hand hygiene and don sterile gloves.
6) Restrain patient; avoid excessive stress to patient. Restrainer should occlude the vessel proximally to ensure maximum visualization but should release occlusion during incision to avoid accidental vessel laceration.
7) If chemical sedation or general anesthesia is not feasible, infuse lidocaine (0.2–0.5 ml) into the subcutaneous region around the intended insertion site.
8) Prepare skin with a final surgical scrub and maintain a sterile field by placing eye drape over prepared area.
9) Make a skin incision immediately parallel to the vessel with a #10 or #15 scalpel blade.
10) Use blunt dissection to expose vein; dissect vein with curved hemostats so minimal fascia remains attached.
11) Slide hemostats under vein to level of handles to create a platform.
12) Tie off vein distally (or cranially if jugular vein is used) using absorbable monofilament suture; keep ends long to use for traction.
13) Place second suture around vein proximally, but do not tie.
14) Insert #11 scalpel blade parallel to vessel length, through center of vessel; turn 90 degrees and cut outward.
15) Using catheter introducer or blunt end of curved needle, open vessel and slide in largest bore catheter possible.
16) Tie second suture trapping vein to catheter; ensure at least 3 mm tissue bumper.
17) Secure catheter to fascia of neck or thigh muscles as appropriate for location.
18) Suture incision (leave partially open if not performed under aseptic conditions).
19) Towel clamps can be used for temporary closure of incision.
20) Place sterile dressing.

saphenous veins, with similar technique for all venous sites (Figure 7.7). Complications of a surgical cutdown can include perforation of the vascular wall, shock or hematoma, thrombosis, venous transection, infection, and cellulitis.

Cutdown incisions should be made (i) in the middle to cranial portion of the jugular groove for catheterizing the external jugular vein; or (ii) on the dorsal antebrachium or lateral aspect of the distal tibia, respectively, for the cephalic and lateral saphenous catheterization.

After clipping and performing an aseptic preparation of the targeted area, a scalpel blade is used to incise the skin directly over, or adjacent to the vein. Either a transverse or a parallel skin incision can be made, using caution to incise only the full thickness of the skin. If the vessel is not immediately identified, it may be necessary to bluntly dissect with mosquito forceps adjacent to the vein (Figure 7.7a). The vein can be identified as a blue to-purple tubular structure within the subcutaneous tissue. Thumb forceps can be used to retract the subcutaneous tissues to expose the vessel. Dissection of adventitial tissue adjacent to the vein can be facilitated by the use of sharp-sharp scissors dorsally and ventrally to the vein; dissecting in a parallel fashion will minimize vessel trauma or rupture.

Once the vessel is isolated from the subcutaneous tissue, a blunt hemostat is slid beneath the vessel through tissue as a type of "platform" for the vessel to lie on top of the instrument (Figure 7.7b). A silk suture is then clasped by the hemostat and drawn beneath the vessel. The suture can be used to retract the vein distally to provide adequate tension for catheterization in the case of peripheral vessels. Alternately, the

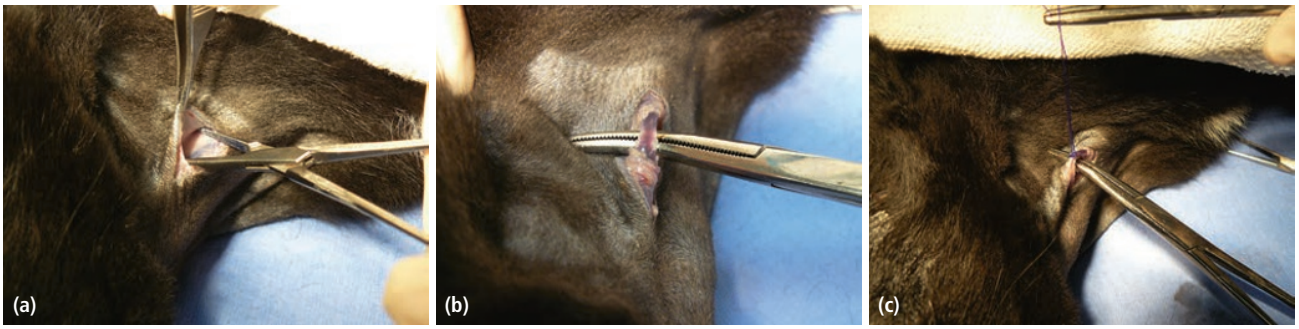


Figure 7.7 (a) The isolation of the vessel can be accomplished by blunt dissection of the subcutaneous tissue adjacent to the vein. (b) A hemostat can be slid underneath the vessel to create a "platform" to facilitate venous entry. (c) Tie off the vessel proximally using absorbable suture.

jugular vein may be sacrificed by ligating it cranially and using the ligature to retract the vessel. A second suture is placed beneath the vessel proximally (Figure 7.7c). The vein is then cannulated through a venotomy (or by venipuncture) between the two ligatures. Following catheterization, the distal suture is tied, securing the catheter in the vein [1].

The catheter should be replaced once the patient is resuscitated and hemodynamically stable. Remove the catheter by using gentle traction and applying direct pressure over the insertion site for several minutes. Allow the incision to heal by second intention if it was not placed under aseptic conditions.

Central Venous Access

Central venous catheters have many advantages over peripheral catheters, including that they allow for a longer dwell time, measurement of central venous pressure, safer administration of hyperosmolar solutions such as total parental nutrition, and serial blood sampling with minimal stress to the patient. The central venous catheter is typically introduced via the jugular vein, but it can be readily inserted into the caudal vena cava via the lateral saphenous in the dog or the medial saphenous vein in the cat. Central venous catheters are available with either a single lumen or with multiple lumens, which allows for the simultaneous delivery of incompatible fluid types as is often needed in the critical patient. Central venous catheters are less likely to be affected by both patient positioning and motion at the point of insertion than peripheral catheters. Centrally situated catheters also allow for cannulation of larger vessels.

Disadvantages of central venous catheters include a longer placement time, greater expense, slight patient discomfort during placement and the longer length may make rapid fluid administration problematic. Monitoring for extravasations with the central venous catheter can also be more difficult than in the peripheral over-the-needle catheter; veterinary technicians should monitor the patient for edema of the neck or sternum, pain with administration of medications, sluggish flow through the catheter, or an inability to aspirate blood back from the catheter. It may be prudent to avoid central venous catheters in patients at risk of thrombosis (e.g. immune-mediated hemolytic anemias) or in animals with known or suspected coagulopathy. In addition, occlusion of the jugular vein may increase intracranial pressure during insertion, which is a relative-to-absolute contraindication in patients with head trauma or other central nervous system disturbances. In patients requiring central venous access and whose intracranial pressure may be elevated, a peripherally inserted central catheter (PICC) can instead be placed via the medial saphenous or lateral saphenous vein.

Catheter Types for Central Veins

Central venous catheter types include long over-the-needle, through-the-needle, and guidewire catheters; all types can be placed via a peripheral or central vessel. Shorter over-the-needle catheters are used primarily for peripheral vein catheterization but can be centrally placed in pediatric patients. One disadvantage of centrally located over-the-needle catheters is patient discomfort, as these items tend to be stiff and can malfunction due to kinking.

Guidewire central venous catheters are placed by the modified Seldinger technique and include single and multilumen catheter systems (i.e. Arrow® Central Venous Catheter, Teleflex Medical, Research Triangle Park, NC; see Figure 7.8). Catheters placed by the modified Seldinger technique are typically soft and flexible and made of a polyurethane material, which is antithrombogenic and can dwell in the patient for a longer period. These catheters are available in a multitude of gauge, length, and lumen number combinations. Certain guidewire central venous catheters are impregnated with antimicrobial substances that may reduce the likelihood of catheter-related sepsis. Disadvantages of the modified Seldinger central venous catheter types are expense, increased risk of local hemorrhage during

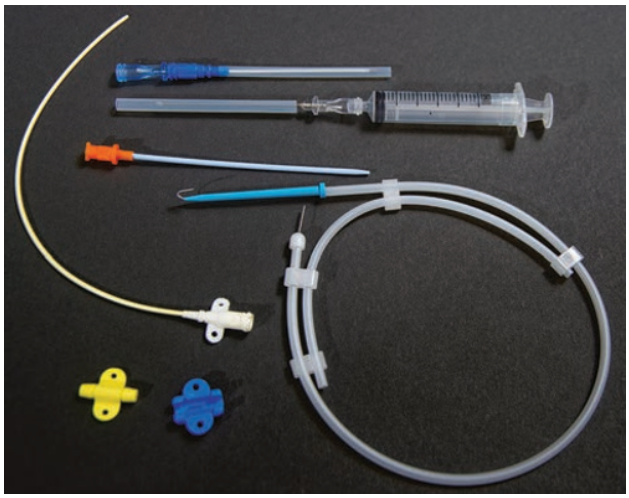


Figure 7.8 Central venous catheter kits using the guidewire (modified Seldinger) technique; such catheters range from single to multilumen devices.

insertion, procedure time, and expertise needed for placement. It is the authors' opinion, however, that the risks and expense of such central venous catheters are far outweighed by the advantages in many critical patients.

Peel-Away® (Cook Medical, Bloomington, IN) sheath introducers can be used to introduce similar catheters to those inserted with the modified Seldinger technique: single to multi-lumen, flexible, and long-term. They can also be used to insert PICC which are very long, thin catheters specifically designed for peripheral insertion (typically saphenous vein). PICCs are usually made from silicone, can be cut to length, can be single or double lumen, and can stay in place for extended periods with proper maintenance. Peel-Away sheaths are a specialized catheter that is placed into the vein and advanced like an over-the-needle catheter (Figure 7.9). The final catheter is inserted through the lumen of the sheath until it is at the desired depth. Tabs on either side of the sheath are pulled, causing the sheath

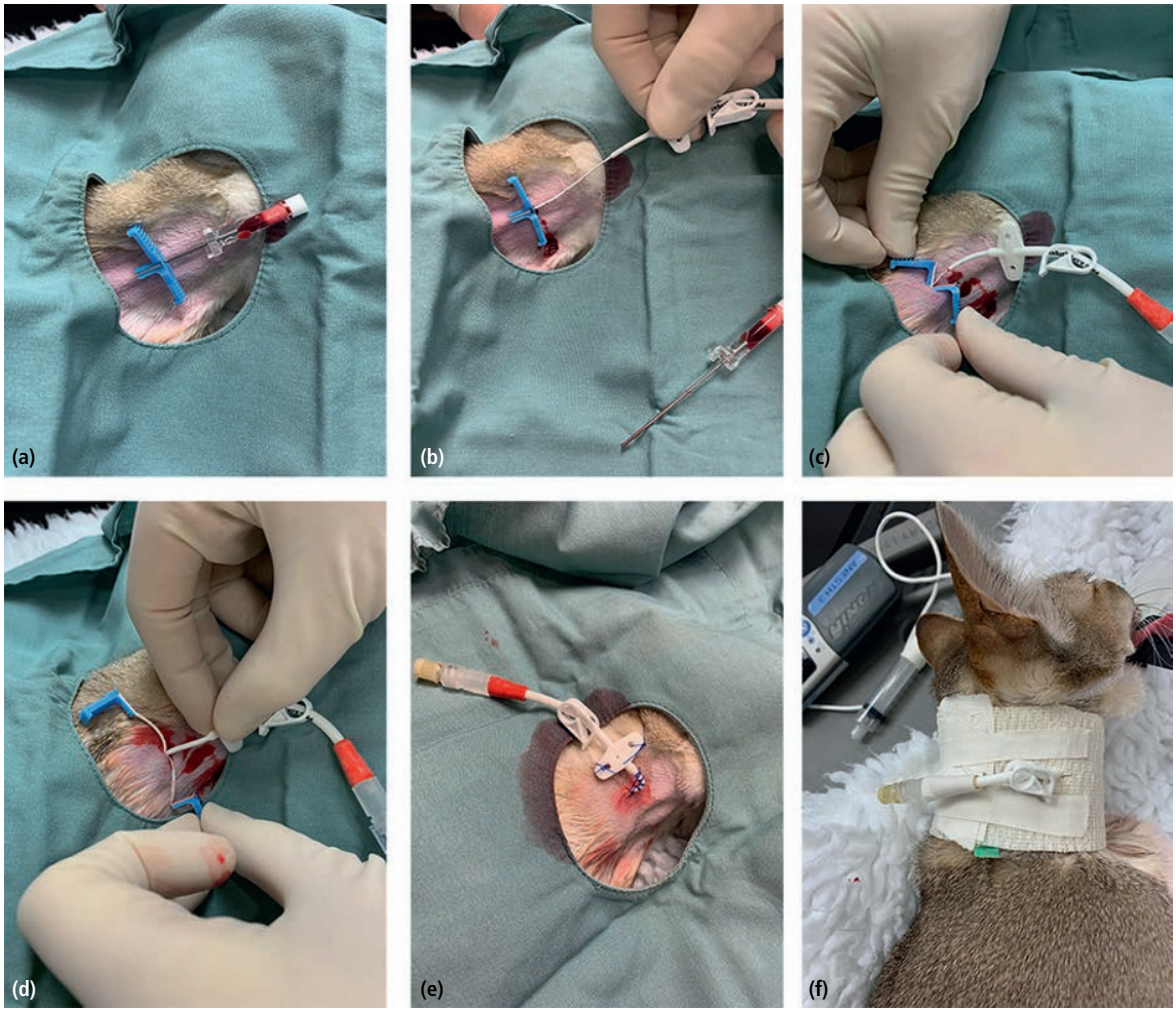


Figure 7.9 The Peel-Away® (Cook Medical, Bloomington, IN) sheath introducer can be used for single or multilumen catheters. (a) Insertion of sheath introducer. (b) Single lumen catheter inserted through the sheath. (c) Activating the peel-away. (d) Removing the peel-away. (e) Affixed catheter with suture. (f) Final bandaged catheter. *Source:* Courtesy of Carolyn Sidenberg.

to split down the middle and allow it to be removed by continued pulling of the tabs. This results in only the final catheter remaining in the vein. This method offers advantages of a high-quality catheter with a relatively simple placement. Disadvantages are the large sheath size (larger than the final catheter), which can be challenging to seat in the vessel.

Through-the-needle long catheters are "all in one" systems that are passed through a needle and are typically longer than over-the-needle catheters (8–12 inch, 20–30 cm) and available in various diameters. These are usually found in the 22–18 G range. Multiple varieties are commercially available (e.g. VeinCath® or Drum Long Line Catheter, MILA International, Erlanger, KY; see Figure 7.10). A plastic sleeve or case around the catheter prevents its contamination during placement. While previous versions of through-the-needle catheters did not allow the needle to be removed, requiring a needle guard to cover the needle, modern versions allow the needle to be disconnected from the system. Through-the-needle catheters can be placed in the jugular, or in a peripheral vessel (canine lateral saphenous or feline medial saphenous) when the jugular vein is not a feasible option, such as in patients with coagulopathy or severe bite wounds to the cervical region. Advantages to the through-the-needle catheter include low cost, and speed and ease of placement. Disadvantages include the potential of shearing of the catheter on the sharp needle edges, and excessive bleeding (the hole made by the needle is slightly larger than the catheter that remains). These catheters are usually only single lumen and require some creativity to keep them from kinking.

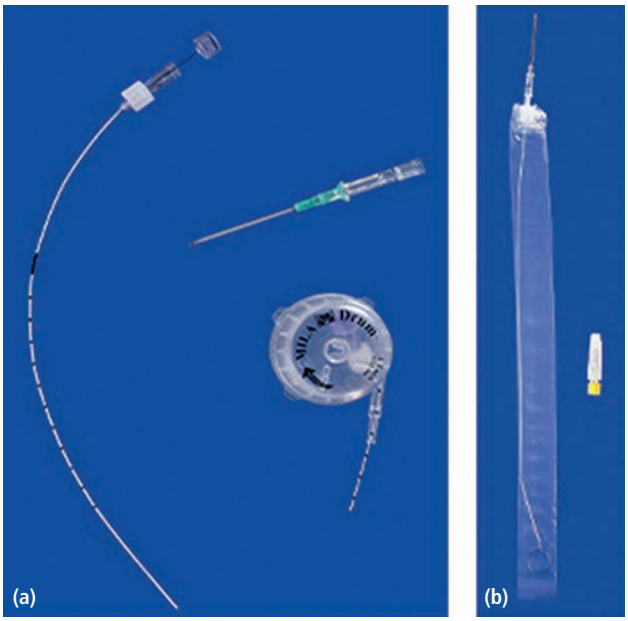


Figure 7.10 Two through-the-needle catheter examples. (a) Drum long line catheter (MILA International, Erlanger, KY). (b) Long-line catheter (MILA International, Erlanger, KY). *Source:* Reproduced with permission from MILA International.

Central Venous Catheter Placement Techniques

Guidewire Central Venous Catheter Technique

Guidewire catheters use the modified Seldinger technique and can be placed percutaneously or via a mini or surgical cutdown (Figure 7.6 and 7.7; Protocol 7.4). Commercially available in single, double, or triple lumen, sizes range from 22- to 16-gauge catheters for peripheral use, and from

Protocol 7.4 Modified Seldinger Technique–Placing a Vascular Catheter over a Guidewire

Items Required

- Clippers
- Surgical scrub
- Sterile gloves
- Sterile drape with small hole (if not included in kit)
- Over-the-needle catheter that fits guidewire
- #11 scalpel blade (if not included in kit)
- Guidewire catheter kit
- Sterile gauze squares (if not included in kit)
- Saline flush
- T-port(s)
- Suture material
- Needle holders
- Thumb forceps
- Bandage scissors
- Bandage material
- At least one assistant

Procedure

1) Generously clip fur and place topical anesthetic such as 4% liposomal lidocaine or lidocaine/prilocaine ointment over the vessel. Cover in a non-absorbent dressing. Allow to sit for 15–30 minutes for optimal analgesia.
2) Analgesia ± sedation should be considered, allow adequate time for optimum results. Collect necessary supplies.
3) Patient should be placed in lateral recumbency with neck extended and thoracic limbs gently pulled caudally. It is recommended that two people restrain the animal, one occluding the vessel and restraining the cranial end of the patient, the other restraining the patient's caudal end.
4) Remove dressing with topical anesthetic and wipe area. Assess clipping and expand as needed to avoid

contamination of insertion site, at least 2–2.5 inches (5 cm) around intended insertion site.

5) Perform hand hygiene, don cap and mask, have restrainer do the same.
6) Aseptically prepare the area per hospital protocol.
7) Perform hand hygiene and don sterile gloves.
8) Prepare skin with a final surgical scrub and maintain a sterile field.
9) Sterilely drape the area with hole centered over insertion site. Many catheter brands provide small drapes for this procedure, otherwise small "eye drapes" work well.
10) Premeasure the central catheter length from the intended insertion site to the intended catheter tip resting point. For jugular catheters, the ideal resting point is in the cranial vena cava at approximately the level of the third to fourth intercostal space. The tip of the jugular catheter should rest in the cranial vena cava just cranial to the right atrium if central venous pressure monitoring is anticipated. Most catheters have markings every centimeter to assist with this placement.
11) Clamp any additional lumens other than the "distal port" (marked, often brown), and remove injection cap if present, from the distal port. Optionally, before clamping additional lumens can be flushed with saline, then clamped. The distal port is not usually flushed, as it remains open and saline will run out.
12) Ask restrainer to put pressure on the jugular vein at the thoracic inlet. They may also be asked to put some tension on the skin to help stabilize the vessel.
13) Place the introducer catheter into the vein, with the tip pointing in the direction of the heart; ensure a "clean" stick with adequate blood flow, advance catheter and remove stylet. At this point, ask the restrainer to remove pressure from jugular vein.
14) Insert the flexible J end of the guidewire into the introducer catheter. Note, the J-wire must first be straightened by pulling it back into the wire housing, then inserting the pointed tip into the catheter hub. Advanced the wire, using the thumb guide and noting the markings on the wire. These markings are at 10 cm intervals, and are visible through the wire housing. Ideally, the wire should be inserted to the length of the catheter being placed (Figure 7.11a). Some brands do not have markings on the wire, and depth must be estimated by watching the end of the wire as it moves through the housing.
15) While holding the wire steady at the insertion site, remove wire housing, then the introducer catheter; ensure that the wire does not inadvertently back out or touch a nonsterile field. Have sterile gauze prepared as the insertion site often bleeds.
16) At all times when the wire is in the patient, it must be held and stabilized. If necessary, have a second assistant with gloved hands hold the wire while completing the next steps.
17) Feed the vessel dilator over the guidewire, to the level of the skin, again, taking care to avoid changing the depth of the wire. Tent the skin at the insertion site, and using a #11 scalpel blade with the cutting edge pointing "up" and the tip entering the same hole as the guidewire, make a stab incision through the skin. Continue to tent the skin and gently rotate the dilator into the vessel (Figure 7.11b). Insert the full length of the dilator into the vessel (depending on patient size), and then remove the dilator from the guide wire. Advancing the full length ensures that the dilator has expanded tissues in the interstitial space and entered the jugular. Expect bleeding from the insertion site, and use sterile gauze to apply pressure while transitioning to the next steps.
18) Insert the catheter over the wire *but do not enter the skin until the wire can be grasped from the distal port* (Figure 7.11c). The wire should be held steady as the catheter is advanced off the wire, and into the vessel. If multilumen catheters are used, the wire will exit the distal port. Once fully advanced to the premeasured depth, remove the wire and leave the catheter in place.
19) Aspirate air from the catheter with a small syringe; blood should easily flow into the syringe. Flush the catheter with sterile saline and clamp and cap port(s) with a T-port or an injection plug. Repeat procedure for each port if a multilumen catheter is used. It is important to always aspirate the catheter first, before flushing, so as not to create an air embolus, even if the catheter port(s) were preflushed.
20) If the entire length of catheter is not used, secure the excess catheter with the plastic catheter holders provided. Secure the catheter in place with sutures between the skin and the designated grooves or holes on the catheter hub and/or the plastic catheter holders (Figure 7.11d). Place a sterile dressing over the insertion site, preferably clear like Tegaderm, so the insertion site can be monitored.
21) Apply a loose neck wrap containing cast padding or stretch bandaging followed by a water-resistant bandage. Tape T-port or multilumen ports on outside of the bandage for easy access. Ensure the bandage is not tight; several fingers should easily slide under the bandage. Recheck patency of the catheter by aspirating blood once the bandage is in place.
22) Label the catheter with size, date, initials and date of bandage change.

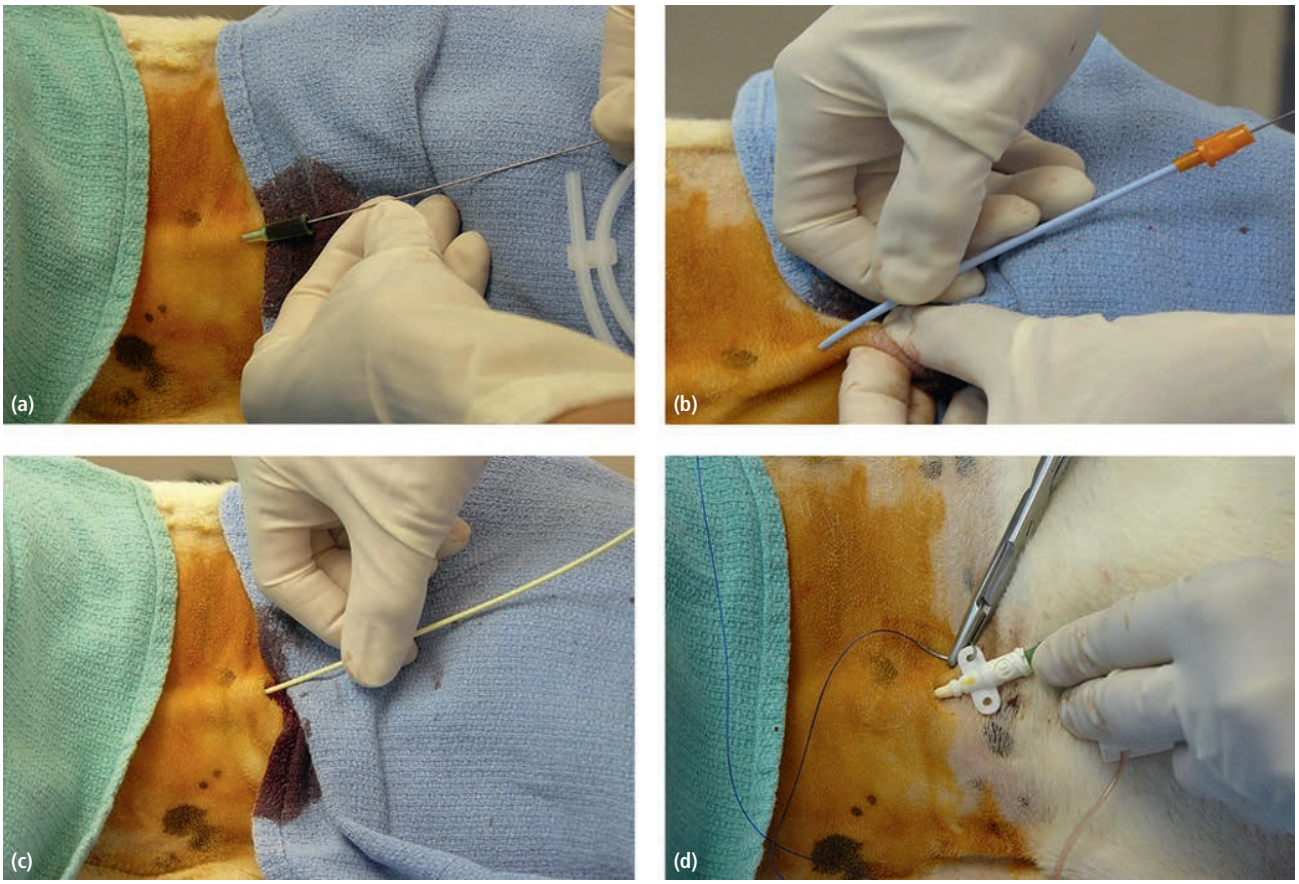


Figure 7.11 (a) After aseptically preparing the area, straighten, then insert the J-end of the guidewire into the vessel via the short over-the-needle catheter. (b) Passage of the dilator may be accomplished by "tenting" the skin and gently but firmly rotating the dilator into the vessel. (c) Insert the catheter over the wire and guide into the vessel. (d) The catheter is secured in place with skin sutures attached to the wings of the plastic applicator.

14- to 24-gauge catheters for central use; lengths range from 16 to 55 cm. Most guidewire catheters come in kits containing the percutaneous introductory short catheter, the central venous catheter, guide wire (typically encased in a protective plastic case for sterile insertion), dilator, flush syringe and anchoring device (Figure 7.11). The authors recommend using a standard over-the-needle catheter that the operator is familiar with as the introducer, rather than the introducer provided in the kit. It is important however to ensure that the introducer size chosen will allow the guidewire to pass through the catheter (not the stylet), so always test this prior to insertion. With correct placement and care these catheters can stay in place for longer periods, usually for the duration of hospitalization. (Table 7.2 summarizes the indications for the different catheter types.)

Care of central venous catheters is critical to maintain patency, patient comfort and to prevent infection and complications (see Chapter 63 for more information). The central venous catheter bandage should be monitored frequently as it may tighten with neck movement and rehydration; the patient should be monitored for airway compromise and facial edema. When the catheter is removed, apply direct pressure to the insertion site for five minutes followed by application of a neck bandage to prevent hemorrhage. The neck bandage may be removed after one to two hours.

Through-the-Needle Catheters

Placement of a through-the-needle catheter requires standard fur clipping and aseptic site preparation. Ideally, a small drape will cover the neck, or an opened sterile gauze will cover the bottom of the prepared site. With either the neck or limb extended, and tension on the skin to prevent needle drag, the needle is inserted at a 30-degree angle with the bevel side up over the targeted vessel. The insertion site should be close to, but not directly on, the targeted vessel. After the needle is passed through the skin, the vein is then punctured directly from the top. Blood will usually flash back into the catheter, which confirms that the catheter is

Table 7.2 Indications for different catheter types.

Catheter type	Indications	Relative or potential complications
Peripheral through-the-needle long catheter	Jugular vein not accessible	Catheter shearing by introducer needle
	Coagulopathy	Excessive bleeding
	Severe bite wounds or edema to the cervical region	
	Frequent blood sampling needed	
	Aggressive cat; restraint often easier for medial saphenous placement than for jugular placement	
	Cervical disease or pain (atlantoaxial luxation or cervical disk disease)	
	Increased intracranial pressure; desire to avoid increasing intracranial pressure through occlusion of jugular vein	
Central venous catheter	Central venous pressure monitoring indicated	Thromboembolic disease
	Infusion of total parenteral nutrition or other hyperosmotic fluid	Coagulopathic disease
	Prolonged hospitalization expected	Respiratory disease (increased placement time)
	Frequent blood sampling required	Immune-mediated hemolytic anemia (due to thromboembolic disease potential)
	Multiple ports required for simultaneous delivery of various fluids and medications	Increased intracranial pressure (actual or suspected)
		Cervical disease or pain (atlantoaxial luxation or cervical disk disease)
Vascular access port	Chemotherapy	Sepsis
	Delivery of sedation for repeated radiation therapy treatments	Thrombosis
	Long-term delivery of intravenous medications or fluids	
	Repeated blood sampling	
	Total parenteral nutrition	
Intraosseous catheterization	Severe hypotension	Pneumatic bones
	Severe dehydration	Metabolic or infectious bone disease
	Extremely small body size (neonates, exotic animals, pocket pets)	Skin infection around proposed catheter insertion site
	Inaccessible venous access (e.g. edema, thrombosis, severe burns)	

situated within the vessel; the catheter is then advanced into the vein. Depending on type of catheter used, the catheter is advanced by pushing it through the plastic sleeve into the vein, or the drum is rotated to feed the catheter into the vein. Resistance to advancement should be minimal. Once the catheter has been fed entirely into the vessel, the stylet, if present is pulled, the needle is removed and the clamshell adapter is attached. The catheter is aspirated to ensure blood flow, any samples necessary are obtained, and the catheter is flushed with 0.9% saline. The catheter should be anchored with skin sutures, covered in a clear dressing such as Tegaderm™ (3M™, St. Paul, MN), and protected with a soft bandage.

Ultrasound-Guided Venous Access

The availability of ultrasound in veterinary medicine is increasing and has gained popularity not only for diagnostics at the bedside, but also for procedures. Ultrasound-guided venous access is a very useful skill, as it can assist in successfully catheterizing difficult veins. Many will use ultrasound when traditional attempts have not been successful, however it is becoming more and more common to use ultrasound for all attempts, particularly for central veins. In human medicine, ultrasound-guided access has become the norm for central venous access. This modality is comprehensively discussed in Chapter 9.

Alternate Vascular Access Options

Intraosseous Catheterization

Intraosseous (IO) catheterization is a procedure in which a needle is placed into the medullary cavity of a bone, typically when the need for vascular access is urgent and venous catheterization cannot be performed quickly (Protocol 7.5). The IO space is easily accessible even in the most dehydrated or hypovolemic animal.

IO catheters are an alternative to IV access in critically ill patients. Once placed, they may be used to deliver almost any drug, fluid, colloid, or blood product to the patient. IO infusions allow for infusion into the medullary cavity, a highly vascular space, and uptake into the systemic circulation is similar to direct IV injection. Advantages of intraosseous catheter placement include speed and ease of placement, minimal complication rates, ease of fluid administration and minimal cost of supplies.

The IO route is most commonly used in cardiopulmonary arrest, cardiovascular collapse (shock), and may also be used in neonates in place of IV access. In many cases, placing an IO catheter is much faster than attempting an IV catheter. The Reassessment Campaign on Veterinary Resuscitation (RECOVER) Initiative has recognized IO to be superior to intratracheal administration of drugs during CPR.

Protocol 7.5 Intraosseous Catheterization

See also Figure 7.12.

Items Required

- Clippers
- Surgical scrub
- 2% lidocaine in a 1-ml syringe with a 23–25 G needle
- Suture material
- Saline flush
- T-port
- Hypodermic needle appropriate for patient size (e.g. 22 G, ¾ or ½ inch long) for small patients or neonates, or spinal needles
- EZ-IO® drill and catheters (MILA International, Erlanger, KY) or bone marrow needles for adult patients whose bones have already ossified.

Note that spinal needles, containing an outer needle and an inner stylet, work very well, as the shafts of hypodermic needles used on the neonate can become clogged during placement.

Procedure

1) Select best placement site, taking into consideration patient comfort, ease of placement and anticipated catheter dwell time.
2) Collect necessary supplies.
3) Closely clip a generous area (2–2.5 inches, c. 5 cm) around the intended insertion site and remove any loose fur.
4) Perform hand hygiene.
5) Aseptically prepare the area as per hospital protocol.
6) Perform hand hygiene; if the patient is immunocompromised, it is recommended to wear sterile gloves.
7) Restrain patient; avoid excessive stress to patient.
8) Administer a small amount of lidocaine to the level of the periosteum.
9) Prepare skin with a final surgical scrub and maintain a sterile field.

For Needle Intraosseous Catheter Placement

1) Place a finger on the long axis of the bone in which the catheter will be placed, to determine its axis and location.
2) If using the femoral trochanteric fossa, adduct the limb slightly (toward ventral midline) and rotate trochanteric fossa laterally to avoid the sciatic nerve.
3) Insert the catheter distally lengthwise into the medulla of the femur or humerus cortex parallel to the finger held alongside the bone, aiming for the trochanteric fossa if the femoral trochanteric fossa is the insertion site. Simultaneously push and twist the needle in a single line (i.e. avoid movement in the third dimension). Expect initial resistance as the needle passes through the cortex. If resistance is felt as the needle passes through the cortex into the medulla, the bevel of the needle may be seated incorrectly, causing blockage. Gently turn the needle 90–180 degrees to dislodge and attempt placement again.
4) Once the needle is in place, push the hub of the needle back and forth, simultaneously moving the limb; the hub of the needle should be seated securely in the shaft of the bone and move along with the bone when the limb is moved.
5) Aspirate needle or catheter; aspiration of bone marrow confirms placement. A radiograph can also confirm the correct anatomical location.
6) Flush gently with a small amount of saline; little resistance should be felt. Attach a T-port.
7) Anchor the catheter either by placing a stay suture through the skin near the catheter hub and attaching that suture to the tubing of the T-port, or by stapling a

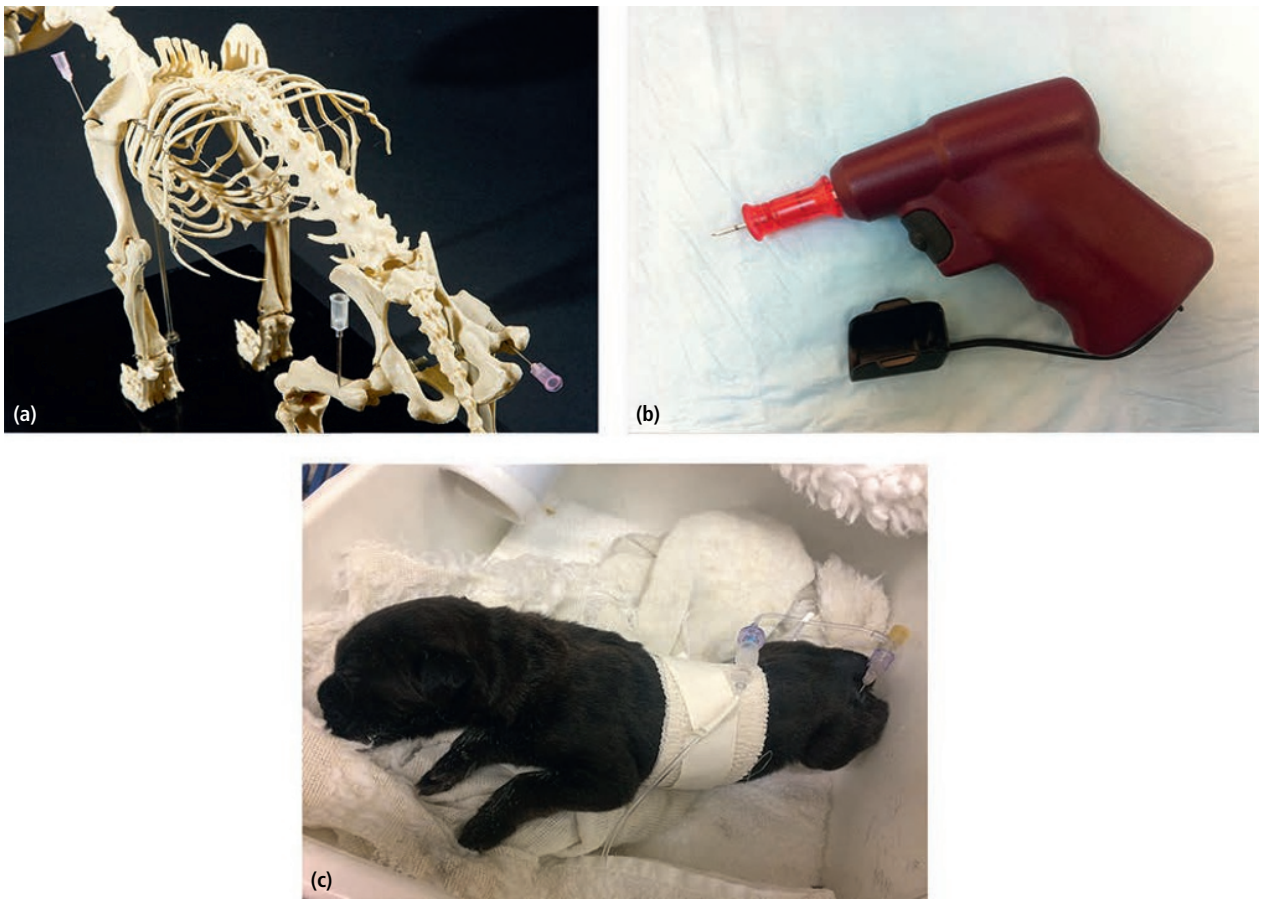


Figure 7.12 (a) Potential locations for intraosseous catheter placement (as indicated by needles) include the trochanteric fossa of the femur, the wing of the ileum, the proximal humerus, and the tibial tuberosity. (b) An intraosseous drill and catheter used for rapid intraosseous access in adult animals (EZ-IO®, MILA International, Ehrlinger, KY). (c) A hypodermic or spinal needle appropriate for patient size or a bone marrow needle can be used as an intraosseous catheter. Ideally, the catheter should be inserted with gloved hands through an aseptically prepared site. *Source:* Courtesy of Kathryn Powell.

small butterfly tape anchor from the catheter hub to the skin.
8) Check for displacement of the catheter at least twice daily.

For Drill Intraosseous Catheter Placement

1) The most common sites are the lateral proximal humerus and the medial proximal tibia.
2) Perform a small stab incision with a #11 blade. Attach catheter to drill and push the needle through the stab incision. Have an assistant hold skin taught.
3) Push catheter against bone (perpendicular to the long axis) and activate drill. Maintain pressure until catheter is at desired depth (it is easy to go too far and into the cortex on the other side of the bone).
4) Disconnect drill and remove stylet.
5) Aspirate and check for flash of blood to verify placement, then rapidly and forcefully infuse 5–10 ml of sterile saline. This will expand the cavity and allow for better fluid flow.
6) Attach fluid line and begin fluid resuscitation.
7) EZ-IO catheter should be removed as soon as vessels are obtainable, within 24 hours. In the author's experience, vessels can usually be obtained within an hour of beginning fluid therapy.

For All IO Catheters:

1) Check surrounding tissue for fluid leakage
2) If leakage is evident, the catheter may need to be removed and another bone tried.

Dosages between IV and IO are identical, and blood samples can often be obtained by aspirating marrow from the medullary cavity.

Clinical indications for IO catheterization include severe vascular collapse (e.g. hypovolemic shock), severe vascular trauma, peripheral edema, thrombosis, small patient size (e.g. neonates, exotics), or even obesity. Mastering IO catheterization, which is simple to perform, can prevent patient death when timely vascular attempts are not feasible or practical. Locations for intraosseous catheter placement include the trochanteric fossa of the femur, the wing of the ileum, the proximal humerus and the tibial tuberosity (Figure 7.12a). Potential complications are minimal; fracture of the bone at the catheter site, infection, osteomyelitis, edema and patient discomfort associated with rapid fluid infusion are the reported complications to intraosseous catheterization [2]. The most common complication is dislodgement of the catheter due to animal movement. When this technique is applied to other species, care must be taken never to introduce fluids into pneumatic bones. Medications targeted for IV administration can be used via the IO catheter or needle. Rate of maximal fluid administration is related to the diameter of the catheter or needle.

Care of Intravascular Devices

Catheter care is important to avoid complications and to ensure patency for the desired duration of any indwelling intravascular device, as it predisposes a patient to device associated infection. All personnel involved with the placement or handling of the catheter should use aseptic technique when placing, changing bandages, administering fluids or medications, withdrawing blood, or while using monitoring devices attached to the catheter. Infection may occur as a result of contaminated IV solutions, contaminated injection ports, catheter caps or T-ports, poor skin preparation or insertion techniques or failure to routinely change the catheter bandage. Monitoring equipment should be cleaned and disinfected between patients. Multiple ports and lines should be labeled appropriately and handled with aseptic technique. Ports should be swabbed with alcohol and allowed to dry prior to introduction of a needle. Insertion sites should be routinely monitored for thrombosis, which causes a "ropey" feel to the vessel; for patient discomfort, redness, heat, or swelling around the insertion site; for catheter migration either into or out of the skin in comparison with its depth at initial insertion; and for subcutaneous extravasation, which can lead to discomfort upon injection and the accumulation of fluids or medications under the skin.

Catheter bandages should be removed fully to allow inspection of the catheter site at least once daily. Adhesive tapes must be examined for tightness, dryness, or blood staining; bandages contaminated with body fluids should be replaced immediately. Another benefit of the sterile adhesive bandage at the insertion site of the catheter is that the final piece of tape need only be removed so the sterile bandage can be lifted to examine the insertion site, before replacing the tape and final bandage.

While sterile technique should be maintained for any vascular device intervention, nursing management of catheters used for PN warrants special focus, since nutritional formulas are excellent media for bacterial colonization. Proper catheter care of a PN catheter includes strict aseptic technique during insertion, including proper skin preparation, placement of a sterile underwrap over the insertion site, and strict aseptic technique when handling PN administration tubing and bags. The IV lines should not be disconnected; if diagnostic testing or frequent walking is necessary, PN administration lines and bags should accompany the patient after clamping lines to avoid accidental rapid infusion. If a multilumen catheter is placed, proper identification of each port is necessary, with one line dedicated to the PN solution only.

Catheter Flushing

Catheters should be flushed with a small volume of sterile saline (0.5–2 ml, depending upon catheter length and size of patient) at least every 8–12 hours. Several studies have indicated that 0.9% saline is as effective as heparinized saline in maintaining catheter (peripheral and central) patency, although most have been conducted in the healthy patient population [3]. The evidence seems clear that routine use of heparinized saline is no longer necessary, and may be reserved for only certain patients, and at the clinician's discretion. The authors recommend flushing any unused central venous catheters or ports every four hours with 1.5–3 ml of flush using sterile technique. See Chapter 63 for more detailed information.

Peripheral Intravenous Catheters

A peripheral IV catheter can remain in place for as long as it is functioning as expected, is clean and dry, there is no redness at the insertion site, and there is no pain on injection or flushing. The exception to this is catheters placed emergently, these catheters should be replaced as soon as possible, as it is likely that steps were missed such as hand hygiene, and proper skin asepsis. Any cutdown incision made during placement can increase the potential for infection; these catheters should be replaced or removed as soon as possible. Any catheter should be removed when no

longer needed [4]. Intraosseous catheters are similar to IV catheters, and it has been suggested that they can stay in place for over three days in veterinary patients if no signs of infection are found [3].

Central Intravenous Catheters

Central IV catheters may dwell for the length of patient hospitalization as long as there is no suspicion of catheter-related infection [4], however, they require daily insertion-site inspection for signs of thrombosis or leakage and daily site maintenance. Evaluation of the patient for nasal or oral discharge, facial swelling, difficulty swallowing, and impaired breathing can be indicators of jugular vein thrombosis [5]. Central venous catheter care also requires daily inspection as indicated above for peripheral catheters. As central catheters are significantly longer than peripheral catheters, signs of extravasation may not be as apparent. The midsternal region should be examined for edema related to any jugular venous catheter and the proximal aspect of the pelvic limb should be examined for edema related to lateral or medial saphenous venous catheter placement. Patency can be evaluated by ensuring blood can be aspirated. Note that neck bandages should follow the "two finger rule," wherein the bandage is loose enough to slide two fingers underneath to help ensure patient comfort. If it is necessary to change a central catheter and catheter-related infection is not suspected, replacement can sometimes be done by passing a sterile guide wire through the existing catheter, removing the catheter and aseptically placing a new one over the wire [4].

Complications

Bacterial contamination of catheter sites can result from skin contamination at insertion, contamination from patient interference, soiled bandages, improper handling, contaminated injectates, blood left inside an injection port or T-port, and from the tops of multiuse IV medication bottles. Personnel should practice proper hygiene protocols, including hand hygiene, swabbing ports and medication bottles with antiseptics as well as frequent bandage changes for any catheter in any anatomical location. The authors recommend the use of commercially available sterile syringes of flush. Any patient with an unexplained fever, pain upon injection, or inflammation at a catheter insertion site should be investigated for a catheter-related infection. Suspicious catheters should be removed immediately See Chapter 62 for in-depth discussion.

Phlebitis is another commonly encountered complication of indwelling vascular catheterization. Phlebitis can occur due to inflammation associated with movement of the catheter in the vessel, irritation of the vessel from the catheter or fluids/medications administered, or due to bacterial infection, which can lead to sepsis.

Air embolism can occur if lines become disconnected or a significant volume of air is present within an administration set. Central venous catheters have the highest risk of incidence as air can be suctioned in due to negative pressure within the thorax [4]. Central venous catheters and large-bore catheters pose the risk of exsanguination if patient interference or disconnection goes undetected. Catheter embolism can occur if a fragment of the catheter becomes free within the vessel either due to placement error or patient interference. Radiopaque catheters can allow radiographic investigation to locate any such fragments and aid in planning for surgical retrieval.

Thrombus formation can occur with any venous or arterial cannulation, particularly if there is endothelial damage within the vessel. Smaller veins with slower blood flow, the use of rigid catheters, insertion over a joint, or pre-existing conditions such as immune-mediated hemolytic anemia, glomerulonephritis, or vasculitis may increase the risk of thrombus formation. Severe thrombus formation could lead to pulmonary thromboembolism.

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*Deceased.

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8

Arterial Puncture and Catheterization

Elisa M. Mazzaferro and Nicole van Sant

Arterial puncture and catheterization aree among the most important techniques required for monitoring of the critically ill small animal during hospitalization and while under general anesthesia. Arterial puncture is most commonly performed to obtain arterial blood samples for blood gas analysis. If repeated arterial blood sampling is required, or if the patient requires continuous direct blood pressure monitoring, the placement of an arterial catheter is necessary.

Arterial Puncture

Arterial puncture is most commonly performed on the dorsal pedal artery (or one of its metatarsal branches) or the femoral artery (Protocol 8.1). To perform an arterial puncture, position the patient in lateral recumbency on the side of the targeted artery. Clip and clean (e.g. with isopropyl alcohol) the area over the artery. In most cases, a full surgical scrub is unnecessary unless an indwelling catheter is going to be introduced into the artery, or if the animal is immunocompromised and at risk of infection.

Procedure

The supplies required to perform arterial puncture for blood sample collection are heparin and a 3-ml syringe with a 22- or 25-G needle attached – or a lithium heparin arterial blood gas (ABG) syringe with its needle attached – as well as pressure bandaging supplies appropriate for the sampling site. Prefabricated syringes that contain a pellet of lithium heparin can be purchased from a variety of manufacturers (e.g. Smith Medical; Vital Signs, Inc.; Becton Dickinson; Cardinal Health). A simpler and less expensive technique, however, is to use equipment that is already stocked in the emergency room or intensive care unit.

If using a standard 3-ml syringe and needle, pull a small amount of liquid heparin into the syringe, and pull the plunger to the 3-ml mark to coat the entire inner surface of the syringe with the heparin. Expel all the heparin and air from the syringe. Pull air back into the syringe to the 3-ml mark and forcibly expel all the heparin and air from the syringe again; repeat this forced air expulsion procedure three times [1]. This syringe evacuation procedure minimizes sample dilution with liquid heparin, which would cause significant preanalytical error in blood gas and electrolyte values. Even using this technique, ionized calcium concentration should not be measured on heparinized samples [1].

Following aseptic preparation of the proposed needle insertion site, palpate for the dorsal pedal pulse over the second and third metatarsal bones, or for the femoral pulse over the cranial medial aspect of the proximal femoral diaphysis. Insert the needle at a 15–20 degree angle with respect to the skin over the point where the pulse is most easily palpable. It is easiest to feel the pulse with the nondominant hand. Advance the needle very slowly in 1–2 mm increments; after each movement, look at the syringe hub carefully for a flash of blood. If the needle has been advanced to what seems a sufficient depth and a flash of blood has not been encountered, the needle should be slowly withdrawn in the plane of entry. As the needle is withdrawn, watch closely for a blood flashback. In some cases, the needle has punctured through the deep portion of the vessel wall and blood will enter the needle and syringe during withdrawal. If the needle must be redirected, pull it to the superficial subcutaneous tissues before redirecting.

Once the needle tip is seated in the artery, gently pull the plunger of the syringe to withdraw blood or allow arterial pressure to fill the syringe. The latter technique confirms that the blood is arterial rather than venous. Gently

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Protocol 8.1 Arterial Puncture for Blood Sample Collection

Items Required

- Clean clippers and blade
- Skin-cleaning supplies: isopropyl alcohol-soaked cotton, or full surgical scrub as appropriate
- Prefabricated lithium heparin ABG syringe or 3-ml syringe, liquid heparin, and a 25- or 22-gauge needle
- Pressure bandage for post-puncture wrap
- An assistant (usually only one is required)

Procedures

If using a prefabricated ABG syringe, begin at step 6.

1) Collect necessary supplies.
2) Equip the 3-ml syringe with the needle.
3) Pull a small amount of the heparin into the syringe, then pull the plunger back to coat the entire inner surface of the syringe.
4) Expel all the heparin and air from the syringe.
5) Pull air into the syringe to the 3-ml mark and forcibly expel contents from the syringe; repeat three times. The syringe and needle hub should appear evacuated of liquid heparin.
6) Closely clip a generous area (2–2.5 inch, c. 5 cm) around the intended insertion site and remove any loose fur.
7) Aseptically prepare the area. Alternate cleansing scrub with isopropyl alcohol; prepare in circular motions, starting from the center and finishing at the periphery with each swab.
8) Perform hand hygiene and don examination gloves.
9) Restrain patient; avoid excessive stress to patient.
10) Palpate for the pulse over the second and third metatarsal bones for the dorsal pedal artery, or over the cranial aspect of the proximal femoral diaphysis for the femoral artery.
11) Insert needle at 15–20 degree angle with respect to the skin where the pulse is most easily palpable.
12) Advance needle very slowly, checking frequently for a "flash" of blood. If the needle has been advanced a sufficient depth and no flash has been encountered, slowly withdraw the needle in the plane of entry, watching closely for a flash.
13) Once the needle tip enters the artery, gently pull the plunger or allow arterial pressure to fill the syringe.
14) Gently withdraw the needle from the skin and apply pressure with a bandage for many minutes to an hour.

withdraw the needle from the skin when an adequate sample has been withdrawn. Apply pressure to the puncture site with a bandage for many minutes to an hour. When a pressure bandage is applied with tape or vet wrap and left on the patient for a period of time, it may be useful to write "remove pressure bandage" on the patient's order sheet so the bandage is not left in place for a prolonged time (Protocol 8.1.)

Following collection, the sample syringe should be capped, and the sample analyzed immediately to ensure accurate results. Samples can be stored for up to one hour if immersed in an ice bath to prevent ongoing cellular metabolic activity within the sample that can result in a change in PaO_2, $PaCO_2$, and pH.

Sublingual Venipuncture

Sublingual veins are also used for sampling in anesthetized patients because oxygenation in this vascular bed closely approximates arterial blood. Thus, samples from this site can be used for ABG analysis. These superficial, soft-walled vessels are usually punctured with a 25 G needle. Firm digital pressure on the vessel must be performed for a minimum of five minutes to prevent hematoma formation after puncture. The sublingual veins are not usually used for catheterization in clinical patients.

Arterial Catheter Placement

Although the placement of an arterial catheter can be more technically difficult than placement of a peripheral venous catheter, the equipment necessary and the procedures performed are largely the same. The materials and supplies required to attach an arterial catheter to a transducer for continuous blood pressure monitoring are discussed in Chapter 12. Special considerations for arterial catheter placement follow.

Site Selection

A variety of arteries can be used for arterial catheter placement, including the dorsal metatarsal artery (commonly called the dorsal pedal artery) and the radial, coccygeal, femoral, or auricular arteries. When selecting a site for placement, it is important to consider the patients' mobility and activity level, and whether the patient has access to

the site and could potentially remove the catheter. As mentioned below, one should also consider risk of contamination and practicality of keeping the area clean when selecting an arterial catheterization site. More peripheral arteries may be preferable in patients with hemostatic concerns. Finally, the operator's experience and expertise with different anatomic sites may impact catheter site selection.

Aseptic Technique

The most important aspect of minimizing the risk of catheter-related infection is strict adherence to aseptic technique when placing an arterial catheter [2]. An important consideration when placing an arterial catheter is thus whether the location has a high risk of contamination. For example, although the dorsal pedal arteries are perhaps the simplest to catheterize, consider whether the patient has diarrhea that could potentially contaminate the arterial catheter site. It is generally recommended that no catheter be placed into an area of damaged skin, abrasion, or pyoderma [3].

In addition to site selection, adherence to cutaneous antisepsis protocols is strongly recommended [4]. Many arterial catheter-related infections are acquired extraluminally, from the invasion of skin microorganisms at the insertion site. Incorporating preventive measures such as skin preparation with chlorhexidine or chlorhexidine impregnated dressings at greater than 0.5% strength has the potential to significantly reduce catheter-related infection rates. Additionally, the use of sterile gauze or a semipermeable transparent dressing to cover the insertion site is preferred over the use of topical antibiotic ointment or creams, as topical preparations may promote fungal infections and antibacterial resistance.

One of the most common causes of hospital-acquired infection is the transmission of disease-causing bacteria on the hands of hospital personnel. Therefore a very basic, and extremely important, tenet of infection control and antiseptic technique is for the operator to thoroughly wash their hands before placing an arterial catheter. The Centers for Disease Control and Prevention has published handwashing guidelines that instruct on proper hand hygiene techniques for healthcare workers [5] (see also Chapter 62 for more detailed information). For arterial catheter placement, after carefully scrubbing the hands the operator should don examination gloves, unless the patient is immunocompromised, in which case sterile gloves should be worn.

Once an acceptable artery has been identified, carefully clip all fur over the artery and circumferentially around the patient's extremity, leaving at least 2 inches (5 cm) between the fur margin and the proposed insertion. Next, perform a surgical preparatory scrub on the area over and around the catheter insertion site.

Analgesia

Placement of an arterial catheter can be uncomfortable, so some patients benefit from sedation or a local anesthetic during placement.

Percutaneous Facilitation

The placement of the arterial catheter through a small hole in the skin helps prevent the tip of the catheter from becoming burred. If a burr develops, catheter insertion will be difficult and there is increased risk of thrombus formation [6].

The concept of percutaneous facilitation involves making a small nick in the skin surface over a proposed site of catheter placement in animals that are extremely dehydrated or have very thick or tough skin. Percutaneous facilitation is most commonly performed with the bevel of an 18- or 20 G hypodermic needle. Locate the artery using gentle, digital palpation as to not occlude the vessel and obscure the pulse. Identify the catheter insertion site. Tent the skin over the catheter insertion site and make a nick through the skin with the needle's sharp bevel, taking care to avoid underlying vessels. If the bevel of the needle nicks the underlying artery, the artery will spasm and the pulse will wane or disappear. When an artery undergoes spasm, it is very difficult to cannulate until a palpable pulse returns.

In patients that are very small, obese, edematous, or hemodynamically unstable, digital palpation may be insufficient at locating arteries in peripheral sites [7]. Ultrasound-guided vascular access may be warranted to identify and evaluate targeted vessels, to provide guidance to improve arterial catheter placement success, and to minimize complications associated with arterial catheter placement [8].

Ultrasound-guiding can be employed using two techniques to image the artery and the surrounding structures. Positioning the ultrasound probe over the artery in longitudinal orientation images the artery as a tubular structure (Figure 8.1a,b) and can offer guidance for establishing the needle path ensuring successful arterial cannulation (Figure 8.1c) Positioning the probe over the artery in transverse orientation or short axis images the artery in cross-section as a circular structure and the needle appears as a dot on the ultrasound screen. Both techniques provide a comprehensive image of the targeted vessel, and when incorporated into placement protocols have been shown to improve overall success rates and reduce complications associated with arterial catheterization.

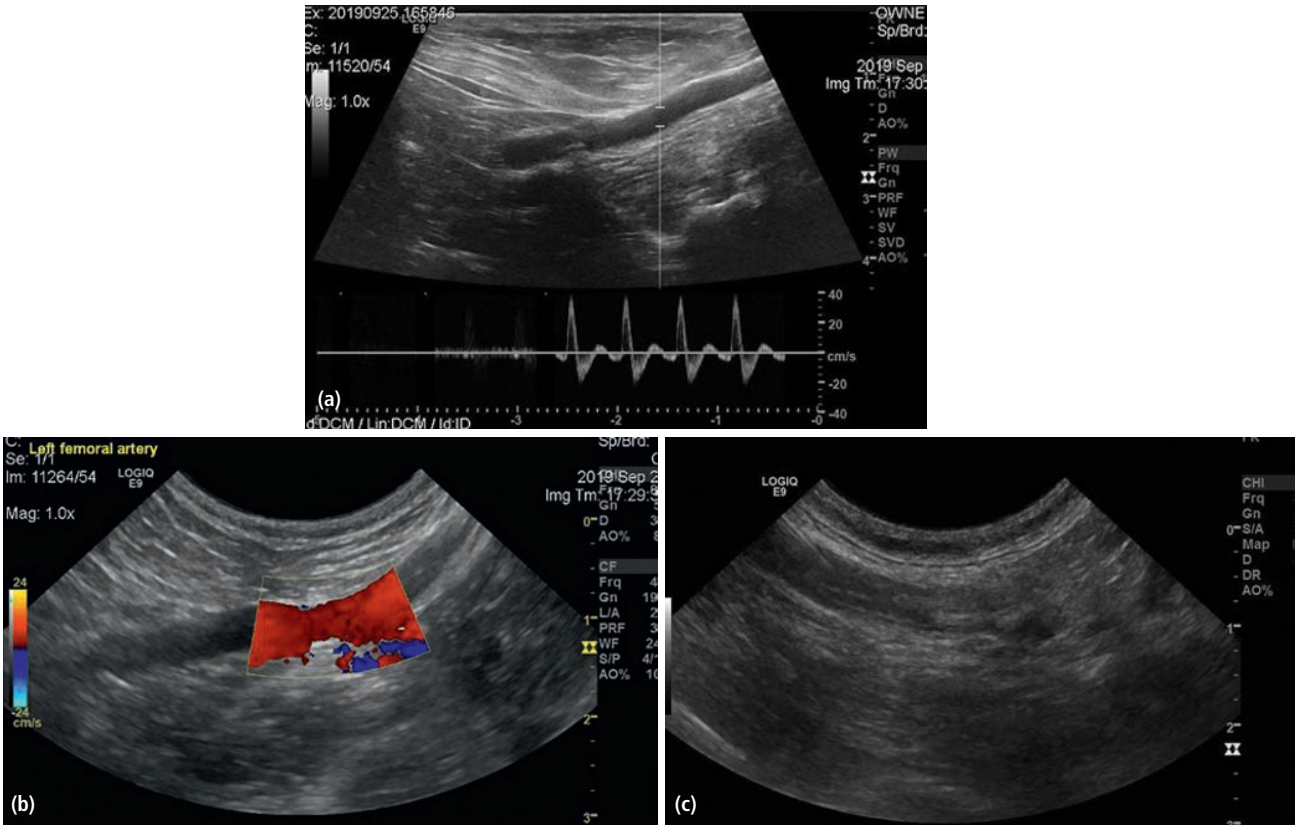


Figure 8.1 Longitudinal of femoral artery (a), with color Doppler confirming arterial blood flow (b). Hyperechoic structure at left of figure inserted into vessel lumen confirms needle insertion into artery (c).

Securing the Arterial Catheter

If an arterial catheter accidentally becomes disconnected, excessive blood loss can quickly occur, which may increase morbidity in an already critical patient. As such, it is recommended that the infusion plug and T-port have Luer lock connections (e.g. BD Luer-Lok™, Becton, Dickinson and Company, Franklin Lakes, NJ). Any attached fluid-filled monitoring system should likewise have Luer lock connections. When securing the arterial catheter itself, some clinicians prefer to use surgical glue to adhere the catheter hub to the patient's skin. However, glue can be difficult and painful to remove. If the catheter hub and patient's skin are dry and free of debris, the catheter can usually be secured adequately with standard white medical tape, as described below.

Dorsal Pedal Artery Catheterization

The dorsal pedal artery and its metatarsal arterial branches are located over the metatarsal bones. The most prominently palpable is usually found on the dorsomedial aspect of the foot between the second and third metatarsal bones, just distal to the tarsus (the hock).

To insert a dorsal pedal artery catheter (Protocol 8.2), place the patient in lateral recumbency on the side in which the catheter is to be placed (i.e. if the proposed insertion is the right dorsal pedal artery, the patient should be positioned in right lateral recumbency). The limb should be extended comfortably for the patient, and the distal limb gently rotated such that the dorsal pedal artery is palpable and on the nondependent, medial surface of the limb. Some people tape the digits to the table or a heavy sandbag to keep the limb in place during catheter placement. This positioning will help the operator to introduce the catheter into the artery. Palpate the paw between the second and third metatarsal bones distal to the tarsus to locate the pulse. Ultrasound-guided assistance can also facilitate locating the artery. Using 70% isopropyl alcohol, prepare the skin and apply the ultrasound probe to the skin over the anatomical landmarks. Hold the probe in transverse orientation to image the artery and evaluate for pulsatile blood flow.

Once the artery has been located, clip the fur at least 2 inches (5 cm) in all directions from the proposed catheter insertion site, as patient size allows. You may choose to clip fur circumferentially from the metatarsal region to maximize tape adhesion when securing the catheter.

Protocol 8.2 Dorsal Pedal (Dorsal Metatarsal) Arterial Catheterization

Items Required

- Clean clippers and blade
- Sandbag for limb positioning, if desired
- Surgical scrub preparation supplies
- Examination or sterile gloves for operator
- Sterile gauze squares
- Surgical tape (½-inch and 1-inch widths)
- Cotton roll gauze
- Water-resistant bandaging material, if desired
- 22–20 gauge needle
- Over-the-needle or over-the-wire intravascular catheter
- Luer lock T-port or Luer lock infusion plug
- Preservative-free heparinized saline flush syringes
- "Not for IV infusion" label or indelible marker
- One or two assistant(s)

Procedure

1) Collect necessary supplies, prepare tape, and prepare and flush the T-port or male adapter with preservative-free heparinized saline.
2) Place patient in lateral recumbency, with limb of proposed catheter insertion adjacent to the table.
3) Assistant should restrain the animal in lateral recumbency.
4) Secure patient's digits to a sandbag or the table's edge with medical tape.
5) Palpate gently for arterial pulse over second and third metatarsal bones to determine proposed insertion site.
6) Clip fur over dorsal aspect of the metatarsus at least (2 inches, 5 cm) from the proposed catheter insertion site. Wipe clipped fur away with a gauze square.
7) Aseptically prepare the proposed catheter insertion site using surgical scrub technique. Allow a minimum of appropriate contact time of the cleanser with the skin, according to manufacturer's instructions.
8) Perform hand hygiene and don examination gloves (sterile gloves if the patient is immunocompromised).
9) Gently remove residual scrub with sterile gauze moistened with sterile water or saline.
10) Place sterile gauze square over the fur on the distal limb, to avoid contamination of the catheter.
11) Use gloved index finger to palpate dorsal metatarsal pulse in the surgically scrubbed area.
12) Once pulse is found, perform percutaneous facilitation with bevel of a 20–22 gauge needle, if needed.
13) Gently insert over-the-needle catheter through the skin, directing the catheter at a 15-degree angle with respect to the skin toward the pulsing artery.
14) Advance the stylet–catheter apparatus in 1–2 mm increments into the area of the pulse. Observe catheter hub for a flash of blood.
15) Once a flash is observed in the hub, insert the stylet 1–2 mm more and push the catheter off the stylet into the artery.
16) Before removing the stylet from the catheter, place sterile gauze squares beneath catheter hub to absorb blood.
17) Remove the stylet, and quickly place the male adapter or T-port into the catheter hub. Take care to avoid removing the catheter from the skin.
18) With sterile gauze squares, wipe away excess blood, making sure that the skin under the catheter hub and around the limb is dry.
19) Secure a length of ½-inch medical adhesive tape around the catheter hub, then around the limb.
20) Place sterile gauze over the insertion site.
21) Secure a length of 1-inch medical adhesive tape under the catheter hub, around the limb, finishing with the tape over the insertion site and catheter hub.
22) Secure a third length of tape around the male adapter or T-port and then around the limb as described for step 20.
23) Bandage the catheter with cotton gauze and an outer layer.
24) Secure a "not for IV infusion" sticker over the catheter bandage or make a note with indelible marker.

Following aseptic preparation, feel again for the dorsal pedal pulse over the second and third metatarsal bones. Remember to palpate the artery gently, so as to not occlude the vessel and obscure the pulse. Once the pulse is located, a nick can be made in the skin to facilitate catheter placement (see Percutaneous Facilitation, above). Insert an over-the-needle catheter through the nick or directly through the skin, at a 15–20 degree angle with respect to the skin over the point where the pulse is most easily palpable. It is easiest to feel the pulse with the nondominant hand, then insert the catheter through the skin under the gloved fingertip (Figure 8.2a). Direct the catheter through the skin and into the artery, taking care to advance the needle and catheter very slowly in 1–2 mm increments. After each movement, one should inspect the catheter hub carefully for a flash of blood (Figure 8.2b). If the catheter has been advanced to what seems a sufficient depth and a flash of blood has not been encountered, the catheter

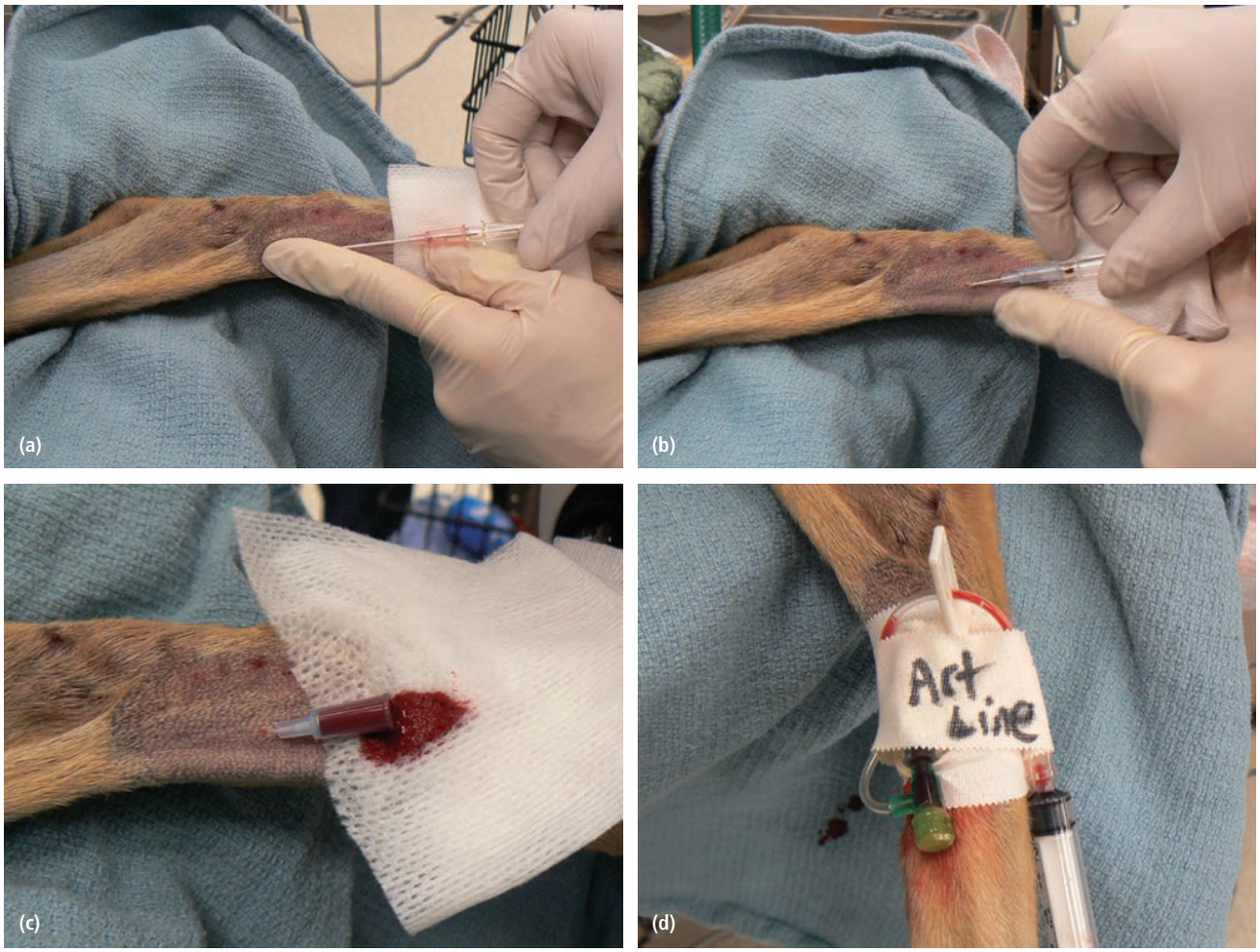


Figure 8.2 (a) Angle of catheter insertion for dorsal metatarsal artery. Note that the operator palpates for the pulse between the second and third metatarsal bones. (b) Watch for blood in the hub of the catheter. (c) Once a catheter is in place, pulsatile blood will flow from the catheter hub. (d) Securing the catheter involves wrapping pieces of tape around the catheter hub, under the catheter hub, and around the limb. The catheter should be labeled as an arterial catheter, so it is not inadvertently used for infusion.

system should be slowly withdrawn. While withdrawing the catheter system, watch closely for a blood flashback. In some cases, the catheter system has punctured through the deep portion of the vessel wall. If there is blood in the catheter hub, advance the needle and catheter another 1–2 mm in the plane of the artery, then gently push the catheter off the stylet into the vessel. If the catheter does not easily advance into the artery, gently withdraw the catheter back over the stylet, and redirect the catheter system in small increments to replace it into the artery. If the catheter does not easily withdraw back over the stylet, it should not be forced back over, as the stylet may perforate the catheter; rather, if the catheter is difficult to pull back over the stylet, the whole system should be removed from the patient's skin and evaluated. In some cases, it is necessary to leave the original catheter and stylet in place and start over, attempting catheterization again proximal to the original site of catheter insertion. If the original catheter system is removed after it has disturbed the artery but before the catheter has been advanced into the vessel, the artery can spasm or bleed and thus complicate further attempts at catheter placement.

Once the catheter is inserted completely into the artery, be careful! Once the stylet is removed, blood should pulse readily from the catheter hub, which helps confirm placement in the artery rather than a vein. Prepare ahead of time by placing several sterile 4 × 4-inch gauze squares under the catheter hub to prevent contamination of the site with blood (Figure 8.2c). Once the stylet is removed, quickly secure a Luer lock male adapter or T-port (preflushed with preservative-free heparinized saline) to the catheter hub, taking care not to inadvertently dislodge the catheter from the artery.

Carefully wipe any blood from the area, then place a length of half-inch white surgical tape around the catheter hub. Squeeze the tape securely around the hub to ensure the hub of the catheter does not spin within the tape, as spinning catheters fall out easily. Once the tape is secured to

the catheter hub, wrap it snugly around the limb. Place a second length of 1-inch tape under the catheter hub, around the limb, finishing with the tape over the insertion site and over the catheter hub. This piece of tape should be snug, but not so snug that it constricts the limb and impairs venous return. Secure a third piece of tape over the Luer lock T-port or male adapter and around the limb, such that it also encompasses the catheter hub. The distal limb and catheter can now be wrapped with gauze rolls of choice. The entire apparatus should be labeled as an arterial catheter to help avoid accidental medication infusion (Figure 8.2d).

Femoral Artery Catheterization

Percutaneous placement of a femoral arterial catheter is almost identical to placement of a dorsal pedal arterial catheter, except for anatomical location. Percutaneous facilitation is required less frequently at this site.

Place the patient in lateral recumbency, with the medial aspect of the patient's limb exposed. The person performing the restraint can pull the nondependent limb proximally, cranially, or caudally, depending on patient comfort. The limb should be clipped and aseptically scrubbed over its medial aspect from the inguinal region to the stifle. The femoral pulse is usually palpable on the cranial aspect of the femur near the inguinal region. The operator should palpate the femoral pulse with their nondominant hand, then insert an over-the-needle catheter at a 15–20 degree angle with respect to the skin, directing the stylet–catheter apparatus toward the point where the pulse is palpable. Watch carefully for a flash of blood in the catheter hub and redirect the stylet with incremental 1–2 mm changes in direction. Once a flash of blood is observed in the catheter hub, the catheter angle should be dropped so that the catheter is in a good plane with the artery before attempting to feed the catheter into the vessel. Once the catheter is seated into the femoral artery, the stylet is removed. Then quickly place a flushed, Luer lock T-port or male adapter onto the catheter hub. Tape the catheter in place as described for dorsal pedal catheters, and flush it again with preservative-free, sterile heparinized saline solution. Depending on patient size and limb conformation, the operator may choose to reinforce the catheter hub's security by suturing the hub's tape to the patient's skin (Protocol 8.3; Table 8.1).

When using ultrasound guidance to facilitate femoral arterial catheterization, the same steps apply as listed above. The patient is positioned in lateral recumbency with the medial aspect of the limb exposed. The limb is clipped and aseptically scrubbed over its medial aspect from the inguinal region to the stifle. The ultrasound machine is draped, and sterile transmission gel is applied to the

Protocol 8.3 Femoral Artery Catheterization

Items Required

- Clean clippers and blade
- Surgical scrub preparation supplies
- Examination or sterile gloves for operator
- Sterile gauze squares
- Surgical tape (½-inch and 1-inch widths)
- Cotton roll gauze
- Water-resistant bandaging material, if desired
- Suture material, needle driver, thumb forceps, and suture scissors, if desired
- Over-the-needle or over-the-wire intravascular catheter
- Luer lock T-port or Luer lock infusion plug
- Preservative-free heparinized saline flush syringes
- "Not for IV infusion" label or indelible marker
- One or two assistants

Procedure

1) Collect necessary supplies, prepare tape, and prepare and flush the T-port or male adapter with preservative-free heparinized saline.
2) Place patient in lateral recumbency, with limb of proposed catheter insertion adjacent to the table.
3) Assistant should restrain the animal in lateral recumbency. The patient should be immobile, and top limb should be well out of operator's field.
4) Palpate gently for arterial pulse on cranial aspect of proximal femoral diaphysis (near inguinal region) to determine proposed insertion site.
5) Clip fur over the femoral artery, from inguinal area to stifle, on medial aspect of dependent limb. If the patient's fur is long, clip limb circumferentially.
6) Aseptically prepare the proposed catheter insertion site using surgical scrub technique. Allow a minimum of appropriate contact time of the cleanser with the skin, according to manufacturer's instructions.
7) Perform hand hygiene and don examination gloves (sterile gloves if the patient is immunocompromised).
8) Gently remove residual scrub with sterile gauze moistened with sterile water or saline.
9) Place sterile gauze square over the fur on scrubbed area's distal margin, to avoid contamination of the catheter.
10) Use gloved index finger to palpate femoral pulse in the surgically scrubbed area.

11) Gently insert over-the-needle catheter through the skin, directing the catheter at a 20-degree angle with respect to the skin toward the pulsing artery.
12) Advance the stylet–catheter apparatus in 1–2 mm increments into the area of the pulse. Observe catheter hub for a flash of blood.
13) Once a flash is observed in the hub, drop catheter angle to align with vessel and push catheter off the stylet into artery.
14) Before removing the stylet from the catheter, place sterile gauze squares beneath catheter hub to absorb blood.
15) Remove the stylet, and quickly place the male adapter or T-port into the catheter hub. Take care to avoid removing the catheter from the skin.
16) With sterile gauze squares, wipe away excess blood, making sure that the skin under the catheter hub and around the limb is dry.
17) Secure a length of ½-inch medical adhesive tape around the catheter hub, then around the limb.
18) Secure a length of 1-inch medical adhesive tape under the catheter hub, around the limb, finishing with the tape over the catheter hub.
19) Secure a third length of tape around the male adapter or T-port and then around the limb as described for step 18.
20) Suture catheter hub in place, if desired, by suturing the hub's tape to the patient's skin.
21) Bandage the catheter with cotton gauze and an outer layer.
22) Secure a "Not for IV infusion" sticker over the catheter bandage or make a note with indelible marker.

Table 8.1 Advantages and disadvantages of various sites for arterial catheter placement.

Location	Advantages	Disadvantages
Auricular	Easy to visualize	Easily dislodged with patient motion
	Easy to catheterize	Can create thrombosis
	Not affected by obesity	Best used for anesthetized patients
		Not for long-term use
Coccygeal	Easy to palpate	Easily dislodged with movement
	Easy to secure	Easily contaminated with fecal material
		Smaller catheters necessary
		Not for long-term use
Dorsal metatarsal	Easy to palpate	May be affected by obesity
	Easiest to cannulate	
	Less danger of hemorrhage in patients with coagulopathies	
Radial	Easy to palpate	Metacarpal pad may interfere with placement
		May easily become thrombosed
		Not for long-term use
Femoral	Easy to palpate	Risk of hemorrhage, particularly if coagulopathy present
		Affected by obesity
		Easily dislodged with movement

proposed catheterization site. The probe is applied to the skin in transverse orientation to visualize the artery. The artery should be centered in the image and the probe should then be rotated 90 degrees into longitudinal orientation to view the entire length of the vessel and catheter. The stylet–catheter is advanced through the skin slightly distal to the probe at approximately a 35 degree angle [7]. Once a flash of blood is observed in the catheter hub, the catheter angle should be dropped so that the catheter is in a parallel plane with the artery before attempting to feed the catheter into the vessel. Once the catheter is seated into the femoral artery, the stylet is removed. The placement of the catheter can be confirmed with ultrasound image if needed.

Auricular Artery Catheterization

Auricular arterial catheters can sometimes be placed in dogs with large, pendulous ears. This technique is generally reserved for patients that are anesthetized, as the pinna is very sensitive to touch and has a twitch reflex that makes its artery difficult to catheterize when the animal is awake. The auricular artery is located on the dorsal aspect of the pinna (Figure 8.3a). Palpate the pulse on the pinna surface and trace it to the ear tip to determine the artery's location. Clip and aseptically prepare the entire dorsal surface of the ear pinna as previously described, while supporting the pinna with four fingers of the nondominant hand; fold the ear tip with the thumb so that it is perpendicular to the main portion of the pinna. Perform percutaneous facilitation if needed and insert the catheter system through the skin directly into the artery in 1–2 mm increments. Once a flash of blood is visible in the catheter hub, gently advance



Figure 8.3 (a) The auricular artery is located approximately on the midline of the pinna's dorsal surface. (b) Once the catheter is in place, the hub should be secured by applying adhesive tape around the hub and extending the tape circumferentially around the pinna. (c) Folded 4 × 4-inch gauze squares or rolls of gauze should be used to splint the pinna with the arterial catheter in place, to enhance security.

the catheter into the auricular artery. Quickly remove the catheter stylet and replace it with a preflushed Luer lock male adapter or T-port. Wipe the area clean; then secure a length of half-inch medical tape to the catheter hub and wrap it around the pinna (Figure 8.3b). Because the ear is floppy and can fold on itself, use several folded 4 × 4-inch gauze squares or a roll of cotton gauze to softly splint the underside of the pinna such that the lateral aspects of the pinna are folded around the rolls (Figure 8.3c). The catheter and rolls of gauze or cotton are taped in place in a manner similar to that used for dorsal metatarsal catheters. Often, the weight of the bandage becomes too cumbersome in an awake patient and stimulates head shaking. This can cause the catheter to become dislodged. Also, the pinna has a relatively high risk of ischemia with prolonged arterial occlusion. Therefore, auricular artery catheterization is often used only in extremely subdued, obtunded, or anesthetized patients for a limited time (Protocol 8.4).

Radial Artery Catheterization

Catheterization of the radial artery is technically more difficult than for other anatomic locations because the radial artery is small. This technique can be performed in larger dogs while the patient is under general anesthesia. To place a radial arterial catheter, the patient is placed in lateral recumbency with the target limb adjacent to the table, and the palmar aspect of the patient's paw is clipped just proximal to the metacarpal footpad. After the aseptic scrub, the patient's paw is held in the operator's hand and the radial pulse palpated with the forefinger. Percutaneous facilitation is often helpful in this location. With the dominant hand, an over-the-needle catheter is inserted through the skin at a 15–20 degree angle, while the operator observes closely for a flash of blood. The size and length of catheter depends on the size of the patient and the artery. Longer catheters (e.g. 1½ inches, 5–7 mm) should be chosen for larger dogs, as skin movement in this

Protocol 8.4 Auricular Artery Catheterization

Items Required

- Clean clippers and blade
- Surgical scrub preparation supplies
- Examination or sterile gloves for operator
- Sterile gauze squares
- 4 × 4-inch gauze squares, or 3-inch roll gauze
- Surgical tape (½-inch and 1-inch widths)
- Cotton roll gauze
- 22–20 gauge needle
- Over-the-needle or over-the-wire intravascular catheter
- Luer lock T-port or Luer lock infusion plug
- Preservative-free heparinized saline flush syringes
- "Not for IV Infusion" label or indelible marker
- An assistant, if needed

Procedure

1) Collect necessary supplies, prepare tape, and prepare and flush the T-port or male adapter with preservative-free heparinized saline.
2) Place the patient in sternal or lateral recumbency.
3) If the patient is not anesthetized, an assistant should restrain so the patient is immobile.
4) Clip the dorsal pinna surface on midline, 2 inches (5 cm) from proposed insertion site in all directions.
5) Aseptically prepare the proposed catheter insertion site using surgical scrub technique. Allow a minimum of appropriate contact time of the cleanser with the skin, according to manufacturer's instructions.
6) Perform hand hygiene and don examination gloves (sterile gloves if the patient is immunocompromised).
7) Gently remove residual scrub with sterile gauze moistened with sterile water or saline.
8) Hold pinna in the nondominant hand, folding the ear over the fingers.
9) The auricular artery should be visible on dorsal midline of the ear pinna.
10) Perform percutaneous facilitation, if desired. Insert the catheter into the auricular artery.
11) Watch for a flash of blood in the catheter hub.
12) Once a flash of blood is visible in the catheter hub, advance the catheter and stylet an additional 1–2 mm.
13) Feed catheter off the stylet into the artery.
14) Before removing the stylet from the catheter, place sterile gauze squares beneath catheter hub to absorb blood.
15) Remove the stylet and quickly place the flushed male adapter or T-port into the catheter hub. Take care to avoid removing the catheter from the skin.
16) With sterile gauze squares, wipe away excess blood, and make sure that the skin under the catheter hub and around the ear is dry.
17) Secure a length of ½-inch medical tape around the catheter hub, then around the ear.
18) Secure a length of 1-inch medical tape under the catheter hub, around the ear, finishing with the tape over the catheter hub.
19) Secure a third length of tape around the male adapter or T-port and then around the pinna as described for step 18.
20) Place a roll of gauze or rolled up gauze squares under the ventral aspect of the ear.
21) Tape the gauze roll in place with several lengths of surgical tape.
22) Secure a "not for IV infusion" sticker over the catheter bandage or make a note with indelible marker.

area can dislodge shorter catheters. Once a blood flash is visible in the catheter hub, the catheter is inserted and a flushed Luer lock T-port or male adapter is attached in place of the stylet. It is the author's recommendation that radial arterial catheters should not remain in place for longer than 24 hours, particularly in cats and smaller dogs, because of the risk of arterial thrombosis and inhibition of perfusion to the distal extremity (Protocol 8.5) [9].

Coccygeal Artery Catheterization

The coccygeal artery can be catheterized in patients under general anesthesia. For coccygeal arterial catheter placement, the patient is positioned in either dorsal or lateral recumbency, and the ventral aspect of the tail base is clipped. Circumferential clips of the tail base should be considered in patients with long fur that could potentially contaminate the catheter site. After an aseptic scrub and proper hand hygiene, the coccygeal pulse is palpated on the tail's ventral midline. The pulse is palpable between coccygeal vertebrae. Once the pulse is felt, an over-the-needle catheter is inserted at a 15–20 degree angle with respect to the skin, into the artery, while the catheter hub is observed for blood. Once a blood flash is visible in the catheter hub, the catheter is inserted and a flushed Luer lock T-port or male adapter is attached in place of the stylet. The catheter is secured to the tail with medical tape and gauze as previously described (see Protocol 8.6 for details). Coccygeal arterial catheters usually clot within 24 hours, so they must

Protocol 8.5 Radial Artery Catheterization

Items Required

- Clean clippers and blade
- Surgical scrub preparation supplies
- Examination or sterile gloves for operator
- Sterile gauze squares
- Surgical tape (½-inch and 1-inch widths)
- Cotton roll gauze
- Water-resistant bandaging material, if desired
- 20–22 gauge needle, if desired
- Over-the-needle or over-the-wire intravascular catheter
- Luer lock T-port or Luer lock infusion plug
- Preservative-free heparinized saline flush syringes
- "Not for IV infusion" label or indelible marker
- An assistant, if needed

Procedure

1) Collect the necessary supplies, prepare tape, and prepare and flush the T-port or male adapter with preservative-free heparinized saline.
2) Position the patient in lateral recumbency, with the limb of proposed catheter insertion adjacent to the table.
3) If the patient is not anesthetized, have an assistant restrain.
4) Palpate gently for arterial pulse on the caudomedial aspect of limb, just proximal to the metacarpal footpad, to determine proposed insertion site.
5) Clip fur proximal to metacarpal footpad, at least 5 cm (~2 inches) from the proposed catheter insertion site in all directions. Wipe clipped fur away with a gauze square.
6) Aseptically prepare the proposed catheter insertion site using surgical scrub technique. Allow a minimum of appropriate contact time of the cleanser with the skin, according to manufacturer's instructions.
7) Perform hand hygiene and don examination gloves (or sterile gloves if the patient is immunocompromised).
8) Gently remove residual scrub with sterile gauze moistened with sterile water or saline.
9) Place sterile gauze square over the metacarpal pad and fur on the distal limb, to avoid contamination of the catheter.
10) Use gloved index finger to palpate radial pulse in the surgically scrubbed area.
11) Once pulse is found, perform percutaneous facilitation with bevel of a 20- to 22-gauge needle.
12) Gently insert over-the-needle catheter, directing catheter at a 20° angle with respect to the skin toward the pulsing artery.
13) Advance the stylet-catheter apparatus in 1- to 2-mm increments into the area of the pulse. Observe catheter hub for a flassh of blood.
14) Once a flash is observed in the hub, insert the stylet 1–2 mm more and push the catheter off of the stylet into the artery.
15) Before removing the stylet from the catheter, place sterile gauze squares beneath the catheter hub to absorb blood.
16) Remove the stylet, and quickly place the male adapter or T-port into the catheter hub. Take care to avoid removing the catheter from the skin.
17) With sterile gauze squares, wipe away excess blood, making sure that the skin underneath the catheter hub and around the limb is dry.
18) Secure a length of ½-inch medical adhesive tape around the catheter hub, then around the limb.
19) Secure a length of 1-inch medical adhesive tape under the catheter hub, then around the limb, finishing with the tape around the catheter hub.
20) Secure a third length of tape around the male adapter or t-port and the around the limb as described for step 19.
21) Bandage the catheter with cotton gauze and an outer layer.
22) Secure a "Not for IV Infusion" sticker over the catheter bandage, or make note with indelible marker.

be watched very closely and flushed carefully. Also, because of the risk of contamination by fecal material, and also because the catheters tend to become dislodged with patient movement, coccygeal arterial catheters should be removed after general anesthesia and/or surgery has been completed.

Monitor the extremity distal to the catheter site for poor perfusion. If the limb distal to the catheter feels cool or cold to the touch, if the limb is painful, if the catheter is not working well or is no longer patent, or if the arterial pulse cannot be palpated, the artery may be thrombosed and perfusion to the limb may be compromised. In such cases, remove the catheter immediately. Ischemic complications of arterial catheterization are especially common in cats, which generally have poorer collateral circulation than dogs.

Protocol 8.6 Coccygeal Arterial Catheterization

Items Required

- Clean clippers and blade
- Surgical scrub preparation supplies
- Examination or sterile gloves for operator
- Sterile gauze squares
- Surgical tape (½-inch and 1-inch widths)
- Cotton roll gauze
- Water-resistant bandaging material, if desired
- 22–20 gauge needle, if desired
- Over-the-needle or over-the-wire intravascular catheter
- Luer lock T-port or Luer lock infusion plug
- Preservative-free heparinized saline flush syringes
- "Not for IV infusion" label or indelible marker
- An assistant, if needed

Procedure

1) Collect necessary supplies, prepare tape, and prepare and flush the T-port or male adapter with preservative-free heparinized saline.
2) Place the patient in lateral or dorsal recumbency.
3) Clip fur circumferentially from tail base, at least 2 inches (5 cm) from the proposed catheter insertion site in all directions. Wipe clipped fur away with a gauze square.
4) Aseptically prepare the proposed catheter insertion site using surgical scrub technique. Allow a minimum of appropriate contact time of the cleanser with the skin, according to manufacturer's instructions.
5) Perform hand hygiene and don examination gloves (sterile gloves if the patient is immunocompromised).
6) Gently remove residual scrub with sterile gauze moistened with sterile water or saline.
7) Using a gloved index finger, palpate ventral midline of tail base, between coccygeal vertebrae.
8) Perform percutaneous facilitation with bevel of a 20–22 gauge needle, if desired.
9) Place sterile gauze square distal to the insertion site, to avoid contamination of the catheter.
10) Insert the catheter at a 20-degree angle with respect to the skin, between coccygeal vertebrae, toward the palpable pulse.
11) Advance the stylet–catheter apparatus in 1–2 mm increments into the area of the pulse. Watch the catheter hub for a flash of blood.
12) Once a flash of blood is observed, align catheter angle with the artery, then push the catheter off the stylet, into the artery.
13) Before removing the stylet from the catheter, place gauze squares under the catheter hub to absorb blood.
14) Remove the stylet, and quickly place the male adapter or T-port into the catheter hub. Take care to avoid removing the catheter from the skin.
15) Wipe away excess blood, and make sure that the skin under the catheter hub and around the tail is dry.
16) Secure a length of ½-inch medical tape around the catheter hub, then around the tail.
17) Secure a length of 1-inch medical tape under the catheter hub, around the tail, finishing with the tape over the catheter hub.
18) Secure a third length of tape around the male adapter or T-port and then around the tail as described for step 17.
19) Secure a "not for IV infusion" sticker over the catheter bandage or make a note with indelible marker.

Arterial Catheter Care

Because significant hemorrhage can occur quickly if an arterial catheter is dislodged, it is important that the catheter be securely placed. The catheter should be labeled appropriately to avoid intra-arterial infusion of drugs, intravenous fluids, or blood products. Except for small amounts of preservative-free, heparinized or non-heparinized flush, no other drugs, blood products, or solutions should be administered through the arterial catheter [5, 6]. In human patients, complications have been associated with inadvertent intra-arterial administration of vasopressors, dextrose, potassium chloride, antibiotics, and insulin [10]. Inadvertent disconnection of the catheter under a bandage can result in significant blood loss.

The patency of arterial catheters is achieved either by intermittent or continuous flushing using either heparinized or 0.9% saline solutions. Although there is evidence to conclude that there is no difference with the use of heparinized saline compared with 0.9% saline solution in preventing catheter occlusion, determining a protocol for maintaining arterial catheters using intermittent flushing requires further investigation [11–13] and should focus on patient safety. Intermittent catheter flushing should be performed every four hours. If heparinize saline is used as a flush solution, care must be taken in small patients not to

over-heparinize or cause intravascular volume overload. Additionally, if 0.9% saline is used, cannula occlusion may occur. If the arterial catheter is being used for continuous blood pressure monitoring, it can be attached to a pressure transducer attached to a bag of heparinized saline under pressure. Most pressure transducers contain a continuous flush system of saline that delivers approximately 3 ml/hour of flush solution, which maintains catheter patency. There is currently no evidence to support the addition of heparin (1–2 iu/ml) for continuous flushing decreases risk of arterial catheter occlusion [12, 13].

The catheter and bandage should be assessed frequently for evidence of moisture, soiling, or blood staining. The catheter bandage should be removed, and the catheter evaluated at least once a day for evidence of redness, swelling, or discharge from the catheter insertion site. If there is pain when the catheter is flushed, or if any of the above abnormalities consistent with inflammation are observed, the catheter should be removed and a pressure bandage secured over the catheter insertion site for at least an hour, to decrease the risk of hemorrhage from the arterial puncture site. See Chapter 63, Care of Indwelling Device Insertion Sites, for more information on regular maintenance of arterial catheters.

Complications Associated with Arterial Puncture or Catheter Placement

Artery puncture or inadvertent dislodgment of a catheter can result in arterial hemorrhage and hematoma formation at the puncture or insertion site. While severe hemorrhage from an arterial catheterization is rare, caution should be exercised with the femoral artery in particular. Basic precautions include inserting a catheter only as large as necessary to avoid premature clotting of the catheter, to obtain adequate blood samples, to obtain a good pressure waveform, and to avoid lacerating the artery during puncture [14].

Arterial occlusion from thrombosis is a potential complication of arterial catheterization, and although rare, can lead to ischemic injury. Catheter size in relation to the artery, the presence of a hematoma and the duration of the catheter in the artery have been identified in human studies as factors that increase risk for arterial occlusion and should be considered when placing arterial catheters in critically ill small animals. Ultrasound-guided placement of arterial catheters can help aid in selection of appropriate catheter size and minimize hematoma formation. Considerations regarding duration of arterial cannulation are made based on individual patient need, although the associated risk of arterial occlusion likely increases with time. A rare complication of arterial catheterization is ischemic necrosis with subsequent infection, which could lead to the need for amputation [14, 15].

Contraindications to Arterial Puncture and Catheterization

Arterial puncture and catheterization can be problematic in patients with hemostatic abnormalities. For example, if an animal has severe thrombocytopenia with a platelet count less than 40 000 platelets/μl [16], or if an animal has vitamin K antagonist rodenticide intoxication, arterial puncture or catheterization can result in hemorrhage from the arterial puncture or catheter site. In the presence of these conditions, placement of an arterial catheter is relatively contraindicated until the platelet count increases or until the coagulopathy has been resolved. The risk of hemorrhage must be outweighed by the need for direct arterial catheterization in very critical patients.

Hypercoagulable states, such as those associated with immune-mediated hemolytic anemia or a protein-losing nephropathy or enteropathy, can have an increased risk of thrombosis; embolism of the artery distal to the catheter site also may be an increased risk [16]. This complication is uncommon, so a prothrombotic state is a relative contraindication to catheter placement. One must weigh the benefits of catheter placement in a prothrombotic animal carefully with respect to the risks involved with its placement. If an animal has a pulmonary thromboembolism and will require numerous ABG analyses during the course of hospitalization, an arterial catheter may be necessary. However, if a catheter is placed simply to obtain continuous blood pressure monitoring, the use of an indirect method such as Doppler plethysmography or use of an oscillometric monitor may be preferable.

It is the author's opinion that arterial puncture or catheterization should not be performed if the skin and tissue overlying the artery are compromised in any manner [3]. Shearing injuries, pyoderma, burns, and even small abrasions potentially pose an increased risk of infection and, as tissue heals, an increased risk of thrombosis and wound contracture. For this reason, alternative anatomic locations should be considered for arterial puncture or catheterization.

Troubleshooting

The arterial catheter should be assessed frequently for patency and cleanliness. If the catheter is not patent, the first step should be to unwrap the catheter to see if the catheter has slipped or is no longer in the artery. It is not uncommon for the catheter to kink at the insertion site, so this should be investigated before aggressively flushing the catheter. Because embolism is a possibility, an arterial catheter that is not flushing easily should always be evaluated (see Chapter 63 for more detail).

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9

Ultrasound-Guided Vascular Access

Søren Boysen and Valerie Madden

In veterinary patients requiring rapid fluid resuscitation, airway management, or injectable medications, the placement of intravenous (IV) devices is essential. In most patients, simple landmark-based blind placement of peripheral vascular catheters is successful (see Chapter 7). However, situations can be encountered where peripheral vascular access is difficult or impossible to achieve due to thrombosis, edema, obesity, limited viable peripheral vessels, or due to marked peripheral vasoconstriction and vascular collapse. Being familiar with ultrasound-guided vascular access, automated intraosseous catheter placement, and vascular cutdown techniques is invaluable in these patients, particularly given unsuccessful attempts at obtaining vascular access may result in hematoma formation and delayed treatment.

The decision to place an ultrasound-guided peripheral catheter, or an intraosseous (IO) catheter, or to perform a vascular cutdown to gain vascular access depends on patient considerations, particularly patient stability, reason for vascular access, equipment available, operator experience, and client financial considerations. In general, either an IO catheter or vascular cutdown is preferred when the patient is unstable (see Chapter 7) while ultrasound-guided catheter placement is reserved for the more stable patient in which vascular access is difficult.

Ultrasound-guided peripheral and central venous catheterization is commonly used in human emergency and critical care settings as it has a higher success rate, lower complication rate, and is faster than blind/landmark techniques in people, particularly when peripheral vascular access is difficult [1, 2]. The success rates and time to place ultrasound-guided catheters in veterinary medicine have not been well studied. Based on the limited evidence available, the complication rate and time to place ultrasound-guided central lines in dogs under anesthesia are similar to those of blind peripheral catheter placement [3]. The success rate of using ultrasound-guided catheter placement in canine cadavers is very high in situations where edema and hematoma make palpation of landmarks difficult [4]. There is also evidence that ultrasound-guided femoral arterial catheter placement has good success and low complication rates in anesthetized dogs [5].

Indications for Ultrasound-Guided Vascular Access

Indications for ultrasound-guided catheter placement include hematoma formation, inability to palpate landmarks for peripheral percutaneous catheter placement, edema formation, obesity, and failure to place a percutaneous catheter after three attempts (defined as difficult vascular access) [1, 2, 4]. Contraindications and complications of ultrasound-guided catheter placement are similar to those of standard percutaneous vascular access [1–3].

An advantage of ultrasound-guided catheter placement is that the depth, radius (which may help in choosing the catheter size), and patency (presence of thrombus) of the vessel can be assessed prior to placing a catheter [6]. Although peripheral superficial vessels can be challenging to access via ultrasound guidance, human studies demonstrate very high success rates with ultrasound guidance when vessels are only millimeters from the skin surface and of very small diameter [6]. Blood sampling, including dorsal pedal arterial access for blood gas analysis, can be performed using ultrasound guidance, which has the advantage of visualizing the vessel of interest to avoid accidental venous sampling.

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Techniques

The two common techniques for ultrasound-guided catheter placement include out-of-plane (Protocol 9.1) and in-plane (Protocol 9.2) techniques (Figures 9.1 and 9.2) [2]. The authors have the greatest success using an out-of-plane technique and sweeping (Figure 9.3) the transducer along the vessel as the catheter is advanced (explanation of transducer movements like "sweeping" are found in Chapter 6). The out-of-plane technique works well for accessing vessels when the vessel remains visible and can thus be centered as the transducer is swept and/or fanned to follow the tip of

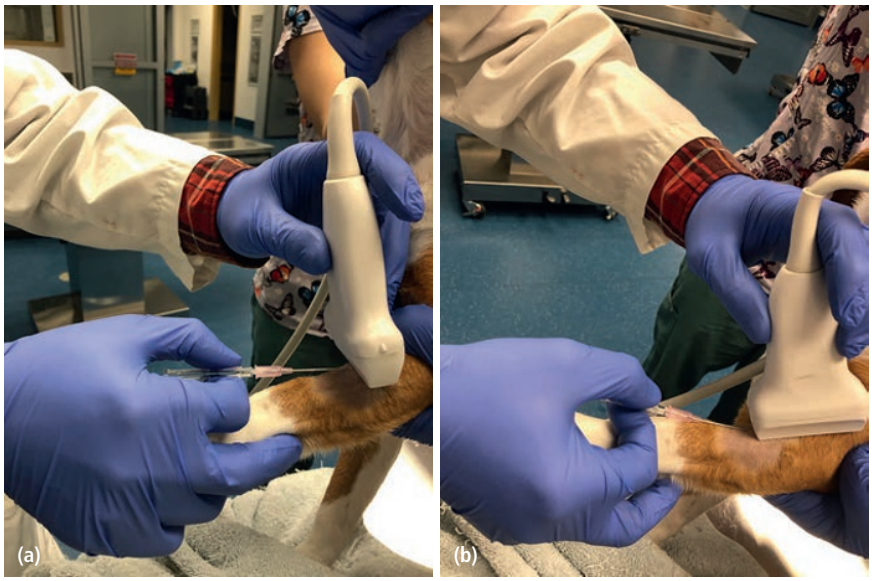


Figure 9.1 (a) Out-of-plane ultrasound-guided vascular access demonstrated on the cephalic vein of a Beagle using a linear array transducer. (b) In-plane ultrasound-guided vascular access demonstrated on a Beagle using a linear array transducer. The large footprint of the linear array transducer makes it more challenging to use an out-of-plane technique in smaller dogs and in cats. The angle of the catheter and stylet should be adjusted depending on the depth of the vessel. In this example, the cephalic vein is quite superficial so the angle of the catheter relative to the skin surface is about 30 degrees.

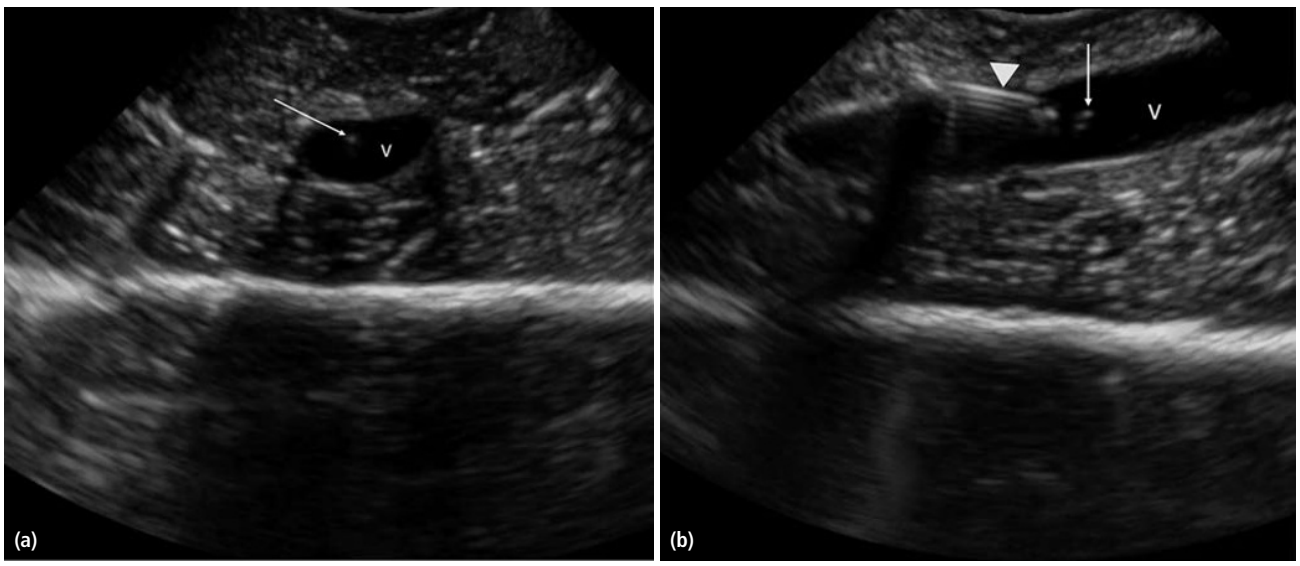


Figure 9.2 (a) Ultrasound still image of a vessel acquired using the out-of-plane technique. The tip of the stylet (white dot) within the vessel lumen (V) with the transducer in short axis (transverse) orientation to the catheter and vessel. Stylet tip identified by the white arrow. The depth is adjusted and the transducer manipulated to center the vessel within the ultrasound image. (b) Ultrasound still image of a vessel acquired using the in-plane technique. The tip of the stylet (white arrow) and catheter (white arrowhead) within the vessel lumen (V) with the transducer in long axis (longitudinal) orientation to the catheter and vessel. The stylet and catheter are visible traversing the proximal vessel wall to enter the lumen of the vessel.

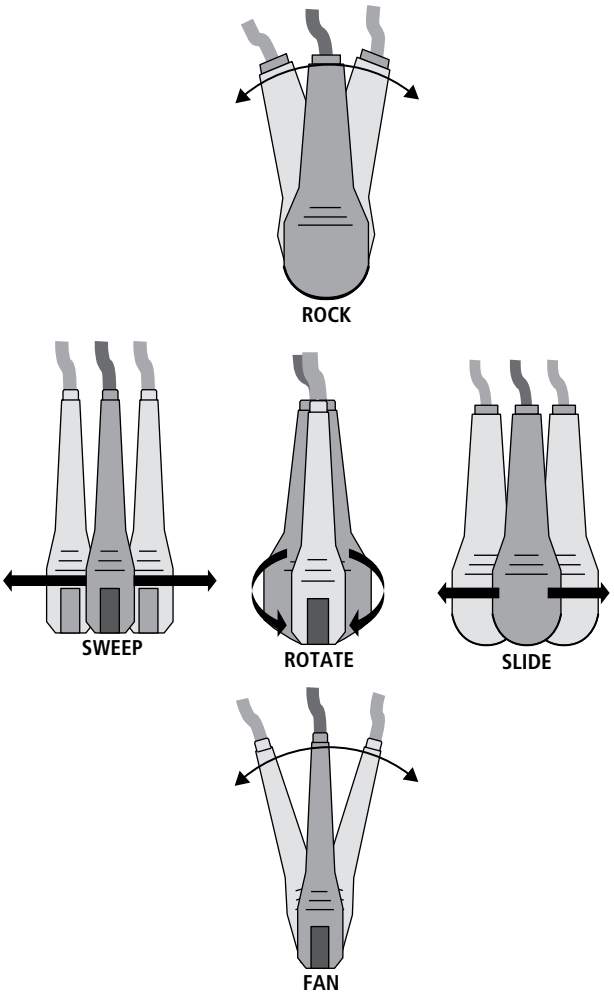


Figure 9.3 Summary of the five different probe manipulations commonly used during point-of-care ultrasound; sweep, slide, rotate, fan, and rock (see Chapter 6 for details).

the catheter stylet (i.e. you do not slide off vessels). Keep the stylet tip visible when using the out-of-plane technique to avoid accidental puncture of the far vessel wall. The catheter tip should be adjusted as needed to keep it centered over the vessel while the tip is still within the subcutaneous tissue (proximal to the vessel wall); such adjustments are easily accomplished with out-of-plane ultrasound-guided catheter techniques.

Aseptic technique should be followed, including the use of sterile ultrasound gel applied in combination with isopropyl alcohol, and if necessary, covering the transducer with a sterile glove or sleeve. Sterile standoff ultrasound pads or jelly pads have been used in some human studies to facilitate ultrasound-guided catheterization, although their application in veterinary medicine has not been evaluated [8].

Complications of Ultrasound-Guided Vascular Access

Complications are uncommon and occur at a similar rate to blind peripheral catheter insertion techniques [2, 3]. In human medicine, reported complications vary depending on which site is used for ultrasound-guided vascular access and include paresthesias, brachial artery puncture, hematoma formation, and IV decannulation [2]. To avoid complications:

- The best target will be the vessel that is the largest and most superficial.
- For deep vessels, angle your catheter at a steeper angle than you would for a superficial vessel (35–45 degrees).

Protocol 9.1 Ultrasound-Guided Vascular Catheterization Using the Out-of-Plane Technique

Machine and patient preparation

1) A high frequency linear array transducer (8–13 MHz) is preferred, although a microconvex transducer can be used if a linear transducer is unavailable.
2) Align the ultrasound machine alongside the patient, in front of the sonographer, so the operator only has to adjust their eye focus from the skin transducer interface to the ultrasound screen (up and down) without having to turn their head or neck to look over their shoulder when changing their focus from the transducer to the ultrasound image.
3) Position the patient in lateral recumbency when accessing the jugular or saphenous veins and sternal recumbency to access the cephalic veins.
4) Depth should be set to maximize the size of the vessel without losing it in the far field of the image. Sweep along the vessel to ensure it remains visible within the ultrasound window.
5) Veins and arteries are easily distinguished by applying pressure to the ultrasound transducer, which will collapse veins at much lower pressures than arteries [2].
6) Avoid applying too much pressure to the transducer when first identifying vessels or during venous access, to avoid collapsing the vessel, particularly veins.
7) "Holding off" the vein helps prevent venous collapse and increases the diameter of the vein.

8) Stabilize the transducer by resting your hand on the patient using the "afternoon tea technique" approach (Figure 9.4) [7].
9) Scan the vessel of interest to gauge its depth from the skin surface, its diameter and patency, as well as the direction of the vessel prior to inserting the catheter.
10) Using a longer (1.8 or 2.5 inch) catheter is often preferred since ultrasound guidance is frequently used in patients with edema, obesity, or hematoma formation where the vessel of interest will be located deeper within the tissues.
11) Fur is clipped over the vessel of interest.
12) Clean the ultrasound transducer by wiping with soap and water followed by a low-level disinfectant (e.g. hydrogen peroxide or diluted bleach) and apply sterile lubricant. A gel-filled sterile glove can be placed over the ultrasound transducer to maintain a sterile field if desired.
13) For venous access, have an assistant occlude the vein or apply a tourniquet proximal to the insertion site.
14) Prepare the skin surrounding the insertion site just distal to the transducer using aseptic technique.

Out-of-plane technique

15) Place the transducer over the vessel of interest at a 90-degree angle of insonation in short axis orientation (perpendicular) to the vessel (Figure 9.4).
16) Adjust the position/depth to center the vessel within the ultrasound image (Figure 9.2).
17) Veins will be thin-walled and easily compressible, compared with arteries, which will be thick-walled and non-compressible (Figure 9.5) [2].
18) When the vessel of interest has been identified, the operator focuses their attention to the ultrasound transducer–skin interface.
19) The bevel of the stylet should be directed toward the ultrasound transducer as this will maximize the reflection of ultrasound waves and enhance needle tip visualization (cut surface reflects the most ultrasound waves).
20) The angle of entry relative to the skin surface varies depending how deep the vessel is within the tissues. Generally, the needle is inserted at an angle of 30–45 degrees to the transducer, just distal to the ultrasound transducer (Figure 9.1) [2].
21) Once the tip of the needle has entered the skin, the operator's focus returns to the ultrasound screen.
22) Slowly sweep the transducer proximally along the vessel as the needle tip moves proximally. This is typically performed by advancing the catheter tip, out of plane, until it becomes visible within the ultrasound image as a "small white hyperechoic dot" (Figure 9.2a, Video 9.1).
23) Failure to stop advancing the catheter as soon as the stylet tip is seen as a white dot on the ultrasound screen increases the risk of accidently traversing the vessel and in the authors' experience, is one of the most common mistakes made using out-of-plane technique. Advancing the catheter tip beyond the ultrasound beam results in uncertainty regarding catheter tip location.

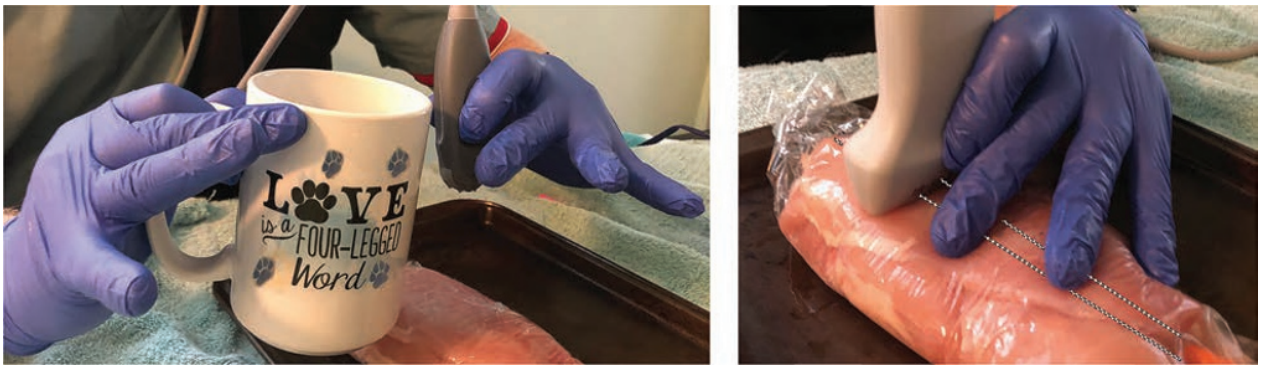


Figure 9.4 Images depicting the "afternoon tea technique" to help stabilize the transducer. The concept involves keeping the little finger extended away from the transducer and in contact with the patient's skin, while the thumb and "pointer" finger are used to grip the transducer head. Left image: the transducer is held like a cup demonstrating proper finger position of the "tea" technique. Right image: the dotted lines represent the walls of a water-filled balloon, which is used to mimic a vessel within a raw chicken phantom model. The transducer is situated perpendicular to the vessel in this example (i.e. out-of-plane). The fingers are gently positioned against the skin (raw chicken breast in this case) to stabilize the probe.

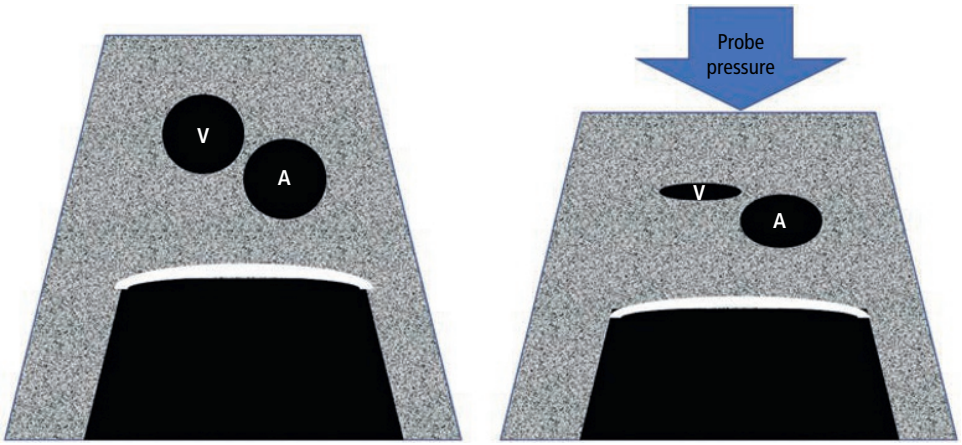


Figure 9.5 Schematic ultrasound image of an artery (A) and vein (V). With application of pressure to the transducer at the transducer-skin interface, the thinner-walled vein collapses, sometimes disappearing completely, while the artery remains visible, compressing less. This "compression" test helps to differentiate arteries and veins that often lie in close proximity.

24) Once the tip is visible discontinue advancing the catheter and sweep the transducer proximally along the vessel until the catheter tip is no longer visible.
25) Failure to slide the transducer completely off the stylet tip (tip completely out of the plane of imaging) can also result in the catheter tip being advanced beyond the ultrasound beam when the catheter is advanced again.
26) The process is repeated until the catheter tip, seen as a white dot, can be visualized within the vessel lumen.
27) Once the catheter tip is visualized within the vessel, a flash of blood will likely be visible in the catheter stylet.
28) The remainder of the procedure proceeds as with the blind technique for vascular catheter placement. Ultrasound guidance can be continued for this step if desired. Decrease the angle of the catheter and stylet approximately 15 degrees relative to the skin surface and advance them together another 1–2 mm to ensure that the tip of the catheter and the stylet are both within the vessel lumen.
29) Hold the stylet with one hand as the catheter is completely advanced off the stylet and into the vessel. If desired, advancement of the catheter off the stylet can be performed by an assistant while the operator ensures the catheter tip remains visible within the lumen of the vessel.

Advantages and Disadvantages

A major advantage of the out-of-plane technique is that it is more forgiving; the operator does not have to maintain both the catheter and the transducer in the same plane to be able to visualize the stylet tip. It is also possible to adjust the stylet tip location within the subcutaneous tissues to recenter the catheter over the vessel if alignment is slightly off (Video 9.2). The disadvantage of out-of-plane technique is the risk of passing the catheter tip beyond the ultrasound beam without realizing this has occurred. A black shadow will often appear below the white dot when this happens.



Protocol 9.2 Ultrasound-Guided Vascular Catheterization Using the in-Plane Technique

1) Follow Protocol 9.1, steps 1–14.
2) The catheter and tip of the stylet are visualized as they enter the subcutaneous tissues within the ultrasound image (the transducer marker or ultrasound image can be set to visualize the catheter entering from the left or right side of the ultrasound image depending on operator preference).
3) The stylet tip and catheter are followed in their entirety as they traverse the subcutaneous tissues and the superficial wall of the vessel to enter the lumen (Figure 9.2b).
4) Once in the lumen of the vessel, the angle between the skin surface and the catheter is reduced, and the catheter and stylet advanced slightly within the vessel, taking care not to traverse the deep vessel wall.

5) Once the catheter is well situated in the vessel, the catheter is advanced off the stylet and down the vessel lumen.
6) The entire procedure can be visualized using in-plane techniques.

Advantages and Disadvantages

The difficulty with the in-plane technique is that it requires more practice to keep the catheter perfectly aligned in the plane of the ultrasound beam, as being off plane by even 1–2 degrees, or failing to maintain the catheter directly in the center of the ultrasound transducer, precludes catheter visualization. Achieving this alignment is more difficult with smaller peripheral vessels, particularly given the narrow width (1–2 mm) of the ultrasound beam projected by linear array transducers. An advantage of in-plane technique is the fact that surrounding structures can always be visualized as the catheter is advanced.

Protocol 9.3 Phantom Chicken Breast Simulator for Ultrasound-Guided Vascular Access Training

1) A phantom model can be made using two raw chicken breasts, plastic kitchen wrap, and modeling balloons (Figure 9.6).
2) One chicken breast is laid in the middle of a flat piece of plastic kitchen wrap that is large enough to wrap around two chicken breasts placed one on top of the other (Figure 9.7).
3) A fluid-filled, tied-off modeling balloon (or similar tubular structure designed to mimic a vessel such as a small-diameter Penrose drain) is placed atop the chicken breast (Figure 9.8).
4) A single balloon or two balloons can be used. Place the balloons parallel to one another, 1–2 cm apart if using a two-balloon model (Figure 9.6).
5) A second chicken breast is placed on top of the balloons to create a "sandwich" with the balloons in the middle of the two chicken breasts (Figure 9.9).
6) The plastic kitchen wrap is then wrapped around the chicken breasts to secure the model.



Figure 9.6 Using short- or long-axis ultrasound guidance (long axis shown in this image) a raw chicken breast vascular access simulator (made from two raw chicken breasts, water-filled balloons, and plastic kitchen wrap) can be used to practice identifying and catheterizing a vessel. The chicken breast creates a more lifelike tissue structure with fascial planes than many other simulator models.



Figure 9.7 One raw chicken breast that has been scored with a scalpel blade is laid in the middle of a flat sheet of plastic wrap. To keep things contained, the model is built within a shallow tinfoil cooking tray.

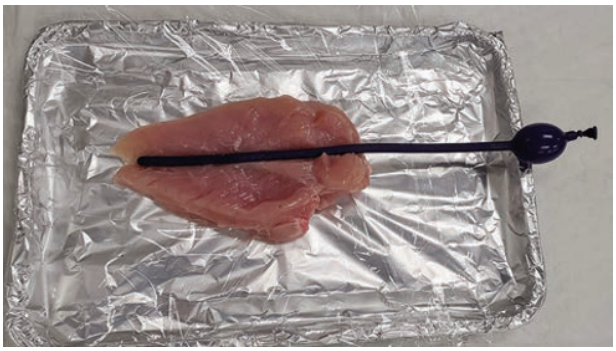


Figure 9.8 A modeling balloon (shown) or small-diameter Penrose drain (not shown) is placed on the chicken breast, within scored grooves created with a scalpel blade.

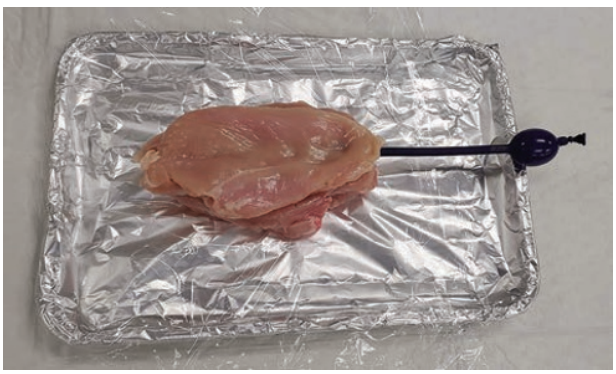


Figure 9.9 A second raw chicken breast is placed over the first chicken breast and balloon. The authors prefer to reverse the direction of the chicken breasts (i.e. one thick side to the left and one thick side to the right), as shown, to optimize consistency of tissue thickness.

Pearls for Making The Raw Chicken Breast Simulator:

- Remove all air from the balloon prior to filling it with fluid (air prevents visualization with ultrasound).
- The balloon can also be slightly overfilled, creating an "aneurysm" that will allow air to be released without losing pressure within the balloon (Figure 9.10). The "aneurysm" also allows any residual air that might remain within the balloon to become trapped in the "aneurysm" which keeps the main vessel that will be catheterized free of gas.
- Avoid overwrapping the chicken breasts with plastic wrap because air can become trapped between the layers of wrap, which hampers tissue and vessel visualization.
- To help keep things nicely situated within the model, score the chicken breast with a scalpel or needle to make a shallow tunnel in the chicken breast before placing the balloons. This helps keeps the balloon in one place within the scored lines when pressure is applied with the transducer to the outside of the model.
- When placing the chicken breasts against each other it is easier to keep objects aligned if the thickness of the chicken breasts is reversed (place the thick side of one chicken breast in one direction and the second thick side of the chicken breast in the opposite direction).



Figure 9.10 After attaching a balloon (shown) or Penrose drain to a fluid filled, 60 cc catheter tip syringe, air is first withdrawn from the balloon. Fluid is then injected into the balloon until it is slightly overfilled, creating an "aneurysm" that will allow air to accumulate without losing pressure within the balloon. Draw back on the syringe a final time to remove air, while still preserving the "aneurysm." Remove the balloon from the syringe and tie off. Food coloring can be used to mimic blood if desired.

- To keep things contained, the model can be placed in aluminum tinfoil trays (Figure 9.7).
- The model also works well to train novices on ultrasound-guided modified Seldinger techniques (Figures 9.11 and 9.12).
- Following the passage of the stylet and catheter for the modified Seldinger technique, with the transducer in long axis to the lumen of the balloon, allows troubleshooting of the technique to see if the J wire becomes caught on the far vessel wall or to see if the catheter tip has accidently traversed the far wall of the vessel (easily corrected with ultrasound guidance by slowly retracting the catheter until the tip is again situated in the "vessel" lumen).



Figure 9.11 After placing an ultrasound-guided catheter into the balloon, a guidewire is passed through the catheter into the lumen of the balloon using a modified Seldinger technique. Ultrasound is not necessary for this step (but it is cool to watch on the ultrasound image!). If ultrasound is used to monitor passage of the guidewire, the transducer should be in long axis to the balloon (or vessel in a live patient) for this step.



Figure 9.12 The catheter is then passed over the guidewire as is normally done with the modified Seldinger technique. Again, ultrasound does not need to be used for this step, but if used, the transducer is oriented in long axis to the balloon to more easily allow the catheter to be seen sliding down the lumen of the balloon, as shown in the image.

Ultrasound-Guided Vascular Access Simulators

Many low-cost simulation models have been used to train clinicians in ultrasound-guided vascular access. The model that the authors prefer is a raw chicken breast model, which uses long, thin, fluid-filled "modeling" balloons or small-diameter Penrose drains (Figure 9.6). Note that you must follow proper hygiene, wear gloves, and wash hands after handling raw chicken to avoid the risk of foodborne illnesses. The technique can also be practiced on cadavers following euthanasia if clients allow.

Video 9.1 Out-of-plane ultrasound-guided vascular access: chicken phantom model.

Video 9.2 Recentering the catheter over the vessel.

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10

Principles of Electrocardiography

Jamie M. Burkitt Creedon

Cardiac Electrical Activity

The heart's main function is to pump blood. The myocardium is composed of muscle fibers linked by conduction system cells. Synchronized electrical stimulation of the cardiac myocytes is necessary for well-coordinated contraction, which optimizes cardiac output. Myocytes are responsible for the heart's contractile function, whereas the conduction system cells deliver the electrical impulse that leads to myocyte contraction [1].

The heart's electrical stimulus originates at the sinus node (also called the sinoatrial node or SA node). The SA node is a group of cells in the right atrium that has the highest intrinsic (spontaneous) rate of depolarization in the normal heart. Because they depolarize more often than other cardiac cells, the SA node is the heart's primary pacemaker. The three internodal tracts (anterior, medial, and posterior) and Bachmann's bundle transmit the electrical impulse to the atrioventricular (AV) node and the left atrium, respectively [2]. By design, conduction through the AV node is slow, to allow a pause between atrial and ventricular contractions. This AV pause allows atrial contraction to push blood into the ventricles before ventricular systole begins. Conduction then proceeds from the AV node to the bundle of His, which is the only conductive pathway between the atria and the ventricles in the normal heart. At the level of the aortic valve, the conduction pathway bifurcates into the left bundle branch and right bundle branch (Figure 10.1). Both bundles divide into a network of Purkinje fibers that are distributed to both ventricles [4]. The wave of myocardial contraction follows the electrical impulse starting in the right atrium, continuing to the left atrium, and then to the ventricles.

The Wave of Depolarization

Cardiac myocytes maintain an electrical gradient across their cell membranes called the "resting membrane potential." This gradient is maintained by multiple systems of active ion transport, including the sodium–potassium (Na^+–K^+) ATPase pump, which pumps sodium out of and potassium into the cell. The intracellular potassium concentration ($[K^+]$) is thus significantly higher than the extracellular fluid $[K^+]$.

A myocyte's resting membrane potential is considered negative because the myocyte's intracellular fluid is more negatively charged than the extracellular fluid. When the myocyte is stimulated by the conduction system or by a neighboring myocyte, its polarity is reduced (the myocyte's interior becomes more positive). The less-negative membrane potential significantly alters sodium and potassium permeability through the cell membrane. Sodium ions rush into the myocyte and K^+ ions move to the outside, creating what is called an "action potential." Because the action potential causes a change in the cell's membrane polarity, this event is called depolarization. One myocyte's depolarization immediately stimulates the depolarization of adjacent cells, and the depolarization continues cell-to-cell throughout the myocardium. This chain reaction of cardiac myocyte depolarization is called the "wave of depolarization."

Coordinated cardiac myocyte depolarization is required for coordinated cardiac contraction [5]. An electrocardiogram (ECG) is a graphic representation of the summation vector of all the action potentials of the heart over time (Figure 10.2).

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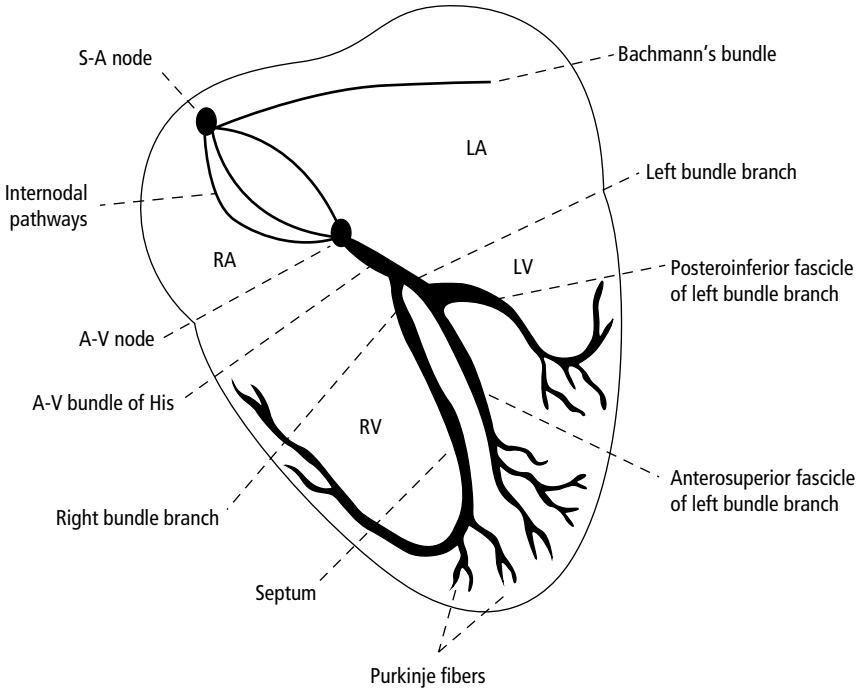


Figure 10.1 Conduction system of the heart. *Source:* Bruce et al. [3]/with permission of Elsevier.

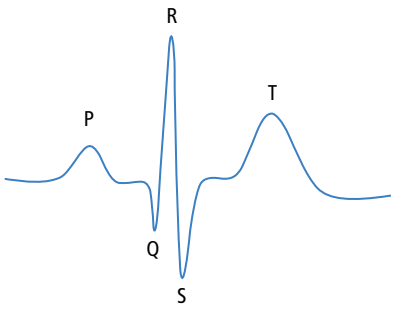


Figure 10.2 Idealized lead II graphic representation of an electrocardiogram from a dog, with the P, QRS, and T waves labeled. The X axis measures time while the Y axis measures the summation vector of the myocardium's electrical activity in the lead being recorded. The P wave is the result of atrial muscle depolarization. The QRS complex is the result of ventricular muscle depolarization. The T wave occurs because of ventricular muscle repolarization; T wave appearance can differ significantly from individual to individual, but it should generally be uniform within the same animal over time. More information is available in Chapter 11.

The Electrocardiogram

The electrocardiograph is a galvanometer or voltmeter that records the electrical impulses between nearby negative and positive electrodes placed in or on the body. A system of two points between which electrical impulses are conducted is called a lead. The electrodes are usually positioned on the animal's limbs (for standard leads), but they can also be placed on the thorax (for precordial chest leads) or in the heart (for intracardiac leads). The graphic representation of the information gathered from an electrocardiographic lead is the ECG. Electrocardiogram setup and monitoring are detailed in Protocol 10.1.

Recording the Wave of Depolarization

If a negative electrode is placed in the vicinity of the right atrium and a positive electrode is placed at the apex of the heart, the normal wave of depolarization travels toward the positive electrode and by convention is represented by a positive (upward) deflection on the ECG [1, 5, 6]. The electrical impulse of a normal cardiac cycle starts at the SA node. (Figure 10.1). The atrial depolarization originates at the SA node, travels through the internodal tracts and Bachmann's bundle and terminates at the AV node. On the ECG, atrial muscle depolarization is represented by the P wave, which is the first positive deflection on the ECG before the QRS complex [7]. Atrial muscle depolarization leads to atrial muscle contraction, and consequently to pumping of the blood from the atria into the ventricles.

There is a physiologic (normal) delay in impulse conduction at the AV node that allows time for blood to flow from the atria into the ventricles prior to ventricular systole. This physiologic delay is the origin of the P–R interval, or the electroneutral "return to baseline" period between the P and QRS waves. Ventricular depolarization starts in the interventricular septum and is represented by a slight negative deflection on the ECG, called the Q wave. When most of the ventricular muscle depolarizes, it generates the

R wave, a large-magnitude positive deflection. The last parts of the ventricles to depolarize are the basilar portions, which create a negative deflection on the ECG that follows the R wave, called the S wave.

After the S wave has occurred, the cardiac depolarization phase is complete. Cardiac repolarization, or the myocytes' re-establishment of their resting membrane potentials, is necessary for another cardiac cycle to start. Ventricular repolarization is represented by the T wave, which in health can be positive, negative, or biphasic [1, 2, 4–8]. For information on ECG waveform interpretation, see Chapter 11.

Einthoven's Triangle and the Principle of Leads

A bipolar lead is the result of the difference in electrical activity between electrodes when a negative electrode is paired with a positive electrode. Each lead "sees" and registers a different view of a single electrical event (such as a cardiac depolarization wave), which allows a more comprehensive understanding of the heart's electrical activity. Imagine that the P–QRS–T complex is an event such as a motor vehicle accident, and that each lead is a witness located in a different position in relation to the event. The witness in the two-story building has a different view than the person across the street, which is again different than that of the witness seated in the coffee shop. All the witnesses saw the same event, but each from a different angle. Thus, we make our interpretation of the event with the advantage of combined observations and not just a single point of view.

Standard ECG Leads

In 1902, Willem Einthoven proposed the first fixed ECG lead system. Einthoven's equilateral triangle illustrates the three standard bipolar leads (Figure 10.3). To obtain these leads, electrodes are placed on the right thoracic limb or "arm" (RA; all ECG limb terminology is in arms and legs, by convention), the left thoracic limb or "arm" (LA), and left pelvic limb or "leg" (LL). The right pelvic limb (RL) is the connection to the ground.

As depicted in Figure 10.3, lead I detects the difference in electrical activity between the right thoracic limb (negative electrode) and the left thoracic limb (positive electrode); lead II detects the difference between the right thoracic limb (negative electrode) and the left pelvic limb (positive electrode); and lead III detects the difference between the left thoracic limb (negative electrode) and the left pelvic limb (positive electrode; Table 10.1) [7]. Standard leads are by far the most commonly used leads in the emergency room and intensive care unit. Augmented unipolar leads and unipolar precordial chest leads are additional leads

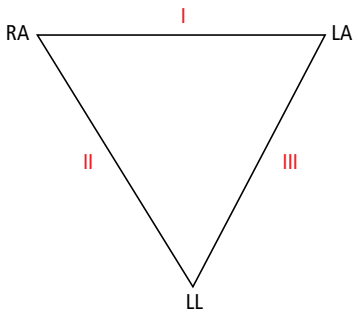


Figure 10.3 Equilateral triangle of Einthoven, oriented as one would view a dorsoventrally oriented radiograph. This figure illustrates standard leads I, II, and III (RA, right arm; LA, left arm; LL, left leg).

Table 10.1 Electrode position for standard bipolar leads: arm, thoracic limb; leg, pelvic limb.

Lead	Positive	Negative/neutral
I	Left arm	Right arm
II	Left leg	Right arm
III	Left leg	Left arm

more commonly used for detailed diagnostic ECG in an outpatient cardiology specialty setting. Augmented leads aVR, aVL, and particularly aVF can be useful when standard limb leads do not answer the operator's questions about ECG rhythm, wave, and complex appearance. More information about these leads is available elsewhere [8, 9].

ECG Measurement in the Emergency Room and Intensive Care Unit

Electrode Placement and Patient Positioning

The patient should ideally be positioned in right lateral recumbency on a nonconductive surface. If the animal is mobile, a handler should rest the right arm over the patient's neck and the left arm over the hindquarters so the limbs are perpendicular to the body, still, and separated (Figure 10.4) [8, 9]. An ECG can be recorded with the patient in sternal or standing position, but this recording should only be used for rate measurement and detection of significant rhythm abnormalities. Fortunately, rhythm investigation is the most common use for ECG measurement in emergency and intensive care settings. Standing technique is especially useful in a patient with respiratory distress. More information about continuous ECG monitoring of acutely and critically ill animals is available in Chapter 11.

The thoracic limb electrodes are usually placed close to the olecranon (elbow) and the pelvic limb electrodes in the



Figure 10.4 Standard patient position for recording an electrocardiogram (right lateral recumbency). Note the four standard electrocardiographic color-coded electrodes (RA, right arm, white; LA, left arm, black; RL, right leg, green; LL, left leg, red).

Table 10.2 Electrocardiograph color-coded cables.

Cable Color	Limb
White	Right arm (RA), right thoracic limb
Black	Left arm (LA), left thoracic limb
Red	Left leg (LL), left pelvic limb
Green	Right leg (RL), right pelvic limb

area of the patellar ligament (stifle). However, the electrodes may be placed at any point distal to the limb's junction with the trunk without significantly affecting the ECG. The limb electrodes are color coded by industry standard, and they should be placed as indicated in Table 10.2 and Figure 10.4. The cables should not be twisted or placed over the patient's body, as this is likely to cause artifacts on the ECG.

Types of Electrodes

Electrodes are attached directly to the patient's skin with alligator clips, smooth clips, electrode patches, or metal plates (Figure 10.5). Alligator clips are the most used electrodes in veterinary medicine since they are relatively simple to apply, are durable, and do not disconnect easily when the patient moves [10]. Smooth clips can improve patient comfort; they can be purchased ready-made or created by flattening the teeth of alligator clips with pliers. Before placing an electrode, a small skin fold should be made in the appropriate location. Isopropyl alcohol or electrocardiographic conducting gel must be applied to the area as a conduction medium; fur clipping is rarely necessary. Alcohol should be avoided in critically ill patients that may be candidates for defibrillation since it is flammable. Electrocardiographic gel is also preferred when ECG recording will be required for longer than 10 minutes. It is preferable to apply the chosen conduction medium before placing the electrodes, as this will minimize interference from the patient's fur. Self-adhesive electrodes are also available (Figure 10.5b), which usually must be secured with a bandage to keep them in place. They can be applied directly to the footpads or in an area of clipped skin.

Electrocardiogram Recording

The electrocardiograph (galvanometer) control settings vary by manufacturer, but there should be an option for paper speed, calibration, and filter settings.

Paper Speed

An ECG can be recorded at any paper speed, but the most used speeds in veterinary medicine are 25 mm/second and 50 mm/second. At a speed of 25 mm/second, 25 mm of paper (five large boxes on the ECG paper) is used to record one second of waves, while at 50 mm/second, 50 mm of paper (10 large boxes) is used to record one second. Thus, the same patient's QRS complex appears wider on the X axis on a 50 mm/second recording than on 25 mm/second because it is recorded faster, which corresponds to more space (twice as much in this example) on the ECG paper.

When performing ECG wave and complex measurements, a lead II recording at 50 mm/second should be used. For rhythm recordings 25 or 50 mm/second can be used, with the choice largely dependent on the patient's heart rate and the desired ECG complex definition. A slower paper speed (e.g. 25 mm/second) will allow a longer recording using the same amount of paper (e.g. as compared with 50 mm/second), which saves ECG paper. A minimum of three complexes should be recorded for each standard lead, and a longer recording is recommended when dysrhythmias are present.

Sensitivity

Standard electrocardiographic calibration is 10 mm/mV, which means that a 1 mV electrical signal generates a 10 mm deflection from baseline on the ECG paper. The operator can recalibrate the electrocardiograph to produce larger (double sensitivity: 1 mV corresponds to 20 mm) or smaller (half sensitivity: 1 mV corresponds to 5 mm) ECG complexes. This feature is especially useful when the ECG complexes are very small, as is common in cats, or are very large, as ventricular premature complexes can be.

The calibration mark is a graphic representation of a selected recording voltage, which is used to gauge the amplitude of the ECG waves. It should appear automatically at the beginning of the recording in the form of a rectangle or line that represents the machine's current calibration (e.g. 10 mm high for standard calibration). The calibration mark precedes the first recorded complex (Figure 10.6).

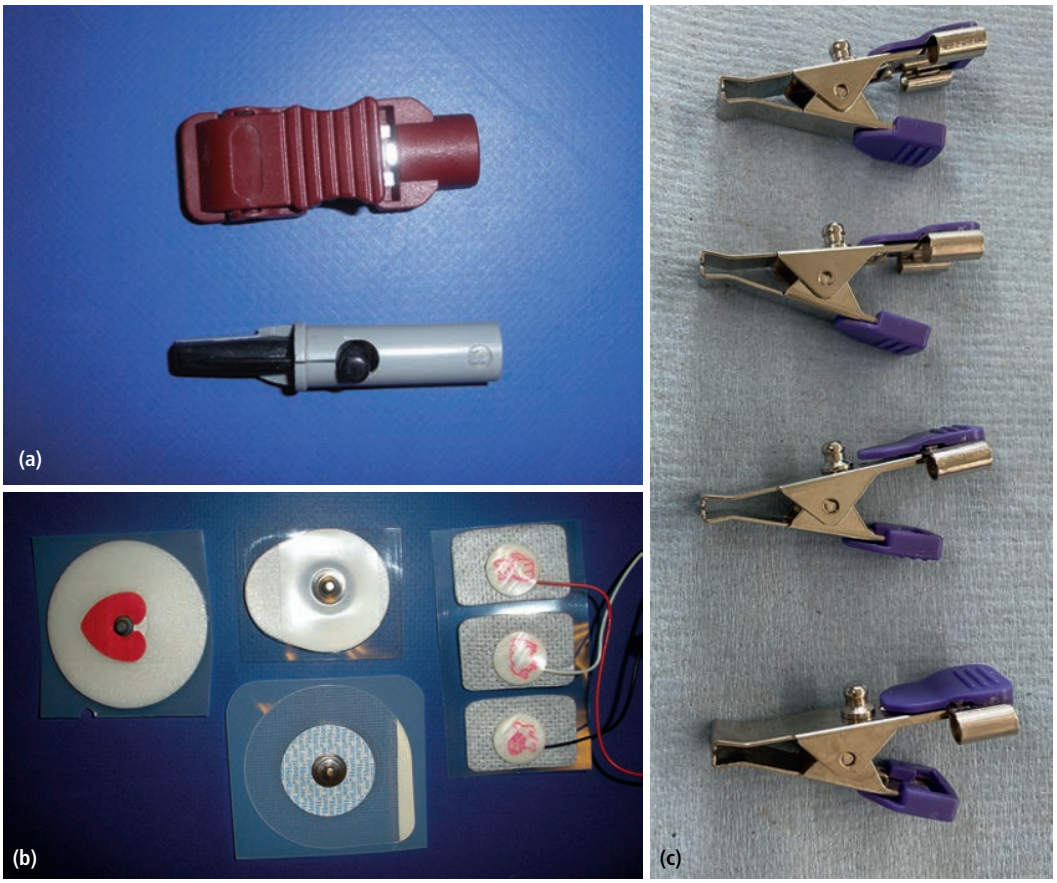
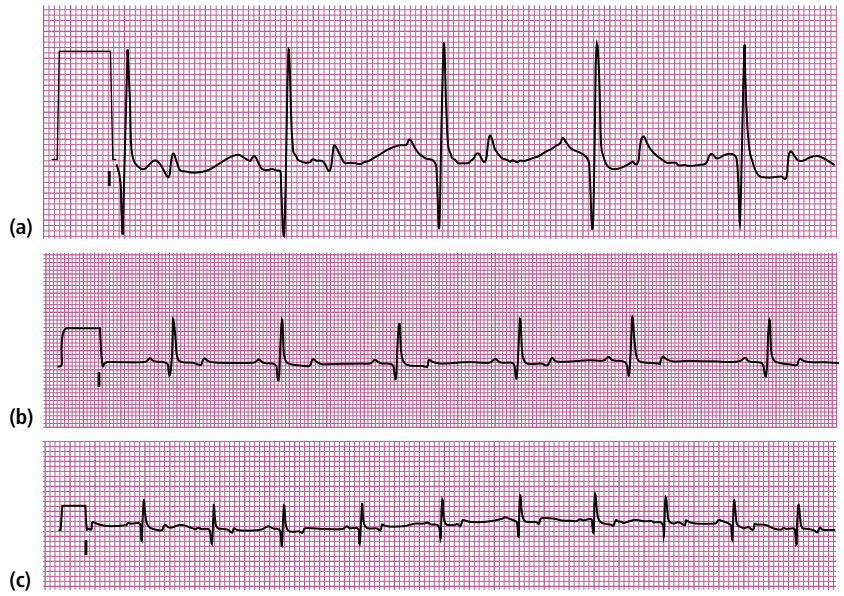


Figure 10.5 (a) An example of an alligator electrode clip (bottom) and a modified metal plate (top). (b) Different types of adhesive electrodes. (c) Alligator style skin clips without teeth. Note the electrodes have integrated snaps to attach to the system's wires.

Figure 10.6 Electrocardiographic recordings of the same dog's ECG at (a) double, (b) standard, and (c) half sensitivities.



Filter Settings

Internal electrocardiographic filters are used to reduce baseline artifacts, but they are not necessary for a good electrocardiographic recording. In dogs, 50 Hz filters are usually appropriate, and 150 Hz filters can be used in cats. Filtering can reduce the amplitude of the complexes on an ECG; therefore, all complex measurements should be performed on an unfiltered tracing.

Ambulatory Continuous ECG Monitoring and Telemetry

Ambulatory Continuous ECG Monitoring

Ambulatory continuous ECG monitoring (Holter monitoring) is an electrocardiographic method that allows recording for longer periods of time, such as 24–48 hours. It is used for the diagnosis, monitoring, and therapeutic evaluation of dysrhythmias. Most of these ECG monitors are powered by batteries and provide a digital recording with multiple channels [9].

Since most dysrhythmias have significant day-to-day variation, there is a considerable advantage to obtaining this longer diagnostic sample. The technique is also useful in the evaluation of syncope and collapse since the patient can go home with the recording device in place to monitor for such sporadic events [9, 11, 12].

ECG Telemetry

Electrocardiographic telemetry is a monitoring technique that helps to monitor hospitalized patients, with digital ECG tracings obtained through a wireless method. This technique allows the patient to move freely in its enclosure without the inconvenience of wires. Telemetry units use a precordial lead system (over the cardiac apex), which usually uses adhesive electrodes that are placed on a shaved area of the thorax.

Equipment Problems Leading to ECG Artifacts

Artifacts can lead to incorrect ECG interpretation. It is thus important to minimize the potential for artifacts such as electrical interference, muscle tremors, patient and system movement, and inappropriate patient or electrode positioning [13]. See Table 10.3 for guidance regarding where to find the source of interference based on affected lead(s). Incorrect electrode placement causes one lead to take on the appearance of another. Many possible lead misplacements can occur, but the most common mistake is switching the electrodes of the thoracic limbs, which will cause negative P waves in lead I and inverted leads II and III. This is the result of lead II becoming lead III, and vice-versa (Figure 10.7). Information about other causes of ECG artifacts is found in Chapter 11.

Table 10.3 One can attempt to determine the location or direction of the source of ECG interference by noting which lead(s) is/are most affected by that interference. This guide may help the operator identify and eliminate the underlying cause of interference.

Interference observed in leads	Direction of source of interference
1 and 2	Patient's right thoracic limb
1 and 3	Patient's left thoracic limb
2 and 3	Patient's left pelvic limb

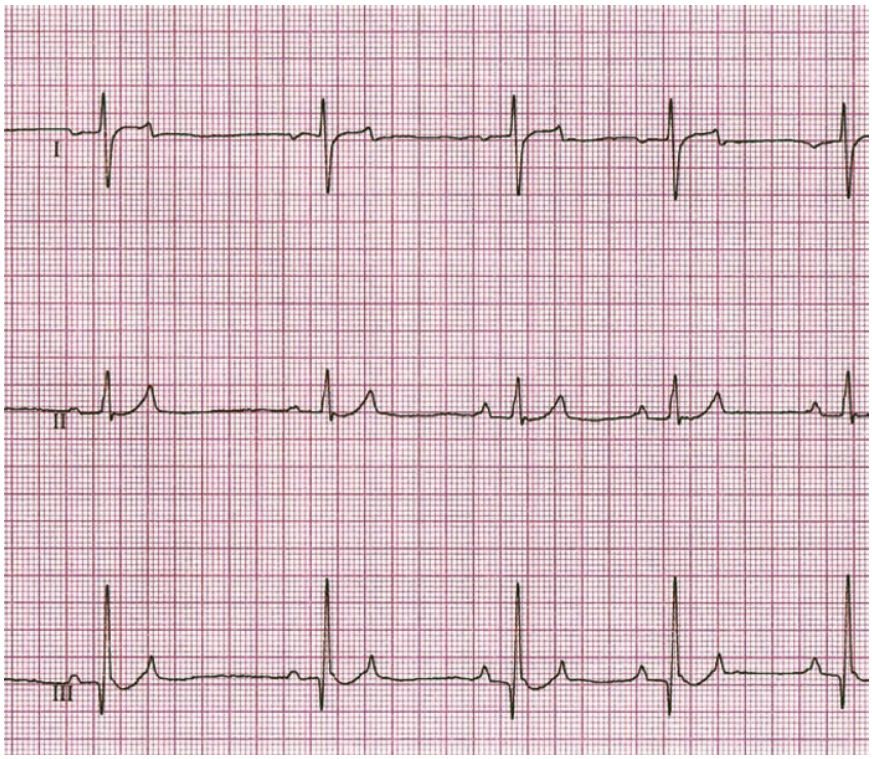


Figure 10.7 Electrocardiogram performed with electrodes placed incorrectly. Top tracing is "lead I"; middle tracing is "lead II"; and bottom tracing is "lead III." The electrodes of the thoracic limbs were switched (the black electrode is on the right and the white on the left thoracic limb). Therefore, the P waves are negative in "lead I" and the readings for leads "II" and "III" are switched.

Protocol 10.1 Electrocardiogram Setup and Monitoring in the Emergency Room and Intensive Care Unit

Items Required

- Electrocardiograph (ECG monitor) with recording paper and appropriate electrode cables
- Clippers
- Electrocardiographic conducting gel or isopropyl alcohol
- Alligator clips (for brief recordings) or adhesive patches (for brief or prolonged recording)
- Adhesive spray (tincture of benzoin spray) if applying electrode patches to skin
- Medical tape if applying electrode patches to paw pads

Procedure

1) Determine whether the patient is a likely defibrillation candidate, and if so, use only conducting gel and not isopropyl alcohol for electrode contact.
2) Decide the patient's positioning and mobility during ECG recording and plan electrode placement accordingly.
3) Connect ECG cables to monitor and ensure cables reach intended patient location.
4) For longer-term recording, adhesive patches work best on the trunk for mobile animals: axillae for white/black electrodes, inguinal regions or caudolateral flanks for red/green electrodes.
   a) Clip the selected sites of fur. Clean away debris at intended electrode site with a swipe of isopropyl alcohol.
   b) Spray with adhesive and allow to dry partially so that the skin feels "tacky" prior to applying electrode patches. Best removed with skin-safe adhesive solvent.
5) For ECG recording in non-ambulatory animals, paw pads work well as electrode sites and do not require fur clipping. Clean away debris on paw pads with a swipe of isopropyl alcohol.
6) Allow alcohol to dry completely, then apply adhesive pad directly to animal's skin. You may consider additional adhesive spray to optimize electrode adhesion.
7) Attach cables to buttons on electrode patches.
8) Turn on monitor to observe ECG tracing.
9) Select lead, screen/paper speed, and sensitivity setting.
10) Observe ECG tracing and record findings in standardized format for your unit.

Acknowledgments

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11

Electrocardiogram Interpretation

Casey J. Kohen

Introduction

Electrocardiography is a valuable diagnostic and monitoring tool in veterinary emergency and critical care (ECC). It provides continuous, real-time information about the cardiovascular and autonomic nervous systems noninvasively. There are few other tools that provide the quantity and quality of clinical information that electrocardiography can provide in such a cost-effective way. Electrocardiography is the gold standard for the detection and classification of arrhythmias and for the assessment of treatment responses. The ECC technician plays a vital role in electrocardiograph use, both in acquiring data and in monitoring and screening for arrhythmias.

This chapter focuses on how to acquire a diagnostic electrocardiogram (ECG) and on specific skills that veterinary ECC staff should strive to master. A comprehensive discussion of ECG interpretation and all possible arrhythmias is beyond the scope of this chapter and interested readers are directed to several excellent texts on those topics (see also Chapter 10).

Acquiring an Electrocardiogram

Electrocardiography is used to display or record the heart's electrical activity. Electrocardiography performed in the emergency room (ER) or intensive care unit (ICU) is "surface" electrocardiography – it measures changes in the overall electrical potential of the myocardium from electrodes placed on the body's surface. This technique can be used to assess cardiac chamber size and conduction pathway function, as well as to monitor heart rate and rhythm. Continuous rate and rhythm monitoring is a common indication for electrocardiography in the ER and ICU. With the widespread availability of radiography and echocardiography, assessment of chamber size based solely on electrocardiography is rarely required.

Electrocardiography can be used as either a diagnostic or a monitoring tool. The proper use of electrocardiography depends on which of those roles the ECG is meant to serve. To obtain a diagnostic ECG, which allows determination of complex amplitude, complex or interval duration, and mean electrical axis for comparison with normal values, the operator must apply standardized methodology (see below). These more rigorous standards are not generally applied when ECG is used as a continuous monitoring tool.

The Diagnostic Electrocardiogram

Patient Positioning

For short-term recording of a diagnostic ECG, it is standard to position the patient in right lateral recumbency. This position is used by convention to assess multiple leads, measure amplitudes of specific wave deflections, and calculate the mean electrical axis. If the animal is to be placed on a metal table or other conducting surface, a blanket or rubber pad should be placed between the patient and that surface to avoid conduction interference and artifacts.

Options and Proper Placement of Electrodes

There are several methods of connecting ECG electrodes to a patient. Alligator clips are a common method for short-term recording and require very little patient preparation. To minimize patient discomfort, the teeth on the alligator clips should be flattened or filed and recording time should be limited. The clips should be placed on the caudal aspect of each elbow near the olecranon, and on each stifle at the level of the patellar ligament. Some ECG machines provide only three electrodes for connection to the patient. The white electrode should be placed at the right elbow, black (or brown) at the left elbow, and red at the left stifle. If a

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fourth (green) electrode is present, it should be placed on the right stifle (see Chapter 10, Figure 10.4). Conducting medium should be placed between the electrode and the patient's skin. A gel designed for ECG electrodes is preferred because these gels are formulated for high conductance to reduce skin resistance and they are generally hypoallergenic. Alcohol and many quaternary ammonia compounds are flammable substances and should not be applied if defibrillation may be required, for example during cardiopulmonary resuscitation.

Adhesive pads are also available and can be applied for longer periods if ECG monitoring is planned to follow the diagnostic ECG. Electrodes are available with snap adapters to connect to the pads. If pads are used, the patient's fur must be clipped, and the skin cleaned and dried to maximize adhesion. The adhesive pads can be placed so that two are on opposite sides of the thorax just caudal to the scapulae (but cranial to the heart), and the third and fourth are placed in the inguinal regions (Figure 11.1). The electrodes are connected in the orientation described previously (i.e. right thorax/axilla, white; right inguinal, green; left thorax/axilla, black or brown; left inguinal, red).

If the waveform is difficult to interpret due to a patient's breathing, the cranial electrodes can be moved more distally to the thoracic limbs; likewise, the caudal electrodes can be moved to the pelvic limbs. Once the electrodes are connected to the patient, the ECG waveform is assessed for quality and absence of artifacts (see below). Although any lead can provide information on heart rate and rhythm, lead II is conventionally used to determine rate and rhythm and to measure waveform amplitude, as well as waveform and interval duration. The determination of mean electrical axis (although rarely done in the ER setting) requires that at least two leads be recorded for analysis.

Recording

A diagnostic ECG should include brief recordings of the three standard leads (I, II, and III) as well as the three augmented leads (aVR, aVL, and aVF) at 25 mm/second paper speed. In addition, 1–2 minutes of lead II at 50 mm/second should be recorded to allow for rhythm and rate analysis. The ECG should be evaluated for the presence of artifacts (see below) and measures taken to remove any artifact noted. See Chapter 10 for more information about leads and diagnostic ECG acquisition.

Continuous Monitoring

Commonly in the ER and ICU setting, the electrocardiograph is used for longer-term monitoring, and maintaining the patient in a standard position (i.e. right lateral recumbency) is not feasible. Emergency and ICU patients are monitored primarily for changes in heart rate and rhythm, in which case specific positioning is less important. In this setting, the lead that produces the most readily identifiable complexes is used. The optimal lead often changes as the patient's position changes.

For continuous monitoring, adhesive pad electrodes offer substantial advantages over alligator clips (see above). If the patient's fur cannot be clipped or the patient requires ECG monitoring only temporarily during anesthesia, the adhesive pads can be applied to the metacarpal and metatarsal pads (Figure 11.1). Tape can be applied circumferentially to better secure the pads and electrodes. Some pads can adhere tightly to the skin and care should be taken when removing them, using adhesive remover as needed to prevent skin irritation and trauma. In patients with ventricular arrhythmias where there is concern for progression to ventricular fibrillation, the placement of larger electrode pads that can be used for defibrillation is advisable (Figure 11.2) [1]. These pads may also be used for external cardiac pacing in patients with complete heart block or who are symptomatic for other bradyarrhythmias (see Chapter 23 for more details) [2]. If the patient is mobile, the electrode wires can be gathered and secured to the patient with a stockinette fitted over the thorax to avoid tangling. Regardless of the electrode



Figure 11.1 (a) Adhesive pads and snap electrodes for long-term monitoring. (b) Placement of adhesive pads on cranial thorax and inguinal region. (c) Alternative placement on paw pads.



Figure 11.2 Using pacing-capable pads for ECG monitoring.

placement method used, after placement the ECG tracing should be inspected for artifacts and measures taken to reduce them.

Continuous ECG monitoring provides both auditory and visual data. Most modern monitors are capable of producing an audible signal synchronized to the QRS complex. This feature allows for qualitative monitoring of rate and rhythm and can alert clinic staff to substantial alterations. When auditory changes are noted, it should prompt a visual inspection of the ECG display. Abnormalities noted after visual inspection should be recorded for analysis and documentation if possible. While recordings made at 25 mm/second allow for more complexes to be recorded on a given length of paper, the authors recommend 50 mm/second recordings since they are generally easier to interpret in small animals.

Electrocardiogram Waveforms

As noted in Chapter 10, the process of myocardial depolarization and repolarization (the re-establishment of resting membrane potential) leads to a series of deflections that are generally recognizable when displayed in sequence over time. Each portion of the normal ECG waveform is associated with a specific portion of the myocardial depolarization–repolarization cycle (Figure 11.3).

In sinus rhythm, in which the electrical impulse begins in the sinoatrial node (also called the SA node or sinus node), the *P wave* is the first deflection noted and reflects depolarization of both atria. As an impulse emerges from the area of the SA node, atrial depolarization begins and progresses from the right atrial myocardium to the left. The direction of this wave of depolarization is such that a small positive deflection lasting less than 0.05 second is observed on lead II. The *P–R interval* is measured from the

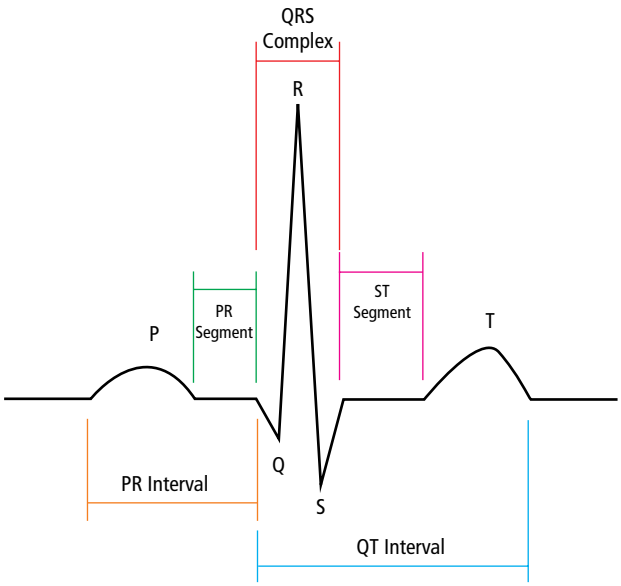


Figure 11.3 ECG waves as seen in lead II.

beginning of the P wave to the beginning of the QRS complex and represents the time required for the electrical impulse to travel from the SA node to the ventricles. A substantial portion of this time is taken up by conduction through the atrioventricular (AV) node. Slower AV node conduction (e.g. when vagal tone is increased) results in a prolonged P–R interval [3, 4]. When the AV node is bypassed (e.g. during ventricular pre-excitation) the P–R interval is substantially shortened and the atria and ventricles depolarize nearly simultaneously; in this case, the P wave and QRS complex are closer together or begin to merge [5]. A P wave with increased duration or increased amplitude may indicate left or right atrial enlargement, respectively [6].

The *QRS complex* is produced as a result of ventricular depolarization. The typical appearance of the QRS is due to the sequential depolarization of different portions of the ventricles such that the wave of depolarization is moving away from a given lead at times (e.g. negative Q and S deflections on lead II) and toward it at others (e.g. positive R deflection on lead II). The duration of the QRS complex indicates how long ventricular depolarization takes to occur. Ventricular depolarization typically occurs quite rapidly (< 0.06 second) due to the presence of specialized conducting tissue (the bundle of His and its branches; see Chapter 10, Figure 10.1) that rapidly conducts the signal to depolarize and distributes it throughout the ventricles. Prolongation of the QRS complex can indicate a conduction disturbance (e.g. bundle branch block, BBB), ectopic origin of the complex (e.g. a ventricular premature complex, VPC), ventricular hypertrophy, or some combination thereof [5]. Increases in the amplitude and duration of

the QRS can be seen with ventricular hypertrophy in some cases [7].

The *ST segment* is a period of relative electrical inactivity. In the healthy myocardium, the ST segment is electrically silent and no differences in potential are detected on any lead. However, in the injured and/or ischemic myocardium an "injury current" may occur between adjacent diseased and healthy sections of myocardium (although more complex pathophysiology likely occurs) [8]. On the ECG this injury current manifests as either elevation or depression of the ST segment compared with baseline (see ECG Skill Set 9 later in this chapter). ST segment alterations can be important indicators of myocardial injury and should prompt further investigation.

The *QT interval* is measured from the beginning of the QRS to the end of the T wave. It represents the combined duration of the depolarization and repolarization processes for the whole ventricular myocardium. Although QT intervals normally vary inversely with heart rate (i.e. longer QT interval when heart rate slows), they are typically less than 0.25 second in duration at normal canine heart rates. Hypercalcemia can result in shortening of the QT interval; hypocalcemia results in prolongation of the QT interval. Hypomagnesemia and hypokalemia can also result in QT interval prolongation. Despite the associations described above, there is minimal veterinary evidence to support the clinical significance of electrolyte effects on QT intervals in dogs and cats.

The *T wave* is associated with ventricular repolarization and the re-establishment of resting membrane potential. T wave conformation is highly variable in populations of normal, healthy dogs and may be positive, negative, or biphasic [9]. However, in an individual patient the appearance of their T wave is generally consistent. An abrupt alteration in the appearance of a patient's T wave should prompt evaluation of serum electrolytes, particularly potassium, and assessment for hypoxemia.

Stepwise Interpretation of the Electrocardiogram

When interpreting electrocardiographic data, it can help to take a stepwise approach and consistently apply it in the same manner every time. While the steps may be taken in any order, there is some benefit to following a specific sequence such that the largest number of rule-outs is removed with each step. For example, determining heart rate first has the advantage of excluding many possible rhythms and allows one to focus on the remaining possibilities. One such stepwise approach used is shown in Protocol 11.1 and further details of each step are provided here.

Determine the Heart Rate

Instantaneous heart rate may be determined by measuring the time between successive P waves or QRS complexes. Mean heart rate may be calculated by determining the number of cardiac cycles over a given length of time (e.g. 3–6 seconds) and multiplying that number to give an average per minute. Arrhythmias involving AV block or ectopic complexes and rhythms may create an overall rhythm in which the atrial heart rate and the ventricular heart rate are not the same. In such cases, it is recommended that both an atrial rate (based on P wave frequency) and a ventricular rate (based on QRS frequency) be determined separately. An example of a situation in which this approach may prove beneficial is given in ECG Skill Set 2 at the end of this chapter.

Evaluate the Overall Rhythm

Next, one may attempt to evaluate the overall cardiac rhythm by inspecting the entirety of the recorded study. Are there specific complexes appearing at unexpected intervals or with P or QRS morphology that differs from the rest? This may indicate ectopic activity such as atrial premature complexes (APCs) or VPCs. The rhythm should be evaluated for whether it is regular or irregular. If the R–R intervals (distance between two adjacent R waves) are evenly spaced throughout the recorded strip, the rhythm is considered regular; if the spacing is variable, the rhythm is termed irregular. However, there may at times be a *pattern* to the irregularity; this is a "regularly irregular rhythm." An example of a regularly irregular rhythm is respiratory sinus arrhythmia wherein the heart rate varies with the phases of respiration (faster during inspiration and slower during expiration). In contrast, an "irregularly irregular" rhythm is one for which there is no discernible pattern to the irregularity. Atrial fibrillation is a classic example of an irregularly irregular rhythm.

Evaluate the Complexes and Intervals

Next, identify the complexes and the intervals, and determine their amplitude and/or duration. Prolongation of a given parameter indicates that whatever process that parameter represents is taking longer than normal. An example is a prolonged P–R interval. The P–R interval is the length of time it takes a signal to travel from the sinus node to the ventricle. The bulk of that time is spent traveling through the AV node, so a prolonged P–R interval indicates slowed AV node conduction (termed first degree AV block). Increased amplitude of a complex may also carry important information (e.g. increased P wave amplitude indicating right atrial enlargement), but this information is generally more heavily scrutinized during a multilead

Protocol 11.1 Stepwise Approach to Electrocardiogram Interpretation

- See accompanying text (Stepwise Interpretation of the Electrocardiogram) for details of each step.

Procedure

1) Determine the heart rate(s):
   a) Atrial rate (frequency of the P waves)
   b) Ventricular rate (frequency of the QRS complexes)
   c) Are they the same?
2) Evaluate the overall cardiac rhythm:
   a) Is the rhythm regular or irregular?
      i) If irregular, is the rhythm regularly irregular or irregularly irregular?
      ii) Are there specific complexes appearing at unexpected intervals or with QRS morphology that is different than the rest?
3) Identify the complexes and the intervals; determine their amplitude and/or duration:
   a) P wave
   b) P–R interval
   c) QRS complex
   d) Assess morphology of the Q, R, and S deflections.
   e) QT interval
   f) T wave
4) Inspect the ST segment for evidence of elevation or depression
5) Compare the amplitudes, durations, and morphology with normal values (Table 11.1) for this species as well as previous values for this individual (if available).

Table 11.1 Normal canine and feline electrocardiogram values [10].

	Canine	Feline
Heart rate	Puppy: 70–220 beats/minute	120–240 beats/minute
	Toy breeds: 70–180 beats/minute	
	Standard: 70–160 beats/minute	
	Giant breeds: 60–140 beats/minute	
Rhythm	Sinus rhythm	Sinus rhythm
	Sinus arrhythmia	
	Wandering pacemaker	
P wave:		
Amplitude	Maximum: 0.4 mV	Maximum: 0.2 mV
Duration	Maximum: 0.04 second (Giant breeds 0.05 sec)	Maximum: 0.04 second
PR interval	0.06–0.13 second	0.05–0.09 second
QRS:		
Amplitude	Small breeds: 2.5 mV	Maximum: 0.9 mV
Duration	Large breeds: 3 mV	Maximum: 0.04 second
	Small breeds: 0.05 second maximum	
	Large breeds: 0.06 second maximum	
ST segment:		
Depression	< 0.2 mV	None
Elevation	< 0.15 second	None
QT interval	0.15–0.25 second at normal heart rate	0.12–0.18 sec at normal heart rate
T wave	May be positive, negative, or biphasic	Typically positive
	≤ 25% of R wave amplitude	Maximum 0.3 mV amplitude

diagnostic ECG study than when electrocardiography is being used as a continuous monitoring tool. Decreased amplitude may occur in a number of settings. Those most relevant to the ECC setting include pleural space filling disorders and pericardial effusion [11].

The normal QT interval varies inversely with heart rate but may be altered pathologically in electrolyte disturbances (most often those involving alterations in calcium or potassium). Nomograms relating QT intervals and heart rates are available but are seldom used [12].

The T wave arises from ventricular repolarization. The process of repolarization is a reflection of how depolarization occurred. If depolarization occurs slowly and atypically, then repolarization will occur abnormally as well. In clinical practice, this phenomenon is most often manifested in the wide and prominent T waves associated with VPCs. Electrolyte disturbances may also alter T wave morphology as evidenced by the tall, peaked T waves seen in hyperkalemia [13]. It should be noted that there is a vast array of T wave morphologies observed in normal patients [9]. T waves may be positive, negative, or biphasic in normal animals [9]. What is important to note is when a patient's T wave morphology changes relative to what had been observed in that same patient previously, as the change may herald the development of myocardial injury [14].

The ST segment should be inspected for evidence of elevation or depression. The significance of these findings is explained further in ECG Skill Set 9 at the end of this chapter.

Compare Measurements with Normal Ranges

Lastly, it is important to note that one should compare measurements with established normal values for this species and with any previous recordings made from this patient. For example, has the QRS amplitude increased relative to the last visit, suggesting progressive left ventricular hypertrophy? Or is this elevation in heart rate something that has been observed every time this patient visits your clinic, suggesting white coat syndrome? [15]

In all cases, abnormalities in rate, morphology, rhythm, or interval durations should be noted in the patient record and brought to the attention of the clinician on duty so that a treatment or monitoring plan can be constructed in a timely fashion.

Recognizing Artifacts

Electrocardiography is prone to several common artifacts that can interfere with interpretation including respiratory artifact, poor-contact artifact, 60-cycle interference, muscle activity artifact, and stylus temperature artifact.

Respiratory artifact is due to electrode motion during exhalation and inhalation. It results in a baseline that cyclically rises and falls rather than remaining stable (Figure 11.4a). Moving the cranial electrodes from the chest wall to the thoracic limbs usually reduces or eliminates this artifact. Poor-contact artifact results in the loss of recognizable complexes and coarse, ultra-high-frequency oscillations (Figure 11.4b). Using contact gel or applying tape to secure electrode pads more firmly can reduce poor-contact artifact.

In most parts of the world, alternating current is used to power devices plugged into outlets. This current alternates polarity (direction) 60 times each second. This alternating current results in an electrical field that can be picked up by ECG equipment. This type of artifact is termed 60-cycle interference and produces a baseline with fine, persistent oscillations (Figure 11.4c); 60-cycle interference can be reduced by making sure that ECG equipment is plugged into a properly grounded outlet and that other devices plugged into this circuit are turned off or unplugged (if possible). Clippers and fluorescent lighting are common sources of 60-cycle interference. Moving the patient and equipment away from walls containing electrical wiring may help reduce 60-cycle interference. Table 10.3 in Chapter 10 provides guidance for locating the source of interference.

Muscle activity can also produce an unstable, oscillating baseline that limits one's ability to interpret the ECG (Figure 11.4d). Muscle tremors and shivering commonly produce muscle activity artifact, as can purring in cats. Removing muscle activity artifact often requires that one address the underlying cause of the muscle activity (e.g. warm a patient that is shivering) or move the electrodes to a different location, or both.

Most ECGs are recorded on thermal paper that darkens when heat is applied to it. The heated stylus moves up and down while the paper is advanced at a predetermined speed (e.g. 50 mm/second). Stylus temperature should be adjusted to provide a tracing with optimal clarity. A stylus that is set at too low a temperature will yield a very faint tracing while an overheated stylus produces a tracing that is overly wide and dark (Figure 11.4e). Either type of artifact can hamper interpretation.

Summary

Electrocardiography is one of the most valuable monitoring tools available to ECC staff. It provides continuous, real-time information regarding cardiovascular status and autonomic tone and defines the nature of cardiac arrest. The ECC technician plays a vital role as a front-line interpreter of ECG data. Ten essential skills for the ECC

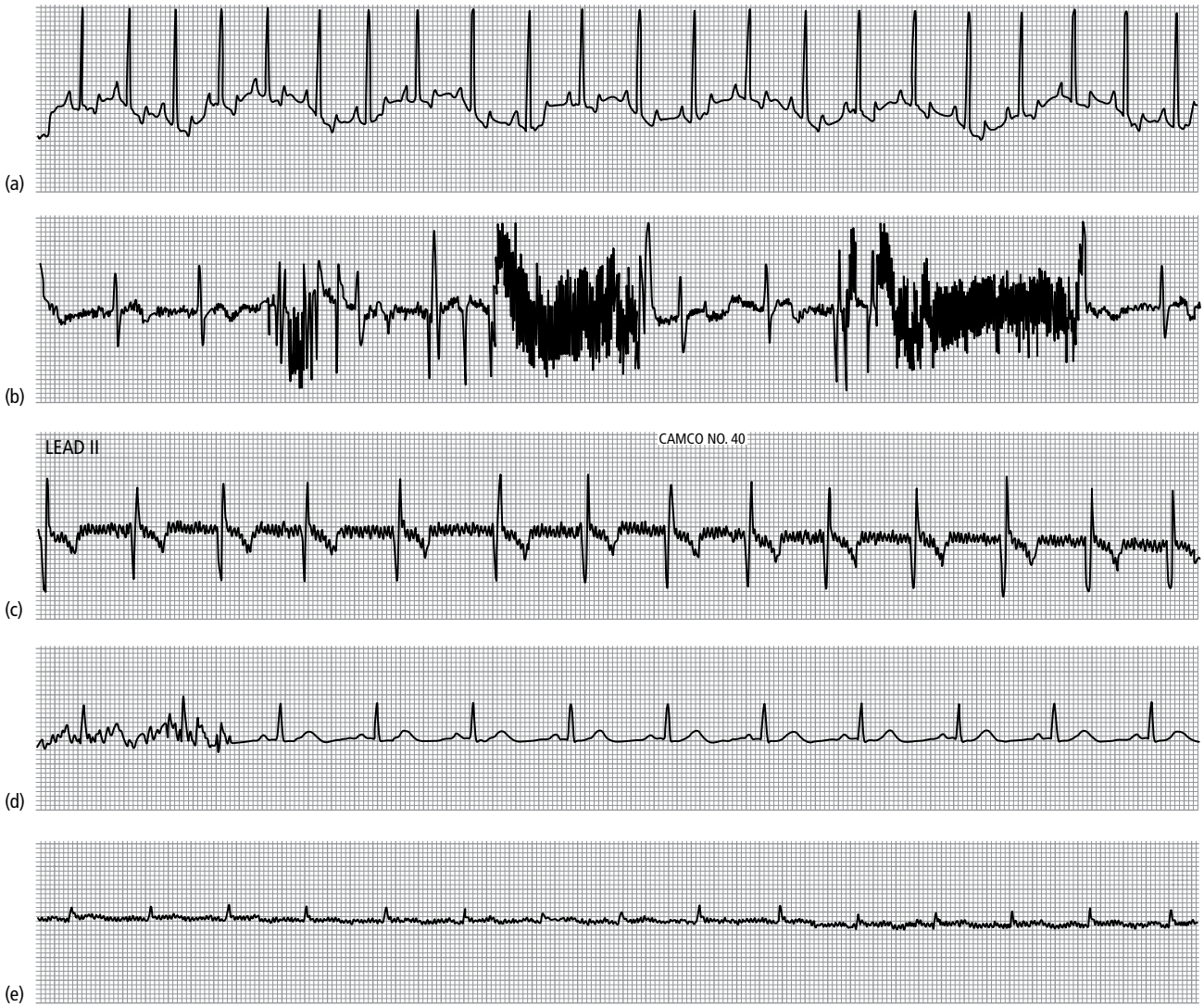


Figure 11.4 ECG recordings showing different types of artifact: (a) respiratory motion artifact; (b) poor lead contact artifact; (c) 60-cycle interference, lead II ECG at 50 mm/second; (d) probable muscle-tremor artifact; (e) excessive stylus heat artifact.

technician to master are discussed in the following "Skill sets" portion of this chapter.

Skill Sets

Skill Set 1: Distinguishing 60-Cycle Interference from Atrial Fibrillation or Atrial Flutter

The ICU and ER environments are frequently busy, crowded places with a great deal of equipment. The pace of clinical practice and the requisite instrumentation frequently lead to poor-quality ECG recordings. One common cause of poor-quality ECG recordings is 60-cycle interference as described above and demonstrated in the first ECG (Figure 11.5a). Note the extreme rapidity with which the baseline oscillates. A key difference between atrial flutter and 60-cycle interference is the rate of the oscillations. The repetitive depolarization of the atrial myocardium that occurs in atrial flutter is much slower than the 3600 oscillations/minute found with 60-cycle interference (60-cycle or 60 Hz = 60 oscillations/second, which is 3600/minute).

Atrial fibrillation and atrial flutter can result in a baseline with an undulating or oscillating appearance. There are, however, key features that allow the technician to distinguish these arrhythmias from 60-cycle interference. Atrial fibrillation is demonstrated in the second ECG (Figure 11.5b). Atrial fibrillation results in uncoordinated atrial electrical activity and thus the baseline undulations are irregular and do not follow a repetitive pattern as seen in 60-cycle interference. An example of atrial flutter is shown in the third ECG (Figure 11.5c). One may note that this arrhythmia does produce a baseline with a repetitive,

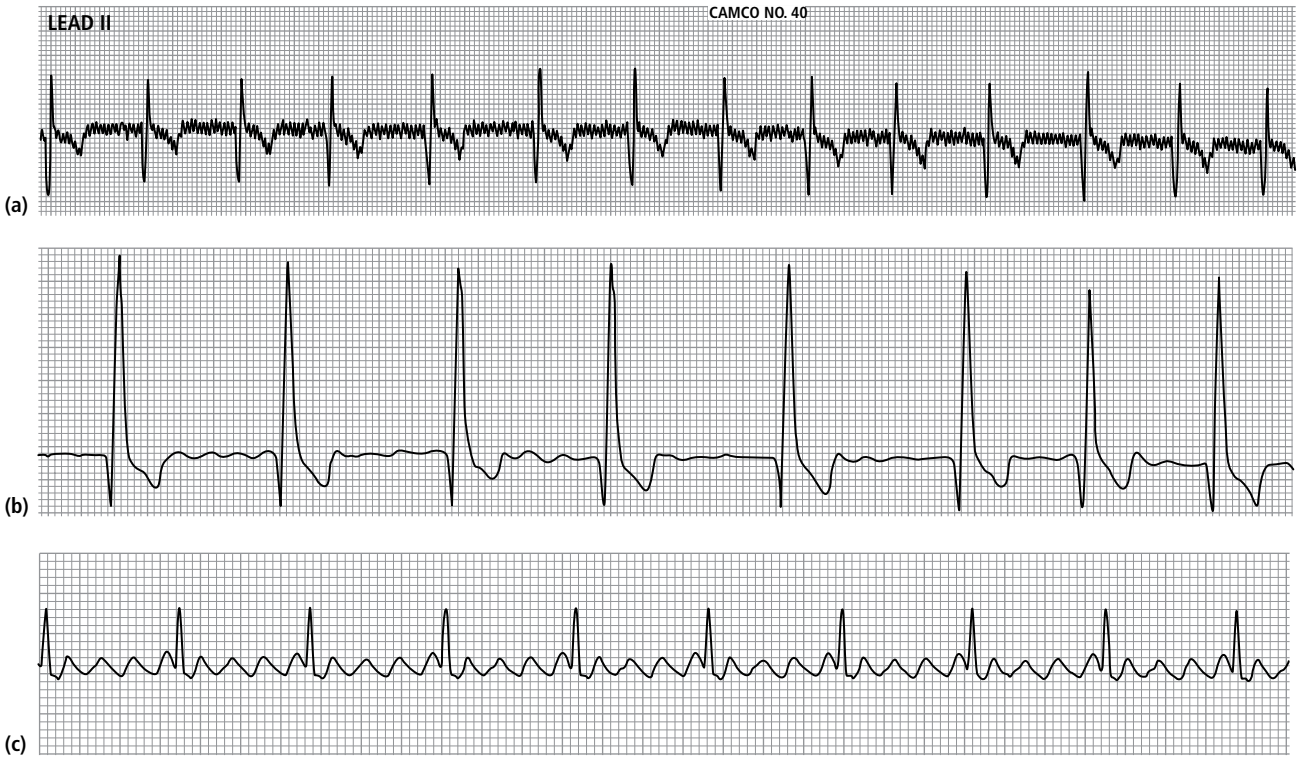


Figure 11.5 (a) Sixty-cycle interference, lead II ECG at 50 mm/second. (b) Atrial fibrillation, lead II ECG at 50 mm/second. (c) Atrial flutter, lead II ECG at 50 mm/second.

oscillating appearance, just slower and with a less uniform morphology than 60-cycle interference. Also note that with both atrial fibrillation and atrial flutter the R-R interval typically is irregularly irregular, which is not expected when 60-cycle interference is superimposed on a sinus rhythm.

Skill Set 2: Distinguishing Third-Degree Atrioventricular Block from Atrioventricular Dissociation

Disorders of conduction through the AV node can result in many different types of arrhythmia, including many forms of AV block. Many of these are easily distinguished from one another based on the nature of the P–R interval and its relation to the QRS complex. However, a complete lack of AV node conduction can result from many causes, including AV node pathology, medications, excessive vagal tone (though this typically results in less severe AV conduction disturbances), or physiologic refractoriness of the AV node due to non-sinus node pacemaker activity. The AV junction contains cells capable of intrinsic pacemaker activity similar to the sinus node cells. However, the activity of the AV junction site is usually suppressed (overridden) by the higher intrinsic rate of the sinus node. On occasion, the discharge rate of the AV junctional pacemaker site can become increased to the point that its rate is nearly equal to that of the sinus node. When this happens, AV dissociation may occur. AV dissociation is a class of arrhythmia wherein the atria and ventricles are controlled by separate, independent pacemakers resulting in two different rhythms. On a surface ECG, these two independent rhythms are superimposed on one another and may appear to represent a single disorganized rhythm. When the rate of the AV junctional pacemaker site is close to that of the sinus node, the sinus node may be unable to pace the ventricles, as the AV node is refractory to conduction because it has already been depolarized by the ectopic pacemaker site. This represents a form of *functional* AV block. No AV node pathology is required for this type of conduction disorder to occur.

The first ECG (Figure 11.6a) is an example of complete heart block (also known as third-degree AV block). The atria are being depolarized by the sinus node and thus regular P waves are noted. The QRS complex is wide, indicating that ventricular depolarization is atypically prolonged. In third-degree AV block, the QRS complexes are the result of a ventricular escape rhythm, and ventricular depolarization frequency (and thus heart rate) is typically slower than normal. This form of AV dissociation is most commonly due to pathology of the AV nodal tissue.

The second ECG (Figure 11.6b) is an example of isorhythmic AV dissociation, which is a category of arrhythmia rather than a specific rhythm diagnosis, much like

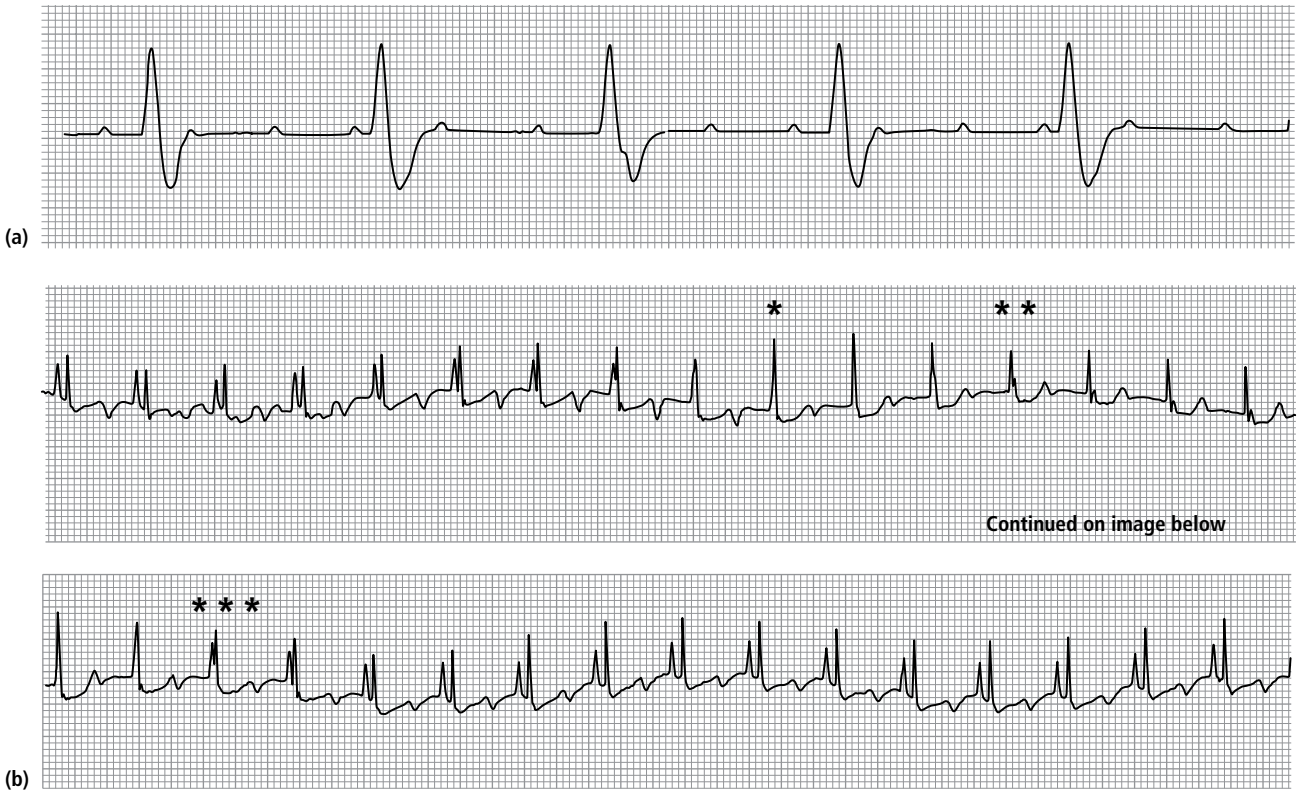


Figure 11.6 (a) Third-degree atrioventricular block, lead II ECG at 50 mm/second (top tracing). (b) An example of isorhythmic dissociation, lead II ECG at 25 mm/second (middle and bottom tracings).

tachycardia is a category not a specific rhythm diagnosis. In the case example provided, one should note that at the beginning of the strip, P waves and QRS complexes are distinct and appear as expected. However, in the center of the upper portion of Figure 11.6b one can see the P waves and QRS complexes merging (*) and summating (QRS amplitude appears increased). The P waves then begin to reappear in the downstroke of the R wave (**) and then ultimately reappear just to the left of the QRS complexes where they are usually found (***). In this case, there are two independent rhythms present: a sinus rhythm depolarizing the atria and an accelerated junctional rhythm depolarizing the ventricles. An important distinguishing factor between the two ECGs (Figure 11.6a,b) is that in third-degree AV block there are many more P waves than QRS complexes, whereas in isorhythmic dissociation rhythms there are typically a few more QRS complexes than P waves. Also, in isorhythmic dissociation rhythms the most common source of ectopic pacemaker activity is the AV junction, and the QRS complexes are thus most often narrow and normal in appearance. Third-degree AV block is typically a significant clinical problem whereas isorhythmic dissociation rhythms may be an incidental finding with fewer adverse effects on cardiovascular performance.

Skill Set 3: Distinguishing APCs from Ventricular Premature Complexes

Ectopic depolarizations (often called premature complexes) may arise from supraventricular or ventricular foci. They represent paroxysmal depolarizations arising from sites other than the sinus node. APCs could more correctly be termed *supraventricular ectopic depolarizations*, which more correctly describes them because (a) APCs may arise from ectopic sites other than the atria, such as the AV junction; (b) they are not always particularly premature; and (c) although APCs do result in depolarization, they may not always result in myocardial contraction. *VPCs* (or ventricular ectopic depolarizations) arise from ectopic sites in the ventricles.

Both APCs and VPCs may be identified in small animal patients in the ER and ICU setting. In dogs, APCs are believed to result more often from primary cardiac disease than are VPCs, which frequently occur due to systemic disease and may be identified in normal dogs on occasion. In contrast, APCs are thought to arise most often from underlying myocardial disease and less often from extracardiac problems in dogs.

Since APCs originate from foci at or above the AV junction, their transmission to the ventricle is typically via the

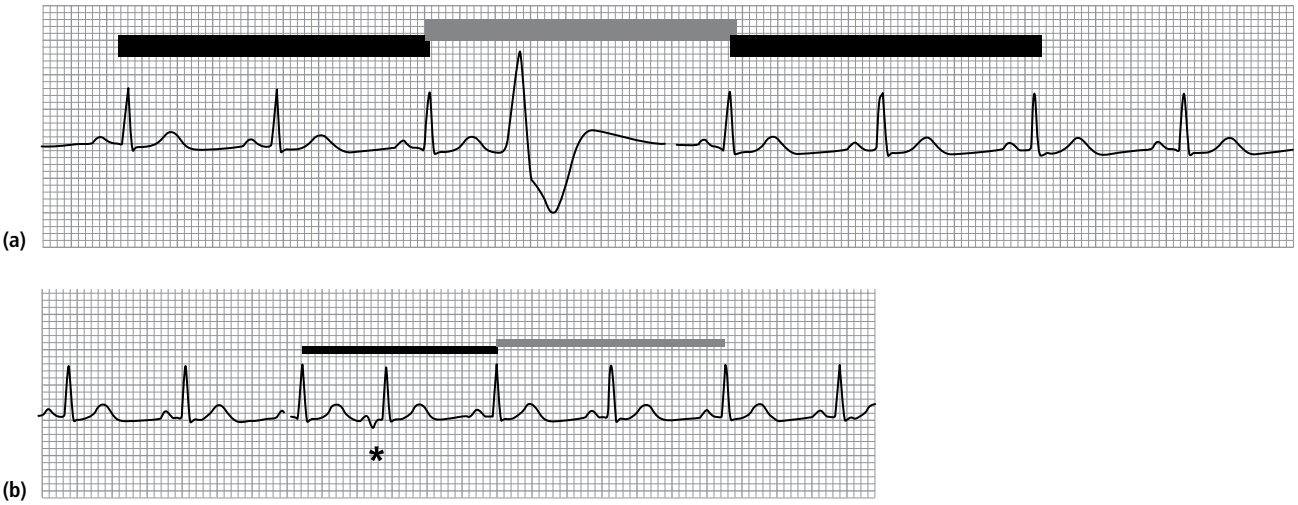


Figure 11.7 (a) VPC, lead II ECG at 50 mm/second; (b) Atrial premature complexes, lead II ECG at 50 mm/second.

Bundle of His and its branches. This normal path of impulse transmission means that ventricular depolarization occurs in the same manner as with a sinus depolarization. Thus, the QRS morphology and duration associated with APCs should be comparable to the QRS complexes observed after P waves (see the second ECG, Figure 11.7b). In contrast, VPCs originate from foci within the ventricles; thus, ventricular depolarization is slower and less organized since the bundle of His does not distribute the impulse as it does with a depolarization that originates from a supraventricular focus. This pattern of depolarization leads to the wide and bizarre morphology typical of VPCs. As repolarization reflects the pattern of depolarization, the T wave is also typically wide and prominent after a VPC. Repolarization of some portions of the ventricle may begin before more distant portions of the ventricle have completed depolarization, leading to slurring of the QRS and T wave together. See the first ECG (Figure 11.7a) for an example of a VPC and note that the ectopic complex both lacks a P wave and has a wide and bizarre morphology.

APCs typically result in atrial depolarization and therefore generate a premature P wave (termed P′ wave) preceding the normal-appearing QRS. An APC with a P′ wave (*) is depicted in Figure 11.7b. P′ waves may have a different morphology than P waves on the same ECG due to different patterns of atrial depolarization. This feature along with the normal QRS morphology is most helpful in distinguishing APCs from VPCs.

APCs and VPCs also differ in that APCs may depolarize and therefore reset the sinus node, whereas VPCs generally do not. When an APC depolarizes and resets the sinus node, that APC is then followed by a "non-compensatory pause" (Figure 11.7b: the gray bar is longer than the black bar) – the complex following the APC occurs after a normal-length R-R interval. Conversely, VPCs are almost always followed by a "compensatory pause," in which the next sinus complex occurs right on schedule (as if the VPC did not occur) because the VPC did not depolarize the SA node (Figure 11.7a: the gray bar is the same length as the black bars). The nature of the pause following an ectopic complex can on occasion assist in proper classification; in cases when the two types of ectopic depolarizations cannot be distinguished readily based on morphology, the differences in the types of pauses that follow them may allow proper characterization.

Skill Set 4: Distinguishing Ventricular Tachycardia from Supraventricular Tachycardia

Distinguishing supraventricular from ventricular tachyarrhythmias accurately is an important skill for anyone performing ECG interpretation. These types of tachyarrhythmias need to be distinguished not only to guide proper antiarrhythmic drug selection, but also because of their different prognoses. Many drug agents that are effective for terminating supraventricular tachyarrhythmias (supraventricular tachycardia, SVT; e.g. calcium channel blockers) are less effective at addressing ventricular tachyarrhythmias. Moreover, rapid ventricular tachyarrhythmias carry greater risk of degenerating into ventricular fibrillation or flutter, which are associated with cardiac arrest. Rapid SVTs also need to be addressed promptly, as they compromise diastolic filling and can result in markedly reduced stroke volumes and poor cardiac output.

SVTs are usually associated with a QRS morphology that is narrow and normal in appearance (unless a conduction disturbance such as BBB is also present). Supraventricular tachycardias include atrial and junctional tachyarrhythmias and this class of arrhythmias is also referred to as narrow QRS tachycardias (this term therefore also includes

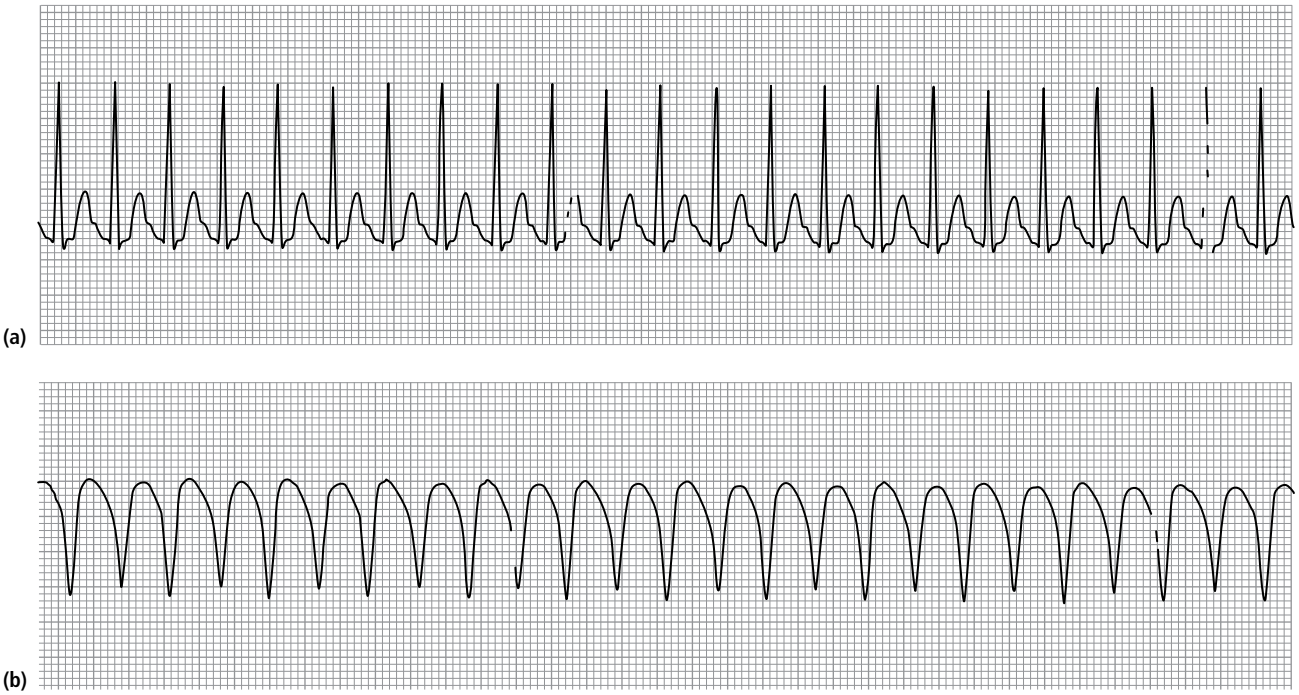


Figure 11.8 (a) Example of a supraventricular tachyarrhythmia, lead II ECG at 25 mm/second; (b) example of a ventricular tachyarrhythmia, lead II ECG at 25 mm/second.

atrial fibrillation and atrial flutter when ventricular rates are rapid). P′ waves (see Skill Set 3) may be identified or may be superimposed or merged with the T wave of the preceding complex (see the first ECG, Figure 11.8a). The heart rate is rapid and often regular, except in the case of atrial fibrillation.

Ventricular tachycardia and ventricular flutter are tachyarrhythmias originating from within the ventricular myocardium. The bundle of His and its branches do not distribute the impulse throughout the myocardium, which results in slower wave propagation and widening of the QRS complex as seen in the second ECG (Figure 11.8b). These tachyarrhythmias may be referred to as wide QRS tachycardias. The occasional P wave may be identified if there is any baseline available for inspection (which is unusual because the QRS rate is very rapid), but they do not have a predictable relationship to the QRS complexes.

The widened appearance of the QRS and the lack of P (or P′) waves is usually sufficient to distinguish SVT from ventricular tachyarrhythmias. However, there are times when the QRS seems only slightly widened and P waves are not identifiable; the classification of tachyarrhythmias becomes more challenging in this setting. Response to a trial IV dose of an antiarrhythmic agent (e.g. lidocaine, diltiazem) and/or response to a vagal maneuver may be required in some cases to accurately identify the rhythm. SVT often respond to vagal maneuvers while ventricular tachyarrhythmias usually do not.

Skill Set 5: Distinguishing Ventricular Premature Complexes from Ventricular Escape Beats

Ventricular ectopic activity is best assessed when considering the context in which it is seen. Ectopic ventricular activity may be pathologic (e.g. VPCs, ventricular tachycardia) or physiologic (e.g. ventricular escape beats, ventricular escape rhythms). VPCs and ventricular tachycardia are discussed in Skill Sets 3 and 4, respectively. Ventricular escape beats are ventricular depolarizations that also arise from a ventricular focus, but under very different circumstances than VPCs. There are cells within the ventricles capable of pacemaker activity at slow rates (20–40 beats/minute), but their activity is usually suppressed (overridden) by the more rapid pacing activity of other sites (i.e. sinus node and/or AV junction). However, when the heart rates initiated by these other sites fall below the intrinsic rate of the ventricular pacing cells, ventricular escape complexes or rhythms should occur. Once the rate of the sinus node or AV junction again exceeds that of the ventricles, the escape complexes will be suppressed once again.

The pacemaking sites in the AV junction and ventricle provide an important "safety net" and likely developed to ensure that a minimum heart rate will be maintained when the sinus node pauses, temporarily arrests, or fails altogether. A similar role is served when AV node conduction disturbances prevent the sinus node's depolarization signal

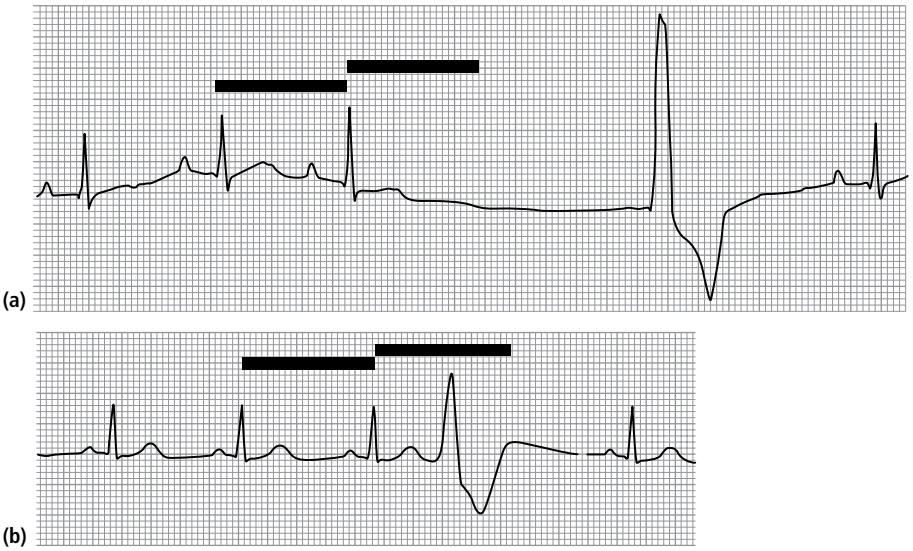


Figure 11.9 (a) Ventricular escape complex (ventricular escape beat), lead II ECG at 50 mm/second; (b) ventricular premature complex, lead II ECG at 50 mm/second.

from reaching the ventricles (e.g. complete or "third-degree" heart block; see Skill Set 2).

The principal feature that distinguishes VPCs from ventricular escape beats is whether the complex occurs before the next QRS complex was expected (VPC) or after a QRS complex would have been expected (escape beat).

In the first ECG (Figure 11.9a), a ventricular escape complex (also called ventricular escape beat) follows the third sinus complex. Once again, the lower black bar indicates the R–R interval between two successive sinus beats. In this case, the wide and bizarre complex lacking a P wave occurs *after* the next sinus complex was expected to occur. Escape beats occur after pauses in sinus node activity as is shown here. Escape beats are important to help maintain adequate cardiac output in the face of inadequate pacemaker activity from the sinus node or disruption of AV node conduction and should not be suppressed with antiarrhythmic therapies.

In the second ECG (Figure 11.9b), a VPC is present following the third sinus complex. The lower black bar indicates the R–R interval between two successive sinus beats. The upper black bar indicates the time interval until the next sinus complex would be expected to occur based on this established R-R interval. The wide and bizarre complex lacking a P wave that occurs before the next sinus complex would be expected (i.e. occurs prematurely) is thus a VPC. This ectopic beat does not reset the sinus node and a compensatory pause follows it (see Skill Set 3). VPCs may be due to cardiac or extracardiac disease in dogs; they often indicate myocardial disease in cats. Frequent or multiform VPCs may require antiarrhythmic therapy.

Skill Set 6: Recognition of Pulseless Electrical Activity

Pulseless electrical activity (PEA) is a rhythm associated with cardiac arrest in small animals. It was previously termed electromechanical dissociation, which is a less correct term, and indicates that myocardial depolarization is not leading to myocardial contraction; although this can occur, there are also situations when depolarization causes contraction but no effective stroke volume is ejected, which is still effectively cardiac arrest. The importance of recognizing the many causes for pulselessness when ECG activity is still present has led to preference of the more general term PEA.

Figure 11.10 shows the simultaneous electrocardiogram (upper tracing) and arterial blood pressure (lower tracing) from a veterinary patient with PEA. The upper tracing demonstrates that repetitive (but not regular) cardiac depolarization (QRS complex, which is widened) and repolarization (T waves) are occurring. The absence of P waves

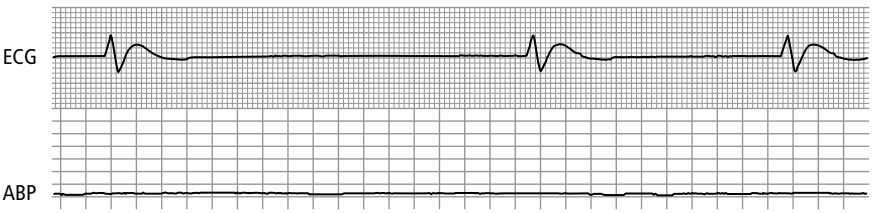


Figure 11.10 Pulseless electrical activity, lead II ECG at 25 mm/second with arterial blood pressure.

suggests atrial electrical activity may be absent, but precordial lead recordings ("chest leads") should be obtained before this conclusion is made. Regardless of the exact rhythm diagnosis, one should note the complete absence of an arterial pulse wave associated with any of the three QRS complexes.

The identification of PEA should prompt the immediate initiation of chest compressions (and other CPR measures) in the unconscious patient (see Chapter 20). The other setting in which PEA is commonly identified is after euthanasia with barbiturate or potassium solutions. Electrical activity often persists for several minutes after meaningful myocardial activity has stopped.

Skill Set 7: Distinguishing Ventricular Tachycardia from Accelerated Idioventricular Rhythm

Ventricular arrhythmias may be due to a number of different mechanisms (e.g. re-entry, enhanced automaticity) and may represent a finding that (a) must be treated (e.g. ventricular flutter); (b) often should be treated (e.g. ventricular tachycardia); (c) may not require treatment (e.g. accelerated idioventricular rhythm [AIVR]); or (d) should not be treated (e.g. ventricular escape rhythms). The decision to treat or not treat a ventricular arrhythmia is often subjective and based on clinical judgment; however, some general guidelines are outlined in Box 11.1.

Excessively rapid heart rates reduce ventricular diastolic filling and can reduce stroke volume and cardiac output while increasing myocardial workload. As such, ventricular tachyarrhythmias with rates greater than 160–180 beats/minute are more likely to require treatment than those of slower rates. Similarly, sustained arrhythmias are more likely to compromise cardiovascular performance than those that are brief and intermittent. Patients whose markers of perfusion are normal often do not require treatment for mild, intermittent arrhythmias. When the ectopic complexes have varying morphology, this may suggest more widespread ventricular myocardial injury and is more likely to prompt the initiation of therapy. In the "R-on-T" phenomenon the QRS complex of one beat occurs before the T wave of the preceding beat has been completed. Ventricular arrhythmias exhibiting R-on-T behavior are at greater risk of deteriorating into ventricular fibrillation (a non-circulating rhythm) and thus warrant therapy. Lastly, all antiarrhythmic agents are inherently pro-arrhythmic so needlessly treating a benign arrhythmia may result in development of a more dangerous arrhythmia.

Box 11.1

Ventricular arrhythmias likely *should* be treated if:

- The rate is rapid (> 160–180 beats/minute).
- The arrhythmia is sustained.
- Cardiovascular performance appears diminished (e.g. poor perfusion parameters, hypotension) as a result of the arrhythmia.
- The ectopic activity has a polymorphic appearance.
- "R-on-T" phenomenon is identified.
- Risk associated with leaving it untreated appears greater than the risks associated with the treatment selected.

The first ECG (Figure 11.11a) shows a simultaneous recording of leads I, II, and III. Paroxysmal ventricular tachycardia is present. The rapid rate, polymorphic appearance, and R-on-T phenomenon all indicate that treatment is warranted.

The second ECG (Figure 11.11b) shows a monomorphic, intermittent ventricular arrhythmia that is termed AIVR. This arrhythmia typically occurs at a rate that is too slow to be classified as a true tachyarrhythmia (as is the case in the example shown). Further, it often causes little impairment in cardiovascular performance and rarely progresses to a more life-threatening form. This arrhythmia is frequently identified in dogs presenting for splenic masses, hemoabdomen, postoperative gastric dilatation-volvulus, or intracranial disease. ECC technicians play an important role in recognizing that this arrhythmia rarely requires treatment other than general supportive care and treatment of the primary problem.

Skill Set 8: Recognition of Ventricular Flutter and Fibrillation

Ventricular flutter and fibrillation represent two of the most immediately life-threatening arrhythmias that the veterinary patient may develop. It is essential that the ECC technician be able to recognize these rhythms. Once it is recognized that ventricular flutter or fibrillation has developed, one should immediately alert the clinician on duty without delay, and chest compressions should be initiated and immediately followed by other CPR measures.

Ventricular flutter (see the first ECG, Figure 11.12a) is a rapid, often regular-appearing rhythm with wide and bizarre QRS complexes that slur into the T waves. The ST segment and other portions of the ECG that would normally appear flat are absent and a sinusoidal appearance to the rhythm is generally noted. Treatment options are similar to those for ventricular tachycardia (i.e. direct current cardioversion, class I antiarrhythmics [e.g. lidocaine], ultrashort-acting class II agents [e.g. esmolol], class III agents [e.g. amiodarone], and/or magnesium sulfate).

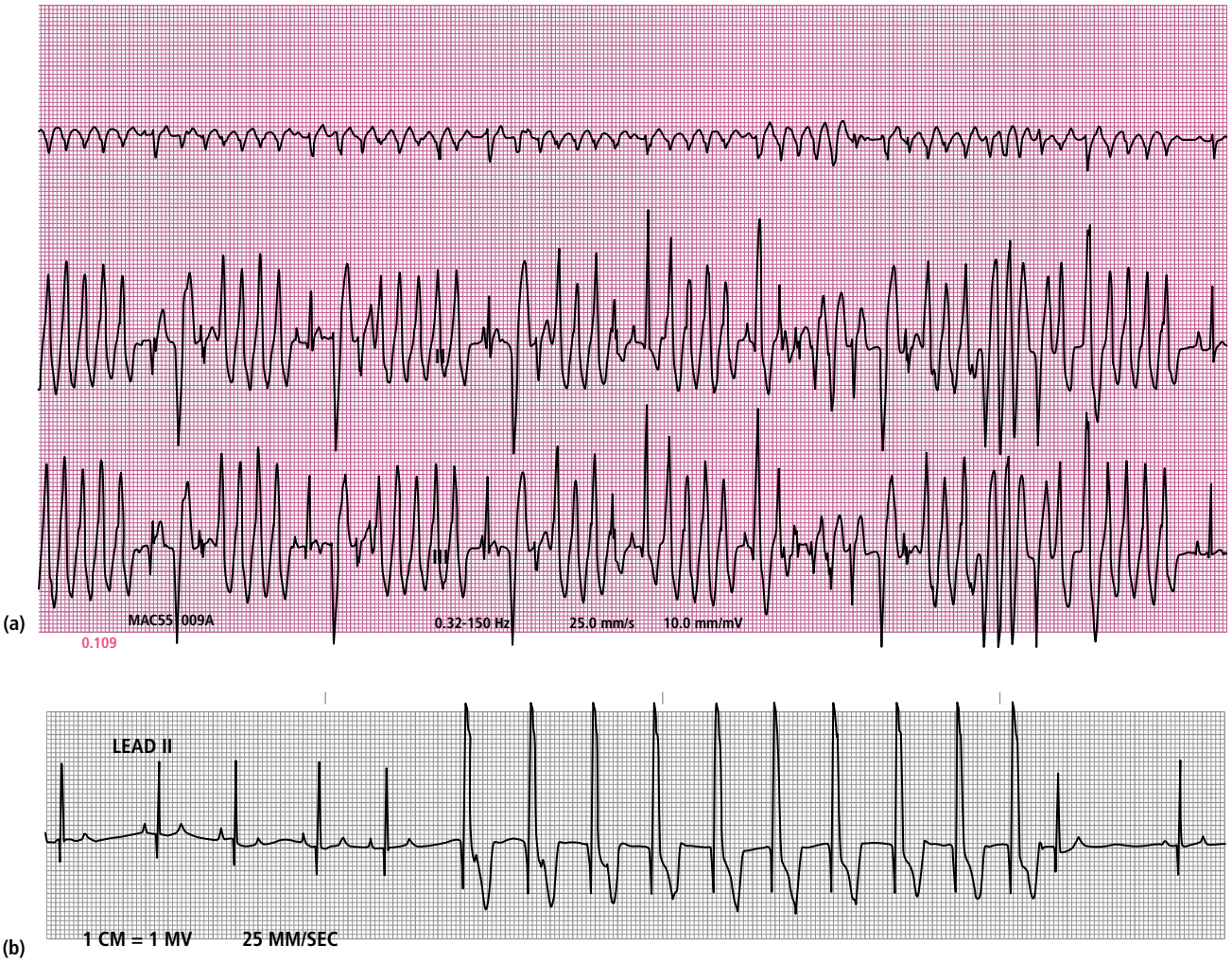


Figure 11.11 (a) An example of ventricular tachycardia, leads I, II, and III ECG at 25 mm/second. (b) Accelerated idioventricular rhythm, lead II ECG at 25 mm/second.

However, in many cases ventricular flutter deteriorates into ventricular fibrillation by the time intravenous antiarrhythmic agents can be administered, and defibrillation equipment should be readied once ventricular flutter is recognized.

Ventricular fibrillation (see the second ECG, Figure 11.12b) represents a chaotic ventricular arrhythmia wherein organized electrical activity of the ventricular myocardium is absent. The lack of organized electrical activity results in the absence of effective myocardial contraction, and cardiac output basically ceases. Ventricular fibrillation is a more common arrest rhythm in people than in veterinary patients, presumably due to the greater human predisposition to myocardial infarction. However, ventricular fibrillation is identified in many cardiac arrest events in dogs and cats. The key features of ventricular fibrillation on an ECG are the lack of recognizable P–QRS–T complexes and the rapid rate coupled with irregular, bizarre waveforms. The example provided in Figure 11.12b is an example of *coarse* ventricular fibrillation. However, it is important to note that *fine* ventricular fibrillation can also occur, in which the waves are quite small in amplitude. Fine ventricular fibrillation may be mistaken for asystole. The only effective therapy for ventricular fibrillation is electrical defibrillation, which should be performed without delay once this arrhythmia is recognized (see Chapter 22). Coarse ventricular fibrillation is thought to respond more readily to electrical defibrillation than fine ventricular fibrillation. Epinephrine may be given prior to defibrillation in an attempt to convert fine ventricular fibrillation to the coarse form before performing defibrillation.

Skill Set 9: Recognition of ST Segment Elevation or Depression

The ST segment on an ECG is typically electrically silent and has a flat appearance. The ST segment encompasses the period of time after ventricular depolarization but prior to the initiation of repolarization. Little or no electrical

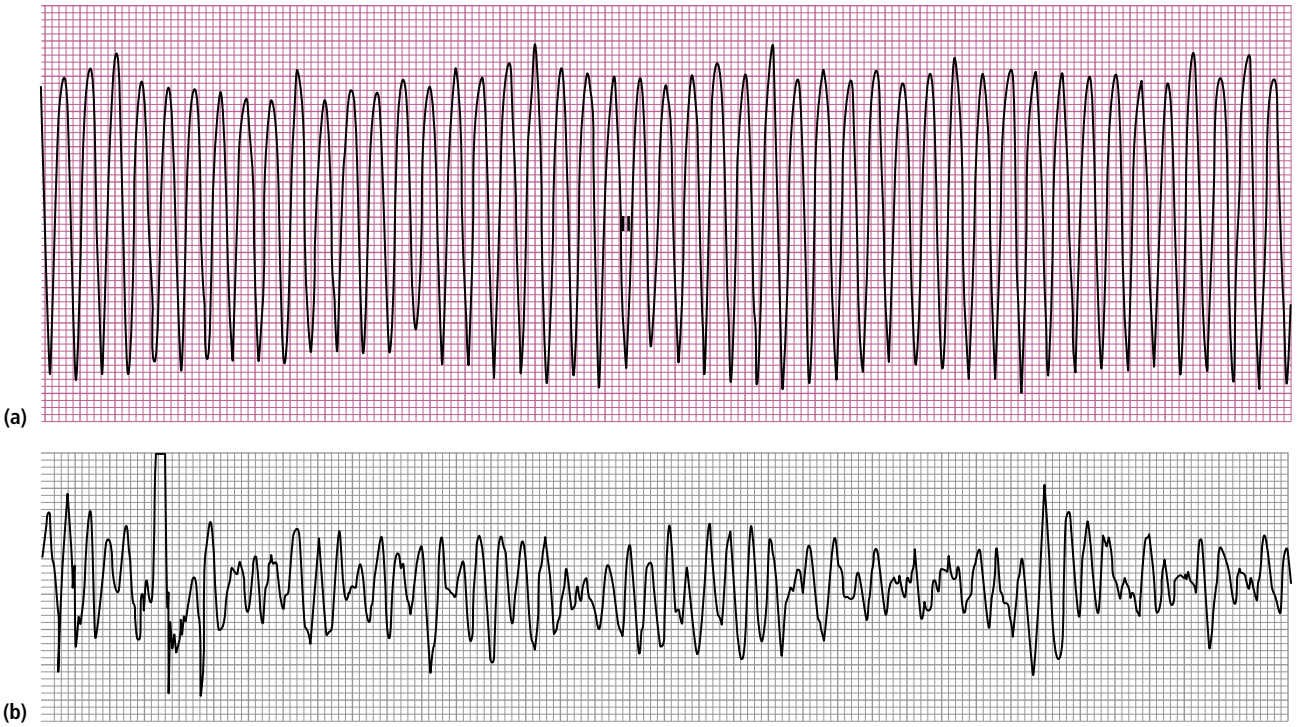


Figure 11.12 (a) Ventricular flutter, lead II ECG at 25 mm/second. (b) Ventricular fibrillation, lead II ECG at 25 mm/second.

potential difference is detectable on any surface lead. However, in the injured myocardium an "injury current" may occur as current flows between diseased myocardium and adjacent, healthier myocardium. This injury current can result in an electrical potential difference being detectable during the ST segment that shifts its position above or below baseline (Figure 11.13). Whether the ST segment becomes shifted upward (elevation) or downward (depression) depends on the relative orientation of the diseased and healthier tissue to one another (e.g. epicardial injury vs. endocardial injury). Myocardial hypoxia can result in ST segment elevation or depression. ST segment changes should always be noted and brought to the clinician's attention when they develop. Investigation of myocardial injury (e.g. echocardiography, arterial blood gas analysis, arterial blood pressure assessment, troponin assay, thoracic radiography) may be warranted once this finding has been detected.

Skill Set 10: Distinguishing Bundle Branch Block from Ventricular Rhythms

The QRS complex may appear wide on an ECG for a number of reasons. Slight QRS widening may occur with left ventricular or biventricular hypertrophy (i.e. depolarization takes a bit longer because there is more ventricular tissue to depolarize). Marked QRS widening is typically due to either (a) dysfunction of one or more of the branches of the bundle of His (BBB); or (b) the process of depolarization bypassing the bundle of His entirely (e.g. ventricular escape beats, premature ventricular complexes). It is important that the ECC technician not assume that all QRS



Figure 11.13 ST segment elevation, lead II ECG at 25 mm/second.

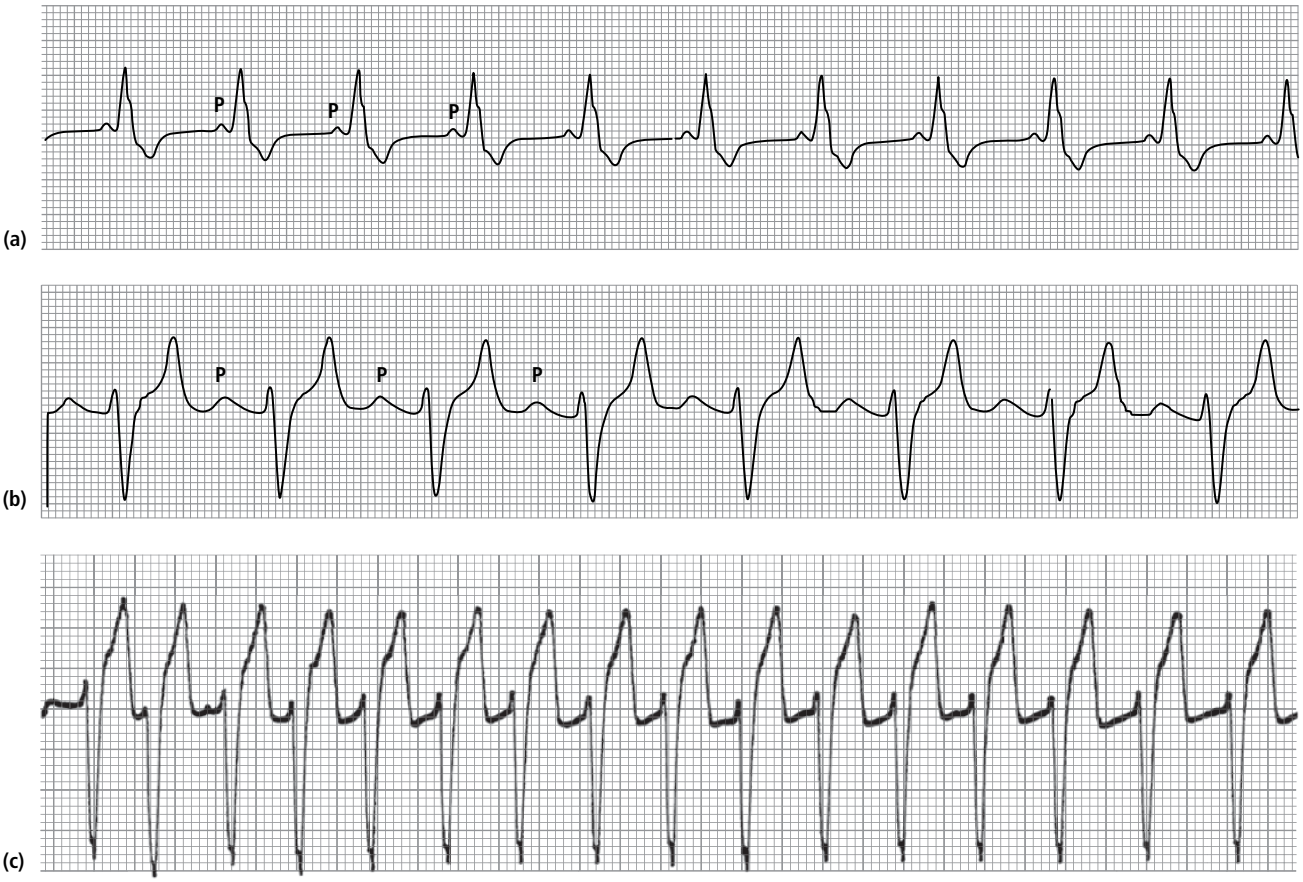


Figure 11.14 (a) Left bundle branch block, lead II ECG at 25 mm/second. (b) Right bundle branch block, lead II ECG at 50 mm/second. (c) A ventricular rhythm that lacks evident P waves, lead II ECG at 25 mm/second.

complexes with a "wide and bizarre" appearance are ventricular in nature. One needs to stop and inspect the ECG tracing for evidence of aberrant conduction (e.g. BBB).

The first two ECGs (Figure 11.14a,b) show examples of block of the left and right branches of the bundle of His (left BBB, LBBB, and right BBB, RBBB), respectively. The most notable difference between LBBB and a normally conducted sinus beat is the widening of the QRS complex. With RBBB, the QRS appearance on lead II exhibits a deep, slurred S wave (the complex appears "upside down" in lead II) in addition to the widened appearance of the QRS complex. If a six-lead ECG study is available, a right-axis deviation of the mean electrical axis may be noted. With both LBBB and RBBB the rhythm originates from sinus node pacemaker activity. Since atrial depolarization precedes ventricular depolarization, P waves are present and the P-R interval is regular and normal (see labeled P waves in Figure 11.14a,b).

Acknowledgments

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12

Fluid-Filled Hemodynamic Monitoring Systems

Jamie M. Burkitt Creedon

Intravascular pressures are commonly measured in acute and critical illness. Intravascular or "blood" pressure is the physical pressure that blood exerts on the vessel wall. This pressure is important because the difference in intravascular pressure at any two points in the vascular network is the driving force for blood circulation. The pressure at the root of the aorta is much greater than the pressure in the vena cava so that blood flows from the arterial to the venous side, delivering oxygen and other nutrients to cells along its path. Intravascular pressures commonly measured in small animals are peripheral arterial blood pressure (ABP) and central venous pressure (CVP). Pulmonary arterial pressure (PAP) and pulmonary arterial occlusion pressure (also called pulmonary capillary wedge pressure or "wedge pressure") can also be directly measured; however, these measurements are less commonly performed in veterinary patients.

Peripheral ABP may be directly evaluated by measuring intraluminal pressure following placement of an intravascular catheter measurement system, or it can be indirectly measured by surface-applied cuff pressure and flow-detection methods such as Doppler ultrasound or oscillometry. CVP can only be measured directly by measuring pressure transmitted from the central vein into a catheter-associated measuring system. This chapter deals specifically with the technical aspects of the direct measurement of intravascular pressure using fluid-filled monitoring systems.

Driving Pressure, Resistance, and Blood Flow

Pressure is defined as a force per unit area:

$$P = F / A \tag{12.1}$$

where P is pressure, F is applied force, and A is the cross-sectional area over which force is applied. To interpret measured pressure, it must be compared with a known pressure standard. For instance, one can only appreciate the effect of pressure applied to the surface of a rubber band if one knows the pressure the rubber band exerts back on the operator – whether the band will stretch depends on the difference between these pressures. In medicine, the accepted standard pressure against which physiologic pressures are compared is barometric pressure (P_B), or the pressure in the earth's atmosphere, which is approximately 760 mmHg (1031 cm H_2O) at sea level. Clinically, we often are more interested in knowing the intravascular pressure at a given site compared with P_B (i.e. "What is the ABP as measured in the femoral artery of this cat?") than in knowing the difference in pressure between two different anatomic sites.

To understand the physiologic determinants and importance of intravascular pressure, one must also understand Ohm's law of hydrodynamics, which is expressed as follows:

$$\Delta P = Q \times R \tag{12.2}$$

where ΔP is the pressure difference between an upstream and a downstream measurement site, Q is the flow of blood between those sites, and R is the resistance between those sites. The ΔP can be referred to as the *driving pressure*, which in the case of intravascular pressures is the pressure differential between the "upstream" and "downstream" portions of a blood vessel. Blood flow (Q) is the volume of blood movement per unit time and is directly correlated with ΔP. Resistance to flow, R, is the force opposing forward fluid movement. In very basic terms, the higher the driving pressure, the more likely blood will flow through the blood vessel; the higher the resistance across the vessel, the less likely blood will flow through that vessel. An important concept herein is that blood flow through a vessel or to an organ is not guaranteed by a "normal"

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driving pressure if high resistance is present; conversely, blood flow may be adequate at low driving pressure if the resistance through the blood vessel or organ is also low.

Determinants of Intravascular Pressure

Determinants of Systemic Arterial Pressure

Systemic ABP is the product of cardiac output (CO) and systemic vascular resistance (SVR) such that

$$\text{ABP} = \text{CO} \times \text{SVR} \tag{12.3}$$

This equation is derived from Ohm's law, where CO is blood flow (Q), SVR is vascular resistance (R), and ABP is driving pressure (ΔP).

Systemic ABP is determined by cardiac factors such as heart rate and contractility, by blood volume, and by systemic (also called peripheral) vascular tone. Blood pressure is under tight minute-to-minute and long-term control by an integrated neural, endocrine, and paracrine system. Full discussion of these physiologic influences is outside the scope of this chapter, and details can be found elsewhere [1, 2].

Determinants of Central Venous Pressure

CVP can be used as a surrogate for right atrial pressure when the tricuspid valve functions normally and there is no blood flow obstruction between the catheter tip and the right atrium. CVP is very close to right ventricular end diastolic pressure; its value represents the filling pressure of the right side of the heart. Factors determining central venous (right atrial) pressure include effective blood volume at the site of measurement, pleural pressure (which influences pressure across the walls of the great veins and the right heart), venous vascular resistance, and right heart function. Changes in CVP were traditionally used to estimate blood volume in critically ill patients. Despite its historical use for this purpose, evidence suggests that there is no reliable relationship between fluid responsiveness and either CVP or change in CVP [3].

Determinants of Pulmonary Arterial Pressure

PAP is the intravascular pressure that drives blood from the right side of the heart to the left side of the heart. PAP is the product of CO and pulmonary vascular resistance (PVR):

$$\text{PAP} = \text{CO} \times \text{PVR} \tag{12.4}$$

PVR is affected by multiple factors such as pleural pressure variation (in disease or secondary to the respiratory cycle) and pulmonary venous pressures. A thorough review of these factors is available elsewhere [4].

Indications for Direct Intravascular Pressure Monitoring

Direct intravascular pressure measurement is an integral part of the critically ill patient's monitoring plan. CVP can only be measured directly via central venous catheterization. Indications for CVP monitoring are discussed in Chapter 15. PAP is commonly estimated in small animals via Doppler echocardiographic evaluation using the modified Bernoulli equation rather than measured directly. However, any time precise, continuous, or frequent measurements of PAP are desirable, it must be measured directly with a pulmonary arterial catheter (see Chapter 16).

Systemic ABP can be measured either by a direct method using a peripheral arterial catheter system or indirectly by using surface-applied pressure from an inflatable cuff partnered with flow-detection methodology (sphygmomanometry). The most common methods of indirect ABP measurement in dogs and cats are Doppler ultrasonic or oscillometric techniques, which are discussed in Chapter 14.

Most human intensive care references consider direct pressure monitoring the "gold standard" against which indirect methods are compared [5–8]. Direct pressure measurement is considered more accurate because detection and measurement of arterial pressure occurs directly in the vessel lumen. Cuff measurement techniques depend on blood flow to provide pressure estimates. Cuff methods are particularly poor in patients with low blood pressure secondary to myocardial failure or in cases where significant alteration in vascular resistance occurs (shock) [5, 7]. In these cases, there can be large differences between values provided by indirect methods compared to gold standard direct measurement. An international consortium has set performance standards for indirect ABP monitors in people [9]. No veterinary studies have yet shown that indirect methods meet these standards in dogs and cats, and study methods have not necessarily matched those set out by the consortium [10–13]. Therefore, continuous invasive pressure monitoring is indicated in many critically ill patients, and particularly in those experiencing shock states (Box 12.1).

Despite the advantages of direct intravascular pressure measurement, the technique is technically challenging and measurement errors occur even when equipment is properly calibrated, leveled, and zeroed (information about these procedures below). Directly measured systolic and diastolic ABP are often inaccurate in dogs and cats; directly measured mean arterial blood pressure (MAP) is more consistent and is a useful value to monitor, as it is the mean

Box 12.1 Indications for Continuous Direct Arterial Blood Pressure Measurement

- Hypotensive states with actual or potential tissue hypoperfusion
- Significant peripheral vasoconstriction
- Severely hypertensive states
- During vasodilator therapy
- Intraoperative and postoperative monitoring of critically ill patients

(continuous) pressure the tissues experience. The inherent pitfalls of even this gold standard pressure monitoring procedure underscore the importance of evaluating the whole patient. Reported values that do not fit the clinical picture should be considered suspect, and the monitoring system investigated for sources of error. System inaccuracy and sources of error are addressed later in this chapter.

Central venous and direct arterial pressure monitoring require large venous and arterial catheterization, respectively, and therefore carry the risks of these techniques. Complications, troubleshooting, and contraindications for central venous and arterial catheterization are discussed in Chapters 7 and 8, respectively.

Types of Direct Intravascular Pressure Monitoring Systems

Direct pressure monitoring systems in veterinary practice usually consist of fluid-filled tubing that connects a vascular catheter at or near the site of interest to a measuring device. Pressure waves move throughout the blood from the area of interest within the body (such as the cranial vena cava for CVP measurement), through the catheter and fluid-filled tubing, either to a water manometer or to a pressure transducer–processor–display system. Fluid must completely fill the system because fluid is relatively noncompressible and therefore transmits pressure waves well; air is too compressible to accurately transmit pressure waves. The fluid column between the site of interest and the measurement system must be unobstructed for the measuring system to provide accurate information.

The *water manometer* is the simpler of the two common measurement systems. The intravascular catheter is attached to a fluid-filled system of tubing and a manometer (Figure 12.1). There is a continuous fluid column between the catheter tip within the patient's vessel or right atrium and the manometer. The pressure at the catheter tip supports a column of fluid within the vertically oriented manometer; the pressure is then reported as the height of the fluid within the column. Therefore, when



Figure 12.1 A water manometer being used to measure central venous pressure (CVP) in a ferret. Note the bottom of the manometer, at 0 cm in height, is being leveled with the ferret's right atrium, establishing the right atrium as the "zero" reference point with which the CVP will be compared.

used properly, this technique allows for direct measurement of the pressure at the catheter tip. Measurements are manually performed intermittently. Water manometers are calibrated in centimeters of water (cm H_2O) and are generally only used for CVP measurement in veterinary practice. Peripheral ABP is too high to allow for practical measurement with a water manometer (ABP is higher than the pressure produced by the standard fluid column, so arterial blood would shoot out the top of the water manometer). Water manometers generally report a single value, which is considered the mean intravascular pressure in the cavity (vessel, cardiac chamber) of interest at the reference height (more regarding reference height, leveling, and zeroing below).

For direct ABP measurement, a fluid-filled catheter system is attached to a pressure transducer–processor–display system. This system is commonly used for systemic blood pressure because it is designed to accommodate the higher pressures generated in arterial blood vessels and permits continuous monitoring. There is a continuous fluid column between the catheter tip within the patient's body and a pressure transducer, which changes (transduces) the physical pressure wave into an electronic signal (see Chapter 13, Figure 13.1). The electronic signal is transmitted via a cable to a processor where the signal is amplified and processed into a real-time display of graphic waveform and numeric pressure values. Digital monitors generally display pressure in millimeters of mercury (mm Hg), and report systolic, mean, and diastolic pressures. Transducer–processor–display systems are often part of multiparameter patient monitors, which simultaneously report multiple physiologic parameters (e.g. temperature, respiratory rate, electrocardiogram, multiple pressures, pulse oximetry;

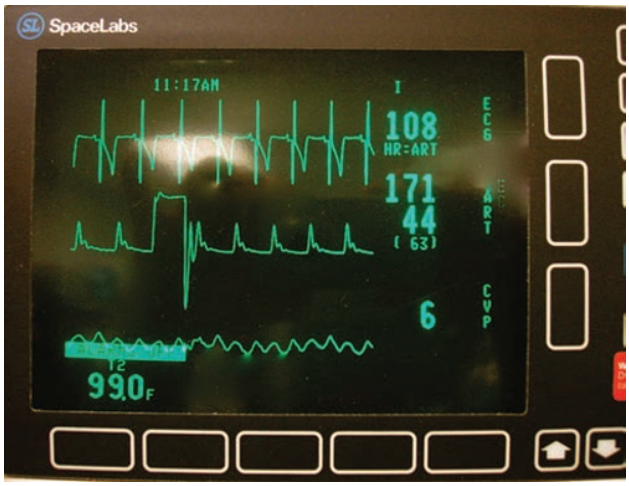


Figure 12.2 The screen of a multiparameter monitor displaying a simultaneous lead I ECG, heart rate, arterial blood pressure waveform, arterial blood pressure values, central venous pressure waveform, mean central venous pressure value, and rectal temperature. A square wave test is visible in the arterial pressure tracing between the second and third arterial pressure waveforms.

Figure 12.2). Blood pressure waveforms are displayed on the monitor screen in addition to numerical pressure values.

Measurement System Components

Both the manometer system and the pressure transducer–processor–display system begin with an intravascular catheter and specialized fluid-filled tubing. Beyond the tubing, the manometer system consists of one three-way stopcock, a water manometer, and a fluid reservoir (source). Beyond the tubing, the electronic system consists of at least one stopcock, a pressure transducer, a pressurized fluid reservoir, a flush device, a cable connecting the transducer to a processor, and the processor–display, which is generally a single unit.

For the pressure (and waveform, with an electronic system) to be reported faithfully, each of the system components must meet certain physical and technical criteria. Even if each of the system components meets the required criteria, combining the components alters the system's overall physical properties such that it may not provide accurate information. The operator should therefore be able to do the following: appropriately set up the system; recognize waveform patterns in electronic systems consistent with system malfunction; and test the electronic system for fidelity.

Intravascular Catheter

The catheter's gauge, length, insertion site, orientation, and proximity to the vessel wall all affect reported pressures. Frictional resistance to fluid movement within the system increases as the catheter's inner diameter decreases or the catheter lengthens. Therefore, the ideal catheter for pressure wave transmission would be short with a large bore. However, because the catheter itself increases resistance within the vessel at the insertion site and thus alters pressure, the catheter would ideally occupy no more than 10% of the vessel lumen [5]. The reality for dogs and cats is somewhere in the middle: in general, the catheter should easily fit within the vessel with minimal risk of vascular occlusion.

The catheter insertion site should be chosen for cleanliness, ease of placement, and maintenance. If a stiff catheter is used, it should remain straight along its entire path to minimize kinks. Catheters for CVP monitoring are usually inserted into the external jugular vein in both dogs and cats and threaded into the cranial vena cava. Alternatively, a long, flexible catheter may be placed into a saphenous vein and threaded cranially into the thoracic vena cava. Because saphenous-inserted thoracic caval catheters yield similar values to those placed through the external jugular vein [14], this is common practice in dogs and cats. Venous catheter insertion is discussed in detail in Chapter 7.

Common insertion sites for arterial catheters in dogs are the perforating metatarsal artery (commonly called the dorsal pedal artery) and the radial, coccygeal, femoral, and auricular arteries. In cats, the femoral artery is often used; the dorsal pedal or coccygeal artery can be used for short durations, such as for anesthetic procedures. Information regarding arterial catheter insertion can be found in Chapter 8. Mean pressure is lower in distal portions of the arterial tree compared with the aorta because pressure wave energy is lost as heat generated by frictional flow resistance along the vessel length. The pressure difference along the arterial tree is minimal and is thus not considered when selecting measurement sites.

Catheter tip orientation in relation to direction of blood flow affects the measured pressure value in both arterial and venous systems. A catheter tip facing into the blood flow (upstream) will measure a slightly higher pressure value than the actual intravascular pressure; a catheter tip facing away from the flow of blood (downstream) will measure a pressure value slightly lower than actual pressure. These differences are due to alteration in kinetic and potential energy at the catheter tip and are relatively small in the vascular catheters commonly placed in veterinary practice.

Noncompliant Tubing

The tubing that connects the intravenous catheter and pressure transducer must be made of specialized material to prevent pressure wave energy from being absorbed (or "dampened") by the tubing wall. This tubing is called

rigid, noncompliant, or high-pressure tubing. The tubing is filled with fluid and all air bubbles removed. Use of standard (softer) fluid extension tubing between the vascular catheter and the pressure transducer causes error in the pressure measurement and the waveform. Noncompliant tubing is less important for CVP measurement than it is for systemic arterial pressure but is still desirable.

Water Manometer

Water manometers are used for intermittent measurement of CVP, which is detailed in Chapter 15. The water manometer is a plastic tube marked in centimeters along its length with a "zero" mark near the bottom of the column height (sometimes the bottom *is* the zero mark, as in Figure 12.1). The zero mark is used as a reference site for aligning the manometer at the level of the right atrium prior to CVP measurements (see Zeroing the Transducer, below). If a commercially produced water manometer is unavailable, one can create a water manometer from standard fluid extension tubing hand-marked with centimeters indicated along its length (see Chapter 15, Figure 15.2). The base of the water manometer fits into the three-way stopcock in vertical orientation with the noncompliant tubing–catheter system on the second port and the fluid reservoir on the third.

Although it is called a "water" manometer, the tubing column is filled with a biologically compatible crystalloid fluid to perform the measurement. The fluid reservoir for the water manometer is usually a 20-ml syringe filled with isotonic crystalloid. Full details regarding assembly and use of the water manometer pressure measurement system are available in Chapter 15, specifically in Protocol 15.1.

Pressure Transducer

When an electronic pressure monitoring system is used, the noncompliant tubing is attached to a pressure transducer (see Chapter 13, Figure 13.1). The transducer has a pressure-sensitive membrane that distorts in response to pressure changes that are created when pressure waves strike it. The transducer converts the membrane distortion into an electronic signal using an integrated electronic circuit that functions by the "Wheatstone bridge" principle [5]. The generated electrical signal is transmitted to the processor–display unit by a shielded electrical cable. Most pressure transducers used in veterinary medicine are classified as "disposable," meaning that they are intended for single use in people. Disposable transducers are sterile in their packaging and electronically pre-calibrated; some models come with high-pressure tubing attached.

Pressure transducers have a female adapter for connection to the fluid reservoir. The fluid reservoir is a bag of sterile isotonic crystalloid and administration set attached to the transducer. The bag of isotonic crystalloid fluid is maintained under constant pressure such that a small volume of fluid continuously flushes through the system, preventing blood from flowing back and contaminating the monitoring system. The infused volume is generally 2–4 ml/hour (see manufacturer's specifications for more precise value). Most transducers have a pigtail or lever protruding from the housing that activates an integrated "flush valve" that allows rapid infusion ("fast flush") of fluid from the pressurized fluid bag through the system to clean the transducer head.

Handled with care, disposable transducers may be cleaned and reused following ethylene oxide re-sterilization. Re-sterilization can lead to damage of the pressure-sensitive membrane, which can lead to inaccurate pressure reporting. When a re-sterilized transducer is used, the operator should always recalibrate it prior to performing clinical measurements (see below for information about calibration).

Assembling the Electronic Direct Pressure Measurement System

A full list of supplies required and step-by-step instructions to assemble the electronic direct pressure measuring system are available in Protocol 12.1. Figure 12.3 shows a portion of a direct arterial pressure monitoring system in use.

Technical Aspects of the Electronic Direct Pressure Measurement System

Direct pressure monitoring is relatively complex and far from foolproof. There are many technical points that must be followed for accurate measurement. It is important that the operator understand the technical principles that affect the system's fidelity, how to properly configure and test the system for fidelity, and how to recognize and correct for system error.

Zeroing the Transducer

To interpret measured pressure, it must be compared with a known pressure standard. The standard pressure against which direct intravascular pressure measurements are compared is barometric pressure (P_B), which is approximately 760 mmHg (1031 cm H_2O) at sea level.

The electronic pressure measurement system is calibrated to standard pressure, or "zeroed," by opening the transducer's stopcock port (see Chapter 13, Figure 13.1) to the atmosphere and depressing the "zero" button on the electronic pressure monitor (see Protocol 12.2 for instructions on zeroing a transducer). Zeroing the transducer to

Protocol 12.1 Assembling the Electronic Direct Pressure Measurement System for Central Venous or Arterial Blood Pressure Monitoring

Items Required

- Indwelling vascular catheter at the appropriate site
- ≤ 2 lengths of high-pressure tubing with locking fittings – keep as short as feasible
- Stopcocks with locking fittings – ≤ 2
- One to two sterile infusion plugs with locking fittings
- Sterile pressure transducer with integrated fast flush device
- Sandbag and medical tape, or another stabilizing device to which to affix the transducer
- Bag of isotonic crystalloid flush solution with standard administration set attached; standard extension set(s) as needed
- Confirm with clinician whether or not to add heparin to flush solution
- Pressure bag of appropriate size for fluid bag used
- Monitor, with its power cord and transducer-to-monitor cable
- Power source

Procedure

1) Collect necessary supplies.
2) Plug the monitor in, turn it on, and attach the transducer-to-monitor cable to the monitor. Configure the monitor per manufacturer instructions to display pressure from the socket into which you have inserted the transducer-to-monitor cable.
3) Perform hand hygiene and don clean examination gloves.
4) Aseptically prepare the patient's catheter port onto which the monitoring system will be attached. Flush the patient's catheter gently to ensure patency.
5) Heparinize the flush solution if the clinician has so ordered. Standard dilution is 4 units heparin per milliliter of flush (1 ml of 1000 iu/ml heparin added to 250 ml 0.9% NaCl). Mark the fluid bag to indicate any additives. Remove all air from the fluid bag by inserting a 22-gauge needle into the medication port and withdrawing gas until none remains. This minimizes the risk of air embolism from the pressurized system.
6) Attach a standard fluid administration set, with the roller clamp closed, to the heparinized fluid bag and squeeze the drip chamber until it is approximately half-filled with fluid. Prime the fluid line with flush using gravity flow, then reclamp the fluid line. Handle fluid line such that the line's end remains sterile.
7) Insert the fluid bag into a pressure bag and inflate to approximately 300 mmHg. Hang the pressurized bag and fluid line near the patient area.
8) Remove the pressure transducer from its packaging and manipulate all moving parts to ensure they move as intended. Handle such that all ports remain sterile. Inspect for any evidence of damage, particularly if the unit has been re-sterilized.
9) Attach the primed flush fluid line to the transducer. If necessary, attach one length of high-pressure tubing to the transducer on the patient side. If necessary, add more high-pressure tubing to reach the patient, to a maximum of two lengths total. Place a sterile, locking injection port to the transducer's zeroing (air) port.
10) The vented cap on a new transducer's stopcock should be thrown away, as its vents may allow contaminants to enter the system and be injected into the patient.
11) If blood sampling from the catheter is desired, place a stopcock between the high-pressure tubing and the catheter or catheter's T-port. The unused port should have a sterile, locking injection port attached to the third port.
12) Use the fast flush device on the transducer to prime the system until fluid drips from the end of the high-pressure tubing. Flush slowly to avoid air bubble formation in the tubing.
13) Attach the monitoring system to the patient's vascular catheter or the catheter's T-port.
14) Tighten all connections and engage all fitting locks to prevent inadvertent disconnection and subsequent blood loss.
15) Plug the monitor cable into the transducer and fix the transducer to its stabilizing device (e.g. sandbag with tape) such that the transducer's zeroing (air) port is at the appropriate height (the level of the right atrium).
16) Zero the system at the appropriate level (see the sections Zeroing the Transducer and Leveling the Transducer for more information).
17) Turn all stopcocks in the system so that they are open to the patient.
18) Perform a fast flush test and make any necessary adjustments to the system to optimize the system's dynamic response. (See text for more information about dynamic response.)



Figure 12.3 Part of a direct arterial pressure monitoring system in a dog. The dog's perforating metatarsal (dorsal pedal) arterial catheter is attached to a T-port, which is connected to noncompliant, fluid-filled tubing by a three-way stopcock. This stopcock is optional to the system and is in place here for serial arterial blood sampling. The noncompliant tubing is attached to the pressure transducer, which is secured to a yellow sandbag with medical tape; the sandbag provides a stable base for the transducer and helps raise the transducer's zero point (top of the transducer's stopcock) to the level of the patient's right atrium. The red pigtail on the transducer can be used to provide rapid system flush from the pressurized fluid bag (not pictured; its tubing leads from the pigtail to the fluid bag out the top of the frame). The gray cable runs from the transducer to the electronic processor-display unit (not pictured).

atmospheric pressure makes discussion of physiologic pressures easier (i.e. "The patient's MAP is 98 mmHg" rather than "The patient's MAP is 760 + 98 = 858 mmHg"). Transducer zeroing should be done at initial system setup, any time system components are removed or replaced, or if any problems occur with system readings. If a transducer system fails to zero, the transducer, the monitor, or the connecting cable may be faulty (most often it is the transducer, especially when using a re-sterilized disposable transducer). These items should be sequentially changed and zeroing re-attempted until the faulty component is identified and replaced.

Water manometers are always open to the atmosphere and are thus inherently "zeroed" to atmospheric pressure. Therefore, no zeroing actions need to be performed on a water manometer, but it must be oriented vertically for measurement and its top has to be open to the air or readings will be inaccurate.

Calibrating the System

After the system is zeroed to the atmosphere, it must be calibrated prior to use. To calibrate a measuring instrument is to compare and align its readings with known standards so that the measuring instrument can provide accurate information. Calibrating a water manometer device simply involves comparing the markings on the manometer against those on a centimeter ruler. Such calibration is unnecessary on commercially available manometers, since they come premarked, but is required if regular fluid extension tubing is used to contain the fluid column.

Calibration is required for electronic systems each time the system is assembled and again any time problems arise. The transducer must be calibrated to confirm that when standard pressure is applied to the transducer, the correct pressure is reported. Calibration should be conducted with the transducer attached to the monitor, as instructed by the monitor's manufacturer. Alternatively, a water manometer

Protocol 12.2 Zeroing a Pressure Transducer

Items Required

- An assembled electronic direct pressure measurement system (see Protocol 12.1), attached to its monitor via the transducer cable

Procedure

1) Plug in and turn on the monitor. Configure the monitor such that it displays an option to zero the transducer (see manufacturer's instructions for specific steps).
2) Perform hand hygiene and don clean examination gloves.
3) Turn the transducer's stopcock "off" to the patient's side of the system so there is a continuous fluid column between the stopcock's injection cap and the transducer's pressure diaphragm.
4) Remove the injection cap from the stopcock, maintaining system asepsis.
5) Depress the "zero" button on the monitor.
6) The pressure tracing should be a flat line at the zero mark on the display.
7) If the pressure reads a number other than 0, or if the pressure tracing is not a flat line at the zero mark on the graph, the transducer may be faulty, in which case it must be replaced with another flushed, sterile transducer. If this does not remedy the issue, the transducer cable or monitor itself may be faulty and may need to be replaced.
8) Once the monitor displays "0" with the transducer's fluid interface open to the atmosphere via the stopcock, aseptically replace the injection cap and turn the stopcock "off" to the injection cap, opening the fluid column between the transducer and the patient end of the system.

filled with fluid to a set height may be attached to the transducer's stopcock and the display's reading compared with the known applied pressure from the fluid column, remembering that 1.36 cm H_2O exerts the same pressure as 1 mmHg. Calibration problems can be due to transducer, monitor, or connecting cable problems.

Leveling the Transducer

A transducer is both zeroed, to eliminate the influence of atmospheric pressure from vascular pressure readings, and *leveled*, to eliminate the influence of *gravity*. For both CVP and ABP, the "level" reference point is the right atrium; thus, the right atrium is called the zero *reference point*. To level the measuring system, the transducer should be placed at the vertical height of the right atrium and zeroed at that point (by opening the three-way valve to air, as above). This step should be performed *prior to every measurement* (Protocol 12.3). An external anatomic landmark that correlates well with the right atrium reference point is the sternum of the cat or dog lying in lateral recumbency. As stated in Chapter 15, in a sternally recumbent animal the right atrium lies at a point roughly 40% the height of a vertical line that extends from the sternum to the top of the dorsal spinous process just caudal to the shoulder.

Once the transducer is leveled (zeroed at right atrial height), it must remain level at right atrial height for every measurement. The reason for this requirement may best be explained by example. In a patient undergoing CVP monitoring, if the transducer falls below right atrial level by 10 cm (approximately 4 inches), a 10-cm blood fluid column is exerting pressure on the transducer in addition to the actual CVP. Though this additional pressure is not a change in CVP, the transducer will "see" both the actual CVP and the additional 10 cm blood column and will report a value equal to the CVP plus 10 cm H_2O. The opposite is true if the transducer sits higher than its zeroed reference point: the pressure reported will be falsely low in such cases (Figure 12.4). For peripheral ABP monitoring, such changes are less likely to create clinical confusion because as a proportion of the pressure of interest, the error is smaller. For instance, while a difference of 10 cm H_2O (7.4 mmHg) in CVP could influence a clinician's decision-making process, that same 7.4 mmHg difference in MAP is less likely to cause an error in clinical judgment. This example underscores the importance of evaluating the whole clinical picture before making treatment decisions,

Protocol 12.3 Leveling a Pressure Transducer at the Zero Reference Point (the Right Atrium)

Items Required

- An electronic direct pressure measurement system, attached to a powered-on monitor and to the patient
- Carpenter's level
- Piece of string

Procedure

1) Configure the monitor such that it displays an option to zero the transducer (see manufacturer instructions for specific steps).
2) Perform hand hygiene and don clean examination gloves.
3) Turn the transducer's stopcock "off" to the patient's side of the system so there is a continuous fluid column between the stopcock's injection cap and the transducer's pressure diaphragm.
4) Remove the injection cap from the stopcock, maintaining system asepsis.
5) Ensure the transducer's fluid–air interface is at the level (vertical height) of the right atrium. Assess this level using the string and carpenter's level for accuracy – this step will enhance accuracy and repeatability from one operator to the next for repeated measures.
   a) With the patient in lateral recumbency, right atrial height is approximately the height of the sternum.
   b) For a dog or cat in sternal recumbency, right atrial height is approximately 40% the distance from the sternum to the dorsal spinous process at the plane just caudal to the scapula.
6) Secure the monitor at this height if performing continuous monitoring and the patient is immobile.
7) Depress the "zero" button on the monitor.
8) The pressure tracing should be a flat line at the zero mark on the display.
9) If the pressure reads a number other than 0, or the pressure tracing is not a flat line at the zero mark on the graph, the transducer may be faulty, in which case it must be replaced with another flushed, sterile transducer. If this does not remedy the issue, the transducer cable or monitor itself may be faulty and may need to be replaced.
10) Once the monitor displays "0" with the transducer's fluid–air interface open at right atrial level (height), aseptically replace the injection cap and turn the stopcock "off" to the injection cap, opening the fluid column between the transducer and the patient end of the system.
11) Allow the system to equilibrate and record the measured value.

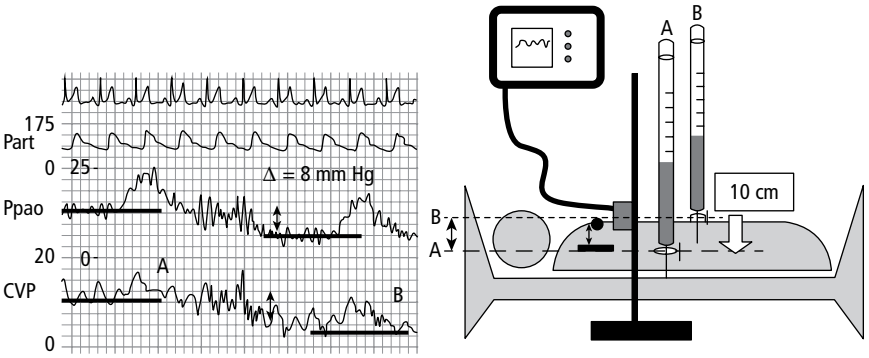


Figure 12.4 Illustration of what happens when the patient's right atrium (level shown by dashed line A) falls below the transducer's zero reference point (at the level of dotted line B) as the bed is lowered: (left) change in recorded pressures (P_{art}, peripheral arterial; P_{pao}, pulmonary arterial occlusion [wedge]; CVP, central venous pressure); (right) water manometers (tubes A and B) that demonstrate how lowering the right atrium relative to the transducer lowers the measured pressures. In this example, the bed was lowered by 10 cm, which means that the patient's right atrium (the proper level) is 10 cm lower than the current zero reference point (B), which translates to a pressure drop of 10 cm H_2O, or approximately 8 mmHg. *Source:* Magder (2007)/with permission of Elsevier. Invasive intravascular hemodynamic monitoring: technical issues.

and the importance of proper transducer leveling at the zero reference point (the right atrium) prior to every measurement, particularly when monitoring CVP.

Dynamic Response of the System

Intravascular pressures are pulsatile in nature. Reflection of pressure waves through the vessel creates multiple oscillating waves of different amplitude and frequency (Fourier series) that summate to create the observed waveform. An intravascular pressure monitoring system must have the physical properties required to measure pressures within the expected range and must be able to respond adequately to physiologic pressure pulsations. The ideal system would report the pressure waves of interest and no others. The ability of a system to accurately display the shape and amplitude of the pulse pressure waveform is determined by the system's *dynamic response*, also called the system's frequency response. The system's dynamic response is determined by its physical properties, specifically its mass, elasticity, and friction [15]. Dynamic response is discussed in terms of natural frequency and damping coefficient, both of which are measurable and have significant impact on a waveform's appearance.

Effect of the Measuring System's Natural Frequency

When stimulated, every structure naturally vibrates at a characteristic frequency, which is expressed in cycles per second or hertz (Hz). This frequency is called the structure's *natural frequency*, fundamental frequency, or resonant frequency. Adding components together (such as connecting a catheter, noncompliant tubing, and transducer) alters a system's natural frequency. It is important that the natural frequency of a fluid-filled monitoring system not coincide with the frequency of physiologic pressure waves, because frequency overlap causes summation (the patient plus the system) and results in exaggerated waveforms and numerical values. Exaggerated results are due to *too low a system natural frequency*, and such exaggeration leads to what is often called overshoot, *ringing*, or resonance of the waveform [15]. Ringing causes pointy, spiked waveforms, falsely high systolic pressure readings, and falsely low diastolic pressure readings.

A fluid-filled monitoring system will have optimal responsiveness if its natural frequency is as high as possible. Though individual materials made for intravascular pressure monitoring are designed with this principle in mind, once a catheter–tubing–transducer system is assembled, the natural frequency drops to minimal requirements for people [5]. Because dogs and cats generally have pulse rates that exceed people's, their pulse pressure waveforms have a higher frequency, and thus almost certainly overlap the natural frequency of most measurement systems. This overlap means that without correction through damping (see below), our patients' pulse pressure waveforms will almost always ring, systolic pressures will be falsely high, and diastolic pressures will be falsely low. One way to maximize a system's natural frequency is to keep the system as simple as possible, for instance with as short a tubing length and as few components as is feasible.

Effect of a System's Damping Coefficient

Damping is loss of the pulse pressure energy between the catheter tip and the transducer. Damping is due to frictional resistance along the system's length, absorption of energy by the tubing and other materials, and larger air bubbles, which are more compressible than fluid. The more damped a system is, the more quickly it returns to zero after an applied stimulus, due to energy loss.

Damping in a pressure system is measured and expressed as the *damping coefficient*; the higher the coefficient, the more significant the damping. An *overdamped* pressure waveform has slurred upstrokes and downstrokes, loss of detail, and a generally flattened appearance; overdamped systems cause a falsely narrowed pulse pressure with falsely low systolic and falsely high diastolic pressure readings. Conversely, *underdamped* waveforms contain nonphysiologic points and spikes, extra waves, and appear exaggerated; they are overshot or have excessive ringing, as discussed previously regarding natural frequency. Underdamping causes falsely high systolic and falsely low diastolic pressure readings. A system with an infinitely high natural frequency does not require damping to produce accurate results (Figure 12.5), but because available systems have natural frequency overlap as noted previously, they usually require some operator-implemented damping.

Determining the System's Dynamic Response

Many catheter–tubing–transducer systems have weak natural frequencies to faithfully reproduce physiologic pressure data; thus, these systems are inherently underdamped. Within a certain range, adjusting the system's damping can help produce more accurate values and waveforms (Figure 12.5). To know whether a system has adequate dynamic response, its natural frequency and damping coefficient should be determined and plotted onto a graph such as Figure 12.5. These properties are measured by performing a fast flush or "square wave" test on the system. When the transducer's fast flush device is activated, the transducer–tubing–catheter system is exposed to the high pressure in the flush fluid bag (300 mmHg). To perform the test, the fast flush device should be opened briefly (< 1 second) and released quickly several times to produce multiple square waves for analysis. The high pressure should appear on the recorder as a square waveform as shown in Figure 12.6. A normal square wave has a nearly 90° upstroke, a flat plateau at 300 mmHg, and a rapid downstroke as the fast flush device is released. The top is squared, as the test's name implies. Protocol 12.4 describes how to determine a system's natural frequency and damping coefficient, and thus the system's dynamic response, using waveforms

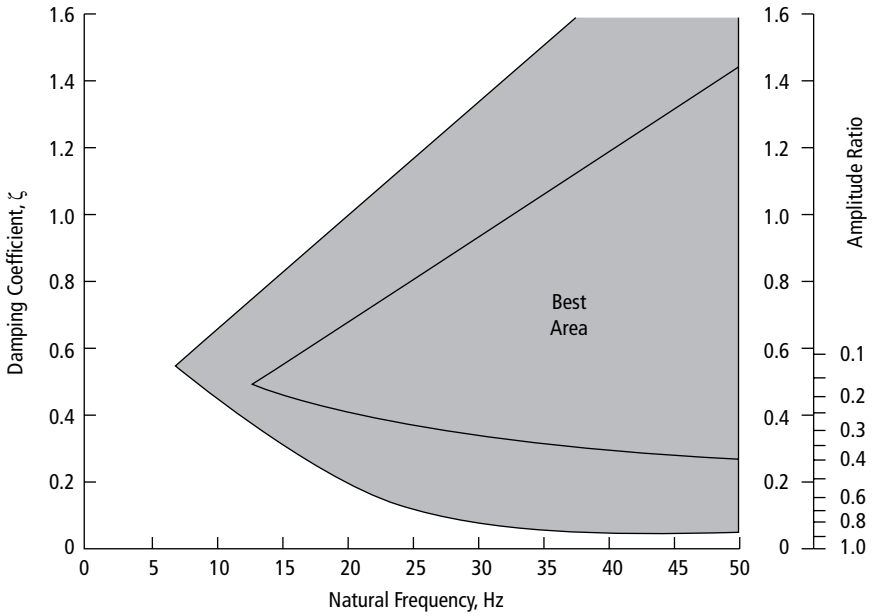


Figure 12.5 Use of natural frequency and damping coefficient to determine whether the catheter–tubing–transducer system is producing accurate pressure waveforms and values. Plot the system's frequency and damping coefficient; the intersection falls in the gray area for an adequately responsive system. Overdamped readings fall above the gray area and underdamped readings fall below the gray area. Note that above approximately 35 Hz natural frequency, the system should be accurate regardless of damping coefficient. *Source:* This figure and its legend were altered and used here with permission from Ahrens and Taylor [16], © Saunders, 1992.

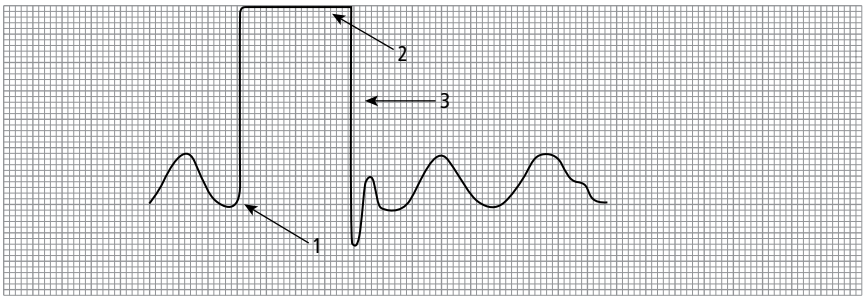


Figure 12.6 Performing a square wave test. The fast flush device is activated at point 1. Squaring of the waveform occurs as the transducer is exposed to 300 mmHg pressure from the flush fluid bag and has a flat plateau (point 2) with right angles on both sides. A rapid downstroke occurs as the fast flush device is released (point 3). This test should be performed several times and the square waves analyzed to determine the system's natural frequency and damping coefficient. These values can then be plugged into the graph in Figure 12.5 to determine whether the system has adequate dynamic response. *Source:* Ahrens and Taylor (1992)/with permission of Elsevier.

Protocol 12.4 Determining the Dynamic Response of a Fluid-Filled Monitoring System

Items Required

- An electronic direct pressure measurement system, attached to a powered-on monitor (with printer) and to the patient
- Calculator
- Straightedge (i.e. ruler)
- Pen and paper

Procedure

1) Ensure the pressure bag containing the flush fluid is pressurized to 300 mmHg.
2) Perform hand hygiene and don clean examination gloves.
3) Perform a square wave test during diastole by activating the transducer's fast flush device briefly (for < 1 second) and quickly releasing the device. A square wave should appear on the monitor. Print this waveform with one to two seconds of strip on either side. Repeat this process three to five times and collect the square waves for analysis.
4) Evaluate the square waves to determine the system's natural frequency. The *natural frequency* is the frequency with which the waveform oscillates after the fast flush device is released. For example: The paper speed in Figure 12.7 is 25 mm/second. Note the number of blocks between oscillation peaks: in this case just over two blocks between oscillations. Divide the paper speed by

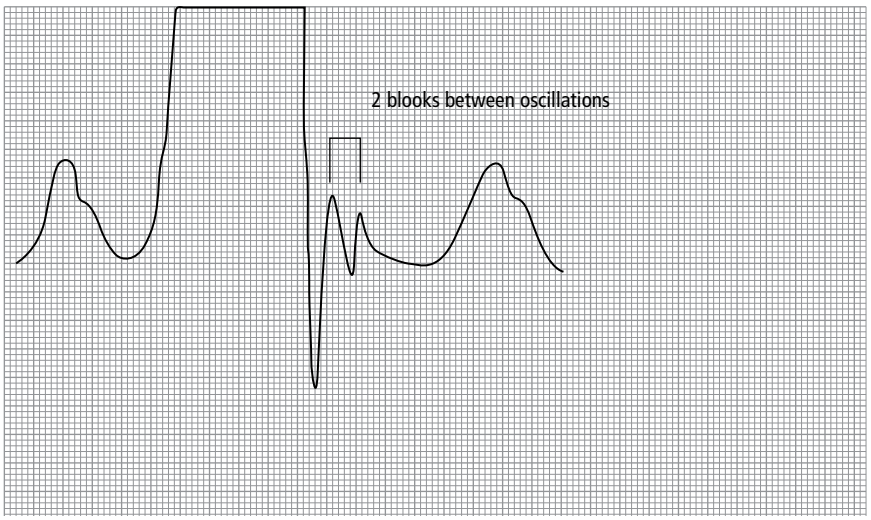


Figure 12.7 Step 4, determine system's natural frequency. Paper speed 25 mm/second. First, note the number of blocks between oscillation peaks – in this case just over two blocks between oscillations. Then divide the paper speed by the number of blocks to determine the system's natural frequency: (25 mm/second) ÷ (2 mm) = 12.5 cycles/second = 12.5 Hz, which is a minimally acceptable natural frequency for measurements in people. *Source:* Ahrens and Taylor (1992)/with permission of Elsevier.



Figure 12.8 Step 6, calculating the amplitude ratio to determine damping coefficient. Paper speed 25 mm/second. First measure the length of two successive oscillations (i.e. from peak to valley, and from that same valley to the next peak). Then divide the smaller oscillation by the larger to determine the amplitude ratio. Here, 6 mm ÷ 28 mm = 0.21, which is the amplitude ratio. *Source:* Ahrens and Taylor (1992)/with permission of Elsevier.

the number of blocks to determine the system's natural frequency: (25 mm/second) ÷ (2 mm) = 12.5 cycles/second = 12.5 Hz, which is a minimally acceptable natural frequency for measurements in people.

5) Determine the frequency on all the square waves collected and average the values to reach the mean natural frequency. Use this mean natural frequency in the dynamic response graphic plot (see step 8).
6) Evaluate the square wave to determine the system's *damping coefficient*. This is done by comparing the length of two successive oscillations and dividing the second (smaller) oscillation by the first (Figure 12.8). The number generated is called the amplitude ratio. For example: The paper speed in Figure 12.8 is 25 mm/second. Measure the length of two successive oscillations (i.e. from peak to valley, and from that same valley to the next peak). Divide the smaller oscillation by the larger to determine the amplitude ratio. Here, 6 mm ÷ 28 mm = 0.21, which is the *amplitude ratio*.
7) Determine the amplitude ratio for all square waves collected and average the values to reach the mean amplitude ratio. Use the graph in Figure 12.9 to determine the damping coefficient from the amplitude ratio.
8) Use a straightedge to plot the damping coefficient against the mean natural frequency on the graph in Figure 12.5. If the intersecting point falls into the gray area, the system is adequately responsive. Overdamped readings fall above the gray area and underdamped readings fall below the gray area.

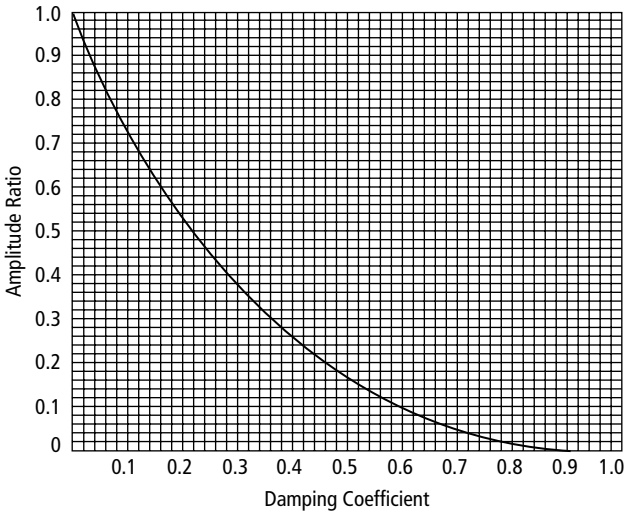


Figure 12.9 Step 7, Use this graph to determine the damping coefficient from the amplitude ratio. *Source:* Ahrens and Taylor (1992)/with permission of Elsevier.

from square wave tests. A square wave test should be performed when the system is initially assembled and any time there are questions about the fidelity of measurement results.

Optimizing Dynamic Response by Altering a Monitoring System's Natural Frequency and Damping Coefficient

There are many steps one can take to optimize the measurement system's dynamic response by either increasing the natural frequency or altering the damping coefficient. Underdamping is far more common in dogs and cats than overdamping. An example from a ringing, underdamped system and one from an overdamped system are shown in the square wave tests in Figure 12.10. Further discussion of the effects of underdamping and overdamping on ABP waveforms and values is found in Chapter 13. Measures to take that may help optimize the system's dynamic response are listed in Box 12.2.

Despite its relevance for accurate pressure readings and interpretation, it is unclear that the systems in clinical use today commonly have adequate dynamic response [15]. It is important to remember both this source of inaccuracy and that pressure waveforms and pressure values are altered by simple changes the operator makes to the system such as alterations in system components and damping. Thus, the gold

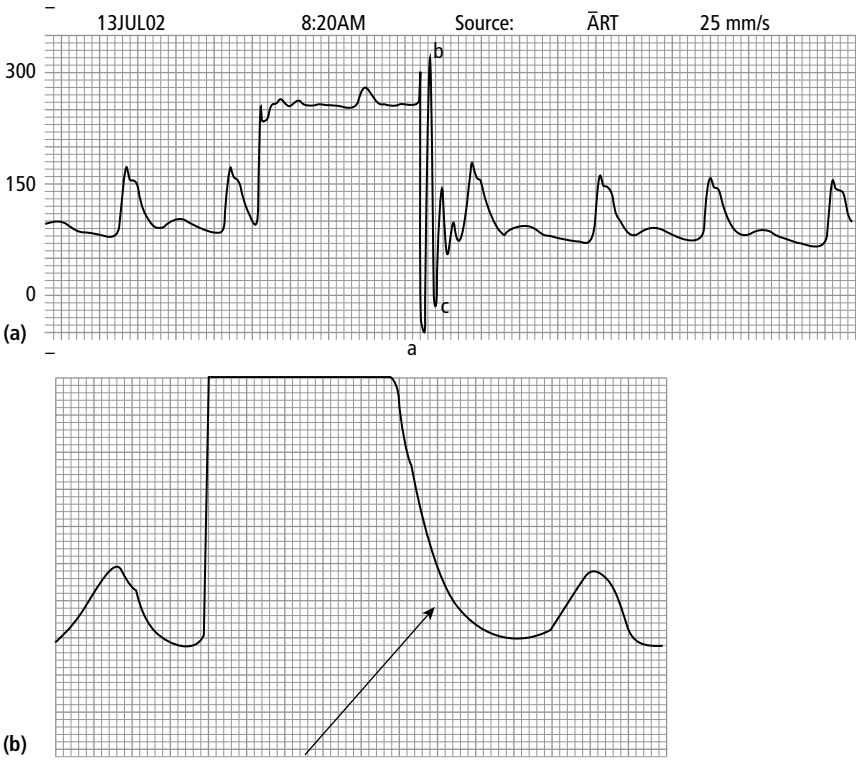


Figure 12.10 Square wave test waveforms: (a) Example of a square wave generated from an underdamped system. Note the spiky, pointy nature of the arterial pressure waveform and the excessive ringing of the square wave test compared with the normal example in Figure 12.6. To confirm underdamping, calculate the amplitude ratio: measure vertically from point a to point b, 37 mm; then from point b to point c, 34 mm; then divide the smaller length by the larger, 34 mm ÷ 37 mm = 0.92. An amplitude ratio of 0.92 corresponds to a damping coefficient of less than 0.1, which is extremely low and corroborates the impression of an underdamped waveform. (b) Example of a square wave generated from an overdamped system. Note the slurred arterial pressure waveform with no apparent dicrotic notch, and the slurred downstroke of the square wave test with lack of oscillations at baseline (arrow). It is very difficult to determine damping coefficient in the absence of any measurable oscillations, but the damping coefficient here is high, probably greater than 0.6 [16]. *Source:* Ahrens and Taylor (1992)/with permission of Elsevier.

Box 12.2 Steps to Help Optimize a System's Dynamic Response

The corrections for overdamped and underdamped systems are similar, so the following actions can be tried in the case of either problem [16, 17].

- Simplify the system as much as possible to increase its natural frequency:
  - Remove as many lengths of tubing between the catheter and the patient as possible such that the tubing does not exceed 3–4 feet (approximately 1 m) in length.
  - Remove any unnecessary stopcocks.
  - Consider removing T-ports or other connections.
- Ensure only noncompliant tubing is present between the catheter and the transducer, particularly for arterial pressure monitoring.
- Check for and remove visible clots or air bubbles.
- Check tubing for kinks or occlusions.
- Gently aspirate and flush the catheter to assess for occlusion.
- If the catheter is <18-gauge (or 7-Fr), compliant, or long, consider replacement with a larger-bore, stiffer, or shorter catheter.
- If the waveform is underdamped, consider insertion of a damping device into the system.

standard invasive pressure monitoring system is also inherently flawed by inevitable operator manipulations.

Summary

Fluid-filled monitoring systems provide important information in patients with cardiovascular instability. Though they are more complicated than noninvasive measurement techniques, they can provide continuous monitoring and may be more accurate. As with any technique, one becomes more proficient with practice and use. A solid understanding of the principles behind fluid-filled monitoring systems allows the clinician to make the most of these systems' capabilities and to avoid technical errors. There is a potential for technical error if equipment is incorrectly configured, inaccurately zeroed, uncalibrated, or poorly leveled. Also, actions we take to optimize the dynamic response of a system (altering natural frequency and damping) can change the reported pressure results and mislead the clinician.

Acknowledgments

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13

Direct Systemic Arterial Blood Pressure Monitoring

Edward Cooper and Stacey Cooper

Arterial blood pressure (ABP) measurement is one of the major hemodynamic monitoring tools used in patient assessment because adequate systemic blood pressure is required to perfuse vital organs. ABP, or more specifically mean arterial blood pressure (MAP), is a function of cardiac output (CO) and systemic vascular resistance (SVR). This relationship is represented by the following equation:

$$\mathrm{MAP} = \mathrm{CO} \times \mathrm{SVR} \tag{13.1}$$

While ABP is often measured to assess whether systemic blood pressure is adequate to perfuse vital organs, as equation 13.1 indicates, a normal blood pressure value does not guarantee adequate blood flow because MAP is affected by vascular tone. The body's compensatory response to homeostatic insult, largely mediated by the sympathetic nervous system, results in tachycardia and vasoconstriction and serves to sustain blood pressure and core organ perfusion. Increasing SVR through vasoconstriction can diminish flow to peripheral tissues, even when ABP is maintained. Therefore, just because blood pressure is normal does not mean blood flow is normal or that global tissue perfusion is adequate. Hypotension occurs only when sympathetic compensatory mechanisms have failed after an insult (decompensated shock).

Other monitoring techniques including serial physical examination, assessment of blood lactate concentration or central venous hemoglobin saturation, the determination of cardiac output, or even direct imaging of the microcirculation potentially offer greater insight into blood flow and tissue perfusion. However, apart from serial physical examination and blood lactate concentration, these techniques are of limited availability and largely impractical for most practitioners. Despite the limitations of the information provided by its measurement, ABP is thus still the most used modality to assess hemodynamic stability (following examination) in veterinary medicine. As the level of care provided to veterinary patients continues to grow, especially in a critical care setting, the value and availability of direct arterial blood pressure (dABP) monitoring has increased significantly. This chapter explores the practical and technical aspects of dABP measurement in small animals.

Indications for Direct Arterial Pressure Monitoring

Blood pressure measurements provide insight into the cardiovascular status of a patient. In patients that are critically ill or have cardiovascular compromise, it is generally accepted that dABP monitoring is more accurate than methods used to obtain blood pressure indirectly (i.e. Doppler or oscillometric monitors) [1–6]. Direct ABP measurement is considered to be the "gold standard" for blood pressure monitoring. There are numerous clinical scenarios in which accurate and continuous dABP monitoring would be beneficial (Box 13.1) [7]. The information obtained using dABP measurement can be used to help tailor administration of medications affecting blood pressure (e.g. titration of vasopressors or antihypertensive medications) or to help guide fluid therapy (e.g. resuscitation of hypovolemic shock) on a minute-to-minute basis [6]. To further understand why dABP measurement might be chosen over indirect blood pressure measurement methods, it is important to understand the benefits and limitations of each modality.

Advantages and Disadvantages of Indirect Blood Pressure Monitoring

As indirect arterial blood pressure (iABP) monitoring is covered in depth in Chapter 14, in this chapter it is only briefly discussed in the context of comparison with dABP

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Companion website: www.wiley.com/go/burkitt/monitoring

Box 13.1 Clinical Scenarios Benefitting from Direct Arterial Blood Pressure Monitoring [7]

- Patients in shock with hypotension or cardiovascular collapse
- Patients requiring vasopressors
- Titration of medications for afterload reduction in patients with congestive heart failure
- Patients receiving pharmacotherapy for severe hypertension
- Patients being mechanical ventilated
- Patients with high anesthetic risk

monitoring. Indirect methods (such as Doppler ultrasound or oscillometry) are noninvasive to the patient and therefore do not require arterial catheterization or setting up a fluid-filled monitoring system. As such, indirect methods are generally less technically demanding. While there is cost associated with acquiring equipment for iABP monitoring, it is typically less expensive than the equipment required for dABP measurement (e.g. pressure transducers, special hemodynamic monitors). For these reasons, iABP is measured more commonly than dAPB in both human and veterinary medicine.

Perhaps the greatest limitation to the use of iABP monitoring is its accuracy. Direct ABP measurement has been shown to be more accurate in both dogs and cats, whether awake or anesthetized [1–6]. This appears to be especially true in hypotensive, hypothermic, and small patients [3, 8]. There are factors such as cuff size, differences in technique, and the possibility of operator error that can further affect the reliability of iABP determination. In addition, iABP measurement provides less information than does dABP measurement. For example, Doppler ultrasound technique only measures *systolic arterial pressure* (SAP), or potentially more so, MAP for cats [5], whereas the direct method measures systolic, diastolic, and mean arterial pressures. While oscillometric machines provide all three pressures, standard oscillometry may be less reliable in cats and small dogs compared with dABP or Doppler ultrasound measurement techniques [2, 4, 6, 9].

Advantages and Disadvantages of Direct Arterial Pressure Monitoring

Direct ABP measurement offers the benefit of beat-to-beat blood pressure monitoring. This allows clinicians and technicians to monitor trends in both blood pressure and arterial waveform, which facilitates rapid recognition of changes in status and prompt intervention. Continuously visible blood pressure values allow the technician to perform other treatments and monitoring, rather than spending time making frequent iABP measurements. The "hands-off" nature of dABP monitoring, once it is established, may also minimize the effect that patient handling can have on the ABP values. In addition to its role in hemodynamic monitoring, placement and maintenance of an arterial catheter also allows for repeated arterial blood sampling to monitor acid–base and blood-gas parameters, which are typically of interest in critical care (see Chapter 8 for more information on arterial catheterization and sampling).

Although the technique may be more accurate in critically ill patients, dABP monitoring is not without drawbacks and complications. Obtaining and maintaining arterial access can be technically challenging. Further, the equipment necessary to monitor dABP can be expensive compared with the techniques used for indirect measurement. While it is considered the most accurate method for blood pressure determination, there are numerous technical and mechanical factors that can interfere with blood pressure signal transduction and overall accuracy of the readings, making even this gold standard prone to error. Technical issues that contribute to inaccuracy (overdamping, underdamping, zeroing errors, etc.) are discussed in detail in Chapter 12. Potential complications associated with arterial catheterization include hematoma or bleeding at the catheter insertion site, infection, arterial thrombosis and associated tissue ischemia, and significant hemorrhage if the transducer system becomes disconnected.

Continuous Direct Arterial Blood Pressure Equipment and Setup

Once the decision is made to measure dABP, all the necessary equipment must be available (see Chapter 12, Protocols 12.1–12.4).

The first step in establishing dABP monitoring is obtaining arterial access. Arterial catheter placement is discussed in depth in Chapter 8; what follows here is a brief overview. Placement of an arterial catheter can be done percutaneously or by a cutdown method. The most common arteries used for dABP monitoring in small animals are the dorsal pedal and femoral, though coccygeal or auricular can also be used. There appears to be no significant difference in the accuracy of pressures obtained from a peripheral compared to a central arterial catheter in people, particularly with regard to MAP [10]; however, there may be significant differences between central versus peripheral blood pressure measurement in dogs, particularly with regard to SAP [11]. The same may be true for cats.

Patient size plays a role in catheter insertion site since dorsal pedal access can be challenging in cats and small dogs. Femoral or coccygeal arteries may be better options in these patients. The catheter site should be clipped and

aseptically prepared. Subcutaneous local anesthetics such as 2% lidocaine can be injected locally prior to the procedure to decrease patient discomfort, especially if a cutdown is performed. Specialized arterial catheters are commercially available; they are generally more rigid, may contain a guide wire to facilitate placement, and are intended for longer-term use. These catheters are more typically used for femoral arterial access. More commonly an over-the-needle peripheral intravenous (IV) catheter is used. Once the area has been prepared, the artery is palpated and the catheter advanced through the skin toward the pulse. Given the relatively small lumen of arteries compared with veins, it is important that small incremental advances are made until there is a flash of blood in the catheter hub. Once this occurs the catheter is advanced off the stylet and into the artery. The catheter is secured with tape and appropriate protective wrap, keeping the insertion site clean and dry. It is further important to label the catheter as "arterial line" so that injections intended for IV administration are not inadvertently introduced arterially (Figure 13.1). The insertion site should be inspected daily to ensure there are no signs of bleeding or inflammation. Warmth of the extremity distal to the insertion should be assessed regularly to monitor for arterial thrombosis.

Once arterial access has been established, the pressure transducer and monitoring system can be attached to the catheter (see Chapter 12, Protocols 12.1–12.4). At one end, the pressure transducer is attached via an administration set to a pressurized 500 ml or 1.0 l bag of 0.9% NaCl to which heparin has been added (to achieve a 1–2 iu/ml concentration). The pressure bag must be inflated to a pressure greater than the patient's systolic blood pressure or blood will flow back into the line. Typically, a pressure between 250 and 300 mmHg is adequate unless the patient has significant hypertension. Heparinized saline is flushed through the system to prime the tubing, making sure to evacuate any air bubbles. When connected to the pressurized saline bag, the transducer will allow a slow forward flow of fluid through the system to decrease the risk of clot formation and catheter occlusion. It is important to check the manufacturer's materials for exact flow rates through a given transducer as this could result in a significant amount of fluid administration for smaller or fluid-restricted patients. Most pressure transducers also have a unidirectional flush valve ("fast flush valve") that can be used to prime the noncompliant tubing (Figure 13.2). At the other end of the transducer is rigid, noncompliant ("high-pressure") tubing that will be attached to the arterial catheter (Figure 13.2). If your transducer system does not come equipped with noncompliant tubing, you will need to supply your own and use it to complete the circuit. It is important that standard extension tubing is not used for this purpose, as its compliant nature will result in signal distortion and affect the accuracy of blood pressure readings (see Abnormal Arterial Pressure Waveforms, below). Once the tubing is connected to the patient, the catheter is flushed using the fast flush valve to verify patency. The use of Luer lock adapters (such as Luer-Lok® from Becton, Dickinson and Company, Franklin Lakes, NJ) throughout the system aids in safety and integrity. Finally, the pressure transducer is connected to the hemodynamic monitor via a transducer cable.

Before you can begin monitoring patient blood pressure you must zero the transducer. This sets a reference point (called a zero point) to which the pressure readings from the system are compared. To zero the system, the transducer should be placed at the level of the right atrium to best approximate central venous pressure. If peripheral pressures are preferred, the transducer should be placed at



Figure 13.1 Labeling arterial catheters so there is not confusion or inadvertent use for administration of fluids or medications.

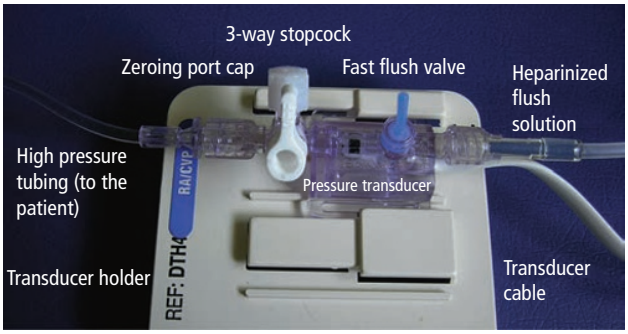


Figure 13.2 Pressure transducer for a fluid-filled hemodynamic monitoring system.



Figure 13.3 Multiparameter hemodynamic monitor screen capture demonstrating standard output of arterial waveform with systolic and diastolic pressures displayed. Simultaneous electrocardiogram and pulse oximetry waveforms are displayed above the arterial waveform.

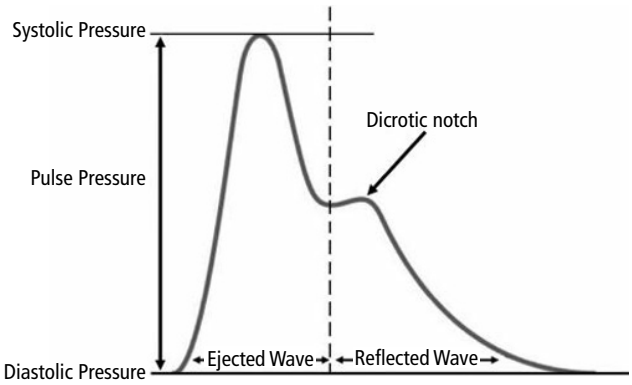


Figure 13.4 Idealized arterial pressure waveform.

the level of the catheter. Once the transducer is positioned, the stopcock is closed to the patient and opened to the atmosphere, and the "zero transducer" or similar button on the monitor is engaged. The waveform line should flatten and the screen should read "0/0 (0)." When zeroing is complete the stopcock is closed to the atmosphere and opened to the patient and the arterial waveform should appear on the screen, providing continuous ABP measurements (Figure 13.3).

Normal Arterial Pressure Waveforms

The waveform generated by the hemodynamic monitor reflects the pressure changes transmitted along the arterial tree and sensed by the transducer. An idealized schematic of an arterial pressure waveform is depicted in Figure 13.4. The "baseline" of the waveform represents *diastolic arterial pressure* (DAP) and indicates the minimum blood pressure, which is present during ventricular relaxation (diastole). DAP is a function of blood viscosity, arterial distensibility, SVR, and the length of the cardiac cycle [12, 13]. The initial upstroke in the waveform represents the rapid rise in arterial pressure from DAP to SAP, which occurs with aortic valve opening and stroke volume ejection. SAP represents the peak blood pressure during ventricular contraction (systole), and its determinants are stroke volume, velocity of left ventricular ejection, SVR, arterial distensibility, and left ventricular preload [13, 14]. The difference between DAP and SAP is called the *pulse pressure*, which is responsible for the intensity of palpated peripheral pulses. As the stroke volume runs off into the arterial tree toward the end of systole, there is a decline in pressure (initial downslope). Once aortic pressure exceeds left ventricular pressure (with left ventricular relaxation), the aortic valve closes. Elastic recoil of the arterial tree in the presence of a closed aortic valve causes a slight rebound (increase) of ABP and results in the dicrotic notch, also called the incisura (Figure 13.4). This notch causes disruption in the downslope of the waveform as pressures return to diastolic values. The presence of the dicrotic notch is largely a function of arterial elasticity and can be diminished or absent due to vasoconstriction [15].

There are changes in waveform appearance as the pressure wave moves from central to peripheral arterial circulation. This phenomenon is referred to as distal pulse amplification. As such there can be slight differences in tracings obtained depending on where the catheter tip is located. In general, the initial upstroke becomes steeper, the systolic pressure increases, the dicrotic notch appears later, and the end-diastolic pressure decreases as the waveform moves from central to peripheral (Figure 13.5) [16]. Despite the higher SAP and wider pulse pressure obtained peripherally, the lower peripheral DAP results in little net effect on the MAP from central to peripheral measurement sites. Preservation of MAP throughout the arterial tree is supported by a study comparing central versus peripheral ABP measurement in anesthetized dogs [11].

In addition to differences in arterial pressure and waveform referable to catheter location, there can also be normal, minor variations in blood pressure seen between inspiration and expiration with spontaneous or mechanical ventilation. During spontaneous breathing, SAP is slightly lower during inspiration than it is during expiration. During mechanical ventilation the opposite is true: SAP slightly increases during inspiration and decreases during expiration (Figure 13.6). Arterial pressure changes during the respiratory cycle because alterations in pleural pressure affect thoracic vasculature and cardiac function, which in turn cause changes in stroke volume [17]. During

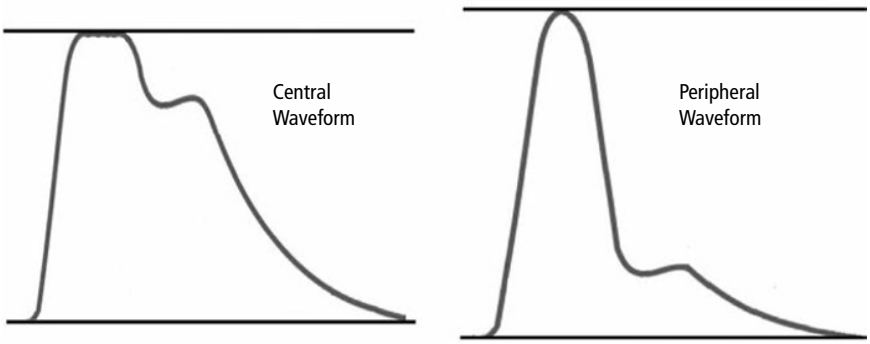


Figure 13.5 Comparison of idealized arterial waveforms from a catheter placed either centrally (left) or peripherally (right).

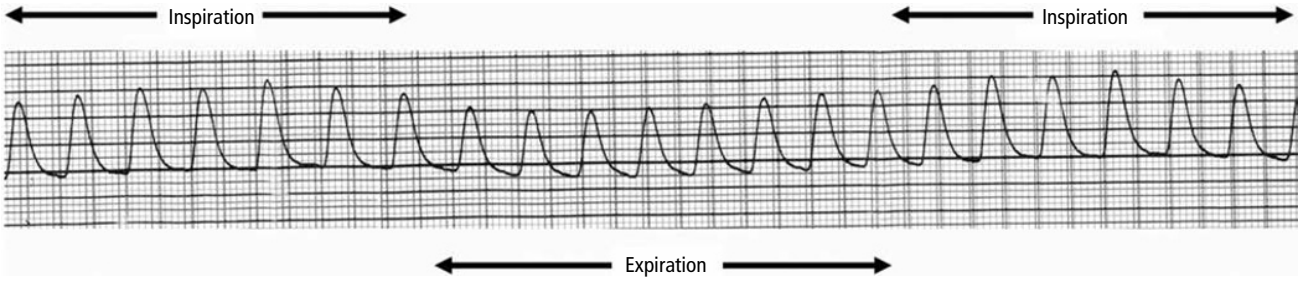


Figure 13.6 Respiratory-associated variation in arterial pressure for a patient undergoing mechanical ventilation.

mechanical inspiration, any positive pressure transmitted across the lung to the pleural space results in an increase in left ventricular preload and a decrease in left ventricular afterload. The net effect is an increase in left ventricular stroke volume and thereby SAP. However, pleural pressure changes result in decreased stroke volume leading to decreased SAP during passive expiration in the mechanically ventilated patient. Under normal circumstances this pressure variation, which typically does not exceed 5 mmHg, is not clinically significant [17]. However, as discussed later, there are certain pathological conditions that can lead to exaggeration of this respiratory cycle-related arterial pressure variation, making the variation more important both diagnostically and therapeutically.

Normal ABPs in dogs have been reported in the ranges of 110–190 mmHg for systolic and 55–110 mmHg for diastolic pressure, whereas cats have systolic pressure ranges of 120–170 mmHg and diastolic pressures ranging from 70–120 mmHg [8]. MAP normally ranges from 80 to 130 mmHg in both species.

Calculations Derived from the Arterial Pressure Waveform

MAP is generally considered superior to systolic pressure as an indicator of true driving pressure for tissue perfusion [18]. In addition, MAP is much less susceptible to variability associated with catheter location and transducer signal distortion. As such, determination of MAP is important for assessing clinical status as well as guiding therapeutic decisions. Using dABP measurement, most hemodynamic monitors calculate MAP by averaging the area under the arterial pressure waveform over several beats. It is also possible to approximate MAP through a calculation based solely on SAP and DAP. Based on the premise that approximately two-thirds of the cardiac cycle is spent in diastole, the equation:

$$\mathrm{MAP} = \mathrm{DAP} + \left[\left(\mathrm{SAP} - \mathrm{DAP}\right)/3\right] \tag{13.2}$$

provides a good estimate of MAP. However, patients with tachycardia have decreased diastolic filling time, and thus equation 13.2 will underestimate MAP (i.e. actual MAP will be closer to SAP than calculated). In addition, this equation may overestimate MAP in patients with narrow arterial pulse pressure waveforms, since narrow waveforms have a smaller area under the curve (and thereby a lower true MAP), regardless of the SAP and DAP.

Calculations of Arterial Blood Pressure Variation

As previously stated, there are normally minor variations in SAP during both spontaneous and mechanical ventilation. Hypovolemia magnifies this effect because during

hypovolemia the heart and the thin-walled intrathoracic vessels (such as the vena cava and pulmonary veins) are more collapsible [17]. Under such circumstances, the changes in pleural pressure that occur during the respiratory cycle can have more significant hemodynamic impact and thus result in greater pressure variation. This phenomenon led to the notion that respiratory cycle-associated arterial pressure variation could be used as an indicator of volume responsiveness in mechanically ventilated patients. Spontaneously breathing patients generally have wide variation in tidal volume, and thereby variable changes in intrathoracic pressures, which unfortunately makes respiratory effects on arterial pressure less consistent and interpretation challenging. Systolic pressure variation (SPV) and pulse pressure variation (PPV), dynamic markers of respiratory cycle-associated arterial pressure variation, have been explored as indicators of volume responsiveness for mechanically ventilated patients and are discussed here briefly:

Systolic Pressure Variation

SPV is either the absolute difference between the maximum systolic pressure (SP_{max}) present during inspiration and the minimum systolic pressure (SP_{min}) present during expiration, or as a percentage indexed against the average systolic pressure:

$$SPV = SP_{max} - SP_{min}\ or \tag{13.3}$$

$$SPV(\%) = (SP_{max} - SP_{min}) / [(SP_{max} + SP_{min})/2] \times 100 \tag{13.4}$$

An SPV greater than 10 mmHg has been shown to correlate to hypovolemia in human patients [19]. In addition, in people SPV has been shown to correlate with pulmonary capillary wedge pressure and left ventricular end-diastolic area, both correlates of intravascular volume status [20, 21]. Recent investigation suggests some potential application in dogs, with one study showing an SPV greater than 4.5% consistent with volume responsiveness [22]. Another study also found reasonable correlation to parameters of volume responsiveness in canine goal-directed therapy [23].

Pulse Pressure Variation

PPV offers yet another way to quantify respiratory variation in arterial pressures associated with ventilation (Figure 13.7). PPV is obtained by dividing the difference between the maximum and minimum pulse pressures (PP_{max} and PP_{min}) over a single breath by the mean of the two values [24]. The PPV, expressed as a percentage, is given by the following equation:

$$PPV(\%) = 110 \times (PP_{max} - PP_{min}) / [(PP_{max} + PP_{min})/2] \tag{13.5}$$

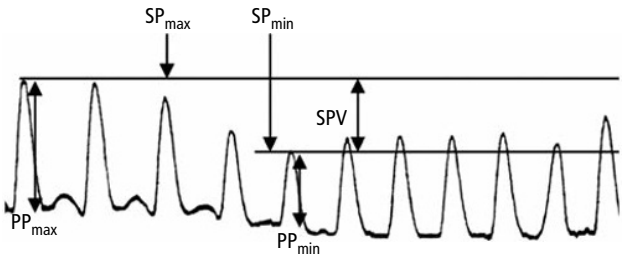


Figure 13.7 Variables used to determine volume responsiveness based on ventilation-associated variation in blood pressure. PP_{max}, maximum pulse pressure; PP_{min}, minimum pulse pressure; SP_{max}, maximum systolic pressure; SP_{min}, minimum systolic pressure; SPV, systolic pressure variation [17]. *Source:* Adapted from Michard 2005.

Compared with SPV, *PPV appears to have stronger correlation to hypovolemia and volume responsiveness,* with higher PPVs correlating to greater degrees of volume responsiveness in human studies [25, 26]. Several studies have also investigated the use of PPV in canine patients, showing good correlation and suggesting values >11–16% being consistent with a volume-responsive state in a variety of clinical circumstances [23, 27–29].

There are several limitations to using these techniques. Perhaps the most significant is that, as previously mentioned, ABP variation equations can only be used in patients undergoing positive-pressure ventilation. This limits the application to patients needing ventilatory support for hypoxemia, hypoventilation, or during anesthesia. In addition, factors such as technical issues, the presence of arrhythmias, effects of chest wall and lung compliance, or right or left ventricular failure could all interfere with the accuracy and utility of these values in determining volume responsiveness [17].

Other Uses for the Arterial Pressure Waveform

In addition to use in assessment of volume status, arterial waveforms have also been used to determine cardiac output through pulse contour analysis. Pulse contour analysis provides beat-to-beat cardiac output values based on computation of the area under the systolic portion of the arterial pressure curve after calibration with a known cardiac output (typically determined by either lithium dilution or thermodilution). Cardiac output determination from the arterial pressure waveform requires additional equipment and software. Available systems include PulseCO™ (LiDCO Ltd., London, UK), PiCCO (Pulsion Medical Systems, Munich, Germany), and Flotrac® (Edwards Lifesciences, Irvine, CA), all of which have been validated in a variety of clinical scenarios in humans [30, 31]. While potentially useful in clinical veterinary medicine, cardiac

output determination by pulse contour analysis has been shown to have poor correlation compared with lithium dilution or thermodilution techniques and frequent recalibration is required [32–34].

Abnormal Arterial Pressure Waveforms

Recognition of abnormal pressure waveforms is an essential component of dABP monitoring (Protocol 13.1). Alterations in waveform morphology could reflect true changes in clinical condition, thereby warranting intervention for the patient. On the other hand, they could indicate a technical or mechanical issue that would require troubleshooting the system rather than the patient.

Technical Problems that Cause Abnormal Arterial Pressure Waveforms

One of the major technical issues that can arise with use of a dABP transducer system is pressure waveform overdamping or underdamping. Damping is the inherent tendency for the system itself to alter the pressure signal as it is transmitted from the patient to the transducer. Underdamping occurs when the resonant frequency of the monitoring system too closely matches the frequency of the pressure waveform. The result is a summation or resonance of the two frequencies, amplification of the signal, overestimation of SAP, and underestimation of DAP. All dABP monitoring systems have some inherent underdamping effects and, as such, tend to report falsely high SAPs and falsely low DAPs. The degree of inaccuracy can be minor or significant. The MAP reported is generally considered accurate. Normal arterial pressure waveforms have no "pointy" or jagged parts – waveforms with points or sharp peaks are therefore likely underdamped. The length of tubing connecting the arterial catheter to the transducer can contribute to underdamping in a direct relationship – increased length of tubing worsens underdamping.

Overdamping, on the other hand, results in attenuation or muting of the arterial pressure waveform, leading to falsely low SAP and falsely elevated DAP. The net effect is a significant reduction in the pulse pressure though the MAP generally remains accurate. Overdamped waveforms are overly smooth with loss of many defining characteristics, such as the systolic upstroke and dicrotic notch (Figure 13.8). Potential causes of overdamping include air bubbles in the line, line occlusion from kinking or clotting, or use of overly compliant tubing. More detail concerning system damping and technical issues of fluid-filled monitoring systems can be found in Chapter 12.

Patient Problems that Cause Abnormal Arterial Pressure Waveforms

Alterations in arterial waveforms can also manifest because of significant changes in patient hemodynamics. For example, various arrhythmias can impact cardiac output and blood pressure, and can result in diminished to completely absent arterial waveforms despite the presence of electrical activity (Figure 13.9).

Significant hypotension secondary to low cardiac output (as from hypovolemic or cardiogenic shock) can result in muted waveforms secondary to small stroke volumes combined with peripheral vasoconstriction. As such, pressure waveforms from patients with hypotension can be difficult

Protocol 13.1 Suggested Step-by-Step Approach for Assessing Direct Arterial Blood Pressure

Procedure

1) Determination of arterial blood pressure:
   a) What is the reported systolic pressure?
   b) What is the reported diastolic pressure?
   c) What is the reported or calculated mean pressure?
   d) What is the calculated pulse pressure?
   e) Do these values reflect hypotension or hypertension?
   f) Do these values match the patient's clinical condition?
   g) Do these values coincide with palpated pulse quality?
   h) Is there significant ventilation-associated variation?
2) Assessment of pulse rate and rhythm:
   a) What is the pulse rate reported by the monitor?
   b) Does this value reflect bradycardia or tachycardia?
   c) Does the rate match the auscultated and ECG heart rate?
   d) Is the pulse rhythm regular or irregular? Does it match changes in the ECG (i.e. is there a pulse waveform for each QRS complex)?
3) Assessment of waveform morphology:
   a) Has the waveform morphology changed significantly?
   b) Is the morphology consistent from beat to beat?
   c) Is a dicrotic notch present?
   d) Has the waveform become muted (significant decrease in pulse pressure)?



Figure 13.8 Arterial waveform from a patient with sudden and marked overdamping of the pressure signal (arrow) associated with catheter occlusion. Note the sudden loss of waveform morphology, slight decrease in systolic pressure, increase in diastolic pressure, and loss of dicrotic notch, all without any change in heart rate.

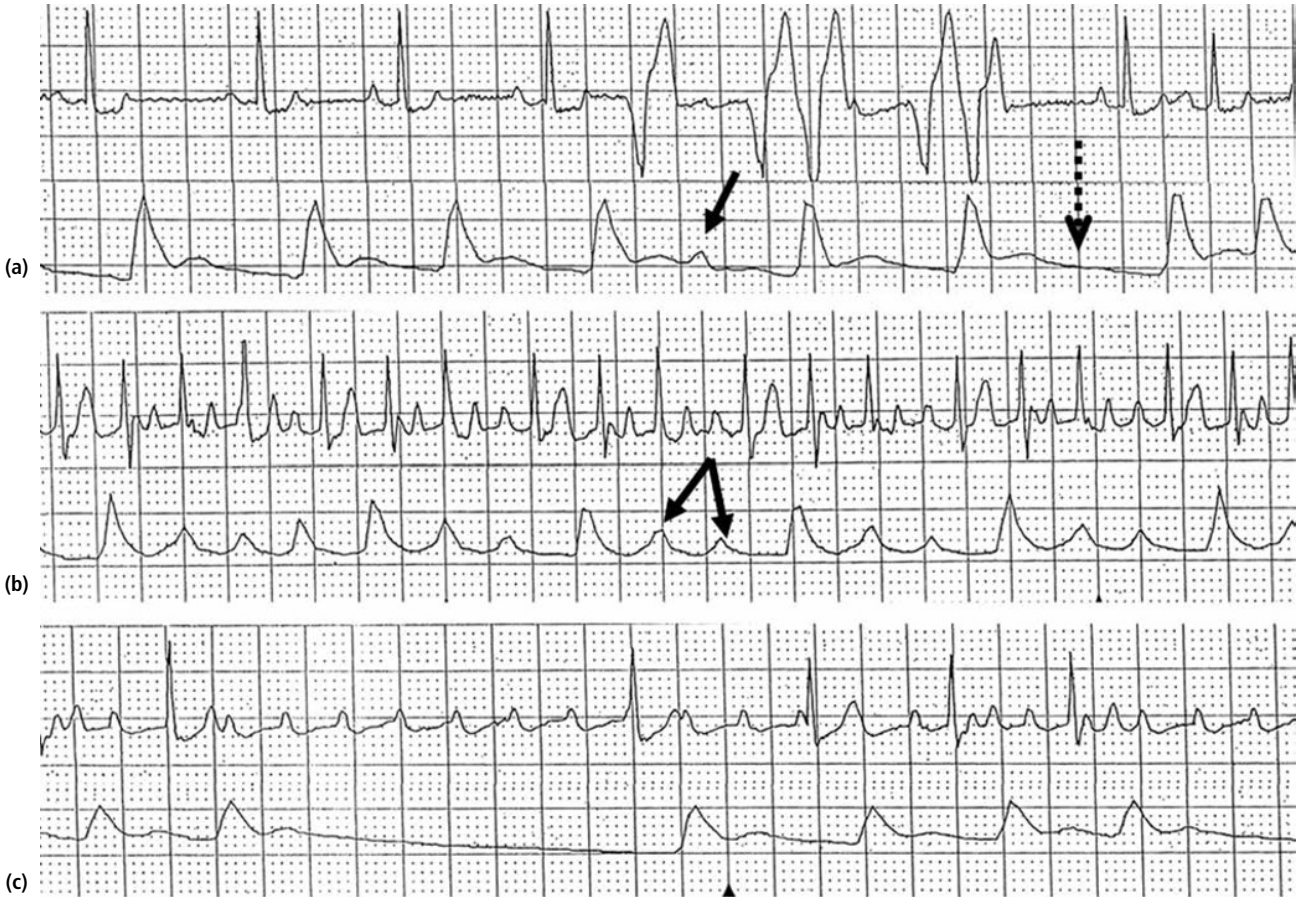


Figure 13.9 Examples of arrhythmia-associated changes in arterial waveform morphology. (a) Ventricular premature contractions associated with diminished (solid arrow) to absent (dashed arrow) pressure tracings. (b) Tachycardia (heart rate 210 beats/minute) resulting in progressively diminished waveforms and blood pressure (arrows). (c) Atrial flutter with prolonged periods of absent ventricular contraction resulting in absent arterial waveforms.

to distinguish from those caused by overdamped systems. Clearly, recognizing the difference is essential to taking appropriate action if the patient is truly hypotensive. The patient's clinical status (mental responsiveness, heart rate, manual palpation of pulses) as well as the MAP (remembering that MAP is typically preserved with overdamping and will be low with hypotension) can be helpful to distinguish between the two. Alternately, a fast flush test (described in Chapter 12) can be performed to assess the system for damping.

Another manifestation of respiratory arterial pressure variation can occur in the form of pulsus paradoxus, most

commonly associated with pericardial effusion that has resulted in cardiac tamponade. As with hypovolemia, the decrease in venous return from increased pericardial pressure results in an exaggeration of the difference between the SAP during inspiration and the SAP during expiration. Provided the patient is breathing spontaneously, SAP will be higher on expiration and lower on inspiration (Figure 13.10a).

Finally, there are certain clinical scenarios whereby a patient has waveforms with increased pulse pressure ("tall") but are of fairly short duration ("narrow;" Figure 13.10b). This morphology is typically caused by an increased SAP and a very rapid falloff to DAP, the latter occurring either because of decreased blood viscosity, peripheral vasodilation, or backward flow of blood. Potential causes of "tall and narrow" waveforms, also referred to as water hammer or Corrigan's pulses, include aortic regurgitation, patent ductus arteriosus, hypertension, sepsis, and dilutional anemia [35].

Thresholds of Concern for Arterial Pressure Value and Waveform Abnormalities

Significant changes in blood pressure or waveform morphology should prompt assessment of clinical condition and intervention as indicated (Table 13.1). Onset or worsening of hypotension, generally defined as SAP less than 80 mmHg or MAP less than 60 mmHg, should be addressed as soon as possible to limit tissue hypoxia and potential for cardiac arrest [8]. Along similar lines, marked hypertension, generally defined as SAP greater than 180–200 mmHg or MAP greater than 140 mmHg, could cause significant end-organ injury and requires intervention [36]. The presence of ventilatory variation in systolic pressure greater than 10 mmHg is consistent with hypovolemia (prompting fluid resuscitation) or pericardial effusion (prompting pericardiocentesis). Arrhythmias that result in a sustained impact on blood pressure or even intermittent hypotension may require antiarrhythmic medications or a pacemaker. This could include tachycardia with heart rate above 180–200 beats/minute

Table 13.1 Guidelines indicating need for clinician intervention based on direct arterial blood pressure monitoring.

Change assessed by dABP	Measurement
Hypotension [7]	SAP < 80 mmHg
	MAP < 60 mmHg
Hypertension [33]	SAP > 180–200 mmHg
	MAP > 140 mmHg
Arrhythmias [35]	Tachyarrhythmias:
	HR > 180–200 (dog)
	HR > 240 (cat)
	Bradyarrhythmias:
	HR < 60 (dog)
	HR < 80–100 (cat)

HR, heart rate; MAP, mean arterial pressure; SAP, systolic arterial pressure.

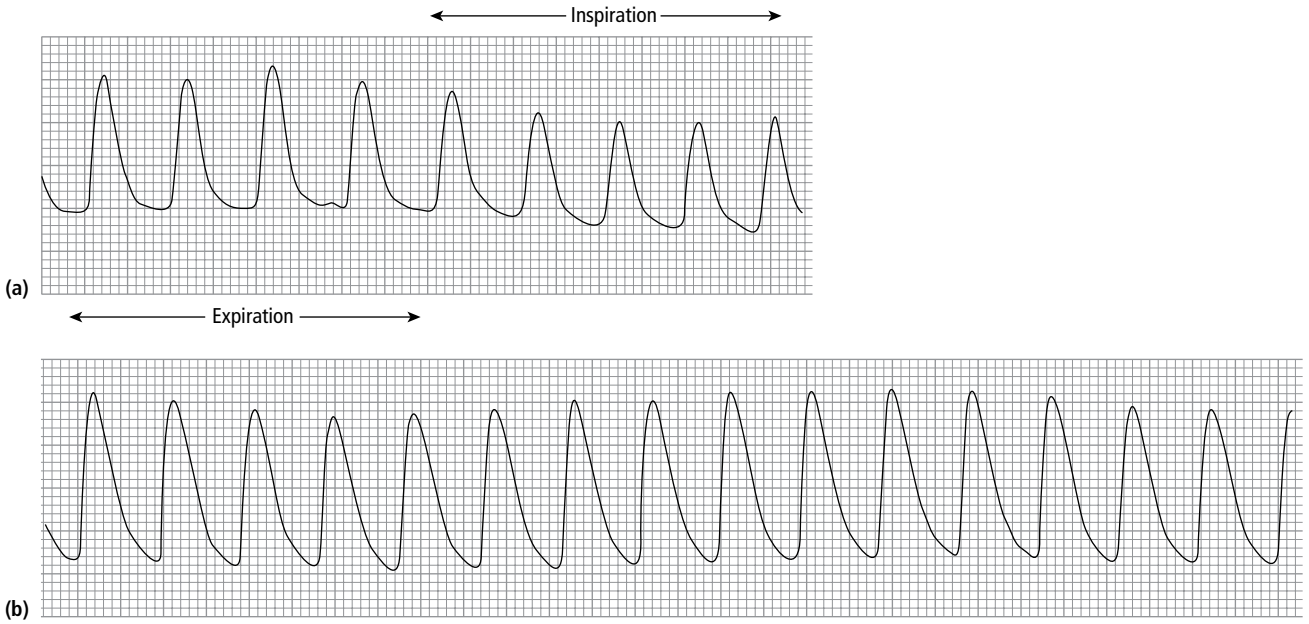


Figure 13.10 (a) Arterial waveforms from a patient with pulsus paradoxus demonstrating respiratory-associated arterial pressure variation. (b) Arterial waveforms from a patient with dilutional anemia demonstrating "tall and narrow" morphology.

(bpm) in dogs or 240 bpm in cats, or bradycardia with heart rate less than 60 bpm in dogs and 80–100 bpm in cats [37].

Troubleshooting Abnormal Waveforms

One of the primary objectives in troubleshooting abnormal waveforms is to determine whether changes in blood pressure or waveform morphology truly reflect changes in patient hemodynamics or if they are due to technical or mechanical issues. These issues arise most commonly with the apparent presence of significant hypotension (and muting of the arterial waveform) or hypertension (and exaggeration of the waveform).

Several steps can be followed to systematically work through the potential causes for these changes (Protocol 13.2). When an abnormal waveform is detected, the first step is to determine whether there are concurrent changes in patient clinical status such as alteration in mentation, heart rate, or palpable pulse quality that might require immediate intervention. In addition, if not already being continuously monitored, an electrocardiogram (ECG) should be obtained to determine whether an arrhythmia is present. If there are no changes in clinical condition or ECG, the transducer setup should be assessed to ensure that the noncompliant tubing is not kinked and that there are no air bubbles present in the fluid column. It is also important to ensure the pressure bag is still inflated to 250 mmHg or higher. If there has been significant change in patient position, it may be beneficial to re-level and re-zero the transducer.

To assess patency of the arterial catheter, the catheter should be aspirated to ensure arterial blood is obtained; any air bubbles or clots should be removed. If the catheter does not readily aspirate, the system can be gently, manually flushed to assess patency and to evacuate any air bubbles or clots. A fast or forceful flush of an arterial catheter that does not readily aspirate carries the risk of introducing air or small thrombi into distal arterial circulation. If the system does not flush or aspirate readily, the arterial catheter should be closely inspected to confirm its position and functionality. If the catheter appears to be at least partially patent then a "fast flush test" (described in detail in Chapter 12) can be performed to assess for the presence of overdamping or underdamping. If underdamping is present, it may be necessary to use shorter noncompliant tubing between the pressure transducer and the arterial catheter. If overdamping is present and does not resolve with flushing, it may be necessary to confirm the presence of noncompliant rather than standard extension tubing between the catheter and the transducer, and to assess all system connections, lines, and the arterial catheter itself for bubbles, kinks, or other abnormalities that might interrupt pressure wave transmission.

Conclusions

Despite the potential limitations and technical difficulties associated with its use, dABP monitoring offers valuable information regarding hemodynamic status. If equipment is available and one becomes accustomed to the process, dABP measurement could be used in any 24-hour veterinary hospital and is especially useful if critically ill patients are routinely seen.

Protocol 13.2 Troubleshooting Abnormal Arterial Pressure Waveforms

Procedure

1) Assess patient for changes in clinical status to explain change in morphology:
   a) Assess mentation, heart rate, pulse quality, mucous membrane color, capillary refill time, and extremity temperature.
   b) Assess ECG for changes in rate and rhythm coinciding with changes in waveform.
2) If patient parameters have not changed:
   a) Assess transducer setup and make sure:
      i) Noncompliant tubing is used between patient and transducer.
      ii) Noncompliant tubing is not kinked.
      iii) There are no air bubbles in the fluid column.
      iv) Pressure bag is inflated to ≥ 250 mmHg.
   b) Re-level transducer to level of the patient's heart base and re-zero the transducer if patient position has changed significantly.
   c) Assess patency of arterial catheter:
      i) Aspirate arterial catheter.
         - Ensure arterial blood is easily obtained.
         - Remove air bubbles and clots from line.
      ii) Flush arterial catheter.
         - Ensure catheter flushes easily – do not force.
         - Evacuate air bubbles or small clots from system.
3) Perform "fast flush test" to assess for system overdamping or underdamping.

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14

Noninvasive Arterial Blood Pressure Monitoring

Christopher L. Norkus and Nicholas L. Rivituso

Oxygen delivery to tissue (DO_2) is required for normal cellular function. Oxygen delivery to tissue may be calculated as the product of cardiac output (CO) and arterial oxygen content (CaO_2):

$$DO_2 = CO \times CaO_2 \tag{14.1}$$

Blood pressure is the pressure that blood exerts on the vessel wall. Cardiac output, not blood pressure, is a constituent of delivery of oxygen to tissue, as shown in equation 14.1. However, the measurement of cardiac output in the clinical setting is labor intensive and challenging. Blood pressure is related to cardiac output in that it is determined by a combination of cardiac output and systemic vascular resistance (SVR). Additionally, blood pressure dictates blood delivery to certain vital organs such as the brain, heart, and kidneys. Because blood pressure is easier to measure than cardiac output and because it is of vital importance physiologically, blood pressure measurement is an important method to monitor the cardiovascular system in small animal patients. Failure to achieve and maintain adequate blood pressure is associated with increased short- and long-term morbidity and mortality in anesthetic and critical care settings [1–6]. As such, the routine evaluation of blood pressure has been integrated into numerous established human medical scoring systems such as the Simplified Acute Physiology Score, Acute Physiology and Chronic Health Evaluation, and the Sequential Organ Failure Assessment.

Blood pressure can be broken down into three distinct reported components: the systolic arterial pressure (SAP), the diastolic arterial pressure (DAP), and the mean arterial pressure (MAP). The SAP and DAP correlate to their respective phases of the cardiac cycle while MAP is the average of the arterial pressure over time. MAP is not equal to the arithmetic mean or "average" of SAP and DAP because more of the cardiac cycle is spent in diastole than systole. This principle is demonstrated by the following equation, which estimates MAP:

$$MAP = \left[\left(SAP - DAP\right)/3\right] + DAP \tag{14.2}$$

Systemic MAP is the product of cardiac output and SVR; see equation (14.3). Cardiac output is blood flow provided by the heart and is the product of stroke volume (SV), the volume of blood ejected with each heartbeat, and the heart rate (HR):

$$MAP = CO \times SVR \tag{14.3}$$

$$CO = SV \times HR \tag{14.4}$$

Stroke volume is determined by cardiac preload, contractility (inotropy) and relaxation (lusitropy), and afterload. SVR is influenced by vasomotor tone (degree of vasodilation or vasoconstriction) and blood viscosity. While increased SVR for a static cardiac output leads to increased blood pressure, the increased vascular resistance decreases blood flow, as described by Ohm's law:

$$Q = \Delta P / R \tag{14.5}$$

In this equation, Q is blood flow, ΔP is the driving pressure of blood from one point in the systemic circulation to another, and R is resistance. When cardiac output is substituted for Q (blood flow); the difference between MAP and right atrial pressure (RAP) is selected as ΔP (driving pressure); and SVR is applied for R (resistance), the equation can be rewritten as follows:

$$CO = \left(MAP - RAP\right)/SVR \tag{14.6}$$

It is important to acknowledge, therefore, that increases in blood pressure do not guarantee improvements in blood flow or oxygen delivery to tissue.

When monitoring blood pressure, it is critical to remember its determinants, since therapy should target the aberrant underlying component, whether that be preload,

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Companion website: www.wiley.com/go/burkitt/monitoring

contractility, heart rate, SVR, or a combination thereof. For example, a patient with hypotension associated with septic shock may need targeted therapy to address preload due to hypovolemia, inotropy due to decreased cardiac contractility, as well as SVR due to systemic vasodilation.

Comparison of Blood Pressure Monitoring Modalities

Blood pressure monitoring is an integral part of modern small animal emergency and critical care practice. The evolution of noninvasive arterial blood pressure (NIBP) monitors has led to easier monitoring and management of both hypertensive and hypotensive states. Invasive, direct arterial blood pressure (dABP) monitoring remains the gold standard in both veterinary and human medicine; dABP monitoring involves intra-arterial catheterization, connective tubing, a pressure transducer, and a monitor allowing continuous reporting of SAP, DAP, and MAP (see Chapter 11). Alternatively, an aneroid manometer can be used in the absence of a pressure transducer and monitor to allow for continuous evaluation of MAP.

In general, dABP monitoring is considered more accurate than indirect methods. Additional advantages of dABP monitoring are that it is continuous, allowing for beat-to-beat monitoring of the most critically ill patients, and that the arterial pressure waveform generated is helpful in monitoring and diagnosis. While it is considered the most accurate method, dABP monitoring has several drawbacks, including that it is technically challenging to establish and maintain, arterial catheterization can be uncomfortable, it poses a risk of catheter-related bloodstream infection and arterial bleeding, and that more equipment and expertise are required than for NIBP measurement. A 2017 retrospective study evaluating the use of arterial catheters in anesthetized dogs and cats did not identify tissue ischemia as an associated complication; however, 22.3% of animals evaluated had a temporary loss of peripheral pulse at the catheterized site following use [7]. Another potential drawback of dABP monitoring is increased cost to the client, as the required equipment and advanced training of nursing staff are more extensive and expensive than for NIBP measurement.

NIBP monitoring techniques depend on detection of blood flow that passes beneath an occluding cuff, though the sensor differs between methods. In general, all NIBP techniques offer the advantages over direct techniques of being faster, noninvasive, easier to perform, and requiring relatively minimal technical expertise. The primary disadvantages of NIBP monitoring are that they have relatively poor accuracy during peripheral vasoconstriction (e.g. hypovolemic shock); are less accurate at high and low pressures and heart rates compared with dABP methods; may fail to perform accurately during arrhythmia; and that they are not truly beat-to-beat continuous. The Doppler method additionally requires that an operator be physically involved during each measurement. Despite these limitations, given the technical requirements and costs associated with dABP monitoring, NIBP has become a commonplace method to obtain arterial blood pressure measurement.

Validation of Noninvasive Arterial Blood Pressure Monitoring

The ease and widespread availability of NIBP monitoring has made it the most common for measuring blood pressure in veterinary emergency and critical care medicine. Therefore, it is important that NIBP monitoring devices are adequately accurate for clinical use. Given that direct blood pressure measurement is considered the gold standard, NIBP devices have been evaluated for accuracy primarily by comparing dABP and NIBP measurements in both human and veterinary patients. The Association for the Advancement of Medical Instrumentation (AAMI) has developed guidelines for validation of NIBP measuring devices in people, which involve comparison of NIBP measurements to dABP measurements. Few indirect blood pressure monitoring devices have met these criteria in people, and to date, no indirect blood pressure measuring devices have been validated by these criteria in conscious dogs or cats [8].

Discrepancies between direct and indirect arterial blood pressure measurements can be attributed largely to the fact that the dABP technique measures blood pressure directly, whereas most indirect techniques measure some variable related to blood *flow*. The NIBP measurement techniques commonly used in small animal practice estimate blood pressure by detecting return of blood flow distal to an occlusive cuff by Doppler ultrasonic technology as the cuff is slowly released, or by sensing oscillometric arterial wall motion beneath an occlusive cuff as the cuff is slowly released. Indirect methods are also somewhat limited by the fact that they require a large superficial artery on a distal extremity that can be occluded by the pressure cuff. The limited presence of these vessels, the variability in size and shape of the limbs, and the potential inaccessibility of these arteries due to trauma or peripheral venous catheters can make it difficult to obtain reliable NIBP readings in dogs and cats.

Comparison with the dABP measurement method should not be the only way in which NIBP measurement devices are judged. Inherent limitations such as patient noncompliance, significant differences in patient size and conformation, and lack of protocol standardization make NIBP measurements in dogs and cats inherently more

difficult to obtain than in people. Thus, validation of veterinary devices should likely take into consideration these inherent difficulties in our unique clinical setting. In 2007 and 2018, a panel of experts on systemic hypertension in the American College of Veterinary Internal Medicine (ACVIM) made recommendations for the validation of NIBP methods in veterinary patients [8, 9]. These recommendations are based on the AAMI guidelines for NIBP monitoring in people and state that the tested NIBP monitoring device should be compared against a dABP measurement device or another NIBP measuring device for which validation has been published in a refereed journal; that a device is validated for only the species and condition in which the validation test was conducted; and that a device may be validated for systolic, diastolic, or both types of measurements [8, 9]. The ACVIM panel stated that the investigational criteria and recommendations of the AAMI should be followed; criteria for validation of system efficacy are detailed and strict [8, 9]. Although there are no validated NIBP measuring devices for use in veterinary patients, NIBP monitoring is nevertheless more commonly used than direct monitoring in clinical practice.

Indications for Noninvasive Blood Pressure Monitoring

There are many indications for monitoring blood pressure in dogs and cats in the emergency and critical care setting. Blood pressure should be measured in patients with known hypotension or hypertension, in patients with diseases or conditions that may lead to hypotension or hypertension, and in all patients undergoing anesthesia. Table 14.1 gives arterial blood pressure values that are considered normal for dogs and cats.

Hypotension

Hypotension, or low arterial blood pressure, is not a primary disease but is rather a clinical manifestation of another problem. Hypotension most commonly results from inadequate SVR, cardiac preload, cardiac contractility, heart rate, or a combination thereof. Common conditions that may result in hypotension include congestive heart failure, sepsis, anaphylaxis, hemorrhage, severe vomiting or diarrhea, trauma, and gastric dilatation–volvulus. Anesthesia itself is also a common cause of hypotension in dogs and cats. Many injectable and inhalant anesthetic agents cause peripheral vasodilation and depression of cardiac contractility, either of which can result in hypotension. The 2009 updated recommendations for monitoring anesthetized veterinary patients by the American College of Veterinary Anesthesia and Analgesia state that blood pressure monitoring should be performed for all patients [11]. NIBP allows monitoring of trends and allows for rapid therapeutic intervention.

Table 14.1 Normal arterial blood pressure values in dogs and cats [10].

Arterial pressure	Values (mmHg)	
	Dogs	Cats
Systolic	90–140	80–140
Diastolic	50–80	55–75
Mean	60–100	60–100

Source: Simmons and Wohl, 2009/with permission of Elsevier.

Hypertension

Hypertension is a sustained increase in systemic blood pressure. Systemic arterial hypertension is becoming more commonly recognized as a complication of several disease processes in dogs and cats. This increased index of suspicion for hypertension has made screening and monitoring with NIBP measurement devices invaluable in clinical practice. There are two clear indications for measuring blood pressure in the context of hypertension: evidence of end-organ damage consistent with a hypertensive episode and the presence of a disease or condition that is known to cause hypertension [8, 9]. The four major end-organs affected by hypertension are the kidneys, eyes, brain, and heart [8, 9]. Sustained or acute increases in blood pressure can have detrimental effects on these organ systems. See Table 14.2 for specific clinical signs of "end-organ" damage. Table 14.3 shows the risk for hypertension-induced target organ ("end-organ") damage.

Systemic arterial hypertension can be either situational hypertension (white coat hypertension), primary or essential (idiopathic), or secondary to another disease. Secondary hypertension can also occur in response to therapeutic agents that are known to cause hypertension, such as erythropoietin, mineralocorticoids, phenylpropanolamine, phenylephrine, ephedrine, pseudoephedrine, toceranib, glucocorticoids, and chronic, high-dose sodium chloride [8]. Additionally, intoxicants such as cocaine, methamphetamine/amphetamine, and 5-hydroxytryptophan have also been associated with secondary systemic hypertension [8]. Both dogs and cats are affected by diseases and conditions known to cause hypertension. In dogs, diseases commonly associated with hypertension include acute or chronic kidney disease, hyperadrenocorticism, diabetes mellitus, obesity, hyperaldosteronism, pheochromocytoma, brachycephaly, and hypothyroidism. In cats, chronic kidney disease, diabetes mellitus, hyperthyroidism, obesity, primary hyperaldosteronism, pheochromocytoma,

Table 14.2 Common target organ ("end-organ") damage secondary to hypertension.

Tissue	Hypertension injury	Clinical findings
Kidneys	Progression of chronic or acute kidney disease	Increased creatinine, SDMA, or decreased GFR
		Proteinuria or microalbuminuria
Eyes	Choroidopathy	Acute blindness
	Retinopathy	Exudative retinal detachment
		Retinal hemorrhage
		Hyphema
Brain	Encephalopathy	Central neurologic signs
	Vascular accident	
Heart and circulation	Left ventricular hypertrophy	Hemorrhage (e.g. epistaxis)
	Left-sided congestive heart failure	Left ventricular concentric hypertrophy
		Gallop heart sound
		Systolic heart murmur
		Increased respiratory rate/effort

GFR, glomerular filtration rate; SDMA, symmetric dimethylarginine.

Table 14.3 Risk for hypertension-induced target organ ("end-organ") damage.

Systolic arterial pressure (mmHg)	Risk classification
<140	Minimal
140–159	Low
160–179	Moderate
≥180	High

and hyperadrenocorticism are conditions associated with hypertension [8]. A clinician is likely to order blood pressure measurements in patients with these conditions for both initial screening and serial monitoring.

Noninvasive Blood Pressure Monitoring Methods

The two most common noninvasive ways to monitor blood pressure include Doppler ultrasound and oscillometric sphygmomanometry. Clues obtained from a detailed physical examination including weakness, decreased mentation, tachycardia in dogs and abnormal heart rate in cats, prolonged capillary refill time, cool extremities, decreased urine production, and pale mucous membrane color all suggest poor perfusion and should raise suspicion for hypotension. Palpation of peripheral pulses and relative pulse "quality" or pressure has also been proposed as a potential method to assess blood pressure. Pulse pressure is an assessment of stroke volume and is the difference between SAP and DAP:

$$\text{Pulse pressure} = \text{SAP} - \text{DAP} \tag{14.7}$$

The difference between SAP and DAP tends to be greater during normotension than during hypotension. Hence, patients with small pulse pressures may be more likely to have low blood pressure whereas those with normal pulse pressure may be more likely to have normal blood pressure. However, many factors, such as peripheral vasoconstriction, can influence this difference. Recent prospective evaluation of the relationship between peripheral pulse quality and Doppler SAP in dogs and cats has found weak correlation between peripheral pulse palpation and an actual blood pressure measurement [12, 13]. In dogs, the median SAP was not significantly different between animals in which femoral pulses were present versus those in which they were absent. In the same study, dogs with absent metatarsal pulses were 7.6 times more likely to be hypotensive, though palpable metatarsal pulses did not rule out hypotension [13]. Cats with absent metatarsal and femoral pulses had a median SAP of 30 mmHg with a range of measured SAPs between 30 and 105 mmHg, whereas cats with strong metatarsal pulses had a median SAP of 135 mmHg with a range of 58–210 mmHg [12]. From these studies it could be broadly said that peripheral pulse quality worsens as blood pressure decreases but that peripheral pulse quality is imprecise and should never be used as a substitute for systemic arterial blood pressure monitoring. Additionally, it is important to note that no correlation can be estimated regarding pulse pressures and hypertension.

Doppler Ultrasound

Principles

Doppler ultrasonic blood pressure monitoring is one of the most common NIBP measurement methods. In this technique, a Doppler probe containing two piezoelectric crystals is placed against the skin overlying a peripheral artery, distal to a pressure cuff applied circumferentially to the patient's limb. The Doppler probe emits ultrasonic waves into the tissue that are reflected to the probe. The difference between the emitted and returning signal frequencies, called the *frequency shift*, is detected by the transducer and converted to an audible signal emitted from the Doppler unit. Pressure is read from a sphygmomanometer connected to the pressure cuff proximal to the Doppler transducer. The cuff is inflated, which occludes the artery, until no sound is audible from the Doppler monitor's



Figure 14.1 Equipment required for Doppler blood pressure measurement including a pressure cuff of appropriate size, ultrasonic gel, Doppler machine, sphygmomanometer. *Source:* Photograph courtesy of Christopher Norkus.

speaker. The cuff is then gradually deflated until the first audible arterial sound returns, which is documented as the SAP. The use of headphones is recommended [14]. Basic equipment required is shown in Figure 14.1.

The Doppler technique is inexpensive, easy to perform, and widely used in dogs and cats. It cannot be used in dogs and cats to determine MAP or DAP. Doppler is a relatively sensitive NIBP measurement method in low-flow states and in smaller patients, compared with standard oscillometry. Doppler ultrasound has been found to have poor agreement with dABP in dogs weighing less than 5 kg; it has high specificity (97%) and poor sensitivity (56%) for detecting hypotension [15, 16]. Work in cats has suggested that measurements from Doppler ultrasound may correlate better with MAP in this species [17]. Another study in cats, however, suggested that measurements were significantly lower than SAP obtained from a femoral artery catheter and thus a calibration adjustment of 14 mmHg should be added to Doppler ultrasonic measurements to better correlate with SAP obtained by femoral dABP [18].

Cuff Selection

The pressure cuff size is key to obtaining accurate results. The cuff width should be approximately 30–40% the circumference of the limb at the point where it will be placed (Figure 14.2). If the cuff size is too small, there can be erroneously high SAP readings, whereas if the cuff is too large, the readings may be erroneously low due to compression of a larger portion of the artery. See Table 14.4 for appropriate pressure cuff sizing.



Figure 14.2 Proper measurement technique to determine size of a blood pressure cuff: approximately 30–40% of limb circumference at site of intended cuff placement. *Source:* Photo courtesy of Christopher Norkus.

Table 14.4 Cuff selection for noninvasive arterial blood pressure monitoring. The cuff width (size) should be 30–40% the extremity circumference at the site of cuff placement [8].

Cuff size (cm)	Limb circumference	
	(inches)	(cm)
1.0	1.0–1.5	2.5–3.7
2.0	1.5–2.4	3.8–6.2
3.0	2.5–3.4	6.3–8.7
4.0	3.5–4.4	8.8–11.2
5.0	4.5–5.4	11.3–13.7
6.0	5.4–6.4	13.8–16.2
7.0	6.4–7.4	16.3–18.7
8.0	7.4–8.3	18.8–21.2
9.0	8.4–9.3	21.3–23.7
10.0	9.4–10.3	23.8–26.2
11.0	10.4–11.3	26.3–28.7

Source: Adapted from Acierno et al. 2018.

Systemic arterial blood pressure is generally referenced to the level of the right atrium, which would require that the site of measurement (i.e. the blood pressure cuff) be positioned at right atrial level. If the cuff is positioned lower than the right atrium, readings will be falsely elevated; the opposite is true if the cuff is positioned above the right atrium [8, 9]. It is suggested that, if the vertical distance between the cuff and the right atrium is 4 inches (10 cm) or more, a correction factor be applied as in Table 14.5. Rather than applying correction factors, it is

Table 14.5 Correction factors for a vertical distance of ≥ 4 inches (10 cm) between the right atrium and the pressure cuff.

If the cuff is . . .	Then . . .	Because . . .
≥ *10 cm below* the right atrium	*Subtract* 0.8 mmHg for every 1 cm the cuff is below the right atrium	The reading from the blood pressure monitor is falsely high
≥ *10 cm above* the right atrium	*Add* 0.8 mmHg for every 1 cm the cuff is above the right atrium	The reading from the blood pressure monitor is falsely low

recommended that the cuff be as close as possible to the horizontal plane of the right atrium. However, these correction factors can be helpful in animals in situations that prevent the operator from repositioning the patient.

Performing Doppler Ultrasound Blood Pressure Measurement

Equipment required for Doppler ultrasonic blood pressure measurement includes clippers with blade, ultrasonic gel, medical adhesive tape, various size pressure cuffs, sphygmomanometer, and Doppler ultrasound machine. See Protocol 14.1 for detailed instructions regarding performance of Doppler ultrasonic NIBP measurements. The environment in which to measure blood pressure should be isolated, quiet, and away from other animals, and ideally the animal's owner should be present [8, 9]. The patient should not be sedated and should be allowed to acclimate to the measurement room for 5–10 minutes before a measurement is attempted [8]. Novelty and anxiety have been shown to falsely elevate blood pressure in dogs [19]. Many of the preceding recommendations are not possible in the emergency room and intensive care unit settings, but should be followed if possible. The same individual should perform all measurements following a standard protocol. Training of this individual is essential.

The patient should be gently restrained in a comfortable position, ideally in sternal or lateral recumbency to limit the vertical distance from the heart base to the cuff [8]. Measurements in dogs, however, appear to be significantly affected by body position, with sitting dogs having higher

Protocol 14.1 Doppler Ultrasonic Blood Pressure Measurement

Items Required

- Clippers with clean blade
- Ultrasonic conductance gel (Figure 14.5)
- Medical adhesive tape
- Various size pressure cuffs
- Sphygmomanometer
- Charged (or plugged in) Doppler ultrasound machine, with headphones
- Assistant, if required

Procedure

1) Collect all necessary supplies.
2) Position the patient in lateral or sternal recumbency while holding the planned cuff site at approximately the level of the right atrium. If positioning the cuff at approximately the vertical level of the right atrium is not possible, a correction factor is then used. If the cuff is ≥ 10 cm *below* the right atrium, then *subtract* 0.8 mmHg for every 1 cm the cuff is below the right atrium. If the cuff is ≥ 10 cm *above* the right atrium, *add* 0.8 mmHg for every 1 cm the cuff is above the right atrium.
3) Clip fur from area over artery.
4) Secure a deflated cuff proximal to the artery and attach the sphygmomanometer. The cuff should be of a size that is approximately 30–40% of limb circumference at the cuff's application site (Figure 14.2). When measuring Doppler on a forelimb, secure the cuff over the antebrachium. When measuring on a rear limb, secure the cuff proximal to the hock. Medical adhesive tape should be applied circumferentially around the limb on the proximal and distal edges of the cuff/limb interface to secure the cuff to the limb (Figure 14.4).
5) Apply ultrasonic gel to the concave surface of the Doppler probe. Electrode gel should not be used as this will destroy the protective coating of the Doppler probe (Figure 14.5).
6) Place gelled probe on the clipped skin overlying the artery, keeping the probe's cord parallel to the limb. Using medical adhesive tape, secure the probe to the patient. Turn on the device and adjust with fine movements until a clear rhythmic "whooshing" sound is audible with the arterial pulse.
7) Inflate the sphygmomanometer to 30–40 mmHg past the point at which the arterial sounds are no longer detectable.
8) Deflate the cuff at a rate of around 2 mmHg/second until the first rhythmic "whooshing" sounds are detected, marking this point as the systolic blood pressure (SAP).
9) Allow the cuff to completely deflate, allowing blood flow to return to the limb.
10) The first measurement should be discarded and the average of five to seven consistent, consecutive readings should be recorded.
11) Record the name of the person making the measurements, the cuff size, the cuff location, the results, and the veterinarian's interpretation in the patient's record.

blood pressure readings than dogs in lateral recumbency [20]. Additionally in dogs, it would appear that blood pressure measurements made in lateral recumbency are less variable than those obtained in the sitting position [20]. The cuff may be placed on a limb or the tail, taking into account animal conformation and tolerance, and user preference [8, 9]. The authors prefer a front limb or tail when possible. If a rear limb is selected, cuff placement above the hock is recommended [21]. If the cuff is to remain in place for some time, medical tape can be used to secure the cuff as long as the tape does not apply pressure to the limb (Figure 14.3).

The first measurement obtained should be discarded. Then a total of five to seven consecutive, consistent values should be recorded. In some patients, measured readings trend downward as the process continues. In these animals, measurements should continue until the decrease plateaus and then five to seven consecutive consistent values should be recorded [8]. If in doubt, repeat the measurement subsequently. Written records should be kept on a standardized form and include the individual taking measurements, the cuff size and location, the values obtained, rationale for excluding any values, the final averaged result, and then interpretation of the results by a veterinarian.



Figure 14.3 The limb of a dog wearing an appropriately sized pressure cuff. Note that the cuff has been secured using medical tape circumferentially around the limb on the proximal and distal edges of the cuff/limb interface without compressing the limb. *Source:* Photo courtesy of Christopher Norkus.

Standard Oscillometry

Principles

The oscillometric measurement technique is less time consuming and requires less technical skill than the Doppler ultrasonic technique. Oscillometry involves the connection of a pressure cuff to a device that detects arterial wall oscillations during blood flow. Inflating the cuff occludes the artery. As the cuff is deflated, the arterial wall oscillations increase at SAP, reach a maximum at MAP, and decrease at DAP. The oscillometric machines generally display MAP, SAP, DAP, and pulse rate. Many oscillometric machines calculate SAP and DAP from the MAP using proprietary algorithms; therefore, MAP is probably the most reliable reading obtained by standard oscillometry. Calibration of the device should be performed semiannually either by the user, when self-test modes are included in the device, or by the manufacturer [8]. Figure 14.4 shows two different types of standard oscillometers used in small animals.

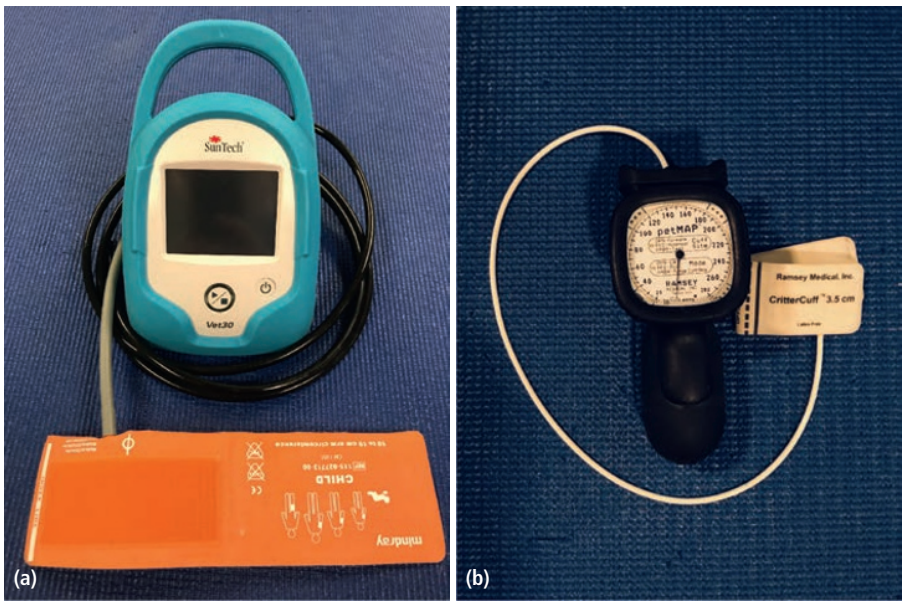


Figure 14.4 (a) One example of a standard oscillometric blood pressure monitor. *Source:* Photo courtesy of Christopher Norkus. (b) An oscillometric sphygmomanometer measurement device. *Source:* Photo courtesy of Maria Battaglia.



Figure 14.5 Use only ultrasound gel (blue, on the right of the image) on Doppler ultrasound probes. Electrode gel (green, on the left of the image) should not be used because it damages the Doppler probe's protective coating and causes the device to malfunction. *Source:* Photo courtesy of Christopher Norkus.

Cuff Selection

For oscillometry, the bladder of the cuff should be positioned over the artery for maximum sensitivity to oscillations. Other instructions regarding pressure cuff size and the pressure cuff position in relation to the right atrium are the same for standard oscillometry as for the Doppler method and are given in the preceding discussion on Doppler ultrasound.

Limitations of Standard Oscillometry

The oscillometric techniques measure pulse rate, which should always be compared with the patient's pulse rate as determined manually [8]. If the oscillometer's reported pulse rate is inaccurate, the blood pressure results may be inaccurate as well. Although oscillometry is routinely used reliably in non-sedated dogs and for monitoring feline and canine blood pressures during anesthetic procedures, this technique may be less reliable in non-sedated cats and small dogs than the Doppler method. This is likely due to smaller peripheral artery size, which may not generate sufficient pulse pressure to generate detectable cuff pressure oscillations. Additional reasons for invalid results using standard oscillometry include cardiac arrhythmia, significant bradycardia or tachycardia, vasoconstriction, hypothermia, and patient movement, regardless of patient size.

Performing Standard Oscillometry

The equipment required for standard oscillometric blood pressure measurement includes various size pressure cuffs, adhesive medical tape, and the oscillometer. See Protocol 14.2 for detailed instructions regarding performance of oscillometric NIBP measurements. As with Doppler ultrasonic measurement, the operator should perform several consecutive blood pressure measurements, discarding the first reading and averaging the following five to seven consecutive, consistent readings [8].

Protocol 14.2 Standard Oscillometric Sphygmomanometry Blood Pressure Measurement

Items Required

- Various size pressure cuffs
- Medical adhesive tape
- Oscillometer, with power cord
- Assistant, if required

Procedure

1) Collect all necessary supplies.
2) Position the patient in lateral or sternal recumbency while holding the planned cuff site at approximately the level of the right atrium. If positioning the cuff at approximately the vertical level of the right atrium is not possible, a correction factor is then used. If the cuff is ≥10 cm *below* the right atrium, then *subtract* 0.8 mmHg for every 1 cm the cuff is below the right atrium. If the cuff is ≥10 cm *above* the right atrium, *add* 0.8 mmHg for every 1 cm the cuff is above the right atrium.
3) Secure a deflated cuff of a size that is approximately 30–40% of the limb at the cuff's application site. When performing oscillometry on a forelimb, secure the cuff over the antebrachium. When measuring on a rear limb, secure the cuff proximal to the hock. Medical adhesive tape should be applied circumferentially around the limb on the proximal and distal edges of the cuff/limb interface to secure the cuff to the limb. The bladder of the cuff (the portion that fills with air) should be centered over the palpable pulse if possible.
4) Push the button on the oscillometer that begins pressure readings. Discard the first reading and record the average of at least five to seven consecutive, consistent readings.
5) Record the name of the person making the measurements, the cuff size, the cuff location, the patient's position, the average SAP, DAP, and MAP, and the veterinarian's interpretation in the patient's record.

High Definition Oscillometry

High-definition oscillometry (HDO) blood pressure monitors are relatively new to the veterinary industry. The developers claim that HDO monitors have many advantages over conventional oscillometric blood pressure measurement devices. Contrary to standard oscillometric sphygmomanometry, in which the MAP is measured and the SAP and DAP are calculated, HDO devices perform real-time analysis of arterial wall oscillations to obtain pressure wave amplitudes. Unfortunately, several recent studies of HDO in dogs and cats have not shown good correlation, accuracy, or precision with dABP or other NIBP measurement methods [22–25]. Some authors of these works have therefore recommended that HDO not be used in patients under general anesthesia and have questioned the clinical value of HDO. Thus, the future role of HDO in small animal emergency and critical care remains uncertain.

Optimizing Reliability of Noninvasive Arterial Blood Pressure Measurements

The ACVIM consensus panel established some guidelines for optimizing the reliability of NIBP measurements [8, 9]. The panel cited operator experience as the most likely cause of inaccurate values; hence, an experienced operator should perform measurements, and appropriate training cannot be overemphasized. The panel's recommendations also include the use of a standardized protocol (Protocols 14.1 and 14.2), allowing the animal to acclimatize to its environment prior to measurement, proper and comfortable cuff placement, and performance of several readings as recommended previously. Although these recommendations are valid and should be followed when possible, some of them are not always possible in the emergency room and intensive care unit. Thus, it is important at the least that an experienced operator measure the blood pressure and to ensure appropriate cuff size and location. The consensus panel also recommends calibrating each NIBP device twice yearly for best accuracy [8].

As stated previously, confirming that the pulse rate reported by the NIBP device matches the patient's actual pulse rate may help ensure more accurate results. Also, NIBP modalities should not be used interchangeably. Specifically, blood pressure readings obtained from different NIBP measurement devices should not be compared (for instance for trending purposes), even in the same patient [26].

Emergent and critically ill patients are often highly stressed and may be in cardiovascular shock. In this patient population, acquiring accurate and reliable NIBP measurements is very challenging even when using all recommended techniques. Therefore, one should reassess a patient's physical examination and vital signs frequently and use additional diagnostic and monitoring tools (e.g. lactate concentration, shock index, mixed venous oxygen saturation, point-of-care ultrasound) to corroborate findings.

Conclusion

Hypotension and hypertension are both common findings in small animal emergency and critical care patients. Numerous disease conditions and clinical presentations warrant NIBP monitoring [8, 9]. Using blood pressure in conjunction with other modalities to help guide fluid resuscitation and specific pharmacologic interventions will help reduce morbidity and mortality in these patients. Familiarizing the nursing staff and clinicians with normal arterial pressure values, along with use of a standardized protocol for NIBP devices, will facilitate their use and optimize their reliability.

At this time there are no machines validated to perform NIBP measurement in dogs and cats, but that does not mean these methods are not useful in clinical practice. Standardization of hospital protocols and staff training for use of NIBP devices is highly recommended. Following the ACVIM guidelines for blood pressure measurement is advised although in emergency and critical situations often it is not entirely possible. Adhering as closely as reasonably possible to standard protocols and using physical examination findings, along with other diagnostic and monitoring modalities, will improve the utility of NIBP devices and their measurement results.

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15

Central Venous Pressure Monitoring

Rosalind S. Chow

Optimizing cardiovascular function is fundamental to the clinical management of anesthetized and critically ill animals. Achieving optimization requires regular assessments of the adequacy of intravascular blood volume and venous return, and the administration of intravenous (IV) fluids to correct fluid deficits. However, determining the ideal volume of IV fluids to administer is not always a simple task, as both insufficient and excessive fluid replacement can be detrimental [1, 2]. Central venous pressure (CVP) is a common method of estimating circulatory filling and cardiac preload in these settings to help guide fluid resuscitation and assess fluid balance in veterinary patients [3–6].

CVP represents the direct measurement of venous blood pressure from a catheter inserted into a large central vein such as the cranial vena cava or caudal vena cava, or the right atrium. In the absence of vascular obstruction, CVP measured from the vena cava is a close approximation of right atrial pressure [7], which in turn is a determinant of right ventricular preload.

However, using CVP to guide fluid therapy can be challenging due to its non-linear relationship to blood volume and poor correlation with cardiac output following a fluid bolus [8], although some argue that these criticisms reflect a misunderstanding of how the results should be interpreted [9]. CVP provides useful information about right-sided cardiovascular function, but a clear understanding of the benefits and limitations of this common monitoring technique is essential to determining the situations where CVP measurement may provide the most benefit in patient management.

Determinants of Central Venous Pressure

CVP is determined primarily by the relationship between venous return and right heart function [10]. Venous return is affected by venomotor tone, venous wall compliance, and circulating blood volume, whereas right-sided cardiac output is determined by heart rate, preload, afterload, and contractility. Therefore, any normal physiological process, disease, or medical intervention that alters one or more of these factors may affect CVP. This includes changes in sympathetic tone that occur during stress and illness, structural and functional diseases of the heart, and vasoactive drugs such as vasopressors, sedatives, and general anesthetics.

In some cases, extravascular forces, such as increases in intrathoracic, pericardial, or intra-abdominal pressures can also change CVP [11, 12]. Increased intrathoracic pressure may develop secondary to a variety of causes including pleural effusion, presence of a space-occupying mass, a forced expiratory respiratory pattern, or positive end-expiratory pressure (PEEP) during mechanical ventilation. A sufficient rise in intrathoracic pressure will compress the vena cava and right atrium and raise the CVP. Similar effects on CVP can be caused by increases in intra-abdominal pressure such as with ascites, acute abdominal syndrome, neoplasia, or increased expiratory abdominal effort. When there is significant disease in the thoracic or abdominal cavities, it is important for the clinician to be aware that CVP interpretation will be more complicated.

Indications for the Measurement of Central Venous Pressure

CVP is commonly used to help guide fluid therapy in animals with abnormalities in circulating blood volume or abnormal right heart function. CVP cannot be used to make inferences about left ventricular preload or function [13]. Specific indications for CVP measurement include the presence of persistent hypotension despite fluid resuscitation, vasopressor therapy, extensive third

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space losses, oliguria or anuria, hemorrhage, trauma, sepsis, burns, and heart failure. Patients undergoing emergency surgical procedures and those with multiple medical problems are predisposed to developing cardiovascular instability while anesthetized. In these animals it may be practical to insert a central venous catheter and begin monitoring preemptively to allow earlier detection of abnormalities in the perioperative period.

Risks

Risks associated with central venous catheterization include thrombosis, thromboembolism, carotid arterial puncture, infection, phlebitis, bleeding, and vascular erosion. Cardiac arrhythmias, or rarely cardiac wall perforation, can develop if the catheter tip is accidentally advanced into the right atrium or right ventricle. There are fewer problems associated with CVP measurement itself although accidental introduction of air or bacteria into the venous system may occur, particularly if poor technique is practiced. The cranial vena cava is a low-pressure system and opening the system to the atmosphere may result in air embolism formation. Fortunately, this is a rare occurrence with intermittent CVP measurement technique and negligible with a continuous monitoring technique as the latter system is closed to the outside environment. Additionally, several straightforward precautions will reduce the incidence of complications. These include following proper hand hygiene measures prior to handling the jugular catheter or CVP system, careful inspection of the tubing and transducer to assess for defects, the use of threaded connectors such as Luer-Lok® (Becton, Dickinson and Company, Franklin Lakes, NJ), and periodic verification of the tightness of these connections, particularly in mobile patients.

Reference Interval

The most frequently used reference range for CVP is around 0–5 cm H_2O in cats [4] and dogs [14, 15] but values of up to 10 cm H_2O can be normal in awake and anesthetized dogs [3, 4, 16, 17]. The broad reference range reflects the wide variability seen between individuals, caused by the collective influence of blood volume, venous tone, and cardiac function on venous pressure.

CVP is measured in millimeters of mercury (mmHg) or centimeters of water (cm H_2O), depending upon the method of measurement. To convert from mmHg to cm H_2O, multiply by a factor of 1.36:

$$\text{CVP in cm}H_2O = \text{CVP in mmHg} \times 1.36 \quad (15.1)$$

Measurement Technique

CVP is the direct measurement of venous pressure within the vena cava or right atrium. It is obtained by inserting a specialized catheter into a peripheral vein (typically the external jugular vein) and advancing the catheter until the tip rests within the cranial vena cava or the right atrium. Right atrial pressure can also be obtained from the proximal lumen of a pulmonary artery catheter. In the absence of vascular obstruction, pressure measurements from the vena cava and the right atrium are generally considered interchangeable at rest [18, 19] although their responses are not identical following fluid bolus or vasopressor therapy [20].

Several alternative catheterization approaches have been described in animals, including catheter insertion into the omobrachial vein in dogs [21] followed by advancement into the cranial vena cava, or catheter placement in the femoral vein and passage into the caudal vena cava in dogs [22] and cats [23]. In human patients, peripheral venous pressure has also been used as an estimate of CVP due to the similarities between centrally and peripherally measured venous pressures [24, 25]. Unfortunately, this technique has shown poor correlation in dogs and cats [26].

More recently in clinical human and experimental canine studies, researchers have investigated the possibility of estimating CVP non-invasively using sonographic measurements of caudal (inferior) vena caval diameter [27–29], vena cava collapsibility [28, 29] or hepatic venous blood flow velocity [27]. These techniques have demonstrated moderate correlation when used to predict broad trends in CVP. However, these methods require specialized sonographic views and their clinical utility in veterinary patients remains unknown. Direct CVP measurement from a central venous catheter remains the gold standard.

Catheter Selection and Placement

Either single-lumen or multilumen vascular catheters can be used if they are of sufficient length to reach the thoracic vena cava from the point of insertion. Selection of a multilumen catheter has the added advantage of allowing continued infusion of IV fluids and drugs through the proximal ports while simultaneously measuring CVP from the catheter's distal port. The rate of fluid administration has no effect on the CVP in people, even during pressurized saline infusions of up to 9120 ml/hour via a double-lumen catheter and up to a combined rate of 14,340 ml/hour through the proximal ports of a triple-lumen catheter [30]. These results are likely similar in veterinary patients although specific experiments have not yet been published.

It is important to determine the appropriate depth of insertion prior to catheter placement. A good estimate of depth can be obtained by measuring the distance between the

planned insertion site and the caudal aspect of the shoulder, which should appropriately position the tip within the cranial vena cava. Following placement, the catheter's position can be verified with radiography as well as by visualization of a characteristic CVP waveform (see Waveform Analysis). All central venous catheters have a radiopaque marker and radiographic confirmation is particularly important when alternative catheter insertion sites, such as the saphenous vein, are used because the proper depth of insertion is more difficult to estimate. Catheters unintentionally advanced into the right atrium should be backed out slightly as they can occasionally generate arrhythmias. Accidental advancement of the catheter tip into the ventricle should be avoided. However, if it occurs, it is not difficult to recognize once a pressure measurement is obtained, as peak right ventricular pressures approach 20–30 mmHg in the normal animal [3]. These large magnitude waveforms are visible as extreme fluctuations of the fluid column of the water manometer during intermittent CVP measurement, or large peaks on the electronic monitor display with continuous CVP measurement.

General Principles

CVP can be measured intermittently or continuously. With both methods, the catheter is connected via a fluid-filled tubing system to a pressure-measuring device that displays the venous pressure. Intermittent measurement involves connecting the catheter to a water manometer, infusing a predetermined volume of saline, and allowing the fluid level to equilibrate with the patient's CVP. The height of the fluid column within the manometer is recorded in cm H_2O. Continuous CVP measurement is obtained by linking the catheter and fluid-filled tubing to a pressure transducer that converts the venous pressure wave to an electrical signal. The monitor displays the venous pressure waveform in real time as well as a mean venous pressure in mmHg. In both cases, the ideal tubing is specialized non-compliant tubing manufactured for blood pressure measurement.

As is commonly the case where more than one method of measurement is available, there are advantages and disadvantages to each technique. The equipment needed to perform intermittent CVP monitoring is simple and inexpensive. However, repeated measurements are relatively time-consuming to obtain and effort must be made to ensure continued sterility of the system between measurements. In contrast, once the system for continuous CVP monitoring is assembled, moment-to-moment changes in CVP are displayed on the monitor without the need for additional intervention. The ability to view CVP continuously is particularly useful for unstable patients in the intensive care unit and in the operating room. Frequent measurements also permit better and timelier information about a patient's response to therapy. However, a higher level of technical knowledge and skill is needed to set up and troubleshoot continuous CVP monitoring, and it also requires the purchase of a specialized monitor. When available, refurbished or used monitors in good working condition may be an economical solution for many hospitals.

Zeroing and Leveling

To obtain reliable pressure measurements, two principles are important. First, blood pressure is measured relative to a standard reference point, atmospheric pressure, which measures approximately 760 mmHg at sea level. Atmospheric pressure is created by the weight of air pressing down on the body and everything within and around it, including the central veins and the CVP measurement system. Since it exerts the same magnitude of pressure on each object, its effects cancel out, allowing atmospheric pressure to be set at 0 cm H_2O (0 mmHg) for purposes of CVP measurement. This allows a CVP of 770 mmHg to be read as 10 mmHg and eliminates the need to adjust CVP measurements for fluctuations in barometric pressure [31]. The process of correcting for atmospheric pressure is called *zeroing*. Pressure transducer systems have an integrated stopcock and port that can be opened to the atmosphere to calibrate, or *zero*, the transducer.

The second principle is the importance of aligning the transducer system (or manometer) with the vascular structure containing the pressure of interest, also known as the *zero reference point*, in order for the measurements to accurately reflect the pressure within the intended vessel. In the case of CVP, the zero reference point is the center of the right atrium. The stopcock (for continuous CVP measurement) or the 0 cm H_2O mark on the manometer (for intermittent measurement) must be positioned, or *leveled*, on the same horizontal plane as the patient's right atrium during zeroing and thereafter during measurement, as demonstrated in Figure 15.1. See Chapter 12 for more information regarding zeroing and leveling transducers.

Due to species- and breed-related variability in thoracic conformation, there is no completely foolproof method of determining where the right atrium lies. However, in general, the sternum is a good approximation for a dog or cat in lateral recumbency. For an animal in sternal recumbency, draw an imaginary vertical line at the caudal aspect of the shoulder that extends from the top of the dorsal spinous process to the sternum. The right atrium lies at a point that is roughly 40% of the height of this line.

Proper determination of the zero reference point is crucial, because failure to level the transducer (or manometer) relative to the right atrium will result in an erroneous CVP reading. The value will be falsely high if the transducer is resting below the right atrium and falsely low if the transducer is above the right atrium. Since physiologically significant changes in CVP exist in a relatively narrow range,



Figure 15.1 Leveling of the manometer with the zero reference point. Although this ferret's heart is caudal to the manometer, measurements will be accurate as long as the zero value rests on the same horizontal plane as the patient's right atrium.

patient harm could result if the error is missed, and the resulting measurements are used as the basis for fluid therapy decisions. For this reason, the height of the transducer and stopcock system should always be evaluated prior to each pressure reading. Their position may need to be adjusted depending on whether the animal is recumbent, sitting, or standing. Gentle restraint in lateral recumbency may be helpful for mobile patients to ensure accurate CVP readings. The transducer should also be periodically re-zeroed – always at the level of the right atrium – due to the potential for drift [32]. Progressive upward or downward trends in CVP measurements are significant, particularly if they fall outside expected or target values.

Intermittent Central Venous Pressure Measurement

Prior to initiating CVP measurement, it is important to verify the patient has a correctly positioned central venous catheter, as described earlier in this chapter. Two types of water manometers can be used for intermittent CVP measurement. The best one is a rigid, narrow, cylindrical tube made of glass or plastic specifically manufactured for this purpose that has centimeter markings along its length. A simpler homemade alternative consists of a short section of IV fluid extension tubing that can be affixed to a ruler. Either standard fluid tubing or noncompliant tubing manufactured specifically for blood pressure monitoring can be used. The equipment required to perform intermittent CVP measurement is shown in Figure 15.2.

To ensure accuracy, the patient should rest in the same position for each measurement period and this position should be recorded on the patient's treatment sheet for future reference. Both lateral and sternal recumbency are acceptable. It may be more difficult to obtain accurate readings from an animal that is sitting or standing. Whenever possible, use the same position for subsequent measurements. In some situations, it will be impractical or unsafe to position the patient in a certain way. If this is the case, it is important to adjust the height of the manometer so that the 0 cm H_2O mark is level with the zero reference point (the right atrium) prior to recording the CVP. Alternatively, as CVP can occasionally fall below zero, it can be helpful to align the 10 cm H_2O mark with the zero reference point to facilitate measurement of negative pressures. However, in this situation, it is necessary to subtract 10 cm H_2O from the height of the fluid column in the manometer to obtain the true CVP measurement.

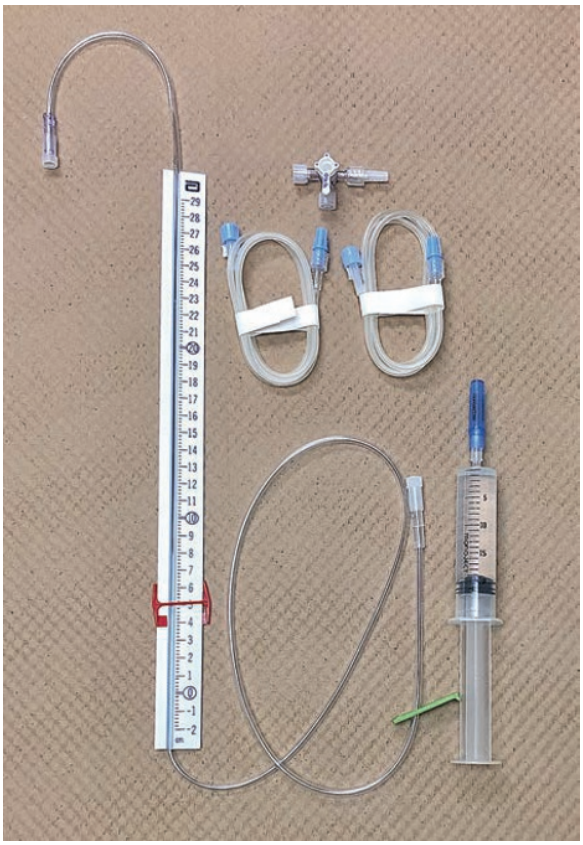


Figure 15.2 Equipment required for intermittent central venous pressure measurement.

The CVP is read at the bottom of the meniscus in the manometer (or at the center of the floating ball in the manometer, if present). Patient heartbeat or respiration may cause millimeter fluctuations in the meniscal level. In a spontaneously breathing animal, the CVP reading will decrease during inspiration and increase during expiration. The pressure reading should be obtained at end-expiration if the patient is breathing normally. If there is pronounced expiratory abdominal effort, the measurement should be obtained at the beginning of the expiratory phase, prior to the onset of active abdominal effort [7, 10]. Detailed instructions on performing intermittent CVP measurement are available in Protocol 15.1.

The time interval between measurements will depend on the patient's cardiovascular status as well as personnel availability, but a reasonable place to start is to space readings one to four hours apart. If more frequent measurements are desired, continuous CVP measurement should be considered.

Continuous Central Venous Pressure Measurement

The list of supplies and equipment needed for continuous CVP measurement is more extensive than it is for intermittent measurement. To better familiarize the reader with the materials and monitoring technique, additional details are provided here.

Protocol 15.1 Intermittent Central Venous Pressure Measurement

Items Required

- Indwelling central venous catheter (inserted to the proper depth)
- Disposable water manometer (calibration in centimeters), or a centimeter ruler and extension tubing
- 0.9% NaCl
- Three-way stopcock with locking fittings
- 20-ml syringe
- Ideally, noncompliant tubing; alternatively, standard fluid extension tubing
- Heparinized saline flush
- One assistant

Procedure

1) Collect the necessary supplies.
2) Perform hand hygiene and put on examination gloves.
3) The patient may lie in lateral or sternal recumbency. This position should be recorded on the patient treatment sheet for future reference.
4) If applicable and safe to do so, have the patient rest in the same position that was recorded in the treatment sheet for prior CVP measurements.
5) Flush the patient's central venous catheter with heparinized saline solution to ensure patency.
6) Assemble the CVP monitoring setup. First orient the manometer vertically. Then connect the three-way stopcock to the manometer, to the saline-filled syringe, and to the fluid tubing.
7) Orient the three-way stopcock valve so that it is closed to the manometer and open to the tubing and fluid-filled syringe. Then, prime (fill) the stopcock and fluid tubing with 0.9% NaCl from the syringe. Connect the fluid tubing to the patient's indwelling central venous catheter. If a multi-lumen catheter is being used, connect the tubing to the central lumen (the most distal port).
8) Orient the stopcock valve so that it is closed to the patient's central venous catheter and open between the manometer and the fluid-filled syringe. Using the 20-ml syringe, fill the manometer with 0.9% NaCl to a level that is approximately 10–20 cm H_2O greater than the patient's expected CVP. Do not allow the manometer to overflow while filling.
9) The manometer may be attached to an IV pole to facilitate height adjustments, held vertically in the operator's hand, or taped to the wall of the cage. Locate the 0 cm H_2O mark and position the manometer so that it is level with the zero reference point (the patient's right atrium).
10) Close the stopcock valve toward the syringe, and unclamp the central venous catheter line, which will open a fluid column between the manometer and the patient. The fluid level in the manometer will initially fall as it flows into the patient and then stabilize as the fluid level equilibrates with the CVP.
11) The operator should ensure consistent readings by performing three or more consecutive measurements and calculating an average. Record the average pressure and patient position on the patient's treatment sheet.
12) When the measurements have been completed, reclamp the central venous line, turn the stopcock off to the manometer and disconnect the pressure tubing from the central venous catheter. Reconnect the line to IV fluid therapy if it is intended to be resumed, otherwise flush the central venous catheter with heparinized saline and cap the port with an injection plug.

Pressure transducer kits are presterilized and typically consist of a disposable pressure transducer, three-way stopcock with zeroing port cap, and a flush mechanism (Figure 13.1). Some kits also include noncompliant ("high-pressure") tubing that is needed to join the pressure transducer to the patient's central venous catheter. The tubing may also be sold separately. Additional tubing can be added between the transducer and the patient if more length is needed. Standard fluid extension tubing is soft and compliant, whereas the specialized tubing made for blood pressure measurements is flexible but non-distensible. In theory, excessively long or compliant tubing could suppress, or *dampen*, transmission of the pressure waveform, resulting in waveform distortion [33, 34]. Owing to the low vascular pressures of the venous system, damping from overly compliant tubing is less of a concern with CVP measurement than it is with direct arterial blood pressure monitoring [34]. Optimization of the CVP waveform will be achieved with noncompliant tubing specifically manufactured for blood pressure monitoring. The reader is referred to Chapter 12 for more details regarding tubing characteristics.

The last element of the continuous monitoring setup is assembly of the flush system, which is pressurized and provides a simple method of flushing the catheter by pulling the rapid flush valve (usually referred to as a *pigtail*) on the transducer. The flush solution consists of heparinized saline and it is prepared by adding unfractionated heparin to a bag of 0.9% NaCl to a final concentration of 4 IU/ml.

Prior to beginning measuring, the transducer must be zeroed at the proper level by exposing it to atmospheric pressure in approximately the same horizontal plane as the right atrium (see Intermittent Measurement instructions for more details). The fluid-filled system is zeroed and leveled by turning the stopcock lever off toward the patient, loosening (or temporarily removing) the zeroing port cap and setting the monitor to zero. The reader should consult the reference manual for their specific monitor for additional details. Once this has been done, the monitor will display 0 mmHg and the waveform tracing should overlap zero on the displayed scale. If the transducer does not zero, the transducer is the most likely culprit, and it should be replaced and the procedure reattempted. After the zeroing step, the zeroing port cap is replaced and tightened, and the stopcock is turned so it points off toward the port cap to create a continuous fluid column between the pressure transducer and the patient's central vein. CVP measurement may then begin.

As with intermittent CVP measurement, it is important to ensure the transducer is at the level of the zero reference point (the right atrium) prior to zeroing or obtaining a reading. This is best achieved by having the patient lie in the same position for each measurement period. Specific instructions for assembling the continuous CVP measurement system are included in Protocol 15.2. Common issues and solutions that may be encountered during continuous CVP monitoring are summarized in Table 15.1.

Protocol 15.2 Continuous Central Venous Pressure Measurement

Items Required

- Indwelling central venous catheter inserted to the proper depth
- Pressure transducer kit
- Bag of 0.9% NaCl heparinized to a concentration of 4 iu/ml
- Standard fluid administration set
- Pressure bag of appropriate size for heparinized saline bag
- Ideally, noncompliant fluid tubing; alternately, standard fluid extension tubing
- Electronic blood pressure monitor and its associated transducer cable

Procedure

1) Collect the necessary supplies.
2) Perform hand hygiene and put on examination gloves.
3) The patient may lie in lateral or sternal recumbency. This position should be recorded on the patient treatment sheet for future reference.
4) Flush the patient's central venous catheter with heparinized saline solution to ensure patency.
5) Assemble the heparinized saline flush system by spiking the heparinized saline fluid bag with the standard fluid administration set and flushing fluid through the tubing. Clamp the tubing and cap the set's open end.
6) Place the pressure bag over the bag of heparinized saline, hang it on an IV pole placed next to the patient, and inflate the pressure bag to 300 mmHg.
7) Assemble the transducer system by connecting it to the noncompliant tubing, to the assembled heparinized saline flush system, and to the transducer cable and electronic monitor, as shown in Figure 11.1.

8) Prime (fill) the transducer and noncompliant tubing system with heparinized saline by pulling the transducer's fast flush valve (pigtail).
9) Connect the fluid-filled pressure tubing to the central venous catheter. If using a multilumen catheter, the CVP measurement should be obtained from the most distal lumen, reserving the other lumens for IV fluid therapy, drug administration, or blood withdrawal. It is not necessary to discontinue IV fluid or drug administration through the other ports during CVP measurement.
10) Position the height of the transducer and stopcock system so that the stopcock is aligned at the same height as the zero reference point (the right atrium). The transducer may be attached to an IV pole, to the cage door, or taped to a stable support that is resting on the floor of the cage.
11) Flush the catheter by pulling on the pigtail and wait for the pressure to equilibrate.
12) The system must be calibrated (zeroed) before any measurements can be interpreted. To perform zero calibration, with the stopcock at the height of the right atrium, turn the stopcock off toward the patient. Remove the zeroing port cap on the stopcock to open it to the atmosphere. Select the zeroing function on the display monitor and wait for it to read 0 mmHg. Replace the zeroing port cap and turn the stopcock toward the zero port, which will allow the pressure to equilibrate between the patient's vena cava and the transducer.
13) Once the pressure has stabilized, record the CVP measurement.
14) For subsequent measurements, verify the transducer height is correctly positioned at the zero reference level before recording the CVP measurement.

Table 15.1 Troubleshooting tips for continuous central venous pressure measurement.

Problem	Possible cause
Pressure is displayed as a flat line rather than a waveform	Complete occlusion of the catheter, stopcock, or fluid line (displayed pressure will be far above the normal reference range)
	Partial occlusion of the catheter, stopcock, or fluid line
	Patient is small (cats and small dogs occasionally lack a visible waveform although the mean pressure can still be used for trending purposes)
	Air bubble or leak in the system
No pressure is displayed on the monitor	Monitor display settings are incorrect
	Transducer was not zeroed
	Transducer cable is malfunctioning or is not plugged into the monitor
Pressure reading is higher than expected	Intrathoracic or intra-abdominal pressure is significantly increased
	Central venous catheter is clamped off or occluded
	Transducer is below the level of the right atrium
	Transducer is defective
Pressure reading is lower than expected	Transducer is above the level of the right atrium
	Transducer is defective
Waveform is "noisy"	Patient movement
	Panting
	Catheter tip is within the heart
	Arrhythmia
Sudden change in pressure	Hemodynamic instability
	Transducer position relative to the zero reference point (right atrium) has changed
System does not flush	Stopcock position is incorrect
	Pressure bag is not sufficiently inflated
	Heparinized saline bag is empty
	Heparinized saline administration line is clamped off or occluded
	Central venous catheter is clamped off or occluded

Maintenance of the Continuous CVP System

For the patient undergoing continuous CVP monitoring, certain maintenance procedures should be included in the patient's treatment orders, listed in Box 15.1. The patient can be disconnected from and reconnected to the CVP measurement system without the need to re-zero the transducer as long as the disconnection occurs between the transducer and the central venous catheter. However, if the transducer cable is disconnected from the monitor, it will be necessary to re-zero the transducer prior to obtaining a CVP reading.

CVP Interpretation

CVP should never be used as the sole monitoring parameter to determine the adequacy of circulating blood volume. It must always be evaluated in conjunction with the patient's history, signalment, and physical examination, and ideally with knowledge of the animal's cardiac and kidney function. Laboratory data and additional markers of hemodynamic status, if available, such as arterial blood pressure, urine output and cardiac output, should also be factored into the clinical assessment.

Due to the number of factors that can influence CVP, an isolated value is clinically meaningless. However, serial measurements over time may document trends in venous blood pressure that can provide useful information to assist in the assessment of circulating volume status. Apart from its usefulness as a diagnostic tool when hypovolemia is suspected, CVP measurement can also be used to monitor the effectiveness of fluid therapy to treat low circulating blood volume. However, in a similar manner, its success or failure should be determined by concurrently evaluating a combination of other clinical markers.

Box 15.1 Maintenance of the Continuous Central Venous Pressure System

- The transducer and catheter system should be periodically flushed by pulling on the fast flush device. This should be performed at least once every four hours.
- The transducer should be re-zeroed no less frequently than every 12 hours due to the potential for drift.
- Change the flush solution and tubing every 48 hours.
- Ensure there are no air bubbles in the fluid line at any time.
- Periodically inspect and reinflate the pressure bag to 300 mmHg as necessary and verify the heparinized saline bag is not empty.

In hemodynamically unstable patients, the primary objective of fluid therapy is to optimize right ventricular preload in an effort to improve cardiac output and tissue perfusion. The rationale for performing CVP measurement is its ability to serve as an estimate of right atrial pressure, which is a major determinant of right ventricular end-diastolic pressure. Right atrial and right ventricular pressures are equal when the tricuspid valve is open and pressures have equilibrated at the end of ventricular diastole. Right ventricular end-diastolic pressure is in turn related to right ventricular end-diastolic volume, which determines end-diastolic myocardial wall stretch, or *preload*.

While the initial temptation would be to assume that low values of CVP correspond to hypovolemia, and high values indicate volume overload, in reality, the association between CVP and preload is not straightforward. Critics note that isolated CVP values do not correlate well with intravascular blood volume, nor does CVP accurately predict stroke volume or cardiac output following a fluid challenge [1, 8]. Despite these limitations, CVP can still provide useful information about preload and right-sided heart function [31]. A review of the physiological principles behind venous return and venous pressure is useful here to highlight the benefits and limitations to using CVP in the clinical setting.

Venous return describes the flow of blood from the systemic circulation back to the heart. Proper flow depends on the maintenance of an adequate pressure gradient, often referred to as the *driving pressure*, between the peripheral and central venous circulation. The magnitude of driving pressure in the venous circulation is small with the peripheral venous pressure averaging only 5–10 mmHg greater than the pressure within the central veins. Homeostatic adjustments ensure continued return blood flow as variables change [11].

The dynamic properties of the venous system that allow it to regulate venous return also govern its other role, which is to serve as a blood reservoir. Veins contain approximately 65% of the systemic blood volume [35]. A major portion of that blood is contained within the splanchnic veins, which function as capacitance vessels capable of significant adjustments in wall compliance to accommodate changes in blood volume. When effective circulating volume is low, constriction of the splanchnic veins increases the circulating pool of blood to help support adequate venous return [11]. CVP may change minimally during this time despite the recruitment of additional volume and clinically the patient may appear to be coping quite well. However, once the blood reservoir has been depleted and other compensatory mechanisms have been exhausted, the patient will decompensate and CVP will fall.

The complex relationship between CVP and circulating blood volume explains why a CVP within the normal reference range cannot distinguish the normovolemic patient

from one with compensated hypovolemia or hypervolemia, since homeostatic mechanisms will attempt to maintain an adequate pressure gradient for venous return for as long as possible. Likewise, a severely elevated CVP often reflects cardiovascular pathology, but it would be difficult to distinguish between normovolemia in the presence of severe cardiac dysfunction, or hypervolemia with adequate cardiac performance on the basis of CVP measurement alone [19]. The complexity of these interactions may explain why studies have consistently failed to find a threshold CVP pressure below which fluid loading will always improve cardiac output [36, 37]. From the opposite vantage point, there is no compelling physiological rationale to support a therapeutic strategy of designing a fluid therapy plan to target a specific CVP goal, due to the inability of CVP to predict fluid responsiveness with a high degree of certainty [8, 9].

Despite these limitations, several generalizations about CVP interpretation can be made that support its continued usefulness in the ICU and in anesthetized patients. First, CVP should be regarded as a probable, rather than an absolute, indicator of volume status. In other words, in the presence of normal cardiac function, patients with a low CVP are more *likely* to respond to volume than patients with a normal or high CVP. In human patients with severe circulatory dysfunction, a CVP less than 5 mmHg has been shown to be an excellent positive predictor of fluid responsiveness [38]. In contrast, patients with a CVP greater than 10–12 mmHg are unlikely to benefit from a fluid bolus, although some still can [7, 31]. Those that do respond may have a condition such as elevated intrathoracic or intra-abdominal pressure that is causing the CVP to overestimate the true transmural pressure [7, 12].

The second generalization that can be made is that rising or falling trends are clinically meaningful. An experimental study of induced hypovolemia and hypervolemia in mechanically ventilated dogs showed that despite considerable variability in CVP between individuals, the directional change in CVP in these dogs paralleled blood volume as it rose and fell [17]. Therefore, a progressive drop in CVP should alert the clinician to the possibility of ongoing and excessive internal or external fluid losses, particularly if supported by the presence of other markers of hypoperfusion. In contrast, a rising CVP with concurrent evidence of worsening tissue perfusion may indicate declining cardiac function as the cause and could suggest that additional fluid loading may be unwise [7]. With the latter scenario, additional caution is warranted if hypoalbuminemia or systemic inflammation is present, as either of these conditions may increase the risk of edema formation with IV fluid therapy. A summary of the possible clinical interpretations of rising or falling trends in CVP is provided in Table 15.2.

Table 15.2 Clinical interpretation of central venous pressure measurements.

Interpretation	Possible cause
Falling trend	Hypovolemia
	Vasodilation
Within normal reference range and static	Normovolemia
	Compensated hypovolemia
	Compensated hypervolemia
Rising trend	Hypervolemia
	Vasoconstriction or systemic hypertension
	Right-sided heart disease: • Tricuspid regurgitation • Tricuspid stenosis
	Pericardial disease: • Pericardial effusion • Constrictive pericarditis • Vena caval obstruction
	Pulmonary disease: • Pulmonary hypertension • Pulmonary thromboembolism
	Increased intrathoracic pressure: • Pleural effusion • Intrathoracic mass • PEEP • Positive-pressure ventilation in the presence of hypovolemia • Pneumothorax
	Increased intra-abdominal pressure
	Occlusion of the catheter, fluid line, or stopcock

Potential Sources of Interpretation Errors

As discussed earlier, extravascular forces such as significant elevations in the pressure within the thoracic or abdominal cavities, can raise CVP. An increase in CVP of approximately 3 mmHg was seen at PEEP levels of 10 cm H_2O in humans [39] and 15 cm H_2O in pigs [40]. However, in the absence of PEEP, an alteration of tidal volume alone (8 ml/kg vs. 16 ml/kg) did not result in a similar effect [39]. The degree to which PEEP would be expected to affect CVP depends on pulmonary compliance, since pressure (e.g. PEEP) applied to less compliant lungs would not affect intrathoracic (pleural) pressures as much as pressure applied to more compliant lungs. Large elevations in intra-abdominal pressure secondary to acute abdominal syndromes, ascites, or a forced expiratory respiratory pattern, can also alter the CVP due to transmission of the increased abdominal pressure across the diaphragm to the thoracic cavity [12, 37].

It is important to recognize that the higher CVP generated by an elevation in intrathoracic or intra-abdominal pressure *does not necessarily result in an increase in driving pressure and venous return*. This is because the physiological variable that ultimately governs distension of the central veins is *transmural pressure*, not CVP [7, 10].

The concept of transmural pressure is best understood by recognizing that venous distension depends not only on the pressure exerted on the vascular wall from the inside (intravascular pressure or CVP), but also on the pressure exerted on the wall from the outside (extravascular pressure, or pleural pressure). The net difference (intravascular pressure minus extravascular pressure) is called transmural pressure. An increase in pleural or abdominal pressure will be transmitted to both intravascular and pleural spaces, resulting in no net change in transmural pressure despite a higher CVP measurement. Venous return depends ultimately on driving pressure, previously described as the difference between peripheral and CVPs. However, it is more accurately defined as the difference between peripheral and central *transmural* venous pressures. In this example, although there has been an absolute increase in measured CVP, transmural central and peripheral venous pressures have remained steady. Therefore, there is no change in the rate of return of blood to the heart.

Performing a Fluid Challenge

When CVP is falling or is extremely low, the index of suspicion for hypovolemia should be high. Usually, an evaluation of other clinical markers will support an assessment of low circulating blood volume and fluid resuscitation can be started immediately. However, on occasion, these findings will be unclear or contradictory. When this occurs, a fluid challenge is the classic method of verifying fluid responsiveness.

The procedure involves giving a small test volume as a rapid bolus and then monitoring for an improvement in clinical perfusion parameters such as patient alertness, pulse quality, pulse rate, mucous membrane color, and capillary refill time. Faster administration reduces the volume needed to achieve a certain magnitude of effect. If a beneficial response is seen following the volume challenge, additional fluid is given until the desired endpoint is reached. Under ideal monitoring circumstances this endpoint would be an increase in cardiac output. However, because cardiac output is difficult to measure without cardiac catheterization or other specialized techniques, several indirect indices are more commonly used, such as improved systemic arterial blood pressure, lower blood lactate concentration, increased urine production, and higher central venous oxygen saturation, as well as improvement in physical examination findings. See Chapter 19 for more information about many of these indirect perfusion indices.

A fluid challenge is performed by rapidly infusing a small volume of crystalloid or colloid using a pressure bag or fluid pump. Useful crystalloid test volumes are 15 ml/kg in the dog and 5 ml/kg in the cat. If a colloid is used, 5 ml/kg in the dog and 2.5 ml/kg in the cat are reasonable. The fluid bolus is given over 10–15 minutes and the animal is monitored for signs of improved perfusion and CVP. The classic response to a fluid challenge in the euvolemic animal is a rise in CVP of 2–4 cm H_2O, followed by a rapid return to the original value within 15 minutes [41]. However, if the starting CVP is low and it rises minimally or rapidly returns to baseline (within 5–15 minutes) following a fluid challenge, hypovolemia is likely [41, 42], particularly when corroborated by other findings as mentioned previously. The fall in CVP is due to the redistribution of fluid from the intravascular to the interstitial space, stress-induced relaxation of venous tone, and pooling of blood within the splanchnic vascular bed [43]. In contrast, a persistent, marked elevation in CVP following a fluid challenge, or a prolonged return to baseline (greater than 30 minutes) may support volume overload, decreased cardiac performance, or restrictive pericardial disease such as tamponade [41].

It should be noted that these guidelines reflect general trends, not absolute rules. A low CVP will not always indicate that a patient has inadequate blood volume, just as a high CVP does not necessarily signify fluid excess or cardiac dysfunction [19]. Normovolemic animals may show a rise in CVP following a test bolus, even though they do not actually require fluid. The concept of CVP serving as a marker of the probability of fluid responsiveness, rather than an absolute predictor of fluid responsiveness, is critical. Therefore, there is no substitute for careful patient assessment and clinical judgment when using CVP measurement to help guide fluid therapy.

The Normal CVP Waveform

Blood pressure in the central veins is pulsatile because of pressure changes in the right heart during the cardiac cycle. The baseline pressure also fluctuates from changes in intrathoracic pressure generated by the phases of respiration. Both intermittent and continuous CVP measurement techniques provide a mean CVP, although in the case of intermittent measurement, small pulsations are often evident in the fluid column [41].

In a similar manner, the CVP reported during continuous measurement represents a mathematical average of

this variable pressure. The pressure waveform is displayed on the monitor and classically consists of three waves (positive deflections from baseline: the *a* wave, *c* wave, and *v* wave) and two descents (negative deflections: the *x* descent and the *y* descent), which correspond to specific right atrial and right ventricular cardiac events (Figure 15.3) [44]. Not all waves and descents will be evident in all CVP tracings and considerable individual variability in waveform appearance can be seen. Waveforms can be small or impossible to discern in cats and small dogs and may be absent if the catheter lumen becomes partially occluded (such as by a blood clot).

The *a wave* is generated by atrial contraction and appears shortly after the P wave on an electrocardiogram. The *a* wave is followed by the *x descent,* which reflects a decrease in right atrial pressure caused by atrial relaxation. The *c wave* is sometimes visible as a secondary peak following the *a* wave and is caused by bowing of the tricuspid valve into the atrium during early right ventricular systole. As atrial diastolic filling proceeds, the *v wave* is created and it is generated soon after the T wave on an electrocardiogram. When ventricular systole ends and the ventricle relaxes, atrial pressure exceeds ventricular pressure, the tricuspid valve opens, and blood flows from the right atrium into the ventricle. Atrial emptying leads to a decrease in atrial pressure, thus producing the *y descent*. The *a* wave is usually larger than or similar in size to the *v* wave [44].

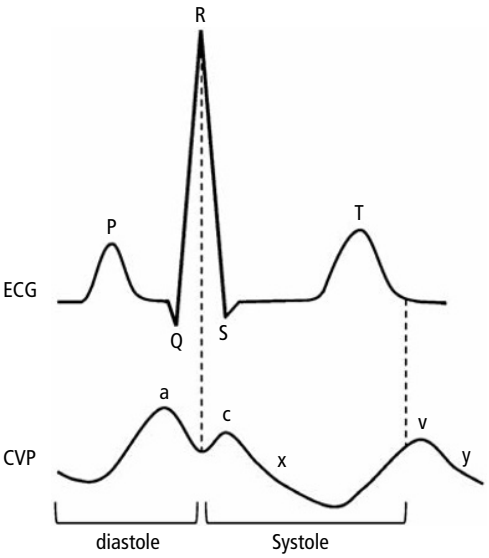


Figure 15.3 The relationship between the waves and descents of the central venous pressure (CVP) waveform and the electrocardiogram (ECG). Under normal circumstances, the mean of the *a* wave provides the best estimate of CVP because it corresponds to the venous pressure at the end of diastole. Ventricular systole begins immediately following the appearance of the QRS complex on the electrocardiogram.

Determining the CVP from the Normal Waveform

During continuous CVP measurement, the monitor displays a single pressure reading that represents an average pressure measurement over time. The mean CVP is often sufficient for clinical assessment in patients lacking significant primary cardiovascular or respiratory disease. However, changes in respiratory pattern, cardiac arrhythmias, and diseases that alter filling, emptying, or compliance of the right heart can lead to an inaccurate estimation of ventricular end-diastolic pressure (preload), resulting in errors in clinical interpretation. When any of these problems are evident, it is important to determine the CVP directly from the printed venous pressure tracing to ensure accuracy. In these situations, the mean of the *a* wave is considered a better estimate of CVP because its timing coincides with ventricular end-diastole [45]. At that brief moment of time immediately before the onset of systole, the tricuspid valve is open and there is a continuous column of blood extending between the vena cava, the right atrium and right ventricle. This allows CVP to reflect the filling pressure, or right ventricular preload, of the heart [45].

To determine the mean of the *a* wave, obtain a printed tracing that simultaneously displays the CVP and electrocardiogram (ECG) waveforms. From the tracing, locate the *a* wave in the CVP waveform, which appears during the PR interval on the ECG. It will be necessary to differentiate the *a* wave from the *c* wave, which is found in the RT interval, and the *v* wave, which appears after the T wave [46]. Once the *a* wave is identified, find the peak pressure (top of the *a* wave) and nadir pressure (bottom of the *x* descent) and calculate the mean of the *a* wave according to the following equation [44]:

$$\text{CVP estimate} = \left(a\,\text{wave peak} + x\,\text{descent base}\right)/2 \quad (15.2)$$

Two alternative methods that have been described to determine the CVP are to locate the R wave [46] or the end of the QRS complex [44] on the ECG. A perpendicular line is drawn extending from here toward the CVP waveform and the point of intersection represents the true CVP.

Abnormal CVP Waveforms

Waveform analysis provides a method of obtaining an accurate estimate of preload in the presence of cardiovascular or respiratory disease. It can also provide important supplementary information about cardiovascular function to help diagnose or confirm the presence of certain abnormalities. Several of these situations are described in the following subsections.

Respiratory Changes

Venous return varies with respiratory phase and respiratory muscle activity. During inspiration, there is a decrease in pleural pressure generated by expansion of the chest wall and caudal movement of the diaphragm [47]. This decrease in pleural pressure is transmitted across the wall of the vena cava and causes a small decrease in CVP, which returns to baseline following passive, unforced expiration. For this reason, in the spontaneously breathing, relaxed patient, the CVP reading (the mean of the *a* wave) should be measured at the end of the expiratory phase to best correspond to right ventricular end-diastolic pressure [7, 10, 47]. As with the method described earlier for normal waveforms, the CVP measurement is determined by calculating the mean of the *a* wave.

When there is increased expiratory effort, which may be seen in patients that are vocalizing or dyspneic, CVP will be overestimated with this method. In that situation, CVP is best obtained during early expiration prior to the onset of active abdominal muscle contraction [10]. Again, the mean of the *a* wave is used but it is necessary to evaluate the printed CVP waveform to determine the most accurate place to obtain this measurement (Figure 15.4). Similarly, positive-pressure ventilation may also raise the displayed mean CVP. The waveform should be examined and CVP measured from a time point corresponding to end-expiration.

Arrhythmias

Figure 15.5a shows a normal CVP waveform. Certain cardiac rhythm disturbances, such as atrial fibrillation and junctional and ventricular arrhythmias, can lead to a lack of atrial contraction and therefore loss of the *a* wave on a CVP tracing. During atrial fibrillation, there is also a prominent *c* wave resulting from overfilling of the right atrium, since it is unable to generate normal contractions (Figure 15.5c) [46]. The most accurate CVP estimate will be obtained by viewing the CVP tracing and ECG simultaneously and selecting the pressure that is present toward the end of the QRS complex, which best represents ventricular end-diastole [34].

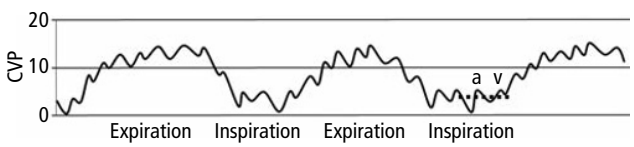


Figure 15.4 Baseline fluctuation in central venous pressure (CVP) waveform seen during forced expiration. The CVP should be estimated by calculating the mean of the *a* wave during early expiration (dashed line). The *a* wave and *v* wave are labeled.

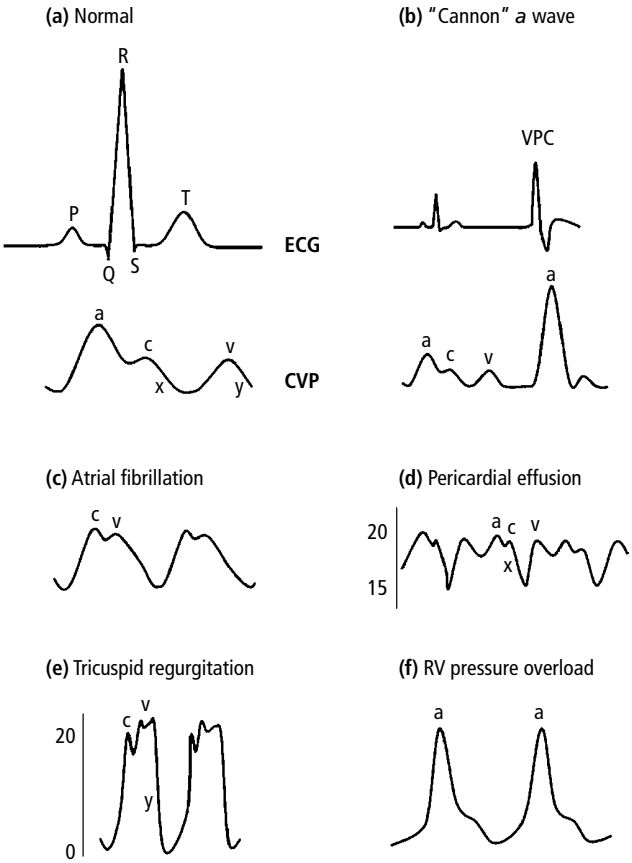


Figure 15.5 Illustration demonstrating (a) normal and (b–f) abnormal central venous pressure (CVP) waveforms. (b) Large "cannon" *a* waves are produced by simultaneous contraction of the right atrium and right ventricle and they can intermittently appear with certain arrhythmias, including second- and third-degree atrioventricular block, and some ventricular arrhythmias such as the ventricular premature complex (VPC) shown here. (c) Atrial fibrillation produces a prominent *c* wave with no a waves. (d) Cardiac tamponade arising from pericardial effusion causes the CVP waveform to become flattened and there is a prominent *x* descent and small or absent *y* descent. (e) A broad and tall *c–v* wave is characteristic of tricuspid regurgitation. (f) Right ventricular (RV) pressure overload arising from pulmonary hypertension or pulmonic stenosis produces large, prominent a waves.

Ventricular premature contractions (seen as ventricular premature complexes on an ECG), atrial fibrillation, atrial premature contractions (seen as atrial premature complexes on an ECG), and second- or third-degree atrioventricular node block can intermittently produce large ("cannon") *a* waves due to atrioventricular dissociation, where there is a transient increase in atrial pressure caused by contraction of the atrium against a closed tricuspid valve during ventricular systole (Figure 15.5b) [48]. When one of these arrhythmias is present, the CVP should be estimated from the normal *a* waves visible on the waveform tracing [47].

Pericardial Effusion with Cardiac Tamponade

Increased pericardial fluid pressure inhibits diastolic filling of the heart. This results in an increase in the mean CVP and flattening of the CVP waveform due to greater equalization of the pressures within the atria and ventricles. A prominent *x* descent is seen due to a rapid reduction in atrial pressure during ventricular systole, and the *y* descent is small or absent (Figure 15.5d) [47].

Tricuspid Regurgitation

Tricuspid valvular disease results in obliteration of the *x* descent during ventricular systole by a large wave created by the backward flow of blood through the incompetent valve [47, 48]. This wave is composed of the merging of the *c* wave and *v* wave. Both may be clearly visible when there is mild insufficiency, but they combine to form a broad wave with a single peak when severe insufficiency is present (Figure 15.5e) [48].

Pulmonic Stenosis and Pulmonary Hypertension

Conditions such as pulmonic stenosis and pulmonary hypertension can result in large *a* waves as the right atrium contracts against the elevated right ventricular pressure (Figure 15.5f) [48].

Alternative Techniques for Assessing Vascular Volume

The gold standard of fluid therapy decision-making would be to assess appropriateness based on its effect on cardiac output. Unfortunately, the technical challenges associated with cardiac output measurement preclude its frequent use in veterinary medicine (see Chapter 14 for more information regarding cardiac output assessment). Owing to its simpler measurement technique and clinical utility, CVP measurement has become a commonly used hemodynamic monitoring tool in veterinary critical care. However, it does not reliably correlate with cardiac output. Therefore, CVP should always be interpreted in conjunction with other clinical and biochemical markers of intravascular volume status as well as with the knowledge of the animal's cardiac and kidney function.

In the critical patient, the importance of performing serial, systematic physical examinations cannot be overemphasized, as these examinations may allow the clinician to detect early changes supportive of low circulating blood volume. Compensatory cardiovascular and renal changes in response to hypovolemia result in centralization of blood volume, renal sodium and water retention, and peripheral vasoconstriction. Initially, few clinical changes will be apparent when homeostatic mechanisms are sufficient to restore tissue perfusion. However, as the volume deficit worsens, these changes typically manifest as the development of mental obtundation, pale mucous membranes, delayed capillary refill time, tachycardia, poor pulse strength, and cool extremities. When physical findings are equivocal, blood lactate concentration can provide a quantitative measure of the severity of impaired tissue perfusion and anaerobic metabolism. As shock worsens, arterial blood pressure will fall, and oliguria or anuria will develop. A high urine specific gravity will also be seen unless a concurrent disorder is present that is impairing renal concentrating ability. See Chapter 17 for more information about many of these other indices of perfusion.

Conversely, in the absence of hypoalbuminemia or cardiac dysfunction, clinical signs suggestive of excessive intravascular blood volume include peripheral edema, chemosis, pleural or peritoneal effusion, serous nasal discharge, and in some cases, development of a new heart murmur or an increase in murmur intensity. Urine output is typically high and urine specific gravity low. If pulmonary edema is present, pulmonary crackles and respiratory difficulty may be evident. These findings, particularly when combined with rising CVP measurements, provide convincing evidence of volume overload.

Conclusion

In summary, the measurement of CVP is a useful adjunctive hemodynamic monitoring tool in critically ill patients and it is readily performed in any patient that has a central venous catheter. However, proper interpretation of CVP requires an understanding of the strengths and limitations of this diagnostic modality. Trends, rather than isolated values, should be followed and the information provided should be considered in terms of probabilities rather than absolutes. A severely low or falling CVP suggests a high probability of hypovolemia. A severely elevated or rising CVP supports hypervolemia. However, similar increases are also seen with increased venous tone, reduced cardiac compliance, diminished cardiac function, or increased intrathoracic or intra-abdominal pressures. In conjunction with other clinical markers of cardiovascular function, CVP remains a useful guide in the assessment and treatment of problems related to intravascular volume status and right-sided heart function.

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16

Cardiac Output Monitoring

Mack Fudge

Hemodynamic monitoring is a cornerstone of critical patient care management. Evaluation of the cardiovascular status of critically ill patients can be broadly divided into parameters that relate to venous return to the heart and those that relate to forward flow from the heart. Parameters that evaluate venous return (preload) include ease of jugular vein distention, central venous pressure (CVP), caudal vena cava diameter (radiography or ultrasonography), and end-diastolic ventricular volume (ultrasonography). Parameters that evaluate forward flow are generally: (i) those related to cardiac output, such as heart rate, stroke volume, pulse quality, and cardiac output; (ii) arterial blood pressure (ABP); (iii) those affected by arteriolar vasomotor tone, such as mucous membrane color and capillary refill time; and (iv) those linked to tissue perfusion, like appendage temperature, urine output, gastric CO_2 tension, oxygen extraction ratio, venous oxygen tension, and metabolic acid–base balance including blood lactate concentration.

Indications for Cardiac Output Measurement

There are various levels of knowledge involved in the evaluation of a patient's cardiovascular status. Clinicians typically start with historical data and a physical examination including mental status, hydration, ease of jugular vein distention, heart rate, pulse quality, mucous membrane color, capillary refill time, and a distal appendage temperature. Based on the findings of this examination, a strong case can frequently be made for hypovolemia, low cardiac output, or poor tissue perfusion; and a therapy plan can be formulated. If the patient responds to therapy, no additional cardiovascular information is necessary. In other cases, the information derived from the initial history and physical examination is insufficient to comfortably define a patient's status, or the patient's response to the initial therapy is insufficient to restore normal-range cardiovascular values. In these cases, subsequent therapeutic interventions require additional information. A second level of cardiovascular information is gained by measuring parameters like CVP, caudal vena cava diameter, end-diastolic left ventricular volume, ABP, and parameters of metabolic acid–base balance. (There are chapters in this textbook describing these techniques.) Often this additional information helps clarify the patient's cardiovascular status and facilitates consequent therapeutic decisions. However, there are a few residual patients that still cannot be clinically defined or do not respond to therapy. In these patients, additional information is required. It is at this point that forward flow information such as cardiac output and oxygen delivery could be helpful.

Cardiac output is typically thought of in the context of being the product of heart rate and stroke volume. Heart rate is straightforward. In a clinical setting, it can be either too high or too low. The therapeutic goal is simply to keep it within a normal physiologic range. Stroke volume is the volume of blood expelled by the heart (right or left ventricle) with each heartbeat. It is influenced by preload, afterload, and cardiac contractility. Preload is effectively the venous return to the heart, or the end-diastolic ventricular filling volume. Afterload relates to arteriolar vasomotor tone or systemic vascular resistance against which the heart works to affect forward blood flow. Contractility can be conceptualized as the intrinsic ability of the individual patient's heart to pump blood. Cardiac output is also influenced by vasomotor tone where blood flow is directly proportional to blood pressure and indirectly proportional to systemic vascular resistance.

Even though cardiac output can be measured many ways, common clinical techniques are somewhat limited in veterinary patients. This is usually due to cost and complexity. In a recent unpublished survey of the 50

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American Veterinary Medical Association accredited colleges of veterinary medicine around the world, an overwhelming majority of schools reported that they do not monitor cardiac output in clinical patients. Of the 46 respondents, only two reported occasional clinical cardiac output monitoring. Others reported cardiac output monitoring as a component of their research efforts, demonstrating that although it may not be practical in the clinical setting, the concept is important in critical patient care.

Cardiac Output Measurement

Classically, clinically applicable techniques used to measure cardiac output involved use of large multiple-lumen catheters connected to expensive, complex monitoring devices. These somewhat invasive techniques were usually done in the intensive care unit or anesthesia setting. Monitoring cardiac output commonly involved indicator dilution. In the past 30 years, there have been many advances in human medicine that have led to less-invasive hemodynamic monitoring techniques [1].

Indicator Dilution Techniques

Indicator dilution techniques involve the injection of a known volume (V_1) of indicator with a known concentration (C_1) into an unknown larger volume (V_2), and then measuring the concentration (C_2) of the indicator in the larger volume of fluid. The unknown volume (V_2) is then calculated by the following formula:

$$C_1 \times V_1 = C_2 \times V_2 \tag{16.1}$$

In a moving fluid such as the cardiovascular system, C_2 is calculated as the average change in indicator concentration over time.

Thermodilution

With indicator dilution techniques, any indicator can be used if it can be measured with a rapidly responding sensor. In thermodilution, the indicator is temperature. A small volume of fluid (V_1) at a known temperature (T_1) is injected into the cranial vena cava or right atrium. This injected fluid flows and mixes with the blood through at least two heart valves. The change in temperature as the blood–fluid mix flows past a rapid-acting thermistor in the pulmonary artery or a peripheral artery is measured (Figure 16.1). The average change in temperature (T_2) is integrated from the change in temperature over time. The unknown volume of blood (V_2, the cardiac output) is then calculated by the Stewart–Hamilton formula:

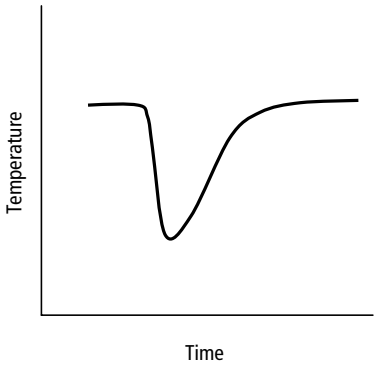


Figure 16.1 Temperature–time curve of a thermodilution cardiac output measurement. (Tracing can be displayed in either an upward or a downward direction.)

$$\text{Cardiac output} = V_1 \times \left(T_b - T_i\right) \times \left(\text{computation constant}\right) \div \left[\begin{array}{l}\text{integrated area of the measured} \\ \text{temperature change over time}\left(T_2\right)\end{array}\right] \tag{16.2}$$

where V_1 is the volume of the injectate, T_b is baseline blood temperature, and T_i is the injectate temperature. The computation constant is calculated from the density and specific heat of the injectate compared with the density and specific heat of blood, and the change in temperature of the injected fluid as it traverses the catheter. The computation constant is provided by the manufacturer of the equipment used. The computer calculates cardiac output in milliliters or liters per minute. This value can be indexed to body size as kilograms of body weight or square meters of body surface area (Table 16.1).

Thermodilution has been demonstrated to be accurate and repeatable in in vitro models [3–6] and when compared in vivo with dye dilution [7], electromagnetic flowmetry [8], and transit-time flowmetry [9].

Transpulmonary thermodilution cardiac output measurements, with a thermistor placed in a peripheral artery, were reported to accurately reflect traditional thermodilution cardiac output measured with a pulmonary arterial catheter [10–14]. This technique appears highly reproducible for monitoring cardiac output in critically ill patients, especially with children [15]. Using the transpulmonary thermodilution method, extravascular lung water (pulmonary edema) can also be estimated.

Cardiac output can be measured continuously with specialized thermodilution catheters that incorporate a thermal filament near the proximal port of a catheter positioned in the cranial vena cava or right atrium. The thermal filament cycles on and off. The change in temperature of the heated blood is sensed by the downstream thermistor in the pulmonary artery. Values for cardiac output, ejection fraction, end-diastolic volume, end-systolic

Table 16.1 Body weight to body surface area and height conversion table.

Weight (kg)	BSA (m^2)	Height (m)	Weight (kg)	BSA (m^2)	Height (m)	Weight (kg)	BSA (m^2)	Height (m)
0.5	0.06	0.30	11	0.5	0.85	31	1.00	1.21
1	0.1	0.38	12	0.53	0.88	32	1.02	1.22
1.5	0.13	0.44	13	0.56	0.90	33	1.04	1.23
2	0.16	0.48	14	0.59	0.92	34	1.06	1.24
2.5	0.19	0.52	15	0.61	0.94	35	1.08	1.25
3	0.21	0.55	16	0.64	0.97	36	1.10	1.27
3.5	0.23	0.58	17	0.67	0.99	37	1.12	1.28
4	0.25	0.60	18	0.69	1.00	38	1.14	1.29
4.5	0.28	0.64	19	0.72	1.02	39	1.16	1.30
5	0.30	0.66	20	0.74	1.04	40	1.18	1.31
5.5	0.31	0.67	21	0.77	1.06	41	1.20	1.32
6	0.33	0.70	22	0.79	1.07	42	1.22	1.33
6.5	0.35	0.72	23	0.82	1.09	43	1.24	1.34
7	0.37	0.73	24	0.84	1.11	44	1.26	1.35
7.5	0.39	0.75	25	0.86	1.12	45	1.28	1.36
8	0.40	0.76	26	0.89	1.14	46	1.30	1.38
8.5	0.42	0.78	27	0.91	1.15	47	1.32	1.39
9	0.44	0.80	28	0.93	1.16	48	1.33	1.39
9.5	0.45	0.81	29	0.95	1.18	49	1.35	1.40
10	0.47	0.83	30	0.98	1.20	50	1.37	1.41

BSA, body surface area. *Source:* Ricco 2020/with permission of John Wiley & Sons.

volume, and stroke volume are generated as an average over the prior 5–10 minutes. This methodology was reported to compare well with intermittent thermodilution cardiac output measurements [16–19], although low-range cardiac outputs may be overestimated while high-range cardiac outputs may be underestimated [20].

Thermodilution cardiac output measurements were traditionally the standard of practice for clinical measurements of cardiac output in people. Balloon-tipped thermodilution catheters are, however, invasive, expensive, and variably difficult to place. Measurements are subject to significant inter-measurement variation. Repeated measures to obtain an average are necessary and take time. Many studies compare the cardiac output estimates of various methodologies with thermodilution. Variance and bias are usually blamed on the compared methodology without regard to the inherent variability of thermodilution. This approach elevates thermodilution to "gold standard" status and perhaps unfairly biases against the tested methodology [21, 22].

While thermodilution catheters permit the measurement or calculation of many important cardiovascular parameters, their use has a variable record with regard to improved patient survival [23–29]. Although seemingly increased monitoring should provide the means for improved patient care, there is no overwhelming evidence for improved outcomes. The lack of statistically significant survival benefit does not, however, prove that the thermodilution catheter is a useless monitoring tool. Thermodilution catheters were used ubiquitously in human critical care and without regard for selecting patients who might truly be benefited. Of course, ultimate survival depends upon the effectiveness of the management of the underlying disease process, regardless of the tools used to monitor patients.

Chemical Indicator Dilution

Lithium and indocyanine green (ICG) indicators can also be used to measure cardiac output. The measurement is made by injecting a known amount of indicator into a central vein and withdrawing blood at a constant rate from an arterial catheter, past a lithium sensor or densitometer, respectively. Newer methods of ICG cardiac output use a transcutaneous finger photosensor [30] or ICG fluorescence [31] rather than an ex vivo cuvette densitometer. With the lithium indicator, cardiac output is calculated from the lithium dose and the area under the lithium concentration-versus-time curve:

$$\text{Cardiac output} = \left[\text{lithium dose}\left(\text{mM}\right)\right] \times 60 \div \left[\text{integrated area under the concentration} - \text{time curve}\left(\text{mM/sec}\right) \times \left(1 - \text{PCV}\right)\right] \quad (16.3)$$

In this equation, PCV stands for packed cell volume. Lithium dilution was reported to compare well with thermodilution in dogs [32], cats [33], and foals [34]. One limitation to lithium and dye dilution techniques is the number of measurements that can be performed before background indicator concentrations start to interfere with subsequent measurements.

Balloon-Tipped Thermodilution Catheter

Balloon-tipped catheters are available as two-lumen catheters for measuring pulmonary artery pressure and pulmonary artery occlusion pressure; as four-lumen catheters for measuring CVP, pulmonary artery pressure, and cardiac output (Figure 16.2); and as five- and six-lumen catheters for measuring CVP, pulmonary artery pressure, and cardiac output, for placement of pacing electrodes, and for continuous reflectance oximetry. Catheters are available in lengths of 80 cm (for pediatrics) or 110 cm (for adults) and in diameters of 4–8.5 Fr, depending upon the number of lumens and intended use. The 4-Fr catheter is the smallest diameter four-lumen catheter with thermodilution capabilities and can be used in cats and small dogs (1–3 kg). The 5- and 6-Fr catheters are suitable for dogs of 3–10 kg; 7-Fr four-lumen catheter can be used in dogs over 10 kg.

Insertion of the Balloon-Tipped Thermodilution Catheter and Complications

Introduction of a balloon-tipped thermodilution catheter can be a technically and technologically intensive procedure. Catheter placement and its later repositioning after placement are facilitated using an appropriately sized introducer catheter. Catheters can be placed via the jugular, saphenous, or femoral vein. Introducer wires, introducer catheters, and thermodilution catheters are long, floppy, and easily contaminated. Sepsis is a major hazard of these catheters. Once placed, they are "busy" catheters, frequently used throughout the day to make measurements and collect blood samples for testing. Aseptic introduction must be assured. Large sterile drapes are used to extend the sterile field to facilitate safe catheter placement.

First, an introducer catheter is placed and secured with sutures. The balloon-tipped catheter is attached to a pressure transducer and a physiologic monitor for pressure measurements to identify the location of the catheter during introduction. As the catheter is advanced toward the right atrium, a CVP tracing should appear on the monitor. When the catheter enters the right ventricle, the tracing changes to a ventricular pressure waveform. Once in the right ventricle, the catheter's balloon is inflated and further advanced until it enters the pulmonary artery. The inflated balloon acts as a sail, aiding the direction of the catheter's forward movement along the route of blood flow from the right ventricle into the pulmonary artery. Once in the pulmonary artery, with the balloon still inflated, the catheter is advanced until it occludes a branch of the pulmonary artery. Here, the pressure tracing will change to a typical occlusion pressure (Figure 16.3). Deflation of the balloon allows the measurement of pulmonary artery pressure. Reinflation of the balloon allows the measurement of pulmonary artery occlusion, or "wedge" pressure.

If during placement the catheter is introduced as far as 50 cm and has not entered the right ventricle, it has likely either coiled in the right atrium or passed into the caudal vena cava. The catheter should be withdrawn and reinserted. There is little directional control over the tip of the catheter, so on subsequent reintroduction, the catheter should be rotated in one direction or another and advanced until it ultimately ("accidentally") falls into the right ventricle. If the catheter end-hole butts up against a vessel or

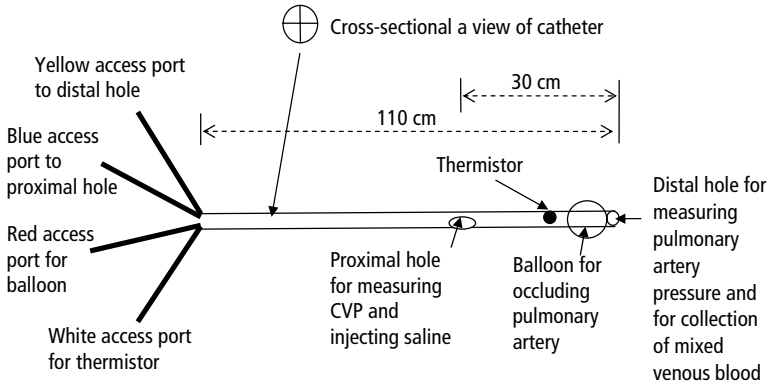


Figure 16.2 Schematic of a four-lumen, thermodilution, balloon-tipped catheter. CVP, central venous pressure.

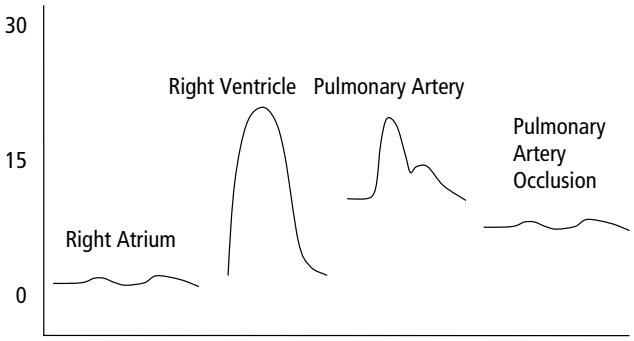


Figure 16.3 Schematic of representative pressure waveforms while introducing the balloon-tipped catheter into the pulmonary artery.

chamber wall, there will be a sudden cessation of pressure waveform on the monitor. If all else fails, fluoroscopy can be used to guide the introduction of the catheter.

When the catheter is first introduced, there is usually a big loop of catheter in the right ventricle. Over time the catheter will migrate further into smaller branches of the pulmonary artery. Full inflation of the balloon could rupture these smaller pulmonary vessels. If less than full balloon inflation occludes a vessel (indicated by the appearance of a typical occlusion pressure waveform on the monitor), the balloon should be deflated, and catheter withdrawn a short distance until it requires the full 1-ml inflation to occlude the vessel. The balloon should be inflated only for the measurement of occlusion pressure and then deflated. It should not remain inflated for an extended duration because of the potential for vessel damage. Pulmonary vessel trauma and rupture has been reported in people due to leaving the balloon inflated or by fully inflating the balloon after the catheter has migrated [35]. Once the catheter is ideally positioned, so that all measurements (central venous, pulmonary artery, and occlusion pressure) can be obtained, it is secured to the patient and bandaged aseptically.

Physical contact of these catheters against the endocardium, especially during introduction, is occasionally associated with arrhythmias. Simply stopping or withdrawing the catheter slightly should fix the problem. Persistent arrhythmia problems may be treated with a common antiarrhythmic.

Catheter-associated clot emboli may occur. There is not much one can do to prevent it. It is not typical to administer anticoagulants to veterinary patients with these catheters or any other type of catheter. Heparin-coated catheters are available that may decrease this problem. Air emboli may occur if air is inadvertently injected into the catheter or into the balloon port if the balloon has ruptured. Rarely reported complications include pneumothorax, hemothorax, and knotting of the catheter (in the right ventricle) during removal.

Thermodilution Cardiac Output Measurement

A pressure transducer and physiologic monitor are required to measure pressures. A compatible cardiac output computer is used for measuring cardiac output. A compatible oximeter is needed for venous blood oxygen saturation measurements.

To make a cardiac output measurement, the thermistor connection is attached to the cardiac output computer. Some systems incorporate an injectate-measuring thermistor at the injection port. The computer will calculate a compensated value for the change in temperature of the injectate as it traverses the catheter, otherwise the temperature of the injectate (room temperature or iced) will need to be measured and entered into the computer. Usually, room temperature fluid is used. Ice water temperature fluid may be necessary for signal detection when smaller volumes of fluid are used in larger patients. Usually, a small volume of an isotonic crystalloid fluid (3–5 or 10 ml; smaller volumes for smaller patients) is used. After recording all the measured pressures, the operator must indicate to the computer that an injection is about to be made. The computer will indicate when it is ready for injection to begin. The designated volume of fluid is injected into the cranial vena cava or right atrium. The operator should try to make the injection at the end of exhalation and as fast as possible. The computer will measure the change in temperature as the fluid–blood mixture passes by the thermistor in the pulmonary artery and calculate the average change in temperature and cardiac output. Typically, outliers are discarded; three to five consistent measurements are taken and averaged to obtain a representative cardiac output value.

The balloon-tipped thermodilution catheter has a useful role in the characterization and management of hemodynamic alterations in critically ill patients. The use of these catheters has decreased in the past 30 years because of recent advances in less invasive hemodynamic monitoring techniques, combined with the results of several randomized trials failing to show improvement in outcomes with their use. Although it is obvious that balloon-tipped catheters should not be commonly used in most critically ill patients, they are still potentially useful in some patients with circulatory and/or respiratory failure, especially when associated with pulmonary hypertension or left heart dysfunction. As with any technique, their use requires expertise in insertion, acquisition, and interpretation of measurements. The decrease in use of the balloon-tipped thermodilution catheters may unfortunately limit exposure of junior doctors and technicians to this device, so that they become less familiar with their use, making it even more complicated and less optimal [1].

Lithium Cardiac Output Measurement

A central venous and an arterial catheter are placed. A lithium sensor is attached to an arterial catheter and to a cardiac output computer. Arterial blood is withdrawn past the sensor at a constant rate and into a collection container. Hemoglobin and sodium concentration are pre-measured and entered into the computer. A sensor constant and the intended lithium dose are also entered into the computer. The calculated dose of lithium is placed into an extension set attached to a jugular catheter. The pump for withdrawal of arterial blood and the cardiac output computer are started. The dose of lithium is flushed into the cranial vena cava after 5–10 seconds. The sensor measures the change in lithium concentration over time and the computer calculates cardiac output. The measurement should be repeated at least once, outliers excluded, and remaining values averaged.

Other Measurements and Calculations

The measurements that can be obtained from the thermodilution catheter are CVP, pulmonary artery pressure, pulmonary artery occlusion pressure, and cardiac output. In addition, mixed-venous blood samples can be obtained from the distal port of this catheter for pH and blood gas analysis. If a separate arterial catheter is placed, ABP and arterial blood samples for pH and blood gas analysis can be obtained. In addition to cardiac output, measurements that can be obtained during lithium cardiac output measurements include CVP and blood sampling, and arterial pressure and blood sampling (for pH and blood gas analysis).

Once cardiac output is measured, heart rate is measured separately, and stroke volume can be calculated. When arterial pressure, pulmonary pressure and cardiac output are measured; systemic arterial and pulmonary vascular resistance and left and right heart work indices can be calculated. When hemoglobin concentration and partial pressure of oxygen (PO_2) are known, oxygen content can be calculated. When cardiac output and oxygen content are known, oxygen delivery can be calculated. When cardiac output and arterial and mixed-venous oxygen content are known, the oxygen consumption and extraction and the venous admixture can be calculated. Table 16.2 contains standard calculations.

Table 16.2 Standard formulas for calculated variables.

Parameter	Formula
Body surface area	$(10.1 \times \text{kg}^{0.67})/100$
Alveolar PO_2 (room air)	[(barometric pressure – 50) × 0.21] – ($PaCO_2$/RQ), where 50 is the saturated water vapor pressure at 38.5°C, 0.21 is the fractional inspired oxygen, and RQ = 0.9
Arterial, mixed venous, and capillary oxyhemoglobin saturation	$([38{,}848/(202 \times PO_2 + 1.17 \times PO_2^2 + PO_2^3)] + 1) - 1 \times 100$[a]
Arterial, mixed venous, and pulmonary capillary oxygen content	$(1.34 \times \text{Hb} \times SO_2) + (0.003 \times PO_2)$, where 1.34 is 100% saturated hemoglobin oxygen content, SO_2 is hemoglobin saturation, PO_2 is partial pressure of oxygen in arterial, mixed venous, or capillary blood
Cardiac index	Cardiac output per square meter BSA or kilogram of body weight
Stroke volume index	CI/heart rate
Systemic vascular resistance index	(ABP–CVP) × 79.92/CI m^2 or (ABP-CVP)/CI kg
Pulmonary vascular resistance index	(PAP–PAOP) × 79.92/CI m^2 or (PAP-PAOP)/CI kg
Left and right cardiac work index	CI × ABPm × 0.0144 CI × PAPm × 0.0144
Left and right ventricular stroke work	SVI × ABPm × 0.0144 SVI × PAPm × 0.0144
Oxygen delivery	$CaO_2 \times (\text{CI } m^2 \times 10)$ or (CI kg/100)
Oxygen consumption	$(CaO_2 - CmvO_2) \times (\text{CI } m^2 \times 10)$ or (CI kg/100)
Oxygen extraction	$VO2/DO_2$ or $CaO_2 - CmvO_2/CaO_2$
Venous admixture	$(CcO_2 - CaO_2)/(CcO_2 - CmvO_2)$
Arterial and venous blood carbon dioxide content	$(2.226 \times 0.0299 \times PCO2 \times (1 + 10^{[\text{pH} - 6.085]})) \times (1 - ((0.0289 \times \text{Hb})/((3.352 - (0.456 \times (SO_2/100))) \times (8.142 - \text{pH}))))$[b]
Carbon dioxide production	$(CaCO_2 - CmvCO_2) \times \text{CI} \times 10$

[a] Reeves et al. (1982) [36].
[b] Douglas et al. (1988) [37].

Other Methods of Estimating Cardiac Output

Electromagnetic flowmetry is the gold standard for experimental blood flow measurement. Flow probes are surgically placed around a suitably size-matched vessel of interest or flow catheters can be introduced into a large vessel. The application of a magnetic field perpendicular to the blood flow induces an electrical potential proportional to the blood flow velocity that can be easily and accurately calculated. Flow is calculated from flow velocity and conduit diameter. Other methods of measuring cardiac output have been recently reviewed [38, 39].

The Fick Method

The *Fick* principle assumes that flow is proportional to the rate of uptake of an indicator gas, and the difference

between the concentration of the indicator gas entering and exiting the organ being studied:

$$\text{Flow} = \left(\text{indicator gas uptake}\right) \div \left[\left(\text{gas conc.in}\right) - \left(\text{gas conc.out}\right)\right] \quad (16.4)$$

Various marker gases have been used: oxygen, carbon dioxide, acetylene, and nitrous oxide. If any two parameters in this equation are known, the third can be calculated. When cardiac output is measured, for instance, arterial and venous oxygen content can be calculated from PO_2 and hemoglobin measurements (Table 16.2). Oxygen consumption (VO_2) can be calculated from the product of flow and arterial–venous O_2 content. The venous blood sample used for these calculations must come from a central vein: pulmonary artery or vena cava. Unfortunately, cephalic or saphenous venous blood oxygen measurements will not suffice for this calculation. By the Fick equation, cardiac output can be calculated from oxygen consumption divided by arterial–venous O_2 content. Oxygen consumption is calculated as the difference between inspired tidal volume and oxygen concentration and expired tidal volume and oxygen concentration. It is usually measured in intubated patients. Fick cardiac output estimates compared well with thermodilution cardiac output measurements in cats at low ($r = 0.89$) and normal ($r = 0.69$) cardiac outputs but overestimated high ($r = 0.75$) cardiac outputs [40]. Fick cardiac output estimates compared well with dye dilution cardiac output measurements in anesthetized dogs [41] and pigs [16].

Arterial–Venous Oxygen Content, Oxygen Extraction, Venous Oxygen, Arterial–Venous Oxygen Saturation

Oxygen consumption is usually not measured in clinical veterinary medicine, so the whole Fick equation (Eq. 16.4) usually cannot be used. However, assuming oxygen consumption has not changed too much, the arterial–venous oxygen content difference alone can be used to estimate the adequacy of tissue perfusion. In clinical situations, this assumption may be flawed since oxygen consumption can easily halve (hypothermia; general anesthesia) or double (increased muscular activity). Similarly, the adequacy of tissue perfusion is directly related to cardiac output only if the animal is not vasoconstricted. When oxygen delivery decreases and oxygen uptake continues at its previous level, a greater proportion of oxygen is removed from the blood (oxygen extraction). This will result in a decrease in venous oxygen and a greater difference between arterial and venous oxygen content. Arterial–venous oxygen content was reported to be 3.6 ± 1.2 ml/dl in normal dogs, increasing to 7.0 ± 1.9 ml/dl in moderately hypovolemic dogs [42]. In this same report, oxygen extraction increased from 21 ± 6% to 42 ± 10%. Oxygen extraction is calculated by the following formula:

$$\text{Oxygen extraction} = \left(\text{Cont}_{\text{Art}}\,\text{O}_2 - \text{Cont}_{\text{Ven}}\,\text{O}_2\right) \Big/ \text{Cont}_{\text{Art}}\,\text{O}_2 \quad (16.5)$$

Central venous oxygen (PO_2 or saturation, SO_2) alone (without calculating oxygen content) can also be used in this same context. Normal central venous PO_2 and SO_2 were reported to be 49 mmHg ± 6 and 78% ± 6, respectively, in normal dogs and decreased to 35 mmHg ± 5 and 56% ± 9 in moderate hypovolemia [42]. In another canine acute hemorrhage model [43], partial pressure of oxygen in mixed venous blood (PvO_2) decreased from 46 to 34 mmHg. Venous PO_2 was reported to be significantly correlated with cardiac output in cats; however, there was notable variability [40]. In a cohort of critically ill people, dobutamine augmentation of cardiac output, decreased oxygen extraction from 48% to 36% and increased mixed-venous oxygen saturation from 49 to 61% [44]. Other studies reported weak correlations between venous oxygen saturation and cardiac output in human patients in intensive care [45] and in a piglet hemorrhagic shock model [46].

Carbon Dioxide-Based Fick

The Fick principle and formula can also be used with carbon dioxide production:

$$\text{Flow} = \left(\text{carbon dioxide production}\right) \Big/ \left(\text{CvCO}_2 - \text{CaCO}_2\right) \quad (16.6)$$

A commercial, noninvasive method for measuring cardiac output in intubated patients is available (NICO, $NICO_2$, and NM3; Philips-Respironics, Murrysville, PA). The method involves the transient partial rebreathing of carbon dioxide (for 50 seconds every 3 minutes). Cardiac output is calculated from end-tidal CO_2 concentrations during normal and CO_2 rebreathing episodes. End-tidal CO_2 (measured) and CO_2 solubility are used to calculate arterial CO_2 concentration. Inspired oxygen and arterial oxygenation are used to calculate a shunt fraction, which is used to correct for the shunted portion of cardiac output [47, 48]. This method of analysis compared favorably with normal-range thermodilution cardiac output measurements (but overestimated cardiac output at low-range cardiac outputs and underestimated high-range cardiac outputs) [20]. The $NICO_2$ system compared acceptably well with ultrasound transit-time flowmetry and thermodilution during and after cardiopulmonary bypass [9]. It also

compared acceptably with lithium cardiac output measurements across a spectrum of low to high cardiac outputs in anesthetized dogs and foals [48, 49]. The NICO family of monitors are noninvasive, easy to use, and provide near-continuous measurements.

Venous–Arterial Partial Pressure of Carbon Dioxide

Carbon dioxide production is not measured in clinical veterinary practice, so the whole Fick equation (Eq. 16.6) cannot be used. However, if CO_2 production has not changed too much, the venous–arterial partial pressure of carbon dioxide (PCO_2) difference alone can be used to estimate the adequacy of tissue perfusion. In common clinical situations, the CO_2 production can easily halve (hypothermia or general anesthesia) or double (increased muscular activity). When blood flow decreases, and CO_2 production continues at its previous level, an increase in venous CO_2 results in a greater difference between venous and arterial PCO_2. Venous–arterial PCO_2 was reported to be 4.2 mmHg ± 1.5 in normal dogs, increasing to 10.7 mmHg ± 3.9 in moderately hypovolemic dogs [42]. In a canine acute hemorrhage model, venous–arterial PCO_2 increased from 5.2 to 12.9 mmHg [43]. Venous–arterial PCO_2 was 4.9 mmHg in critically ill people with normal cardiac output measurements and was 7.4 mmHg in patients with low cardiac output [50]. Venous–arterial PCO_2 decreased from 9 to 5 mmHg with dobutamine augmentation of cardiac output [44].

Pulse Contour Methods

The area under the pulse pressure waveform bears some correlation to stroke volume. The pulse pressure waveform can be qualitatively characterized by digital palpation of an arterial vessel. A tall, wide pulse is likely associated with a large stroke volume. A short, narrow pulse is likely associated with a small stroke volume. The pulse pressure can be measured by indirect sphygmomanometry or by direct arterial measurement.

Pulse contour methodologies calculate stroke volume from measured pulse pressure waveforms using algorithms that consider arterial impedance, compliance, and resistance. At a given arterial compliance there is a directional relationship between the change in the area under the pulse pressure waveform and the change in stroke volume. Unfortunately, arterial compliance is not measured, so cardiac output must be periodically and independently verified. Once this measured cardiac output is fed into the computer, it back-calculates correction factors that will be used in future pulse contour assessments (until it is again recalibrated). There are several commercial devices that continuously measure and assess the pulse pressure waveform.

The PiCCO2™ (pulse index continuous cardiac output) system (Pulsion Medical Systems, Irving, TX) requires a central venous and an arterial catheter, typically placed peripherally. The PiCCO2 system uses transcardiopulmonary thermodilution to intermittently measure cardiac output. Saline is injected into a jugular catheter and the change in temperature is measured by a thermistor in a special arterial catheter. The system integrates a wide array of both static and dynamic hemodynamic data through a combination of trans-cardiopulmonary thermodilution and pulse contour analysis. The PiCCO2 system also estimates cardiac filling volume, intrathoracic blood volume, and extravascular lung water. The requirement for intra-arterial and central venous catheterization limits the use of PiCCO2 to those patients with critical illness or at high risk of severe hemodynamic derangement. While the accuracy of transcardiopulmonary thermodilution as a measure of cardiac output is well established, several other PiCCO2 measurements require further validation within the context of their intended clinical use. As with all advanced hemodynamic monitoring systems, efficacy in improving patient-centered outcomes has yet to be conclusively demonstrated [51].

The $PiCCO_2$ system compared well with thermodilution cardiac output measurements in critical human patients [11, 12, 14, 52–54], a piglet hemorrhagic shock model [46], and in healthy dogs both with thermodilution and lithium dilution techniques [55, 56]. Another study showed poor trending ability when compared to pulmonary artery thermodilution and suggested that it not be used in critically ill patients [57].

The LiDCO™ Plus system (LiDCO Ltd., Cambridge, UK) uses a pulse power analysis algorithm (PulseCO™) that mathematically calculates changes in stroke volume to provide a real-time continuous assessment of hemodynamic status. Independent lithium indicator dilution cardiac output measurements are needed to calibrate the system. There is good agreement with thermodilution cardiac output measurements, but with large variability. Accuracy falls off with time and periodic recalibration by remeasuring cardiac output may be necessary [14]. The PulseCO system compared well with thermodilution and lithium cardiac output measurements; but it overestimated cardiac output at low-range cardiac outputs and underestimated high-range cardiac outputs [20, 39, 58, 59]. Early canine studies of pulse contour cardiac output estimates reported good correlation and accuracy compared with implanted electromagnetic flowmeter measurements [60, 61] and thermodilution cardiac output measurements [62]. Several subsequent canine studies have reported a high variability

and poor correlation between pulse contour cardiac output estimates and lithium calibration cardiac output [63–65].

The MostCare™ device (Vytech Health, Padova, Italy) uses the pressure recording analytical method (PRAM), via perturbation theory, to estimate cardiac output just from the analysis of the pulse pressure wave profile. It requires only an arterial catheter. The PRAM algorithm characterizes the elastic properties of the arterial system from the analysis of the pulse pressure profile. The PRAM system also provides a parameter called cardiac cycle efficiency, which is an index of heart–vascular response coupling (+1 = best; −1 = worst). Cardiac cycle efficiency is a ratio between myocardial work and energy consumed and represents an index of heart stress. PRAM compares well with thermodilution cardiac output in people [66], and with electromagnetic flowmetry and thermodilution cardiac output measurements in pigs across a wide range of cardiac outputs [67].

The Flo Trac™ sensor and Vigileo™ monitor system (Edwards Lifesciences, Irvine, CA) employ user-entered anthropomorphic data (sex, age, weight, height, and surface area) to assign a value to compliance and vascular tone independent of external cardiac output measurements and system recalibration. The sensor can be used with any arterial catheter and no external calibration is required. For use in veterinary medicine, either the algorithms would need to be changed or false information would have to be entered to enable correct surface area calculations. Although Flo Trac compared well with thermodilution cardiac output measurements in people [39, 54], it was reported to be unsuitable for use in dogs as it overestimates cardiac output with a high rate of error [68, 69].

Pulse wave transit time (PWTT) is the time it takes for a pulse to travel from the aorta to a peripheral artery. It is a non-invasive parameter that can detect changes in blood pressure. PWTT can be used to calculate an estimated stroke volume, which subsequently can be used to calculate an estimated cardiac output. PWTT is measured continuously by monitoring the electrocardiogram (ECG) and a peripheral arterial pulse wave. It is the time from the peak of an ECG R-wave until the rise point on a distal pulse wave [70]. The peripheral pulse wave can be measured from either a distally placed SpO_2 probe or directly from an arterial catheter. This method showed a good trending ability to detect 15% changes in stroke volume. However, it offered an unacceptable agreement of estimated cardiac output measured by traditional thermodilution in anesthetized dogs [71].

Transthoracic Impedance and Bioreactance

The transthoracic impedance method uses two pairs of electrodes to measure changes in transthoracic electrical impedance during the cardiac cycle and then calculates an estimate of stroke volume. An electrical current is passed through the thorax, and the voltage change with each heartbeat is measured. This change in voltage is considered the result of a change in thoracic impedance. The current is introduced by a pair of electrodes placed on both sides of the neck and both sides of the lower thorax. The drop in voltage is detected by another pair of electrodes placed below the current introducing electrodes on the neck, and another pair above the current injecting electrodes on the lower thorax [72]. Over a cardiac cycle, the only intrathoracic fluid volume change is intrathoracic blood volume. The magnitude and rate of change reflects myocardial contractility. Baseline impedance may be affected by other fluid accumulation diseases such as pleural effusion and pulmonary edema. A meta-analysis of thoracic impedance technology concluded that the technique may only be sufficiently accurate as a trend monitor [73]. There are several commercial devices that measure the thoracic impedance.

The BioZ™ impedance cardiography system (Cardiodynamics, San Diego, CA, subsidiary of Sonosite, Bothel, WA) evaluates heart rate, blood pressure, cardiac output, systemic vascular resistance, systolic time ratio (an index of myocardial contractility), and thoracic fluid content. Studies report moderate correlation with thermodilution cardiac output measurements [74, 75] and between thoracic fluid content and the amount of fluid removed by hemodialysis [76].

Electrical cardiometry (Icon™ and Aesculon™; Cardiotronic, Inc. La Jolla, CA) uses electrical velocimetry (changes in thoracic conductivity caused by the alignment of red blood cells) to calculate heart rate, stroke volume, cardiac output, systolic time ratio, and thoracic fluid index. Electrical velocimetry estimates of cardiac output compared favorably with transpulmonary thermodilution cardiac output measurements in piglets [13].

The RheoCardioMonitor™ (ACMA, Ltd., Singapore) assesses cardiac output and compared well to thermodilution cardiac output measurements in pigs [77] and people [78].

The Niccomo™ instrument (Medis, Ilmenau, Germany) measures heart rate and blood pressure. It uses a physiologic adaptive signal analysis (PASA) algorithm to calculate stroke volume, cardiac output, systemic vascular resistance, thoracic fluid content, left cardiac work index, several indices of contractility, and systolic time intervals. The company also produces computer-based impedance cardiography interfaces, using the same technology as the Niccomo, that can run on any personal computer (Cardioscreen 1000 and 2000). The PASA algorithm-derived cardiac output correlated well with thermodilution in people [79]. Other studies reported modest to poor correlation between impedance-estimated cardiac output and transthoracic Doppler cardiography [80], thermodilution [81], and lithium dilution cardiac output (LiDCO) [82].

Whole-body impedance estimates of cardiac output correlated well with thermodilution cardiac output measurements in one study [83] but not in another [84].

Bioreactance (NICOM and Starling SV, Cheetah Medical, Newton Center, MA) uses time delay, or phase shifts, that occur when an alternating electrical current is passed through the thorax to evaluate the flow of blood in the thorax. Proprietary algorithms interpret the signal to provide stoke volume. Findings in various studies using the NICOM have been equivocal and have varied including feasible, satisfactorily approximating, and acceptably accurate [85–87]. At least one study found that the NICOM device cannot accurately estimate cardiac output in critically ill patients and could not predict patients' response to saline volume expansion [88].

Doppler Ultrasound

Standard echocardiography, operated by experienced individuals, can be used to measure aortic or pulmonary valve diameter and the velocity–time integral, from which stroke volume can be calculated. Accurate assessment of the diameter of the outflow tract can be challenging. Measurements are intermittent and not always a readily viable option for critical care settings. Echocardiographic estimated cardiac output did not correlate well with thermodilution cardiac output measurements in cats [40]. In a hemorrhage model in dogs, the pulmonic valve, compared with the aortic valve, and proximal and distal aorta, was the site that generated the most repeatable Doppler measurements [43]. None of the echocardiographic cardiac output determinations compared very well with thermodilution.

The USCOM (Ultrasound Cardiac Output Monitor) device (Uscom Ltd., Sydney, Australia) uses continuous-wave Doppler and human anthropomorphic aortic and pulmonary valve data to calculate stroke volume. The extrapolation of animal to human data would prove a substantial challenge in using this technology. The USCOM was reported to provide reliable measurements of cardiac output over a wide range of values in a study of six anesthetized dogs [89]. There are no reports of its clinical use in dogs or cats.

Doppler probes can be placed into the esophagus and positioned to face the descending thoracic aorta (Hemosonic™ 100, Arrow International, Reading, PA; Dynemo™ 3000, Sometec, Paris; CardioQ-ODM™ Doppler Monitor, Deltex Medical Ltd., Chichester, UK). The phase shift in the reflected ultrasound waves as they are carried downstream by the flowing blood is used to calculate blood flow velocity. Flow velocity and time are used to calculate stroke distance. The product of stroke distance and aortic cross-sectional area (measured or anthropomorphically determined) is used to calculate stroke volume. Cardiac output monitoring, assessment of volume status, and evaluation of left ventricular function are examples of transesophageal-derived information [90, 91]. A major assumption of this technique is that a constant proportion of the cardiac output flows through the descending aorta with the remainder going through the brachiocephalic and coronary arteries. Unfortunately, this is variable. Hypovolemia decreases and vasodilation increases the proportion of the cardiac output entering the descending aorta. Transesophageal probes are large, and anesthesia is required to introduce them. They cannot be fixed in place for continuous measurements. Although some reports in people suggest good correlation with thermodilution cardiac output [53, 92–95], other reports suggest a variable correlation [96]. Although less reliable during hypotension, some studies suggest that it can serve as a minimally invasive alternative to monitoring cardiac output in dogs during anesthesia [95, 97], while others report that it is not accurate in measuring cardiac output in dogs [98, 99]. It has shown an acceptable trending ability and could be a valuable tool in guiding therapy, including in cats [100–102].

Other Methods

Velocity encoded phase contrast magnetic resonance imaging is an accurate technique for measuring flow in large vessels [103]. Nuclear scintigraphy can also be used to evaluate cardiac output [104]. The equipment is very expensive, requires considerable training to operate, and the measurements are intermittent. Thus, these techniques do not lend themselves to use in the intensive care setting.

Interpreting Measurements

There is a large amount of cardiopulmonary function information that can be derived when cardiac output is measured (Figure 16.4 and Table 16.3). This allows for broader characterization of patient status and may improve survival opportunities in some patients. Since forward flow depends on venous return, it is appropriate to first evaluate preload parameters. CVP represents preload pressure to the right heart (0–10 mmHg), and pulmonary artery occlusion pressure represents preload pressure to the left heart (2–12 mmHg). Low pressures suggest that there is room for additional blood volume augmentation if other parameters corroborate hypovolemia. High pressures may represent hypervolemia or heart failure and suggest that additional volume loading may be unwarranted. Preload pressure has a variable relationship to preload volume, depending upon ventricular compliance. High preload pressure does not necessarily define high preload volume, but it always necessitates a conservative fluid plan.

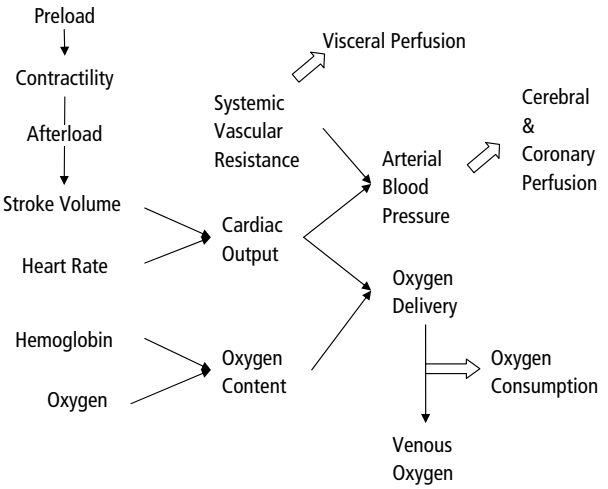


Figure 16.4 Integrated cardiopulmonary function: preload determines diastolic filling of the heart; contractility is the strength of load-independent contraction of the ventricles; afterload is the pressure against which the heart must contract to generate a stroke volume; stroke volume and heart rate determine cardiac output; hemoglobin concentration and the amount of oxygen loaded onto it determine oxygen content; cardiac output and systemic vascular resistance determine arterial blood pressure; arterial blood pressure is primarily important as a determinant of cerebral and coronary perfusion; systemic vascular resistance is primarily important as a determinant of visceral tissue perfusion; cardiac output and oxygen content determine oxygen delivery; oxygen delivery minus oxygen consumption determine venous oxygen.

First, evaluate ABP. Low mean ABP (< 60 mmHg) could be caused by hypovolemia (check preload parameters), poor cardiac output (measure cardiac output and check for cardiac disease), or vasodilation (calculate systemic vascular resistance and check clinical vasomotor tone signs). High mean systemic arterial pressure (> 140 mmHg) is usually attributable to high vasomotor tone (iatrogenic hypervolemia is possible; high cardiac output and arteriosclerosis are unlikely).

Second, evaluate cardiac output. Low cardiac output could be caused by the following: hypovolemia (check the preload parameters); cardiac disease (atrioventricular insufficiency, aortic stenosis, fibrosis, pericardial tamponade); poor contractility (if not measured, then presumed if preload parameters are high and forward flow parameters are low, in the absence of anatomic cardiac disease). Low cardiac output should be treated if it is associated with hypotension or evidence of poor tissue perfusion.

Third, evaluate systemic vascular resistance. Low vascular resistance (vasodilation) may be associated with hypotension, in which case it should be treated by administering a vasoconstrictor. Low vascular resistance associated with acceptable blood pressure does not need to be treated. High vascular resistance (vasoconstriction) may be associated with poor tissue perfusion. If associated with high blood pressure, the situation may benefit from

Table 16.3 Cardiopulmonary values in normal dogs.

Parameter	Units	Baseline n = 97 (mean ± SD)	95% Confidence interval
Body weight	kg	20.5 ± 6.9	
Body surface area	m^2	0.74 ± 0.17	
Temperature	°C	38.4 ± 0.6	38.3–38.5
pHa	iu	7.381 ± 0.025	7.376–7.387
$PaCO_2$	mmHg	40.2 ± 3.4	39.5–41.0
HCO_3a	mEq/l	23.1 ± 2.0	22.7–23.5
BDa	mEq/l	−2.1 ± 2.3	−1.7 to −2.6
pHmv	Units	7.362 ± 0.027	7.356–7.367
$PmvCO_2$	mmHg	44.1 ± 3.8	43.3–44.9
HCO_3mv	mEq/l	24.2 ± 2.1	23.7–24.6
BDmv	mEq/l	−1.9 ± 2.3	−1.4 to −2.3
a–mv pH	iu	0.020 ± 0.012	0.018–0.022
a–mv PCO_2	mmHg	−3.9 ± 1.6	−3.6 to −4.2
a–mv HCO_3	mEq/l	−1.1 ± 0.7	−0.9 to −1.2
a–mv BD	mEq/l	0.2 ± 0.7	0.1–0.4
PaO_2	mmHg	99.5 ± 6.8	98.1–100.8
SaO_2	%	96.3 ± 0.9	96.1–96.5
Hb	g/dl	13.6 ± 1.8	13.3–14.0
CaO_2	ml/dl	17.8 ± 2.3	17.4–18.3

(*Continued*)

Table 16.3 (Continued)

Parameter	Units	Baseline n = 97 (mean ± SD)	95% Confidence interval
$PmvO_2$	mmHg	49.3 ± 5.8	48.2–50.5
$SmvO_2$	%	77.1 ± 5.5	75.6–78.2
$CmvO_2$	ml/dl	14.2 ± 2.2	13.8–14.7
$Ca–vO_2$	ml/dl	3.6 ± 1.0	3.4–3.8
PAO_2	mmHg	105.8 ± 3.7	105.1–106.9
$A–aPO_2$	mmHg	5.5 ± 6.9	3.6–7.4
ScO_2	%	96.9 ± 0.5	96.8–97.0
CcO_2	ml/dl	18.0 ± 2.3	17.5–18.5
Venous admixture	%	3.6 ± 4.1	2.8–4.4
$CaCO_2$	ml/dl	45.8 ± 4.3	44.9–46.6
$CmvCO_2$	ml/dl	48.5 ± 4.4	47.6–49.4
$Ca–vCO_2$	ml/dl	2.7 ± 1.4	2.5–3.0
CVP	cm H_2O	3.1 ± 4.1	2.3–4.0
PAOP	mmHg	5.5 ± 2.9	4.8–6.2
Heart rate	bpm	87 ± 22	83.0–91.8
ABPm	mmHg	103 ± 15	99.9–106.0
PAPm	mmHg	14.0 ± 3.2	13.4–14.7
CO	ml/minute	3360 ± 1356	3086–3633
CI	l/minute/m^2	4.42 ± 1.24	4.17–4.67
	ml/minute/kg	165 ± 43	156–174
SVI	ml/beat/m^2	51.9 ± 13.5	49.2–54.7
	ml/beat/kg	1.93 ± 0.46	1.84–2.02
SVRI	dyne·second·cm^{-5}/m^2	1931 ± 572	1815–2045
	mmHg/ml/minute/kg	0.641 ± 0.173	0.606–0.676
PVRI	dyne·second/cm^5/m^2	196 ± 78	179–210
	mmHg/ml/minute/kg	0.065 ± 0.026	0.060–0.070
LCWI	kg·minute/m^2	6.6 ± 2.3	6.2–7.1
	mm Hg/ml/minute/kg	17045 ± 5393	15957–18132
LVSWI	g·minute/m^2	76.7 ± 24.5	71.7–81.6
	mm Hg/ml/minute/kg	199 ± 54	188–210
LVRPP	Beats/minute mmHg	9057 ± 2937	8465–9649
RCWI	kg·min/m^2	0.91 ± 0.41	0.83–0.99
	mm Hg/ml/minute/kg	2353 ± 981	2156–2551
RVSWI	g·minute/m^2	10.4 ± 3.9	9.6–11.2
	mm Hg/ml/min/kg	27.1 ± 9.1	25.2–28.9
RVRPP	Beats/minute mmHg	1247 ± 510	1144–1350
DO_2	ml/minute/m^2	790 ± 259	737–842
	ml/minute/kg	29.5 ± 8.8	27.7–31.3
VO_2	ml/minute/m^2	164 ± 71	148–181
	ml/min/kg	6.0 ± 2.6	5.5–6.5
O_2 extraction	%	20.5 ± 5.7	19.4–21.7
VCO_2	ml/minute/m^2	128 ± 46	114–136

a, arterial; A, alveolar; ABPm, mean arterial blood pressure; a–v, arterial–mixed venous; A–a, alveolar–arterial; BD, base deficit; c, capillary; bpm, beats/minute; C, content; CI, cardiac index; CO, cardiac output; CO_2, carbon dioxide; CVP, central venous pressure; DO_2, oxygen delivery; Hb, hemoglobin; HCO_3, bicarbonate; LCWI, left cardiac work index; LVRPP, left ventricular rate pressure product; LVSWI, left ventricular stroke work index; m, mean; mv, mixed venous; O_2, oxygen; PAPm, mean pulmonary arterial blood pressure; PAOP, pulmonary artery occlusion pressure; PCO_2, partial pressure of carbon dioxide; PO_2, partial pressure of oxygen; PVRI, pulmonary vascular resistance index; RCWI, right cardiac work index; RVRPP, right ventricular rate pressure product; RVSWI, right ventricular stroke work index; SO_2, hemoglobin saturation with oxygen; SVI, stroke volume index; SVRI, systemic vascular resistance index; VO_2, oxygen consumption; VCO_2, carbon dioxide production. *Source:* Haskins et al. (2005)/American Association for Laboratory Animal Science [105].

judicious vasodilator therapy. However, if associated with marginal blood pressure, the condition should not be specifically treated because it is probably compensation for hypovolemia or marginal cardiac output. In this instance, vasodilator administration will likely cause hypotension.

Fourth, evaluate oxygen content, delivery, consumption, and extraction. Low oxygen content is most likely caused by anemia. Low oxygen delivery may be caused by low oxygen content and/or low cardiac output. Low oxygen consumption may be caused by low oxygen delivery or impaired metabolism. High oxygen extraction usually indicates low oxygen delivery. Low oxygen extraction may be caused by impaired cellular metabolism or peripheral arterial–venous shunting. Venous oxygen is low when oxygen extraction is high, and vice versa.

Conclusion

Advanced hemodynamic monitoring remains a keystone in the management of critical illness. Determination of cardiac output and systemic oxygen delivery can be helpful in patients with perfusion status that is poorly defined by other methods or in patients that do not respond well to therapy. In past years, the more common clinical techniques involved indicator dilution, usually either thermodilution with the balloon-tipped pulmonary arterial catheter or lithium dilution. While rates of pulmonary artery catheter use have declined greatly, there has been an increase in the number of alternatives for monitoring cardiac output as well as greater understanding of the methods and criteria with which to compare devices [51]. Widespread use of these alternatives has not been seen in veterinary medicine. This is likely due to cost, technical complexity, requirement for large or specialized equipment, or no easy extrapolation of the technology into animal patients. Perhaps more commonly in veterinary medicine is the measurement of routinely monitored parameters influencing cardiac output such as CVP, caudal vena cava diameter, end-diastolic left ventricular volume, subjective ultrasonographic assessments of cardiac contractility, ABP, and parameters of metabolic acid–base balance. Though it is unclear whether use of advanced cardiac output monitoring improves outcome, certainly outcome can only be improved if the technique is performed properly, the results are interpreted correctly, and appropriate therapy is instituted in a timely manner.

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17

Point-of-Care Cardiac Ultrasound

Valerie Madden and Søren Boysen

Cardiac veterinary point-of-care ultrasound (VPOCUS), also referred to as focused cardiac ultrasound or FOCUS, or a two-minute screening echocardiogram, is an extension of and often used in conjunction with thoracic (Chapter 27) and abdominal (Chapter 39) VPOCUS scanning. As with other VPOCUS examinations, cardiac VPOCUS is used to obtain clinically relevant patient data that cannot be determined on clinical examination alone. It is not a replacement for a consultative echocardiogram, which is often performed by cardiologists, because it is not intended to provide highly detailed cardiac measurements [1, 2]. Rather, cardiac VPOCUS is applied by the attending clinician at the bedside in light of clinical and historical findings to help discern a preliminary diagnosis (e.g. cardiac vs. noncardiac cause of dyspnea) and/or to direct further diagnostic and therapeutic interventions [1, 2]. As such, it tends to be a time-sensitive examination most often performed in symptomatic or at-risk patients [3]. It is indicated in cats and dogs presenting with respiratory distress, pleural effusion, significant arrhythmias, hemodynamic instability, suspected pericardial effusion, and to assist with the diagnosis of heart failure.

The specific clinical question to be investigated and the cardiac abnormalities that can be ruled in or out with cardiac VPOCUS are few. In human medicine, it is often limited to the following abnormalities: left-ventricular enlargement, left-ventricular hypertrophy, left-ventricular systolic function, left-atrial enlargement, right-ventricular enlargement, right-ventricular systolic function, pericardial effusion, and inferior vena cava size [1, 2]. Although clinical studies are lacking, most of these variables have also been evaluated with cardiac VPOCUS in companion animals. Cardiac VPOCUS should be distinguished from "limited echocardiography," in that limited echocardiography refers to a reduced number of images that are often still interpreted by a specialist/cardiologist, whereas cardiac VPOCUS refers to a narrowly focused ultrasonographic examination often performed by a nonspecialist clinician [1, 2, 4]. With cardiac VPOCUS, interpretation is often subjective and involves one or a few preselected targets to interpret, typically classified as present or absent by using a predefined specific imaging protocol (e.g. right parasternal short-axis left atrial-to-aortic root ratio). The practitioner focuses on making a specific diagnosis or answering a certain, often binary, question or series of questions [2]. Cardiac VPOCUS generally requires less training and expertise than performing limited echocardiography, and much less training than consultative echocardiography [1, 2]. The results of cardiac VPOCUS are used in conjunction with other point-of-care and clinical findings, such as the physical examination, to formulate a diagnostic impression and guide appropriate early patient management. Evidence suggests that cardiac VPOCUS can be performed by non-specialist clinicians to help direct further patient care [4–6].

Clinical studies in cardiac VPOCUS have involved primarily the interpretation of key aspects of a patient's cardiovascular status, including but not limited to:

- Volume status
- Cardiac function
- Pericardial effusion.

Equipment, Patient Preparation, and Image Acquisition for Cardiac Veterinary Point-of-Care Ultrasound

- Although a phased array transducer is advantageous in obtaining echocardiographic images, the authors perform all cardiac VPOCUS using only a micro-convex curvilinear transducer.

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- Conventional echocardiographic modalities applicable in the emergency setting include two-dimensional (2D) and M-mode, although subjective assessment of 2D images is used to answer the vast majority of cardiac VPOCUS questions.
- Doppler and color mode are rarely used to interpret cardiac VPOCUS questions and are not covered in this chapter.

The majority of views interpreted are obtained from the *right side* of the patient with either the patient positioned in sternal/standing or right lateral recumbency, the latter often on a table with the transducer applied from underneath the patient along the right thoracic wall (Figure 17.1).

Although the size of the cardiac window varies with respiratory effort and patient positioning, all images of the heart are obtained where lung tissue is not interposed between the heart and the chest wall (i.e. the cardiac notch), which is in the region of the fourth to sixth intercostal spaces, near the sternum.

Patients that are dyspneic are often scanned in sternal recumbency or while standing and receiving oxygen supplementation and anxiolytics. Although it is more difficult to obtain good quality images in dyspneic patients, most cardiac VPOCUS specific questions can still be answered in these cases. Extending the right forelimb cranially often makes identification of the heart easier (Figure 17.1).

Good quality images can usually be obtained without clipping fur; this is achieved by applying isopropyl alcohol to the skin after parting the fur and applying a generous amount of ultrasonographic coupling gel to the ultrasound transducer head. This creates an air-free zone between the skin and the transducer.



Figure 17.1 (a) Patient positioning and "cutout" tabletop. The patient is restrained in right lateral recumbency with the thoracic limbs extended cranially. (b) An "L-shaped" table can also be used to obtain the right parasternal views or can be created by placing two tables together at 90-degree angles and positioning the patient such that the heart is positioned over the gap in the tables with the thoracic limbs extended cranially. (c) In less stable patients, and most commonly used, the heart can be scanned with the patient in a sternal or standing position, with the right forelimb extended forward. In all cases, pulling the thoracic limb forward facilitates image acquisition of the heart.

Two-Dimensional Echocardiography

Two-dimensional echocardiography is used for nearly all cardiac VPOCUS specific questions. A 2D echocardiographic image is generated from the data obtained by fanning the ultrasound beam across the tomographic plane [7–10]. For each patient and echocardiographic view, optimal image quality depends on transducer selection and instrument settings [8, 9].

In addition to patient positioning and good contact, two settings commonly altered to obtain optimal images include gain and depth [10, 11]. The depth should be adjusted to allow the entire heart, particularly the far wall, to be visualized in the near around two-thirds of the ultrasound image. Set the focal point at the level of the far field wall of the heart. Gain is adjusted to allow the cardiac free wall and lumen to be easily differentiated from one another. Cardiac presets use higher contrast, which makes cardiac chamber assessment easier. Becoming familiar with the freeze function and scrolling the cine loop through frames is helpful in optimizing the cardiac image during cardiac VPOCUS.

M-Mode Echocardiography

M-mode is the modality in which ultrasound beams are aimed manually at targeted cardiac structures to give a graphic recording of their positions and movements [7, 8]. M-mode recordings allow quantitative measurement of cardiac dimensions and analysis of motion patterns depending on transducer positioning [12]. These analyses are not traditionally part of cardiac VPOCUS and often require more training to master.

Cardiac Veterinary Point-of-Care Ultrasound Windows and Views

Cardiac VPOCUS incorporates right parasternal short- and long-axis views, the subxiphoid view, and occasionally left-sided views (Protocols 17.1–17.3). Acquiring all views is often unnecessary. For example, even if only the left atrial-to-aortic ratio can be obtained evidence suggests that cardiac VPOCUS can still have diagnostic value [4, 13, 14]. Furthermore, although left-sided views provide nice windows to assess spontaneous echogenic contrast and/or intracardiac thrombi in cats, they are not required to answer most cardiac VPOCUS questions and are not covered in this chapter.

Two-Dimensional Right Parasternal Views

There are eight standard 2D echocardiographic views obtained from the right parasternal window: two long-axis and six short-axis views [15]. The two long-axis views include (i) the four-chamber view (Figure 17.2), and (ii) the left ventricular outflow tract view (five-chamber view (Figure 17.3), while the short-axis views, from apex to base (Figure 17.4), include (i) the apex; (ii) left ventricle and papillary muscles (mushroom view); (iii) left ventricle at the level of the chordae tendinae; (iv) mitral valve (fish mouth view); (v) left atrium-to-aortic root ratio (peace sign and the whale) with the right ventricular outflow tract and the pulmonic valve; and finally, (vi) the pulmonary artery branches, the right auricle and caudal vena cava (CVC; not shown in Figure 17.4). Although all eight right parasternal views are useful in evaluating cardiac structure and function, there are three short-axis views that are most often used during cardiac VPOCUS: [4, 5]

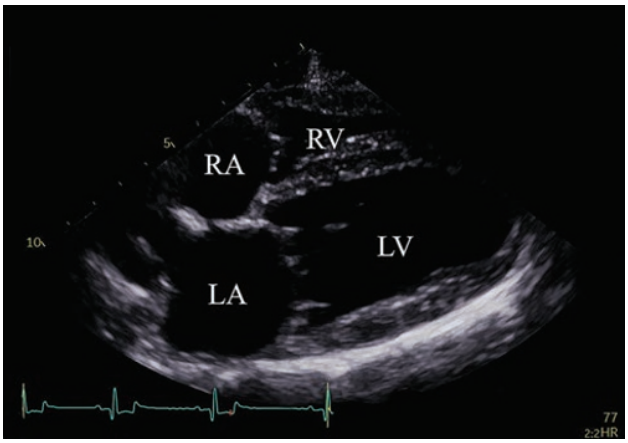


Figure 17.2 Two-dimensional long axis view, right parasternal window. Four-chamber view of the heart showing the left ventricle (LV), left atrium (LA), right atrium (RA) and right ventricle (RV).

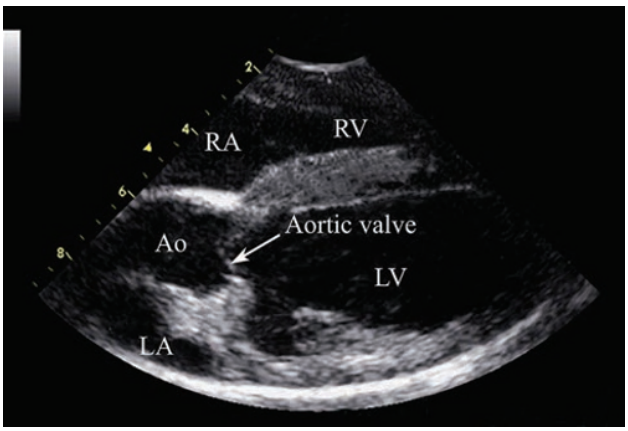


Figure 17.3 Two-dimensional long axis view, right parasternal window; five-chamber left-ventricular outflow tract view of the heart showing the left ventricle (LV), aorta (Ao), aortic valve (white arrow), left atrium (LA), right atrium (RA) and right ventricle (RV).

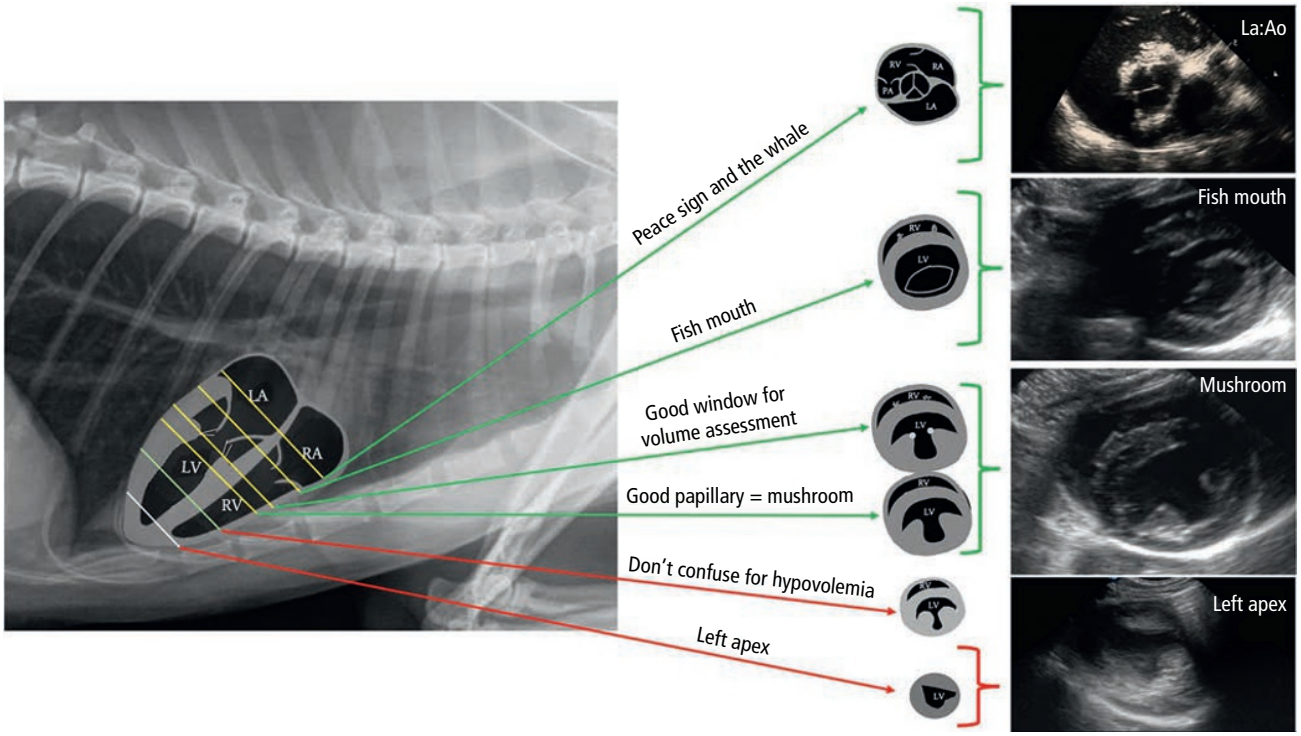


Figure 17.4 Schematic image of the different right parasternal short-axis views starting at the apex level and moving toward the heart base through the papillary muscle level, mitral valve, and finally the left atrium-to-aortic root. The corresponding cardiac still images are labeled; "left apex", "mushroom view" at the level of the papillary muscles where chordae tendinea are just becoming visible (small lighter dots at the tips of the papillary muscles represent the chordae tendinae), "fish mouth view" at the level of the mitral valves, and LA : Ao represents the "Peace sign and the whale" at the heart base level. The heart base image above the left atrial-to-aortic root is not shown in this figure. LA, left atrium; RA, right atrium; RV, right ventricle; PA, pulmonary artery; LV, left ventricle.

1) The "Mushroom view" (Figure 17.5): Often the first view obtained and a common site to estimate volume status and contractility at the left midventricular/papillary muscle region just below the mitral valve.
2) The "Fish mouth view" (Figure 17.6): Serves as a landmark to obtain other views and allows assessment of the mitral valve.
3) The "Peace sign and whale view" (Figure 17.7): Allows assessment of the left atrium-to-aortic root ratio (La : Ao), which is helpful in estimating volume status and deciding if heart failure is present.

Being able to confidently identify the fish mouth view provides the reference point above and below which the two key views used to answer most cardiac VPOCUS questions can be found: the peace sign and whale view, and the mushroom view, respectively.

Subxiphoid View

Because of the liver, the subxiphoid view provides a nice acoustic window into the caudal thorax, which is often used to determine if pericardial and/or pleural effusion are present. The CVC can also be evaluated to assess patient volume status at the subxiphoid site.

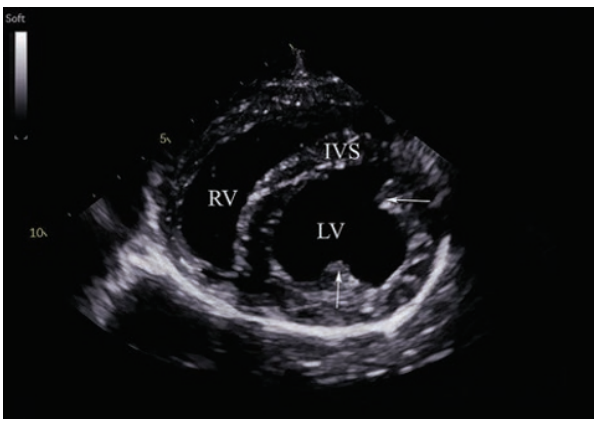


Figure 17.5 Two-dimensional short-axis view, right parasternal window; mushroom view. The initial view obtained is often the midventricular region of the right parasternal short-axis window at the level of the papillary muscles (white arrows), referred to as the "mushroom" view. LV, left ventricle; IVS, intraventricular septum; RV, right ventricle.

Interpretation of Cardiac Veterinary Point-of-Care Ultrasound Findings

All cardiac VPOCUS findings should be interpreted in light of clinical examination and other VPOCUS findings.

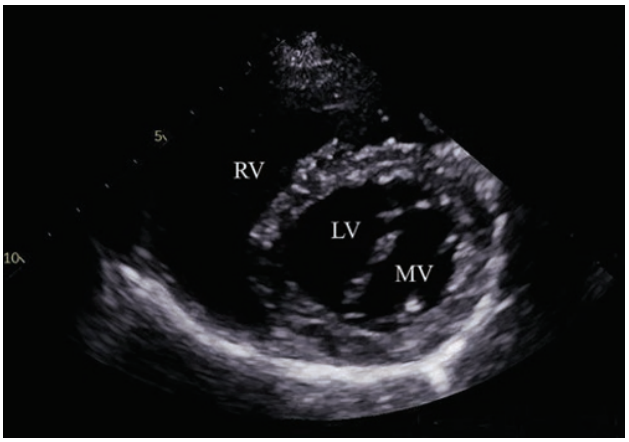


Figure 17.6 Two-dimensional short-axis view, right parasternal window; fish mouth view. After obtaining the "mushroom" view with the transducer in the right parasternal short-axis plane, slowly fan the transducer dorsally directing the ultrasound beams toward the base of the heart until the mitral valve (MV) becomes apparent within the left ventricular lumen (LV). This is the "fish mouth view". RV, right ventricle.

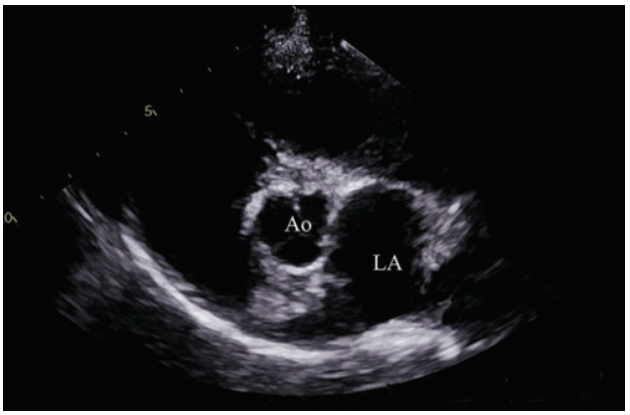


Figure 17.7 Two-dimensional short-axis view, right parasternal window; Peace sign and the whale view to evaluate the left atrium-aortic root ratio (La : Ao). Obtained by fanning the ultrasound beam toward the base of the heart from the mushroom/fish mouth view while in the right parasternal short-axis plane. Image contains a short-axis view of the left atrium (La, whale) and aortic root (Ao, peace sign).

Protocol 17.1 Cardiac Point A-of-Care Ultrasound to Obtain the Right Parasternal Short-Axis Views

1) Place the transducer on the area of the right thorax where the strongest apical heartbeat can be palpated or where the point of the flexed elbow meets the thorax. This will be between the fourth to six6th intercostal spaces, at the level of the costochondral junction or slightly closer to the sternum (Figure 17.8).
2) Orientate the transducer at a 30–45 degree angle to horizontal, with the marker directed cranially and toward the elbow (Figure 17.9). This will align the ultrasound beam parallel and in short-axis to the left ventricle.

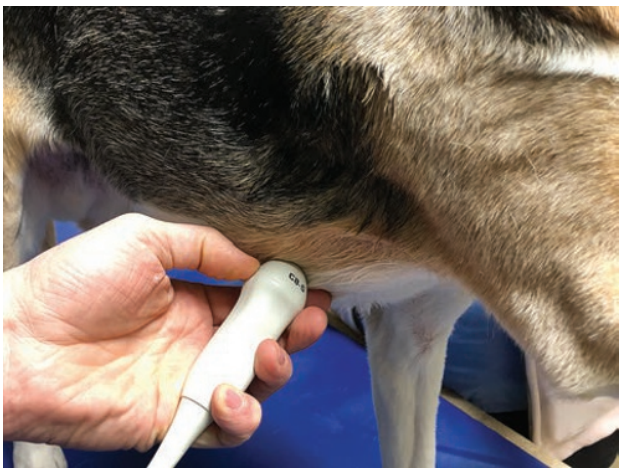


Figure 17.8 The transducer is placed on the right thorax where the strongest apical heartbeat can be palpated; between the fourth to sixth intercostal spaces, at the level of the costochondral junction or slightly closer to the sternum.

Note: Transducer marker orientation varies by operator preference. The right parasternal short-axis images presented in this chapter are obtained with the transducer marker directed toward the elbow and the corresponding marker on the ultrasound image to the left of the ultrasound screen.

3) The initial view obtained is often at the level of the ventricular apex or the midventricular region, which yields an image referred to as the "mushroom" view (Figure 17.5). Adjusting the depth and gain, as well as using standard transducer manipulations such as sweeping, sliding, and rocking (Figure 17.10) are used to obtain the best acoustic window of the heart. If

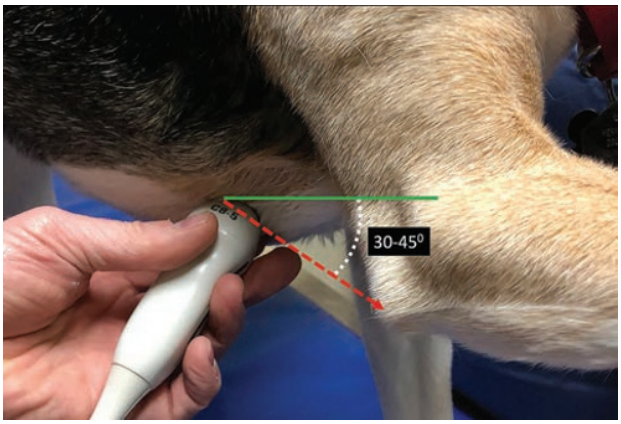


Figure 17.9 Orientate the transducer at a 30–45 degree angle (red dotted arrow) to horizontal (green solid line), with the marker directed cranially and toward the elbow.

image quality remains poor try applying more pressure to the transducer, particularly in obese patients, or clipping the fur. Details of specific transducer movements can be found in Chapter 6.

4) The transducer may need to be rotated slightly in either a clockwise or counterclockwise direction until circular symmetry of the left-sided ventricle is achieved.
5) Once the optimal acoustic cardiac window is obtained keep the transducer relatively static on the thoracic wall and use smaller subtle transducer manipulations (e.g. rotation and fanning, Figure 17.10) to complete the cardiac VPOCUS exam. It is often easier if only a single transducer manipulation is used at one time (e.g. avoid sweeping and fanning simultaneously).
6) By moving the plane of the ultrasound beam from the apex to the base of the heart (slow sweeping and/or fanning depending on the size of the patient), the operator can examine the successive two-dimensional planes with their related cardiac structures (Figure 17.4).
7) Final image acquisition of the La : Ao is often obtained by making small "fanning" transducer movements from the mushroom view (left ventricle and papillary muscles) or fish mouth view (mitral valve) to the peace sign and whale view (La : Ao).
   - In cats and smaller dogs, the transducer is kept at the initial mushroom view and fanned (tilted) from this fixed external thoracic location toward the head of the patient to image the left atrial-to-aortic root ratio (avoid sweeping movements because the cardiac notch is small and the transducer will rapidly transition from the heart to lung). This often requires the transducer to be "angled" from the cardiac notch below the lung toward the spine, particularly in sternal or standing patients (Figure 17.11).
   - In larger dogs, the transducer can often be swept dorsally in small increments between the ribs until the fish mouth view is obtained. From the fish mouth view the transducer is then fanned dorsally from a fixed external thoracic location until the left atrium-to-aortic root ratio is visualized.
   - In some cases, the transducer will need to be advanced cranially one or two intercostal spaces

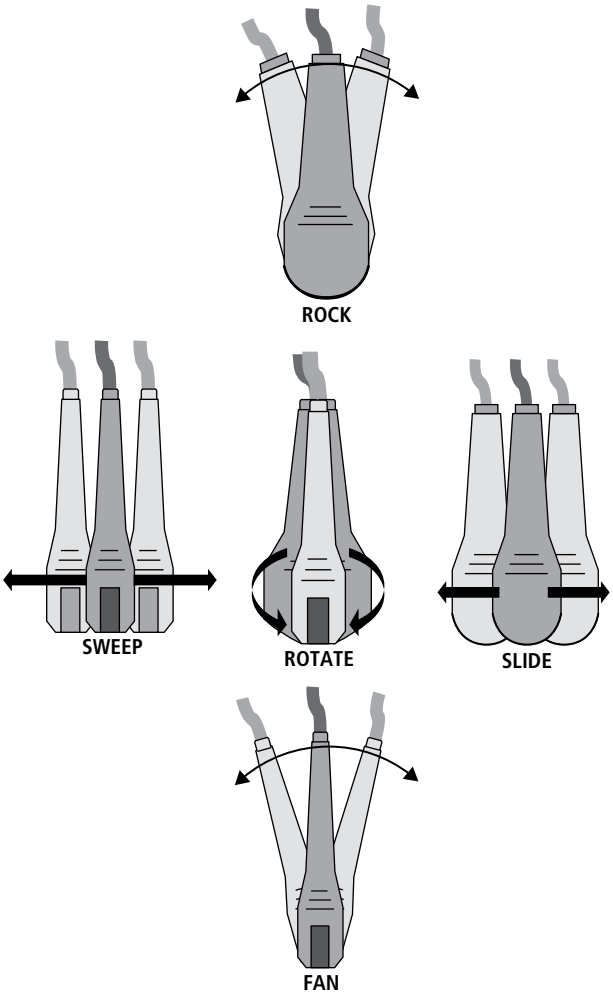


Figure 17.10 Summary of the five different probe manipulations commonly used during point-of-care ultrasound; sweep, slide, rotate, fan, rock. See Chapter 6 for details.



Figure 17.11 Figure of the transducer on the right thorax angled (fanned) toward the heart base to obtain the La : Ao ratio. Owing to the small size of the cardiac notch in standing patients, the ventral lung border (lung in pink, lung border in red) is superimposed over the heart base (heart not shown). To be able to find the La : Ao it is often necessary to fan the head of the transducer dorsally from a fixed external chest point (solid green line) below the ventral lung border until the left atrium and aorta are visible (dotted green line). The initial starting point to fan is often the mushroom view in cats and small dogs.

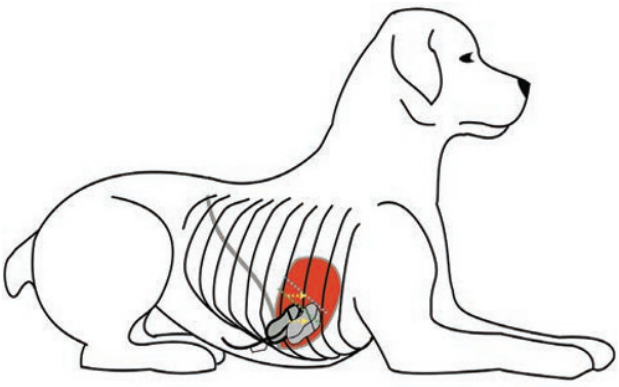


Figure 17.12 In some patients it is necessary to advance the transducer cranially a rib space or two (depicted by yellow dotted arrows). Given that the heart is "tilted cranially," moving a rib cranially often "shifts" the image obtained toward the heart base. For example, if the transducer is over the mushroom view of a large dog, green dotted line, and then advanced horizontally a rib space without performing any other probe manipulations, the image obtained at the more cranial rib space will often be closer to the heart base (blue dotted line).

from the starting position (maintaining the same transducer angle and orientation), and then fanned in order to obtain the La : Ao (Figure 17.12).

8) When the correct La : Ao window is obtained, the aorta should be round to three-leaf clover shaped, and during diastole the three cusps of the closed aortic valve should form the Y shaped "peace sign." The left atrium should be "tear drop" or "whale" shaped (Figure 17.7).
   - Note that the aortic cusps may be less visible when a microconvex vs a phased array transducer is used.
   - If the aorta is not "closed" and part of the outflow track is visible to the left of the aorta, the transducer is likely under rotated (slowly rotate the transducer clockwise) to "close" the aorta, and if part of the outflow track is visible to the right of the aorta, the transducer is likely over rotated (slowly rotate the transducer counterclockwise).
9) Once an acceptable image is obtained the size of the left atrium should be evaluated (see interpretation of views for diagnosis of pathology).
   - Although precise caliper measurements can be made, the left atrial size and La : Ao ratio are often subjectively assessed.
   - If desired, the image can be frozen, and calipers used to determine the size of the left atrium and aorta individually (Figure 17.13).
   - If a pulmonary vein enters the left atrium at the desired measurement point, the measurement is made at an extrapolation of the atrial border or immediately medial or lateral to the vein.
   - The left atrial diameter is divided by the aortic root size to obtain a numerical value representing the La : Ao.
   - Alternatively, a subjective "eyeball" assessment of how many "aortas" fit inside the left atrium can also be made (Figure 17.14).

In summary, the key transducer movements to locate the ideal cardiac VPOCUS windows involve broader movements to identify the heart followed by finer rotation and fanning movements once the fish mouth view identified; rotating the transducer transitions between long and short-axis views, and fanning (tilting) the transducer provides multiple cross-sections of the structure while maintaining a particular axis orientation.

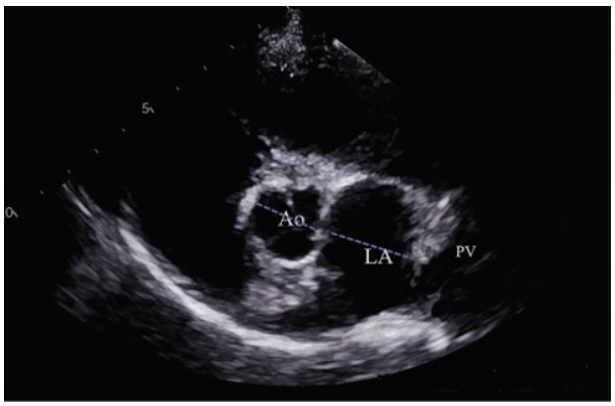


Figure 17.13 Two-dimensional short-axis view, right parasternal window, left atrial-to-aortic root ratio measurement. The image is frozen to obtain a still image from which measurements (blue dotted lines) can be obtained. A caliper is used to obtain a diameter based measurement from one side of the left atrium across to the other. If a pulmonary vein enters the left atrium at the desired measurement point, the measurement is made at an extrapolation of the atrial border or immediately medial or lateral to the vein. Ao, aorta; LA, left atrium; PV, pulmonary vein.

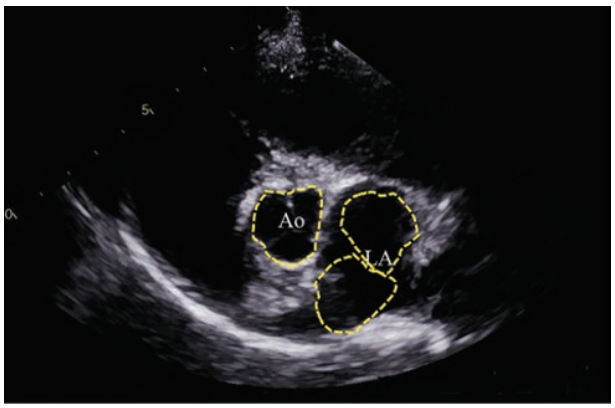


Figure 17.14 Two-dimensional short-axis view, right parasternal window, subjective left atrial-to-aortic root ratio measurement. A subjective estimate of the number of areas of the aorta (yellow dotted line) that can fit into the left atrium has been validated in cats. Ao, aorta; LA, left atrium.

Protocol 17.2 Cardiac Point-of-Care Ultrasound from the Right Parasternal Long-Axis View

1) From the right parasternal short-axis mushroom or fish mouth view, rotate the transducer 90 degrees clockwise to lie almost parallel to the ribs at or just below the costochondral junction.
2) If the ventricular lumen size appears short and narrow with very thick ventricular walls, the ultrasound beam might be traversing the ventricles obliquely. This may be corrected by slowly fanning the ultrasound beam caudally and then cranially until the ventricular lumen diameter is maximized. If the lumen diameter remains small and the walls remain thick pathology should be suspected (see interpretation below). The chamber size and wall thickness should be compared with the short-axis views for consistency.
3) To advance from the four-chamber to the five-chamber view, use a slow steady movement to rotate the transducer 10–15 degrees clockwise until the left ventricular outflow tract is visible. Gentle fanning of the probe cranially may be necessary in some cases.

See also Ware (2007) [9]; Boon (2002) [10].

Protocol 17.3 Cardiac Point-of-Care Ultrasound to Obtain the Subxiphoid View

1) To obtain this view, the transducer is positioned at a 45-degree angle just caudal to the xiphoid process in either a long or short-axis plane.
2) In the long-axis plane the marker is directed cranially while in short-axis it is directed toward the patient's right.
3) It is often easier to start in long-axis orientation (Figure 17.15a).
4) The caudal thoracic cavity, including the pleural and pericardial spaces are assessed. The liver should be visible in the near field and the apex and

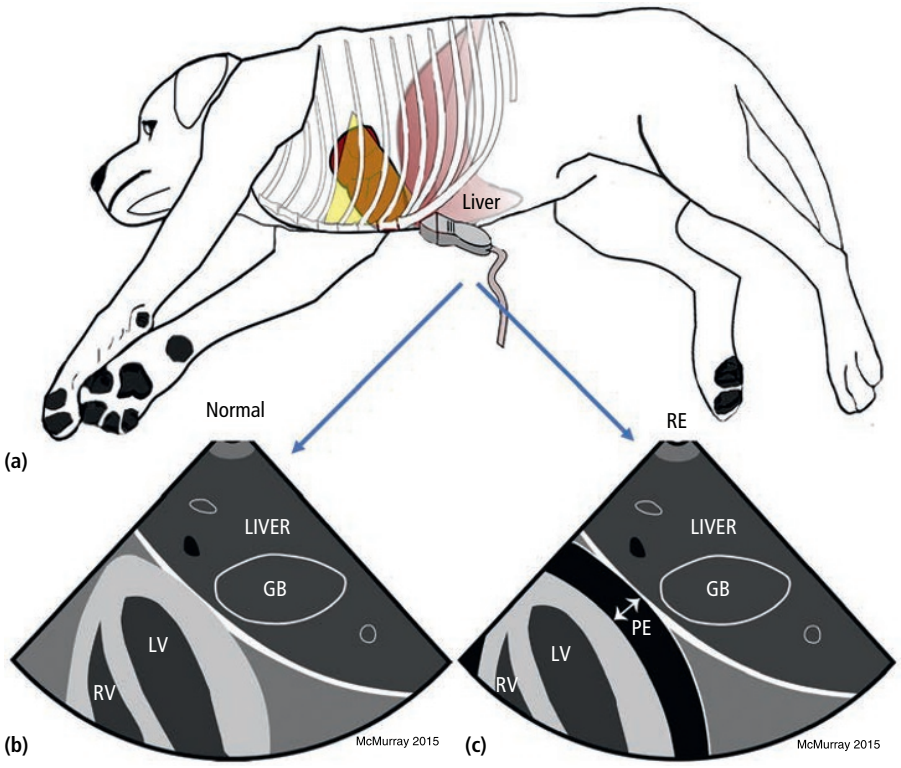
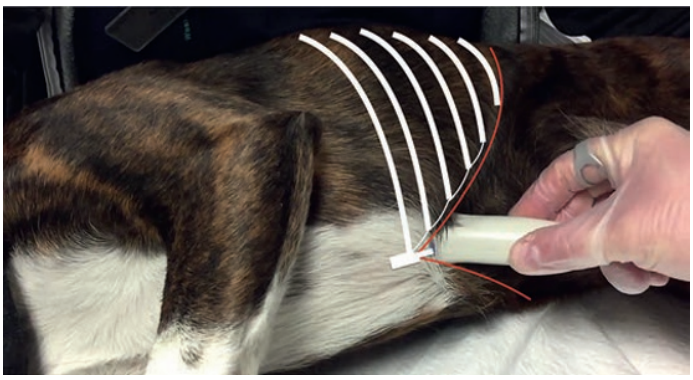


Figure 17.15 (a) Schematic image of transducer location to image the pericardial space and heart from the subxiphoid location. The dog is in right lateral recumbency in this figure, and the probe is placed just caudal to the xiphoid process. The depth is increased, and the transducer is rocked more parallel to the spine to be able to image the apex of the heart, where it is more likely to come in contact with the diaphragm. The heart may lie just left or right of midline (just left in this figure) which also necessitates fanning the transducer in long axis to identify it. (b) Schematic ultrasound image of the heart in contact with the diaphragm in a healthy dog. (c) Schematic ultrasound image of a dog with pericardial effusion obtained at the subxiphoid location. This window can also be used to assess the heart for cardiac contractility in some animals during external cardiac compressions during cardiopulmonary resuscitation. GB, gall bladder; PE, pericardial effusion; RV, right ventricle; LV, left ventricle. *Source:* Images courtesy of J McMurray DVM.

left-ventricular free wall of the heart will be visible in the far field contacting and appearing to "blend" into the diaphragm and liver (Figure 17.15b).

5) If the heart is not initially visible the transducer should be fanned left and right of midline and rocked cranially in the long-axis plane until the heart becomes visible. This often results in the transducer head being "tucked" under the xiphoid process and orientated almost parallel to the spine (Figure 17.15a).
6) In the majority of normal canine patients, the pericardium contacts the diaphragm, and the heart can be visualized; this is not necessarily the case in cats where air-filled lung may occupy the space between the diaphragm and the heart, hampering cardiac assessment from the subxiphoid site.
   - With pericardial effusion and cardiomegaly, a greater percentage of the heart tends to contact the diaphragm, making it visible in both cats and dogs.
7) The CVC can also be identified and assessed at the subxiphoid site in the longitudinal plane where it crosses the diaphragm (Figure 17.16) [15, 16].
   - With the transducer in long-axis, and at roughly a 45-degree angle, slowly fan the probe to the right and left of midline while watching for a "break" in the diaphragm.
   - In most dogs and cats, the CVC can usually be identified just to the right of the heart, often in the same plane the gall bladder is visible.
   - Once the CVC is located the transducer should be slowly fanned off either side of the CVC to locate its widest diameter.
   - Keep the ultrasound beam at the widest diameter of the CVC through several respiratory cycles to determine the CVC index: calculated by measuring the widest and narrowest diameters of the CVC during expiration and inspiration respectively (see Caudal Vena Cava section).
8) Avoid applying too much pressure to the transducer or pressure artifact may result in a "falsely" collapsed CVC.



(a)

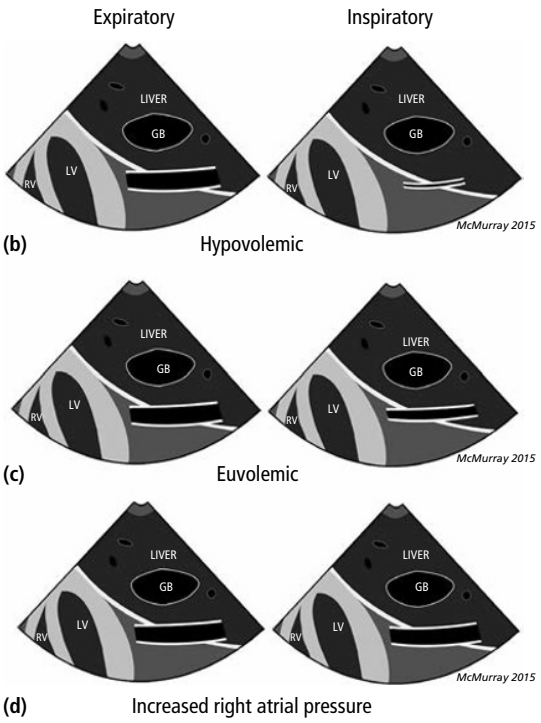


Figure 17.16 (a) Image of the subxiphoid site and transducer angle used to locate the caudal vena cava where it crosses diaphragm. (b)–(d) Schematic images depicting the caudal vena cava collapsibility index in a spontaneously breathing hypovolemic, euvolemic, and a patient with increased right atrial pressures. A subjective assessment is made by comparing the widest diameter of the caudal vena cava (CVC) during expiration to the narrowest diameter of the CVC during inspiration. GB, gall bladder. *Source:* Images courtesy of J McMurray DVM.

Interpretation of the Left Atrium–Aortic Root Ratio

The La : Ao is primarily used to evaluate the size of the left atrium relative to the aorta. Although objective left atrial measurements can be made, in the emergency and critical care setting, evidence suggests that a subjective assessment of left atrial size is sufficient for most cardiac VPOCUS applications [4, 13].

The La : Ao ratio reference range varies slightly depending on reference used, where measurements are made, and the angle at which it is measured, which may vary between patients in lateral vs. standing, and by the operator skill level. In general, a normal La : Ao should be between 1 and 1.5 in cats and dogs (slightly higher in cats) [5, 9, 10]. There is a gray zone regarding the cut off La : Ao to determine left atrial enlargement, up to 1.7 in cats. Fortunately, assessment of left atrial enlargement during cardiac VPOCUS tends to be well outside the gray zone and reference limits, meaning most patients presenting with respiratory distress secondary to congestive heart failure have La : Ao ratios ≥ 2.

Interpretation of an Increased La : Ao

If the left atrium is enlarged, it suggests increased left atrial volume and/or pressure which is often associated with left-sided congestive heart failure or iatrogenic fluid overload. Patients presenting with respiratory distress, an enlarged left atrium, and B lines on lung ultrasound (Chapter 27) should elicit concern for congestive heart failure or iatrogenic volume overload. Patients who present to the emergency department in respiratory distress are unlikely to be experiencing iatrogenic volume overload (in the absence of recent fluid administration), as opposed to patients in intensive care who have received high fluid volumes or several days of fluid therapy.

Novice sonographers should begin with La : Ao ratios ≥ 2 : 1 in cats and dogs. Such a ratio is likely to be present in patients presenting in respiratory distress secondary to congestive heart failure. Therefore, the finding of an La : Ao ratio ≥ 2 : 1 should elicit consideration for left atrial enlargement and severe cardiac pathology or volume overload, which are easily assessed subjectively and is valid regardless of patient position Figure 17.17. Alternatively, in cats, if more than three aortas can be subjectively fit within the left atrium, the left atrium should be considered enlarged (Figure 17.18) [4]. Using both subjective methods is a good way to double check if the left atrium is truly enlarged. (This subjective means of assessing atrial enlargement in dogs has not yet been investigated.)

History, assessment of cardiac function (see Interpretation of Ventricular Contractility below), and left-ventricular lumen size and wall thickness (see Interpretation of the Left Ventricular Mushroom View, below) can often distinguish between left-sided heart failure and iatrogenic fluid overload. The absence of B lines on lung ultrasound in patients presenting with respiratory distress makes left-sided congestive heart failure unlikely [13]. When using the left atrial size to assess volume status, it should be interpreted in light of other clinical examination findings and complimented with CVC assessment (see Interpretation of Ventricular Contractility below).

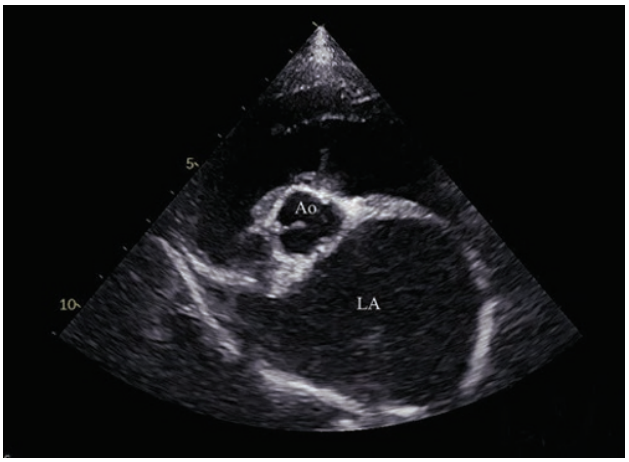


Figure 17.17 Two-dimensional short-axis view, right parasternal window; mushroom view. A patient with significant left-atrial enlargement reflective of advanced cardiac disease and congestive heart failure. Ao, aorta; LA, left atrium.

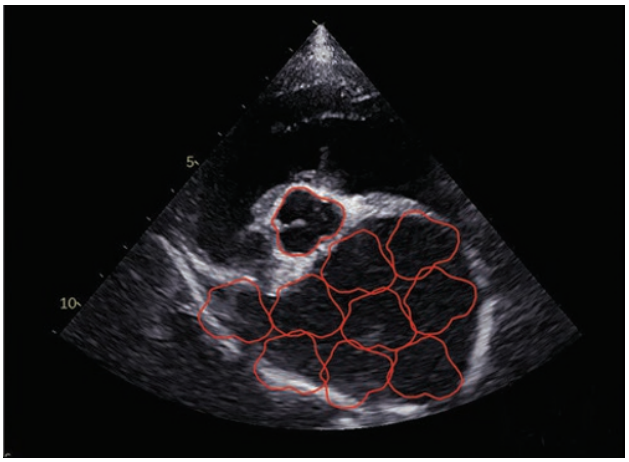


Figure 17.18 Same image as Figure 17.17 with estimation of the number of aorta areas (red circles) that can be fit within the left atrium.

Interpretation of a Decreased La : Ao

A decrease the left-atrial size should be suspected when the La : Ao is less than 1.0. A small La : Ao suggests hypovolemia due to a phenomenon referred to as pseudohypertrophy.

Pseudohypertrophy results from hypovolemia and decreased ventricular filling, which causes decreased left atrial and ventricular chamber size, and the appearance of a thickened intraventricular septum and ventricular free

wall [17, 18]. This resolves with restoration of effective circulating volume. A loss of the visible ventricular chamber during systole, known as ventricular chamber obliteration, may reflect severe hypovolemia due to markedly decreased ventricular filling pressures and represents severe hypovolemia and potential impending cardiovascular collapse.

To help differentiate pseudohypertrophy from hypertrophic cardiomyopathy (HCM), left atrial size should be evaluated: left atrial size is normal to small in patients with pseudohypertrophy and is often enlarged in patients with cardiomyopathy.

Interpretation of the Left Ventricular Mushroom View

The "mushroom" view should be evaluated for left ventricular chamber size, contractility (2D and M-mode), and wall thickness. Less commonly, right-ventricular size and ventricular septal flattening are assessed.

Interpretation of Increased Left-Ventricular Lumen Size

Specific measurements of the left ventricular chamber size are not commonly performed during cardiac VPOCUS. The subjective assessment of an enlarged left-ventricular lumen in the emergency and critical care setting may suggest volume overload or conditions causing myocardial dysfunction (e.g. dilated cardiomyopathy). With volume overload ventricular contractility is generally increased giving the heart a hyperkinetic appearance on ultrasound (see Interpretation of Ventricular Contractility below).In contrast, myocardial dysfunction secondary to dilated cardiomyopathy is associated with decreased contractility giving the heart a hypokinetic appearance on ultrasound (see Interpretation of Ventricular Contractility below). Both volume overload and dilated cardiomyopathy can cause an enlarged left atrium and increased B lines on lung ultrasound; assessing the contractility of the left ventricle is important to differentiate the two conditions.

Interpretation of Decreased Left-Ventricular Lumen Size

The subjective finding of a decreased ventricular lumen size with thickened ventricular walls may suggest pseudohypertrophy secondary to volume depletion or true myocardial hypertrophy. The two can be differentiated by assessing the left atrial size (see Interpretation of the Left Atrium–Aortic Root Ratio, above).

Interpretation of Increased Left-Ventricular Wall Thickness

In the absence of systemic diseases (e.g. hyperthyroidism, systemic hypertension, subaortic stenosis, and acromegaly), increased left-ventricular wall thickness is often associated with HCM in cats, but may also be seen with pseudohypertrophy in both dogs and cats. With both HCM and pseudohypertrophy, there will be wall thickening, enlarged papillary muscles, and/or left ventricular cavity obliteration at the end of systole. With HCM, the left atrium is often enlarged while it tends to be unchanged or small with pseudohypertrophy.

Interpretation of Ventricular Contractility

Left-ventricular systolic function can be assessed through fractional shortening. Fractional shortening (FS) is the percentage change of the short-axis of the ventricular chamber during systole [7, 11]. It is reported to be 30–50% in dogs and 40–60% in cats. It can be subjectively assessed by estimating the change in size of the ventricular lumen during diastole and systole, or objectively assessed using the following formula obtained using M-mode (in mm):

$$FS = \left(LVEDD - LVESD\right) / LVEDD \times 100$$

where LVEDD is the left ventricular end diastolic diameter and LVESD is the left ventricular end systolic diameter.

With practice and by scanning healthy animals, a subjective evaluation of ventricular contraction is usually sufficient to determine whether cardiovascular abnormalities are present. This skill does require becoming familiar with normal values, which can be achieved by evaluating healthy animals. M-mode requires more skill to obtain accurate repeatable measurements and is less frequently used by novice sonographers.

Conditions that increase FS include:

- Hypervolemic patients without myocardial dysfunction.
- Patients with a history of aggressive fluid therapy and combined cardiac VPOCUS findings of a hyperkinetic heart (increased FS), an enlarged left atrium, distended CVC, with or without increased B lines on lung ultrasound should prompt consideration of fluid overload. (Note that hypervolemic patients with normal FS (as opposed to increased) and increased ventricular systolic dimensions likely have some degree of myocardial failure.)
- Patients that have decreased afterload (e.g. arterial vasodilation) without myocardial dysfunction.

Conditions that that decrease FS include:

- Hypovolemia in the absence of myocardial dysfunction (due to decreased myocardial stretch).
- Conditions associated with poor myocardial contractility (e.g. dilated cardiomyopathy or sepsis).
- Increased afterload (e.g. severe arterial vasoconstriction).

Interpretation of Interventricular Septal Flattening

Flattening of the interventricular septum combined with an enlarged right ventricular chamber, and/or increased right ventricular wall thickness suggests increased right ventricular pressures or volume. These findings arguably

take more skill to assess and interpret. However, if this triad of right heart findings is present, causes of pulmonary arterial hypertension should be considered (e.g. heartworm disease, chronic bronchopulmonary disease – e.g. dynamic airway disease/obstruction, chronic bronchitis, or pulmonary fibrosis – and pulmonary thromboembolism).

Interpretation of Right Parasternal Long-Axis Views

From the four-chamber right parasternal acoustic window, the sonographer can subjectively evaluate the size of all cardiac chambers; left atrium, left ventricle, right atrium, and right ventricle. The right atrium and the left atrium should be of similar size (1 : 1 ratio). The intra-atrial septum should be relatively neutral and should not bulge into either atria. The right ventricular chamber is approximately one-third of the left ventricular internal diameter. The free wall of the right ventricle is equal to one-third or one-half the thickness of the left ventricular free wall. The interventricular septum and the left ventricular free wall are normally similar in thickness.

Interpretation of changes in chamber size, wall thickness and contractility in the right parasternal long-axis views is similar to interpretation of changes in the right parasternal short-axis views.

Interpretation of Pericardial Effusion

Pericardial effusion appears sonographically as an anechoic space adjacent to cardiac structures. Sensitivity and specificity for detection of a pericardial effusion is high regardless of patient position. To avoid missing loculated pericardial effusion, multiple acoustic windows should be assessed.

Right Parasternal Views

The right parasternal approach demonstrates the extent of pericardial fluid accumulation in both long- and short-axis views (Figures 17.19 and 17.20). Pericardial effusion appears as a circular accumulation of fluid adjacent to and surrounding the cardiac chambers, with the pericardial sac often being visualized as a hyperechoic (white) line on the side of the anechoic effusion opposite the cardiac structures. Be sure to extend the depth to visualize the entire heart. In the short-axis view, fan from the cardiac apex to the base through all planes to ensure all cardiac anatomy is correctly identified and pericardial effusion is not confused for cardiac chambers.

If it is unclear whether anechoic fluid accumulation is pericardial or pleural in origin, assess the pericardiodiaphragmatic (PD) windows; parasternal transthoracic windows found on both the right and left side of the thorax

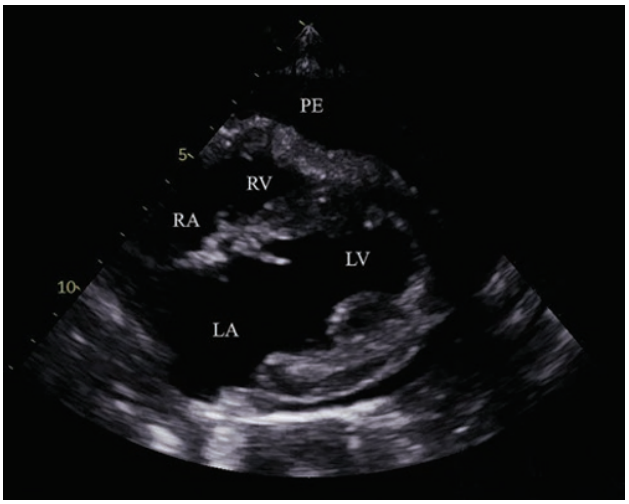


Figure 17.19 Two-dimensional short-axis view, right parasternal window; pericardial effusion. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle; PE, pericardial effusion.

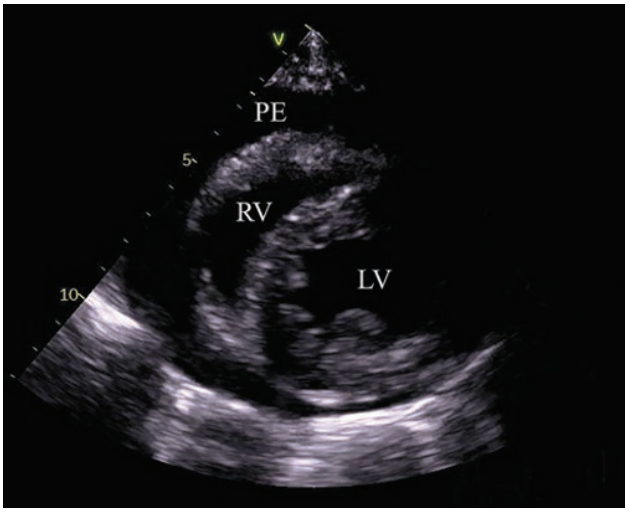


Figure 17.20 Two-dimensional short-axis view, right parasternal window; pericardial effusion. Obtaining a cardiac view in the short-axis and fanning the transducer to move from apex to base helps differentiate heart chambers from pericardial effusion. Pericardial effusion will appear as a circular accumulation of fluid adjacent to the cardiac chambers, with the pericardial sac often being visualized as a hyperechoic line on the side of the anechoic effusion opposite of the cardiac structures. LV, left ventricle; RV, right ventricle; PE, pericardial effusion.

(see Chapter 27). The PD window is identified by locating the abdominal curtain sign caudally (transition from the lung to the soft-tissue abdominal structures at the costophrenic recess) and then following it ventrally until the heart and abdominal structures are visible within the same sonographic window, or sliding caudally off the heart until the diaphragm and soft-tissue structures of the abdomen are identified (Chapter 27). This site often allows

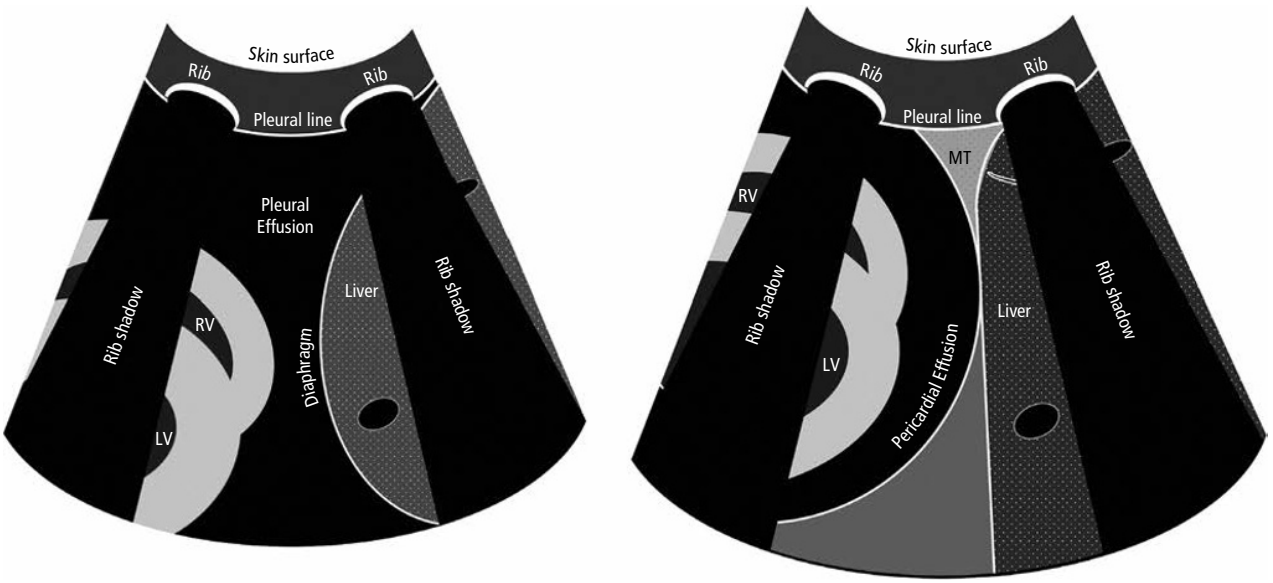


Figure 17.21 Schematic images of the pericardiodiaphragmatic window; from a dog with pleural effusion (left) and from a dog with pericardial effusion (right). In dogs with pericardial effusion the mediastinal triangle (MT), which is normally found in healthy dogs, is often still visible but tends to be lost in dogs with significant pleural effusion. RV, right ventricle; LV, left ventricle; MT, mediastinal triangle.

pericardial and pleural effusion to be differentiated (Figure 17.21). Pericardial fluid is contained and tracks around the heart (away from the diaphragm). Pleural effusion is uncontained and tracks along the diaphragm filling the costophrenic recess, which allows the diaphragm to be visualized curving away from the chest wall in the near field of the ultrasound image.

The Subxiphoid Window

Pericardial effusion may be detected between the diaphragm and cardiac apex via the subxiphoid view (Figure 17.22). If the left-ventricular free wall and apex are directly adjacent to and appear to blend into the diaphragm and liver, significant pericardial effusion is ruled out (Figure 17.15b). If pericardial effusion is present, it will be visible between and separating the left ventricular wall from the diaphragm, arching around cardiac apex away from the liver and diaphragm (Figure 17.15a and 17.22). This is different from pleural effusion, which can also be identified at the subxiphoid view; pleural effusion tends to track along the diaphragm (Chapter 27).

Interpreting Tamponade

Tamponade occurs when the pressure in the pericardium exceeds the pressure in the cardiac chambers, particularly the right atrium, resulting in impaired cardiac filling. It may be easiest to diagnose using the right parasternal four-chamber long-axis view and appears as a compression of the right atrial free wall into the atrium, intermittently reducing atrial chamber size (right-atrial wall compressed inwards during systole; Figure 17.23).

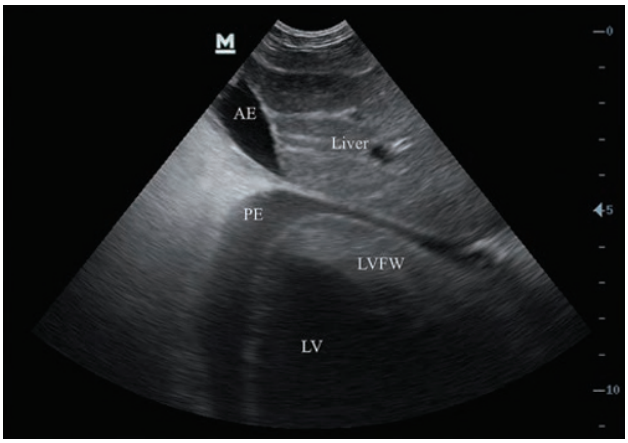


Figure 17.22 Still ultrasound image of pericardial effusion seen form the subxiphoid view of a dog. LVFW, left-ventricular free wall; PE, pericardial effusion; AE, abdominal effusion; LV, left ventricle.

However, it should be kept in mind that tamponade is a clinical diagnosis and the findings of shock in a patient with identifiable pericardial effusion should prompt a diagnosis of tamponade.

Caudal Vena Cava Assessment at the Subxiphoid Window

Although somewhat controversial in human medicine, the diameter of the inferior vena cava and collapsibility index have become well-established as a means of assessing volume status in people [19]. In dogs and cats, the CVC diameter (CVC_d) and collapsibility index (CVC_{CI}) have been

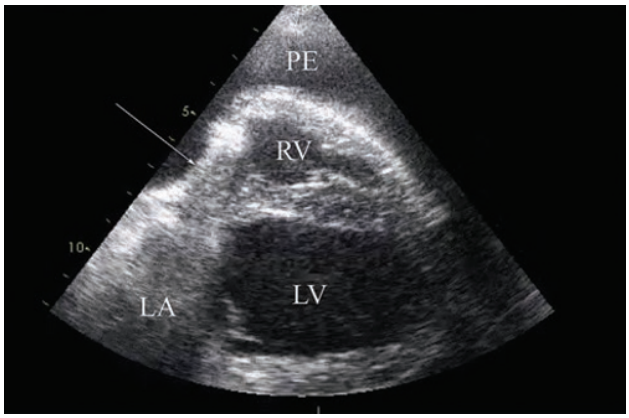


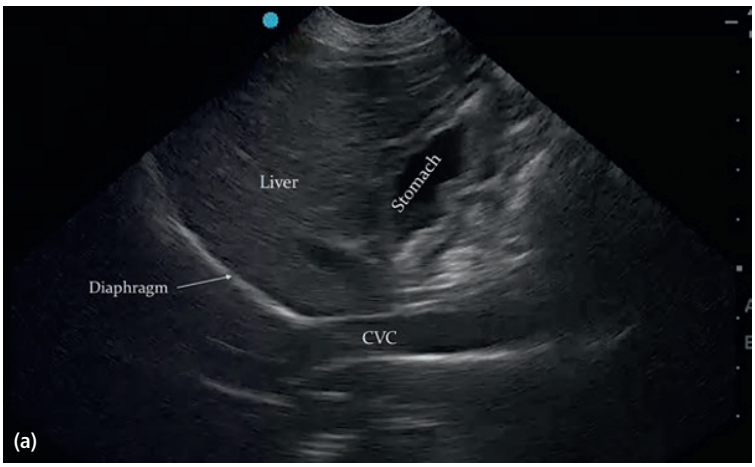
Figure 17.23 Still ultrasound image from a dog with tamponade; the right atrial wall (white arrow) is visible collapsing into the right atrium. LV, left ventricle; LA, left atrium; RV, right ventricle; PE, pericardial effusion.

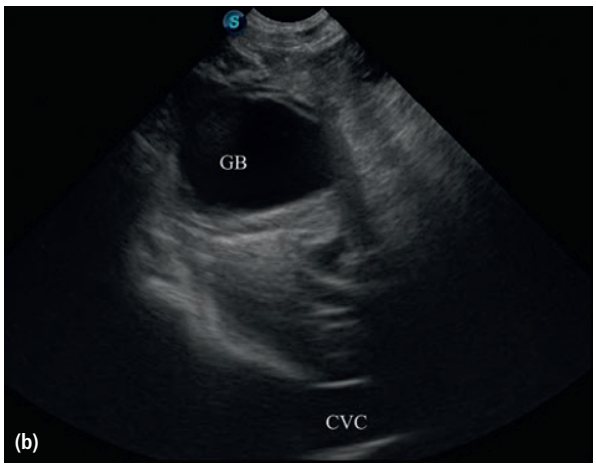
described at several VPOCUS windows, including the subxiphoid site (Figure 17.24a) [16, 20].

Although small animal research is limited, a small clinical study ($n = 27$) in spontaneously breathing dogs with compromised hemodynamics or tissue hypoperfusion suggested that CVC_{CI} can accurately predict fluid responsiveness, although the authors also concluded that research is necessary to extrapolate their results to larger populations of hospitalized dogs [21].

Caudal Vena Cava Collapsibility Index

Breathing changes intrapleural pressures, generating heart–lung interactions, thereby influencing the intravascular volume within the thorax and abdomen [22]. Owing to the elastic nature of the CVC, changes in the intravascular volume with the respiratory cycle result in a dynamic





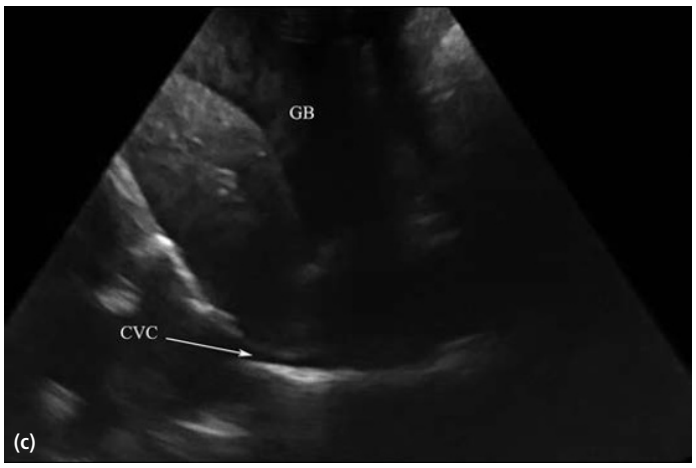


Figure 17.24 (a)–(c) Still ultrasound images depicting (a) a euvolemic patient with a normal caudal vena cava (CVC). (b) A patient with increased right atrial pressures and a "fat" CVC with almost no collapsibility index (in this case due to pericardial effusion [not visible]). Also note there is gall bladder (GB) wall edema (halo sign) in this example, which is very common in patients with pericardial effusion. (c) A hypovolemic patient with a "flat" CVC and subjectively narrow collapsibility index. Although a large CVC collapsibility index is suggestive of hypovolemia if the CVC is close to collapsed and very "flat" the change from flat to fully collapsed can be difficult to see creating the impression of small CVC collapsibility index.

change in the CVC diameter in proximity to the diaphragm [22]. This change in size of the CVC between inspiration and expiration is referred to as CVC collapsibility, and can be expressed as the collapsibility index (CVC_{CI}) using the following formula:

$$CVCCI = (CVCd\ max - CVCd\ min)/CVCd\ max\ [22].$$

Although considerable variation exists for cats and dogs, the reference interval for the CVC_{CI} at the subxiphoid site is roughly 20–45% [16, 20]. An enlarged or "fat" CVC with less than a 20% change in the CVC_{CI} suggests increased right atrial pressures, which may be secondary to hypervolemia, congestive heart disease or cardiac tamponade (Figure 17.24b).

Assessment of other cardiac VPOCUS findings should differentiate causes of an enlarged CVC (see above). In contrast a thin "flat" CVC with no change in the CVC_{CI}, or a change in the CVC_{CI} of more than 50% suggests hypovolemia (Figure 17.24c).

In people, increased respiratory effort is reported to cause a greater change in the CVC_{CI}, which is also likely present in small animals. It is possible to confuse normovolemic dyspneic patients as being hypovolemic.

Increased intra-abdominal pressure (e.g. abdominal masses or effusions) can decrease the size of the CVC. Avoid confusing patients with increased abdominal pressure and normal right-atrial pressure and intravascular volume status as being hypovolemic. Patients with conditions resulting in increased right atrial pressure and abdominal effusion are still likely to have a distended CVC (e.g. patients with right-sided heart failure and/or pericardial effusion).

Cardiac Veterinary Point-of-Care Ultrasound for Assessment of Mechanical Cardiac Activity During Cardiac Arrest

Cardiac VPOCUS may be helpful during cardiopulmonary resuscitation (CPR) to determine whether mechanical cardiac activity is present. However, it should not interfere with CPR efforts. In the authors' experience, the subxiphoid window can often be assessed in dogs and cats during active chest compressions, without interfering with efforts, and does allow rapid assessment of mechanical activity when exchanging chest compressors. Pulseless electrical activity is characterized by cardiac electrical activity without a palpable pulse. However, weak cardiac contractions may not generate peripheral pulses. Echocardiography may help differentiate pseudopulseless electrical activity (cardiac contractions that do not generate a pulse) from pulseless electrical activity. Video 17.1 depicts a period of cardiac VPOCUS during external chest compressions with spontaneous cardiac activity visible during a pause in compressions. Video 17.2 demonstrates cardiac VPOCUS being used to show pulseless electrical activity in a cat with cardiopulmonary arrest.





Video 17.1 A period of cardiac VPOCUS during external chest compressions with spontaneous cardiac activity visible during a pause in compressions.

Video 17.2 Cardiac VPOCUS being used to show pulseless electrical activity in a cat with cardiopulmonary arrest.

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18

Pericardiocentesis

Simon P. Hagley

Pericardiocentesis is a lifesaving yet daunting procedure, primarily required to improve cardiovascular stability in patients with an accumulation of fluid or air in the pericardial space. This chapter reviews the origins of such conditions and provides a step-by-step approach to enable the reader to perform this procedure safely and successfully.

The Pericardium

The pericardium is composed of an outer fibrous layer made up of collagen and elastin, and an inner serous layer comprised of a single sheet of mesothelial cells. This serous layer can be divided into parietal and visceral components that are continuous with each other; the parietal layer is adhered to the outer fibrous pericardium but reflects back, at the base of the heart, to lie on top of the myocardium and form the visceral layer (also known as the epicardium). The space between the two layers of this inner serous pericardium is the pericardial cavity, which in health contains a small volume (approximately 0.25 ± 0.15 ml/kg) of a clear, low-protein fluid [1]. This fluid, derived from plasma, serves to lubricate the layers and drains via the lymphatic system into the mediastinum and right side of the heart. Abnormal or excessive accumulation of fluid in this area is termed pericardial effusion and can lead to profound effects on a patient's circulation.

Although the pericardium can be partially removed or perhaps congenitally malformed, it serves several functions including stabilizing the position of the heart within the thoracic cavity, reducing resistance during cardiac contraction, balancing left and right cardiac output, and acting as a barrier against extension of infection or malignancy from surrounding tissues [2].

Pericardial Space Disease

Congenital malformation of the pericardium is uncommon and infrequently results in complication. Partial defects or agenesis may be an incidental finding on postmortem examination [1, 3]. Peritoneal–pericardial diaphragmatic hernia (PPDH) is one congenital defect that may result in complication if abdominal organs become entrapped in the pericardial cavity, causing varying clinical signs, and requiring surgical intervention. The prevalence appears to be higher in cats than in dogs [4].

Pneumopericardium has been reported in several dogs secondary to trauma, esophageal foreign body penetration, spontaneous rupture of communicating pulmonary bullae, and following exploratory laparotomy in a dog with a previously undiagnosed PPDH [5–7]. However, the most frequently encountered pericardial space disease is pericardial effusion, with commonly reported causes in dogs being neoplastic and idiopathic in origin [8].

In feline patients, congestive heart failure was the most prevalent cause of pericardial effusion with feline infectious peritonitis also being common [9, 10]. A comprehensive list of etiologies can be found in Box 18.1. Pericarditis can occur in association with pericardial effusion, either as the instigating cause of the effusion, or following repeated pericardiocentesis over several weeks.

Hemodynamic Changes Associated with Pericardial Effusion

The elasticity of the pericardium means it has little effect on cardiac chamber filling [1, 11]. Complications arise when the intrapericardial pressure increases, equilibrates with and then ultimately exceeds right-atrial and

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Companion website: www.wiley.com/go/burkitt/monitoring

Box 18.1 Causes of Pericardial Effusion in Dogs and Cats

- Idiopathic
- Neoplasia (e.g. hemangiosarcoma, chemodectoma, mesothelioma)
- Congestive heart failure[a] (more commonly cats)
- Coagulopathy
- Left atrial rupture secondary to mitral valve disease
- Septic pericarditis (e.g. bacterial, fungal, foreign body-associated)
- Feline infectious peritonitis[a]
- Systemic disease[a] (e.g. uremia, hypoalbuminemia)
- Trauma
- Iatrogenic hemorrhage
- Thyroid disease[a] (thyrotoxicosis in cats, hypothyroidism in dogs)
- Nicotine toxicosis[a]

[a] Unlikely to result in cardiac tamponade.

right-ventricular diastolic filling pressures (0–3 mmHg and 0–5 mmHg, respectively), which is typically a result of pericardial fluid accumulation [12]. This results in diastolic collapse of the right side of the heart, reducing venous return and increasing central venous pressure. Such a phenomenon is referred to as cardiac tamponade. A reduction in right-sided cardiac output results, via the Frank–Starling mechanism, in reduced stroke volume and explains many of the typical clinical signs. The pericardial pressure may continue to increase and reach a point where it exceeds left cardiac chamber filling pressures, resulting in more profound cardiovascular compromise. Compensatory neurohumoral mechanisms are stimulated in an attempt to maintain cardiac output by increasing cardiac contractility, heart rate, and systemic vascular resistance, and by retaining sodium and water; however, these processes are readily exhausted [1].

Several factors influence the onset of tamponade including the rate of fluid accumulation and distensibility of the fibrous pericardium. In an acute process such as hemorrhage, even a small volume of pericardial effusion rapidly increases the intrapericardial pressure since there has been inadequate time for the pericardium to stretch or hypertrophy [13]. This will result in acute cardiovascular collapse. However, with slower, more chronic effusion accumulation, gradual hypertrophy of the pericardium can occur, allowing a larger volume of fluid to accumulate. This increased pericardial compliance delays the onset of tamponade and any associated signs of right sided heart failure [1, 14].

Clinical Signs Associated with Pericardial Effusion

In those patients with a low volume of effusion, clinical signs will more likely be related to the underlying disease process with pericardial fluid identified incidentally on imaging. As an example, cats with congestive heart failure may present to the clinic due to respiratory distress from pulmonary edema and have evidence of a heart murmur or gallop sound on examination. However, if cardiac tamponade is present then signs consistent with low-output cardiac failure will be evident. Acute disease is often associated with cardiogenic shock, syncope, hypotension, respiratory distress, or even death [15]. Chronic presentations usually are more related to increases in systemic venous pressure and signs include exercise intolerance, weight loss, anorexia, lethargy, abdominal distension, tachypnea, or a cough [15, 16]. A study showed that 51% of canine patients with pericardial effusion had vomited recently, with this symptom being more common in severely hyperlactatemic dogs, regardless of the underlying etiology of the pericardial effusion [17].

Physical Examination

Physical examination usually reveals evidence of compromised perfusion such as tachycardia, reduced pulse quality, pallor, and reduced mentation, with the possible addition of tachypnea and muffled heart sounds. Chronicity may be implied by the presence of hepatomegaly, ascites, and jugular venous distension as recognized in right sided congestive heart failure, though it should be noted that these patients can also present after acute decompensation with signs of poor effective circulating volume [15, 16].

Pulsus Paradoxus

Pulsus paradoxus may or may not be present in either the acute or chronic presentation. This term describes the exaggeration of a normal physiological variation in arterial pulse pressure depending upon the phase of respiration. During spontaneous inspiration there is a slight decrease in systemic arterial blood pressure due to reduced left ventricular preload from pulmonary venous pooling; the result is a weaker pulse quality. Conversely, the palpable pulse pressure is stronger during expiration. This cyclical variation is more prominent in the presence of pericardial effusion due to reduced venous return to the heart. Pulsus paradoxus is defined as an inspiratory decrease in systolic arterial blood pressure of greater than 10–12 mmHg [18].

Diagnosis of Pericardial Effusion

The clinician may have a suspicion for the presence of pericardial space disease based on a patient's signalment, history, and physical examination findings. To confirm their suspicion and identify the underlying etiology, diagnostic imaging, blood testing, and pericardial fluid analysis may be required.

Echocardiography

It is now commonplace for veterinary practices to have access to ultrasonography. A point-of-care ultrasound is a quick and easy method to identify pericardial effusions and has demonstrated improved sensitivity and specificity compared to radiography [19]. This procedure is also less invasive in that the patient may choose its own body position, which is beneficial when the animal is already cardiovascularly compromised. Pericardial effusion appears as an anechoic or hypoechoic, rounded space between the heart (epicardium) and an often clearly demarcated fibrous pericardium (Figure 18.1; refer to Chapter 15 for additional information regarding point-of-care ultrasonography). A more detailed assessment is required to determine the presence of cardiac tamponade, evidenced by collapse of the right atrium and sometimes right ventricle during diastole.

Echocardiography is the reference standard diagnostic modality for pericardial effusion in human and veterinary medicine, with the ability to detect even small volumes of fluid accumulation. In addition, it can evaluate for the presence of a cardiac mass, valvular endocardiosis with or without left atrial rupture, hypertrophic cardiomyopathy, myocarditis, and concurrent pleural effusion.

Cardiac masses might be more easily identified in the presence of pericardial effusion; however, if the patient is unstable due to cardiogenic shock, therapeutic pericardiocentesis should take precedence over a complete echocardiogram, which can be delayed until the patient is more stable [8]. When attempting to identify the presence of a cardiac mass, particular attention should be paid to the right atrial area; however, there is only moderate correlation between the presumptive tumor type based on echocardiographic tumor location and histopathology [20].

Radiography

Thoracic radiographs may reveal a spherical cardiac silhouette without evidence of the movement blur commonly associated with cardiac contractions (Figure 18.2) [1, 15]. Cardiac enlargement is reported in up to 87% of dogs and 95% of cats depending on the volume of pericardial effusion [15, 21]. In those patients with cardiogenic shock, pulmonary vessels may appear smaller. The presence of cardiac tamponade may also reveal distension of the caudal vena cava and hepatomegaly [22]. Pleural effusion may be evident and abnormal lung patterns have been reported in up to 60% of cats, likely due to the high incidence of congestive heart failure as a cause of pericardial effusion in feline patients [10]. Dorsal tracheal deviation may be noted in association with a heart based tumor and pulmonary metastases may

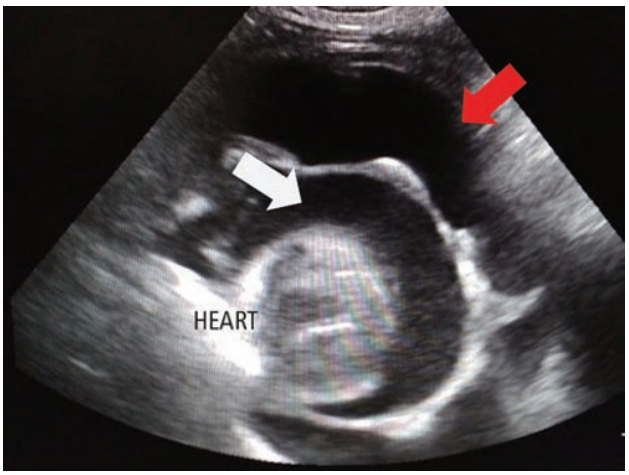


Figure 18.1 Pericardial effusion (white arrow) identified as an anechoic space between the epicardium and the fibrous pericardium in a cat with congestive heart failure. Pleural effusion (red arrow) can also be seen external to the pericardium.

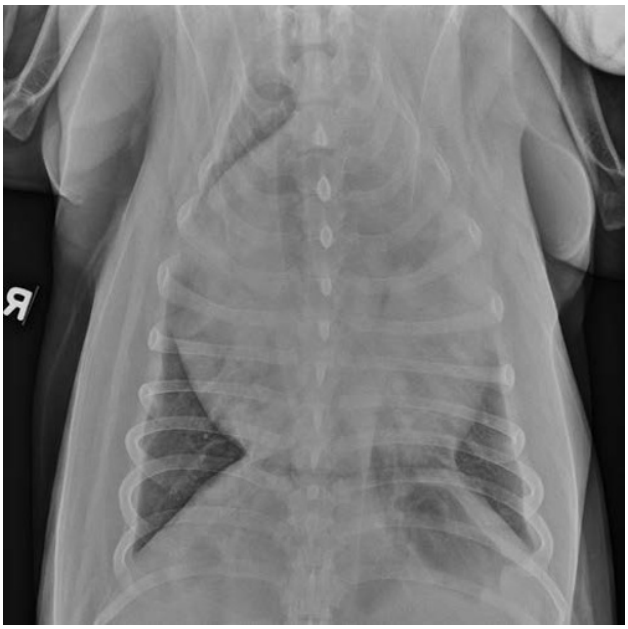


Figure 18.2 Dorsoventral radiograph of a dog with a pericardial effusion evidenced by the enlarged, globoid cardiac silhouette.

be noted when pericardial effusion is secondary to a neoplastic process [8]. Lastly, it is important to consider that in the acute presentation, thoracic radiography may be normal.

Alternative Imaging Modalities

Less commonly employed diagnostics include computed tomography (CT), magnetic resonance imaging (MRI), and non-selective venous angiography. Cardiac MRI yielded useful descriptive information regarding extent, anatomical location, and potential tumor type in a case series of eight dogs but did not substantially improve the diagnosis of a tumor compared with echocardiography [23]. Similarly, multidetector CT did not improve detection of cardiac masses in 11 dogs; however, it was considered advantageous in confirming the presence of pulmonary metastases and extra-cardiac lesions using a single imaging modality [24].

Electrocardiography

Although not a sensitive diagnostic test compared with echocardiography [10], it is important to assess these patients for the presence of dysrhythmias. Most commonly, the electrocardiogram (ECG) is normal or a sinus tachycardia is present; however, atrial and ventricular tachydysrhythmias (including atrial/ventricular premature complexes) can be seen [25]. ST segment elevation and low-amplitude QRS complexes are commonly noted [1, 15].

Electrical alternans refers to a beat-to-beat variation in amplitude of the QRS complexes, caused by the phasic swinging of the heart within a fluid-filled pericardial cavity, shifting the heart's electrical axis in relation to the electrocardiograph leads (Figure 18.3). This is more prevalent in large volume effusions [1, 26].

Blood Testing

If the patient's clinical condition permits, it is recommended to obtain a packed cell volume (PCV), serum total protein concentration, and coagulation profile (prothrombin time, activated partial thromboplastin time or activated clotting time) prior to performing pericardiocentesis. A baseline hematology and biochemistry evaluation may help elucidate the underlying etiology of the effusion but are often low yield. Serum cardiac troponin I (cTnI) has been evaluated in a series of dogs and was significantly higher in dogs with pericardial effusion compared with those without. Studies have also suggested patients with hemangiosarcoma have a higher cTnI (2.77 ng/dl; range 0.09–47.18 ng/dl) than dogs with idiopathic effusion (0.05 ng/dl; range 0.03–0.09 ng/dl) [27].

Pericardial Fluid Analysis

Cytological evaluation of pericardial fluid is generally low yield, especially in hemorrhagic effusions [28]; however, submission for fluid analysis is recommended as it can be beneficial for the diagnosis of lymphoma or bacterial and fungal agents [8]. Given the variation in underlying etiologies, cytological analysis may be more rewarding in cats. Diagnosis of a malignancy is difficult since most types of cardiac neoplasia exfoliate minimally into the fluid, and

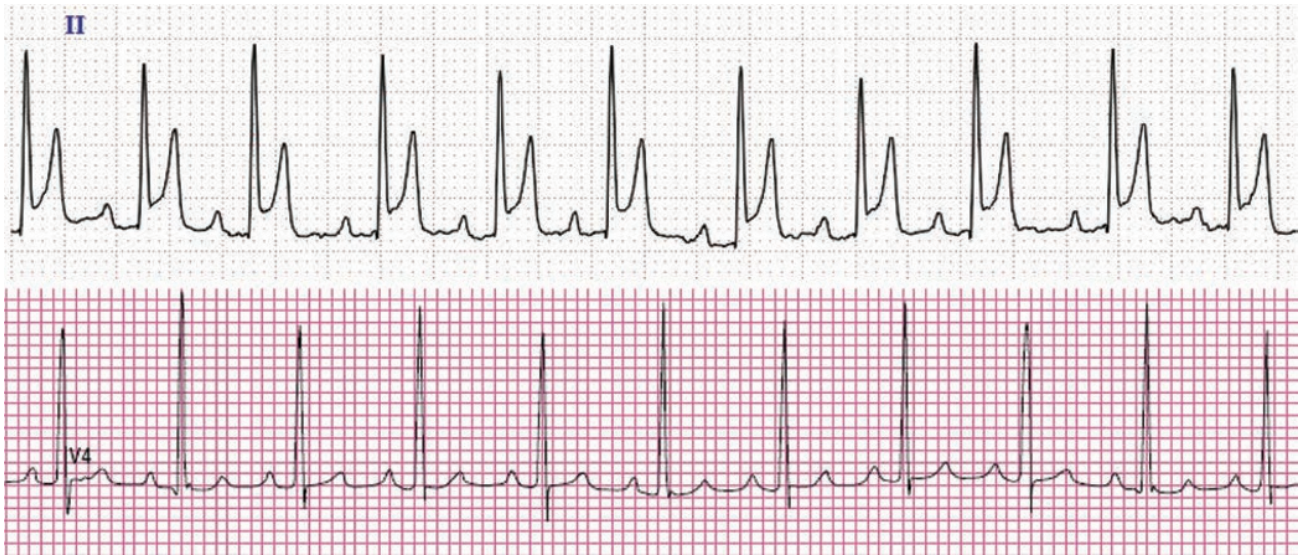


Figure 18.3 Two electrocardiogram leads from different patients with pericardial effusion showing elevated ST segment (top) and electrical alternans (bottom). *Source:* Courtesy of Dr. Elizabeth Bode and Dr. Meredith Daly.

the discrimination between reactive and neoplastic mesothelial cells is challenging. The diagnostic yield is improved if the PCV of the effusion is less than 10% [28]. Some studies have compared other markers in the pericardial fluid such as pH, lactate, and bicarbonate to peripheral whole blood in an attempt to identify the underlying cause, though study results are conflicting [29, 30].

Culture of the sample may be based upon cytological findings and clinical suspicion, and can help target antimicrobial therapy. Samples should be collected in sterile tubes without anticoagulant for culture and in EDTA for cytology.

Emergency Treatment of Pericardial Effusion

Patients with cardiovascular compromise from cardiac tamponade require immediate intervention. Pericardiocentesis as outlined below should be attempted together with shock-dose intravenous fluid resuscitation. If the patient has a known coagulopathy, then fresh frozen plasma or whole blood may be the fluid of choice. Given the high prevalence of cardiac disease in cats, the use of fluids should likely be avoided; fortunately, in this species tamponade is rare.

Pericardiocentesis

An intravenous catheter should be placed to facilitate fluid administration, chemical restraint if required, and as a precaution in case of complications, such as cardiac arrest or ventricular dysrhythmias. Some clinicians advocate for having a pre-drawn syringe of 2 mg/kg lidocaine in canine patients, so that it is readily available should it be needed. Sedation is rarely required in the compromised canine patient, particularly if local anesthetic is used. However, if necessary (as is the case for most cats), the author recommends the use of an opioid possibly in combination with a benzodiazepine, as these sedative agents have minimal hemodynamic impact. Alfaxalone or etomidate are additional considerations should further restraint be required. Continuous ECG and frequent blood pressure monitoring should occur throughout the procedure to ensure rapid identification of any complications, and flow-by oxygen should be provided. Care should be taken to ensure that all equipment potentially needed for the procedure is available prior to commencing (Box 18.2).

Pericardiocentesis can be performed from either the left or the right side of the thorax with the patient in sternal or lateral recumbency. Many clinicians prefer to perform the procedure with the patient in left lateral recumbency, approaching the pericardium from the right side to avoid the coronary circulation and reduce iatrogenic lung injury, taking advantage of the larger cardiac notch. In lateral recumbency, it is thought the apex of the heart may also "fall away" from the site of catheter entry due to gravity. Proponents of a left-sided approach reason that inadvertent puncture of the left ventricle would yield brightly colored oxygenated blood that would grossly contrast the classic "port" colored hemorrhagic pericardial effusion typically seen in dogs. Sternal recumbency may be preferred in a less stable patient.

The best location for catheter insertion into the pericardium must be identified. Echocardiography can be used to visualize the largest volume of fluid between the thoracic wall and the heart and with a sterile cover over the probe, which can also be used to guide insertion of the catheter. If bedside ultrasound is unavailable, pericardiocentesis can be performed blindly at the fourth or fifth intercostal space just ventral to the costochondral junction, where the precordial pulse is most readily palpable. Either the left or right lateral thorax must be clipped and aseptically prepared, between the second and eighth intercostal spaces encompassing at least two-thirds the height of the chest. Hand hygiene should be performed, and sterile gloves used throughout the procedure. The procedure site should be surgically draped. A local block of 2% lidocaine can be administered at the intended site of catheter insertion,

Box 18.2 Items Required for Performing Pericardiocentesis

- Clippers with clean blade
- Surgical antiseptic solutions for skin preparation
- Surgical drape
- Sterile gloves
- 14- to 22-gauge, over-the-needle catheter
  or
- Pericardiocentesis multifenestrated catheter with guidewire
- Scalpel blade #11
- Three-way stopcock
- Fluid extension tubing
- 5–50 ml syringe
- Receptacle for collecting removed effusion
- Sterile sample tubes (EDTA for cytology, without anticoagulant for culture, as needed)
- 2% lidocaine for local anesthetic block (safe dosage)
- Lidocaine 2 mg/kg intravenous preparation
- Electrocardiogram
- Non-invasive blood pressure monitor
- Portable ultrasound if available
- Chemical restraint as needed
- Oxygen supplementation
- Two or more assistants

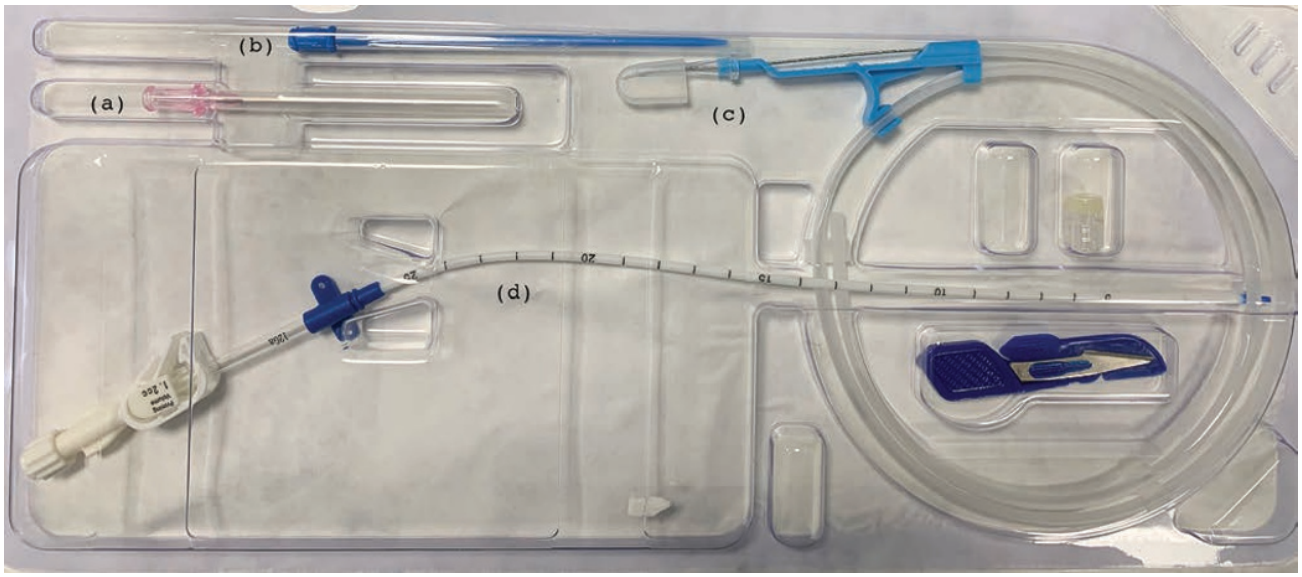


Figure 18.4 An example of a pericardiocentesis catheter set. (a) Needle. (b) Dilator. (c) Guidewire. (d) Pericardiocentesis catheter.

ensuring infiltration into the underlying intercostal musculature and pleura. A stab incision through the skin using a number 11 scalpel blade should be made at the location of the local block.

There are a few different catheter choices available. A standard 14- or 16-gauge, 5.5-inch over-the-needle catheter for medium and large sized dogs, or an 18- or 20-gauge, 1- to 1.5-inch over-the-needle catheter for small dogs and cats, is readily available in most practices. Alternatively, commercially available pericardiocentesis catheter sets may be preferred and typically include a needle, a guidewire, and a large bore multifenestrated silicone catheter (Figure 18.4). Advantages of the catheter include comparatively less irritation to the epicardium and better stability within the pericardial space during drainage. However, one study suggests that placement of pericardial catheters may increase the rate of dysrhythmias requiring treatment [31].

The chosen catheter should be inserted perpendicular to the skin, on the cranial aspect of the rib so that the intercostal vessels and nerves near the caudal rib margin are avoided (Figure 18.5a). The needle is directed toward the heart and advanced slowly until penetration into the pericardial space is suspected (Figure 18.5b). A syringe may be attached to the catheter prior to insertion into the patient and a small amount of negative pressure applied as the catheter is advanced. In the absence of pleural effusion, entry into the pericardial space may be evidenced by the appearance of fluid in the catheter hub or syringe. In patients with pleural effusion, a yellow to serosanguinous fluid may be obtained upon entry into the thorax and should not be confused with pericardial fluid, which is more commonly hemorrhagic. Subtle scratching on the tip of the catheter may be felt as it contacts the fibrous pericardium, and the catheter should be advanced through into the pericardial space. If scratching persists once inside the space, the catheter should be retracted slightly and the ECG evaluated for abnormal complexes, which may appear if the catheter is touching the epicardium or within the ventricle. Ultrasound can also be used to confirm location.

If using an over-the needle catheter, once confident the stylet is in the pericardial space, advance the catheter off the tip and remove the stylet. The catheter can then be attached to extension tubing, a three-way stopcock, and a 5–60 ml syringe based on patient size.

If using a pericardiocentesis set, once the needle is positioned within the pericardial space, the guidewire is passed through the needle into the space and the needle subsequently removed (Figure 18.5c, d). The multifenestrated catheter is then placed over the guidewire and into the pericardial space, with the guidewire being subsequently removed (Figure 18.5e). As previously, a three-way stopcock and syringe can now be attached to the catheter and the effusion can be drained (Figure 18.5f). No more than 2 ml of negative pressure should be placed on the syringe.

Once a small volume of pericardial fluid is obtained, it is important to evaluate it to ensure draining of the correct space. Observing the fluid in a glass tube (containing no anticoagulant) or a syringe for several minutes to assess for clotting is essential. Pericardial fluid should not clot (unless very recent hemorrhage) whereas whole blood from unintentional cardiac puncture will clot readily (unless effusion is due to severe coagulopathy). The PCV of the effusion is often lower compared with the peripheral PCV [30], whereas blood from inside the heart would be similar; again, this may not hold true in acute hemorrhage. Lastly, evaluation of the

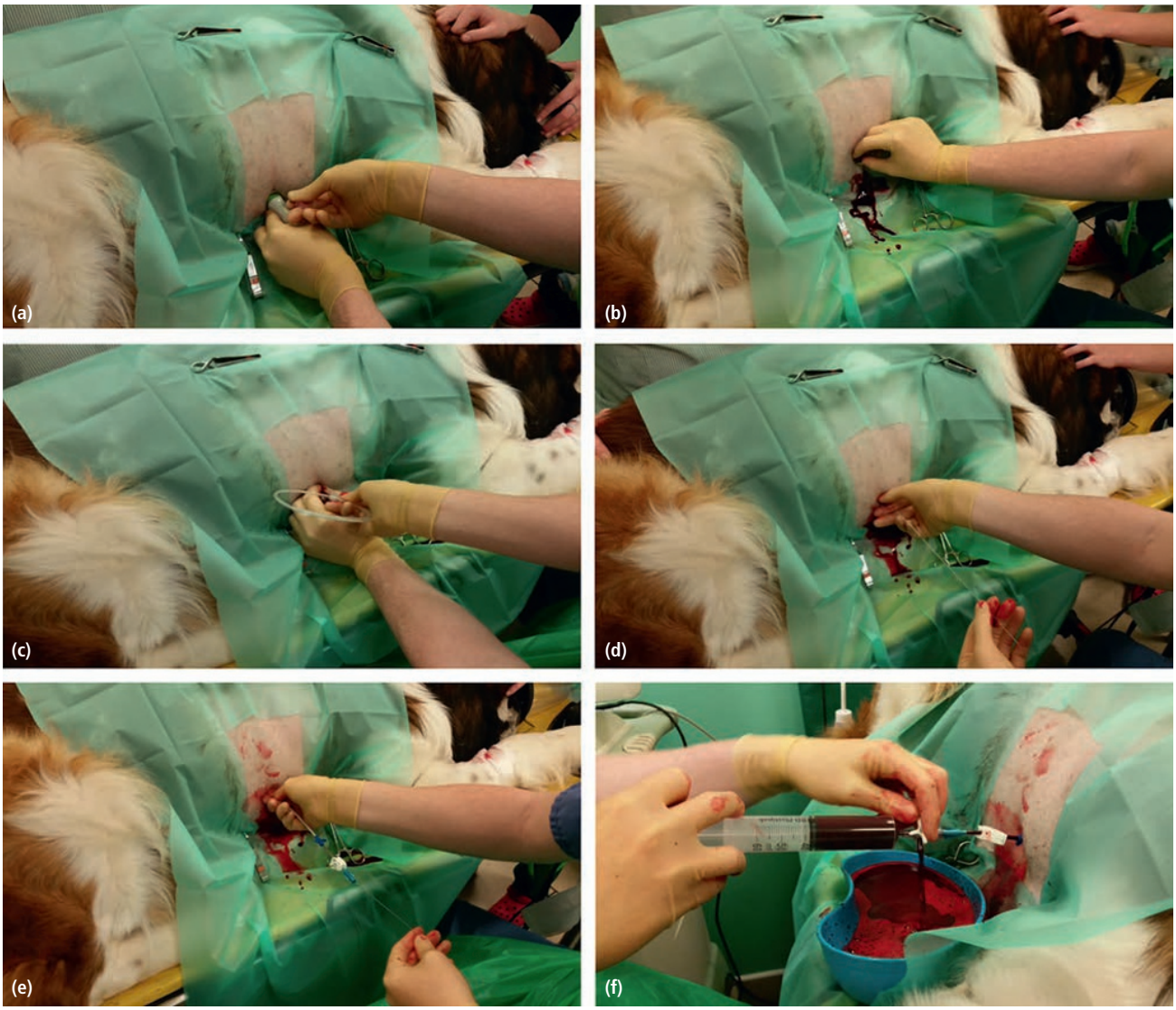


Figure 18.5 Pictorial representation of various stages of pericardiocentesis. The patient has been aseptically prepared and the operator is wearing sterile gloves. (a) Insert catheter in the fourth or fifth rib space, cranial to the rib *or* use ultrasound guidance. (b) Successful insertion into the pericardial space evidenced by the presence of hemorrhagic fluid. A three-way stopcock and syringe could be attached at this stage and the effusion removed. No further steps required. If using a pericardiocentesis catheter set, (c) Insert guidewire through the needle. (d) Remove the needle, leaving the guidewire in place. If required, the dilator could be passed over the guidewire, tunneled through the thoracic wall, and then removed. (e) Place the pericardiocentesis catheter over the guidewire always keeping hold of the wire. Once in place, remove the guidewire. (f) Attach a three-way drainage system and remove the effusion. *Source:* Courtesy of Dr. James McMurrough.

supernatant (formed from centrifugation for five minutes at 3400 rpm) of pericardial effusion will often be xanthochromic or yellow in color, due to the by-products of hemoglobin breakdown from previous bleeding. Supernatant from whole blood is more classically clear. In an unstable patient it may be prudent to continue slow aspiration of the fluid pending the results of these tests.

Following confirmation of correct catheter placement and evaluation of pericardial fluid, continued drainage should proceed until as much fluid as possible is retrieved, so long as active bleeding is not suspected. Hemodynamic improvement in the patient is often noted during the procedure with a reduction in heart rate and improved arterial pressure. Once all the effusion is drained the catheter can be carefully removed. A dressing, skin staple, or tissue glue can be applied over the skin incision if necessary.

The final volume of fluid should be quantified and resolution of tamponade and effusion confirmed with echocardiography. Persistence of significant pericardial effusion following the procedure may imply active hemorrhage or puncture of the myocardium. Refer to Protocol 18.1 for step-by-step instructions.

Protocol 18.1 Pericardiocentesis

1) Collect necessary supplies.
2) Obtain baseline peripheral blood samples and patient vitals.
3) Aseptically assemble drainage tubing, three-way stopcock, and syringe and/or separate pericardiocentesis set so each component is readily available.
4) Place patient in preferred recumbency.
5) Determine optimum site for pericardiocentesis with ultrasound or by palpation.
6) Connect patient to ECG and blood pressure monitor.
7) Clip and aseptically prepare pericardiocentesis site.
8) Perform hand hygiene and put on sterile gloves.
9) Drape the patient.
10) Administer local block of 2% lidocaine through the subcutaneous tissues and intercostal musculature.
11) An assistant should administer chemical restraint if needed.
12) Make a stab incision through the skin with scalpel blade.
13) Incrementally advance the catheter–stylet pair (or pericardiocentesis needle ± syringe) through the skin, subcutaneous tissues, and intercostal musculature cranial to a rib, directed toward the heart (Figure 18.5a).
14) Once pericardial fluid is seen in the hub of the catheter or syringe, either:
    a) Advance catheter and stylet an additional 0.5 cm, then feed catheter off and remove the stylet.
    b) If using a pericardiocentesis kit, remove syringe and advance guidewire through the needle into the pericardial space. Subsequently remove the needle and insert the catheter over the guidewire into the pericardial space. Remove the guidewire (Figure 18.5b–e).
15) Attach catheter hub to available three-way stopcock port and aspirate fluid (Figure 18.5f).
16) Aseptically place fluid samples in plain and EDTA tubes. Examine aspirated fluid for evidence of clotting; determine PCV and serum total protein concentration of fluid and compare with peripheral blood to confirm appropriate catheter placement in pericardial space.
17) Deposit remaining fluid into collection bowl. Quantify volume of retrieved fluid and store samples in refrigerator prior to submission for cytologic analysis.
18) Once all fluid is removed, withdraw catheter from pericardial space (or suture in place if indwelling catheter was chosen).
19) Verify reduction of the effusion via echocardiography if possible.

Longer-Term Treatment of Pericardial Effusion

Treatment and prognosis depend on the underlying cause of the effusion. Approximately 50% of idiopathic effusions will resolve following initial pericardiocentesis; however, the remainder will recur within hours to years [15]. Depending on the rate of fluid accumulation surgical intervention may be considered, such as a subtotal pericardectomy, thorascopic pericardial window, or a fluoroscopy-guided balloon pericardiotomy [32–34]. These surgical procedures are especially beneficial in the treatment of infectious causes, alongside long-term antimicrobial therapy. The treatment for neoplastic effusions will vary based on tumor type though in most cases chemotherapy alone provides a similar mean survival time compared with the combination of chemotherapy and mass resection, which is infrequently feasible [10, 15, 35]. Lastly, appropriate management of congestive heart failure should result in resolution or delayed progression of associated small volume pericardial effusion.

Complications

Pericardiocentesis is a relatively safe procedure if appropriate precautions are taken to reduce potential risks. Commonly recognized complications include ventricular or supraventricular dysrhythmias, inadvertent ventricular puncture, cardiac or coronary artery laceration, hemorrhage, dissemination of infectious agents or neoplasia, and cardiopulmonary arrest [25].

Penetration of the ventricular wall may occur if the catheter is advanced too far into the pericardial space and will likely result in a ventricular dysrhythmia on surface ECG. In addition, the catheter may move with each cardiac contraction. This is often not a serious complication and resolves when the catheter is withdrawn from the myocardium. If not recognized and corrected, however, it could lead to inadvertent removal of large volumes of blood from circulation. Manipulation of the catheter within the ventricular wall increases the risk of cardiac laceration and fatal hemorrhage.

As described previously, continuous ECG monitoring, blood pressure evaluation, ultrasound guidance, monitoring

of the retrieved pericardial fluid for clotting, and readily available anti-arrhythmic medication should allow rapid recognition of complications and immediate intervention. If coronary artery laceration or cardiac rupture occurs, the patient is unlikely to survive.

The clinician must consider relative contraindications to performing pericardiocentesis, such as the presence of a coagulopathy, atrial rupture, actively bleeding neoplasia, or a small volume effusion in the absence of tamponade.

Post-Pericardiocentesis Monitoring

Following the procedure patients should be closely monitored for recurrence of the pericardial effusion, ongoing hemorrhage, or development of ventricular dysrhythmias. Fluid can leak from the pericardial space into the pleural cavity resulting in life-threatening blood loss. These will most likely manifest as hemodynamic instability, meaning that evaluation of cardiovascular parameters, respiratory rate and effort, demeanor, and urine output for a minimum of 24 hours is advisable. Ventricular dysrhythmias can range from mild to severe and may necessitate antiarrhythmic therapy. Serial or continuous ECG, heart rate and blood pressure monitoring, thoracic auscultation, and intermittent echocardiography will facilitate prompt recognition of complications.

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19

Monitoring Tissue Perfusion

Clinicopathologic Aids and Advanced Techniques

Alexandra Nectoux and Guillaume L. Hoareau

Cells depend on oxygen delivery for energy production to maintain homeostasis. Failure to achieve sufficient tissue oxygen delivery results in cell dysfunction and ultimately death. Insufficient oxygen delivery to cells can be focal (e.g. ischemic limb, infarcted spleen) or systemic, which is then termed shock.

Whether hypoxia is focal or systemic, restoring adequate tissue oxygen delivery is a cornerstone of resuscitation. Oxygen delivery to tissues depends on arterial oxygen content and tissue perfusion. This chapter focuses on the importance of monitoring tissue perfusion (Table 19.1).

Clinical Monitoring of Tissue Perfusion

Physical Examination

Physical examination is an inexpensive and rapid way to monitor a patient's perfusion serially. Assessment of global perfusion, a core part of a triage examination, should include evaluation of mentation, heart rate, pulse quality, extremity-to-core temperature difference, mucous membrane color, and capillary refill time. This focused assessment of a patient's circulatory function provides a global evaluation of tissue perfusion and assists in the diagnosis of shock. The evaluation of circulatory function by physical examination may also help in the diagnosis of focal perfusion disturbances such as an ischemic limb if the femoral pulse is absent, for instance.

Arterial Blood Pressure Monitoring

Systemic arterial blood pressure measurement is also commonly used to monitor a patient's cardiovascular status. Unfortunately, normal blood pressure does not indicate adequate capillary perfusion and thus cannot guarantee adequate oxygen delivery to cells. First, microcirculatory disturbances may persist despite normal macrocirculatory parameters such as blood pressure (i.e. hemodynamic incoherence). Second, normal systemic arterial blood pressure does not equate to normal blood flow since profound vasoconstriction, for instance, may result in normal or even high systemic blood pressure in the face of reduced blood flow. Conversely, vasodilation may result in increased blood flow despite a reduction in blood pressure. Such decrease in systemic vascular resistance is often associated with increased cardiac output in the hyperdynamic phase of sepsis.

Blood pressure measurement can be either non-invasive (oscillometric, Doppler, or pressure wave analysis technologies; see Chapter 14) or invasive. Invasive blood pressure monitoring depends on direct pressure measurement within the lumen of an artery. Most systems rely on pressure wave transmission through a column of water to a pressure transducer (see Chapter 12), though solid-state blood pressure transducing catheters are also available.

Regardless of the method of acquisition, arterial blood pressure can be used to calculate cavity or organ-specific perfusion pressures. Clinical interventions or research applications often rely on myocardial, cerebral, abdominal, or spinal perfusion pressures, which are the driving pressures for blood from the arterial to the venous sides of organs. The use of such perfusion pressures in clinical practice as resuscitation endpoints has been studied in various settings. Myocardial perfusion pressure (diastolic aortic pressure minus central venous pressure) and cerebral perfusion pressure (CPP; mean arterial pressure [MAP] minus intracranial pressure) are critical prognostic indicators in cardiopulmonary resuscitation (CPR). Data summarized in current veterinary CPR guidelines suggest that myocardial perfusion pressures above 20 mmHg are associated with increased likelihood of return of spontaneous circulation [1]. Post-cardiac arrest CPP was higher in

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Table 19.1 Summary of commonly available tissue perfusion monitoring tools.

Clinicopathologic aids		Advanced techniques	
Physical examination		Near-infrared spectroscopy	
Arterial blood pressure	Diastolic, mean, systolic	Microcirculation visualization	Orthogonal polarized spectroscopy
	Perfusion pressures		Sidestream dark field
Urine output			Incident dark-field
Clinical imaging	Transcutaneous ultrasound	Transcutaneous O_2 and CO_2	
	Transesophageal echocardiography	Regional capnography	
Metabolic biomarkers	Lactate concentration	Thermography	
	Lactate clearance	Cutaneous laser Doppler	
	Oxygen extraction ratio	Urine oxygen partial pressure	
	$SmvO_2$	Microdialysis	
	$ScvO_2$		
	PCO_2 gradients		

PCO_2, partial pressure of carbon dioxide; $SmvO_2$, mixed venous hemoglobin oxygen saturation; $ScvO_2$, central venous hemoglobin oxygen saturation.

survivors than nonsurvivors following cardiac arrest [2] and CPP optimization is an area of active research. Abdominal perfusion pressure (MAP minus intra-abdominal pressure) is a significant focus for the management of abdominal compartment syndrome, although studies suggest that it should not be used as a resuscitation endpoint [3]. Spinal cord perfusion pressures (MAP minus intraspinal pressure, between the dura and the surface of the cord) over 50 mmHg have been associated with favorable neurological outcomes following traumatic spinal cord compression [4, 5]. The concept of perfusion pressure further supports why a resuscitation strategy solely based on MAP may not maintain adequate perfusion to individual organs (e.g. two patients may have identical MAPs but vastly different intra-abdominal pressures, and hence will have different abdominal tissue perfusion pressures).

Urine Output

Quantification of urine output reflects renal perfusion through glomerular filtration, tubular reabsorption, and patency of the urinary tract. Urine output is part of the essential monitoring of critically ill patients, especially those with particular risk for acute kidney injury. Urine output can be quantified hourly or less frequently, depending on the patient's status. Urine output is an integral factor in fluid therapy planning and overall patient care. It can be easily and precisely measured by placing a urethral catheter connected to a closed collection system. Normal urine output is 1–2 ml/kg/hour in dogs and cats. Oliguria is defined as a urine output less than 0.5 ml/kg/hour [6] and anuria is the absence of urine production. A urine output above the normal range is considered polyuria. In human medicine, plasma renin concentration is well correlated to urine output and represents a useful tool to assess tissue perfusion [7]; however, it is not always increased in response to ischemia–reperfusion injury. For instance, pigs subjected to hemorrhagic shock and endovascular aortic occlusion developed increased serum angiotensin II concentration without significant changes in circulating levels of renin [8].

Clinical Imaging

Imaging tools routinely used in the hospital can be used to some extent to monitor cardiovascular status. Various approaches may assess cardiac filling and systolic function. Different point-of-care ultrasound protocols have been described in the veterinary literature [9]. Tissue perfusion to a specific organ can be confirmed using Doppler ultrasound, but this remains a diagnostic rather than a monitoring tool. Transesophageal echocardiography (TEE), whereby an ultrasound probe is inserted near the heart through the esophagus, is gaining popularity in human emergency medicine and has been used extensively in people undergoing anesthesia. In the human emergency room, TEE has proved a valuable tool in CPR and can assist in improving hand or chest compression device placement to increase stroke volume and optimize perfusion [10]. TEE is also often used to monitor and optimize perfusion in patients undergoing extracorporeal membrane oxygenation [11].

Standard Clinicopathologic/Metabolic Markers of Tissue Perfusion

Metabolic markers of tissue perfusion are another crucial part of patient management and provide information regarding systemic or organ-specific perfusion derangements.

Lactate

Plasma lactate concentration has long been used for the monitoring of systemic or focal (e.g. limb) ischemia. Lactate production has traditionally been classified as type A (insufficient oxygen delivery) or B (increased lactate production in the face of adequate oxygen availability) [12]. Type A hyperlactatemia has been further characterized as relative, from increased oxygen demand (e.g. in exercise, shivering, seizure) or absolute, from decreased oxygen delivery (e.g. in hypoperfusion, severe anemia, severe hypoxemia). Type B hyperlactatemia has been observed with certain diseases (e.g. lymphoma, pheochromocytoma), toxins or drugs (e.g. glucocorticoids, xylitol, metformin), or congenital metabolism disorders. Spurious readings can be observed with ethylene glycol intoxication [13]. Much of the evidence about the usefulness of plasma lactate concentration in veterinary medicine has originated from studies of gastric dilation volvulus. While measurements at single time points do not always provide meaningful information, serial plasma lactate concentration measurements may better correlate with outcome. Lactate concentration-derived parameters such as time to lactate less than 2 mmol/l and lactate clearance can differentiate between survivors and non-survivors in dogs diagnosed with shock [14, 15].

Lactate measurement may also reflect locoregional hypoxia in patients with various conditions. Venous lactate concentration measured from a limb with suspected ischemia may be higher than that of the systemic circulation [16, 17]. Similarly, lactate concentration measured in cerebrospinal fluid reflects the magnitude of spinal cord [18, 19] or cerebral ischemia [20].

Blood Gas Parameters and Oxygen Extraction Ratio

Under baseline physiologic conditions, oxygen is delivered to cells in excess of metabolic demand. When tissue oxygen delivery is decreased, cells and therefore organs can increase the fraction of oxygen they consume from the arterial supply to a higher percentage of the oxygen being delivered. Thus, oxygen consumption remains independent of oxygen supply over a wide range of normal to lower-than-normal-but-adequate oxygen delivery. This principle can be quantified by calculating the oxygen extraction ratio (OER; Box 19.1)

A mixed venous sample is obtained from the pulmonary artery via a pulmonary artery catheter and comprises a combination of cranial and caudal vena cava blood along with coronary sinus blood; thus, mixed venous blood is the most globally representative venous blood sample obtainable. This mixed venous sample better reflects systemic venous oxygen content than central venous blood acquired from the jugular vein or vena cava.

Box 19.1 Calculating the Oxygen Extraction Ratio

$$OER(\%) = (CaO_2 - CmvO_2) \div CaO_2$$

$$CaO_2 = [1.34 \times Hb \times SaO_2] + [0.003 \times PaO_2]$$

$$CmvO_2 = [1.34 \times Hb \times SmvO_2] + [0.003 \times PmvO_2]$$

CaO_2, arterial oxygen content, ml/dl
$CmvO_2$, mixed venous oxygen content, ml/dl
Hb, hemoglobin concentration, g/dL
OER, oxygen extraction ratio
PaO_2, partial pressure of arterial oxygen, mmHg
$PmvO_2$, partial pressure of microvacular venous oxygen, mmHg
SaO_2, oxygen saturation
$SmvO_2$, mixed venous oxygen saturation

While OER increases in shock states to compensate for reduced oxygen delivery, in more severe shock states, cells have extracted nearly all oxygen in the capillary blood, OER stops increasing, and oxygen consumption becomes dependent on oxygen delivery. Scientists have argued that the relationship between oxygen consumption and delivery is actually linear in certain conditions, which would dictate that oxygen consumption would always depend directly (linearly) on oxygen delivery [21, 22]. Extraction ratios can be calculated for specific tissue beds via selective sampling of arterial and venous blood. For example, renal arterial and venous blood can be sampled to calculate the renal OER. This approach is generally limited to the research setting.

The balance between oxygen delivery and oxygen consumption is often evaluated via analysis of central venous (cranial caval, most often) saturation of oxygen ($ScvO_2$), as it is a single number and thus eliminates the need for calculations. $ScvO_2$ can be measured via blood gas analysis or continuous fiber tip spectrophotometry. $ScvO_2$ was part of the early goal-directed therapy protocol for the management of septic shock in people [23]. In a study of dogs with sepsis or septic shock associated with pyometra, a goal-directed resuscitation therapy guided by physical examination, $ScvO_2$, lactate concentration, and base deficit showed that $ScvO_2$ and base deficit were superior to lactate in predicting survival [24]. Importantly, in critically ill people, there is no uniform correlation between lactate concentration and $ScvO_2$ [25, 26]. In isolated studies, resuscitative efforts in patients with early sepsis aiming at improving

lactate clearance or normalizing $ScvO_2$ did not yield significant survival benefit [27]. However, a meta-analysis reported superiority of lactate clearance-driven algorithms when compared to those aiming at improving $ScvO_2$ [28].

Partial pressure of carbon dioxide (PCO_2) can be measured on mixed or central venous blood to calculate the tension difference with that of arterial blood ($Pv\text{-}aCO_2$) [29]. $Pv\text{-}aCO_2$ depends mostly on cardiac output and systemic CO_2 production and is not reflective of tissue hypoxemia; rather it reflects the adequacy of venous blood flow to remove the CO_2 produced in the tissues, an approximation of tissue perfusion. $Pv\text{-}aCO_2$ could be used to guide resuscitation in conjunction with other markers of tissue perfusion, in particular $ScvO_2$, to match oxygen delivery to CO_2 production. A $Pv\text{-}aCO_2$ greater than 6 mmHg may suggest insufficient resuscitation.

More sophisticated calculations leveraging arterial and venous O_2 and CO_2 contents may refine the specificity of tissue perfusion monitoring. In patients with septic shock, following restoration of MAP, increased $Pv\text{-}aCO_2$ correlated with lower tissue oxygen saturation, a reflection of global flow. $PcvaCO_2/CavO_2$ ratio (where $PcvaCO_2$ is the central venous-to-arterial carbon dioxide difference and $CavO_2$ is the arterial–venous oxygen content difference) was a good indicator of local oxygen consumption and microvascular dysfunction [30].

Advanced Tissue Perfusion Monitoring Techniques

The previously described techniques may not reflect changes in microcirculation. Direct microcirculation assessment tools can provide important information to guide resuscitation. Unfortunately, these techniques sometimes require expensive, large, or impractical tools, and are thus primarily used in laboratory or research settings at this time.

Near-Infrared Spectroscopy

Spectroscopy is optical monitoring that relies on the application of different wavelengths of light to tissue. In healthy tissues, hemoglobin, myoglobin, cytochromes, melanins, carotenes, and bilirubin absorb light in a concentration-dependent manner. Tissue oxygenation measurement relies on the distinct light wavelength absorption characteristics of hemoglobin in its oxygenated and deoxygenated forms.

Near-infrared spectroscopy (NIRS) is an optical monitoring technique that relies on the Beer–Lambert law correlating the concentration of a substance to its light absorption. NIRS assesses oxygen saturation of hemoglobin in the capillaries of a tissue (StO_2) and can detect oxygenation disorders before pulse oximetry, plasma lactate concentration, or physical examination [31]. Assuming no significant change in arterial oxygen content, a decrease in tissue oxygenation directly reflects reduced perfusion. StO_2 is mostly measured in peripheral muscle or brain with dedicated probes; since it measures local oxygen saturation, readings depend on probe location [32, 33]. Moreover, changes in blood volume in brain tissue, peripheral vasoconstriction, pain, hypothermia, as well as hypovolemia, meaningfully influence the intravascular compartment and alter readings. In the human literature, several studies showed that tissue desaturation in the frontal cortex of the brain and peripheral muscles (thenar, leg, and masseter muscles) was associated with poor outcomes [34, 35]. However, these results remain controversial [36]. In pigs exposed to various cardiovascular derangements, NIRS readings from the pelvic limbs showed good agreement with more invasive parameters [22]. NIRS evaluation of cerebral blood flow has been described in dogs in an experimental setting, where the probe was applied directly onto the skull [37]. Non-invasive NIRS readings have been reported in healthy conscious and unconscious Chihuahuas [38]. StO_2 declines in various states of shock in canine patients [39, 40]. StO_2 readings were associated with disease severity but did not predict survival [40]. Care providers should consider establishing clear protocols, as various factors such as probe location may introduce variability in the readings. For instance, in healthy dogs, the sartorius muscle provides a good assessment of tissue oxygen saturation and could be consistently used [41]. Similar to many other monitoring devices, there can be significant variability in readings when using different NIRS devices [42].

Microcirculation Visualization

Direct observation of the microcirculation is a dynamic field of research that has rendered this approach more available for clinical use, even in veterinary medicine. There are various technologies to visualize the microcirculation. The following devices are listed from oldest to newest:

Orthogonal Polarized Spectroscopy Imaging

Two polarizers oriented perpendicularly to one another are respectively used to emit and collect light on a specific tissue region. The light collected passes through a spectral filter to isolate the wavelength region and linearly polarize it. A beam splitter reflects the light toward the target tissue, and an objective lens focuses the light onto a region of approximately 1 mm in diameter. The device then generates an image of the illuminated region. Contrast is obtained from the absorption of linearly polarized light by both oxygenated and deoxygenated hemoglobin in the blood. Subsequently, red blood cells in the microcirculation

appear black on the white background of the surrounding tissue, which creates a high-contrast image. This technique was first described in the human literature in comparison with a standard fluorescence method [43]. A study established baseline values in 15 healthy dogs and assessed the reproducibility of this technique [44], but it offers a limited field of view, as only a focal area of tissue can be visualized.

Sidestream Dark Field Imaging

Sidestream dark field (SDF) imaging uses the same concept as orthogonal polarized spectroscopy imaging. In this case, the illuminated light and the reflected light travel via different pathways to not interfere with the image quality. Using a videomicroscope, a green light-emitting diode produces a light beam, which is absorbed by hemoglobin. This technique is limited to experimental use because it requires constant user interactions and long measurements to obtain only a few seconds of video for analysis [45, 46]. Despite those limitations, SDF imaging allows real-time imaging of the microvasculature and was able to demonstrate altered microcirculatory variables in dogs with hemorrhagic shock, when compared to normal dogs [47]. SDF shows an increase in vessel density in dogs receiving various rates of fluid under general anesthesia, which demonstrates the link between SDF findings and perfusion changes [48]. Finally, whereas SDF is a reliable bedside tool to assess microcirculation, it did not correlate with current standard analysis for different perfusion parameters in healthy dogs [49].

Incident Dark Field Imaging

First described in 1970, incident dark field imaging (IDF) is similar to SDF imaging in that it uses light-emitting diodes arranged circumferentially to illuminate a target tissue. In this method, the strobe speed is significantly decreased, which induces less distortion of the red blood cells' images. The device is a small, light, handheld camera, which makes it easier for the operator to manipulate compared to the previous devices. This camera also has a higher optical resolution and a wider field of view that enable the user to identify suitable areas of microcirculation more rapidly. Additionally, in contrast to the previous device, the IDF imaging device has a dedicated integral automated software, decreasing the time needed to acquire images [50, 51]. In comparison with SDF technique, IDF showed comparable vessel detection and significantly better vessel contrast in pigs undergoing a shock state [52].

Several microcirculatory indices can be calculated by semi-automated software using the images acquired by the three previous techniques. Care providers can assess and monitor the microcirculatory system in a peripheral region using the following parameters: (i) Total vessel density, which is a measure of total vessels' length over the area within the field of the camera; (ii) proportion of perfused vessels, which represents the proportion of perfused vessels in the field; and (iii) perfused vessel density, which is a measure of perfused vessels' length within the field. The potential clinical benefits of perfused vessel density, which is derived from total vessel density and proportion of perfused vessels, are outlined by experimental data. A normal perfused vessel density, for example, strongly correlates with tissue survival in rodents [53]. Hemodilution can decrease the perfused vessel density [54]. Finally, (iv) the microcirculatory flow index quantifies the velocity of microcirculatory perfusion.

Transcutaneous O_2 and CO_2 Monitoring

Transcutaneous blood gas monitoring is a noninvasive and reliable technique to approximate oxygen and carbon dioxide tensions in a tissue. It detects early changes in tissue blood gases compared to invasive methods [55]. This technique is more reliable and more accurate than capnography in human patients [56]. Gas tension is measured via polarography. The skin is heated to 43–45°C to increase transcutaneous gas diffusion. Accuracy of this technique can be affected by skin thickness, peripheral vasoconstriction, hypoperfusion, or peripheral edema [57]. Consistent measurement protocols are therefore paramount for reliable measurements. Moreover, a decrease in cardiac output reduces the ability of transcutaneous partial pressure of oxygen ($PtcO_2$) to approximate partial pressure of oxygen (PO_2) [58], which is a significant limitation when monitoring tissue perfusion in critically ill animals. Recently, research in critically ill dogs showed that transcutaneous blood gas monitoring overestimated PaO_2 and $PaCO_2$ and should not be used in those patients [59]. In ventilated patients, an oxygen challenge test is performed to evaluate the change in $PtcO_2$ value in response to an increase in fraction of inspired oxygen. This test predicted outcome in patients with septic shock [60].

Regional Capnography

The tissue-to-arterial PCO_2 gap is a reliable marker of tissue hypoperfusion [61, 62]. This gap can be measured using a tonometer that assesses PCO_2 in a tissue based on the gas's partial pressure equilibrium. Gastric, esophageal, sublingual, cutaneous, and urinary bladder tissues have been studied in experimental animals and human patients [62, 63]. The sublingual-arterial PCO_2 gradient has been described to be a better prognostic indicator than physical markers of hypoperfusion (cardiac index, DO_2, plasma lactate) [64]. In healthy dogs under anesthesia, gastric and bladder tonometry both correlated to sevoflurane-induced hypotension in dogs [65]. In that study, gastric tonometry was a better reflection of global hemodynamics when compared to bladder tonometry.

Thermography

Thermography is a reproducible, non-invasive, rapid imaging technique to measure the heat emitted by a surface. This method is used in human medicine to diagnose venous and arterial thromboembolism [66]. A recent publication in cats showed that infrared thermography had a high accuracy in diagnosing arterial thromboembolism [67]. The technique could also be used to evaluate reperfusion, as evidenced by an increase in thermal signature with return of blood flow [68]. This is a promising technique for clinical practice application to assess peripheral macrocirculation; however, further studies are needed to evaluate its ability to monitor microcirculation.

Cutaneous Laser Doppler

This technology can be used to monitor both blood flow and endothelial dysfunction. The laser Doppler probe (LDP) is placed on an area of clipped and cleaned skin with adhesive tape. The LDP emits laser beams and records signals generated by refracted beams. Signal refraction is created by the flow of blood cells, which is a function of blood flow in the tissue under the probe. The machine provides a computer-generated flow unit, which is proportional to how much blood flow the probe is sensing. The probe evaluates flow at a depth of approximately 1 mm, therefore providing information about arteriolar, capillary, and venular flow. After a few minutes, the probe is heated to a temperature of 42 °C (107.6 °F). In health, this rise in temperature leads to an increase in blood flow, mostly mediated by NO [69], which is reflected as an increase in flow units. If the endothelium is dysfunctional, this flow rise is blunted as a result of decreased NO synthesis or bioavailability. It can be argued that this technique only provides information about the microcirculation immediately under the skin rather than throughout the body. In the research setting, cutaneous laser Doppler flowmetry has been used in dog models for assessing wound healing and grafts. Laser Doppler technology has also been applied to non-cutaneous tissues. Clinical reports of the use of laser Doppler flowmetry (LDF) in dogs are limited. LDF has been used to measure capillary flow in the gastric mucosa of dogs with gastric dilation volvulus [70]. Intra-operative LDF technology has successfully measured spinal cord blood flow in canine patients with intervertebral disc disease. Spinal cord blood flow increased immediately following decompression but did not correlate with degree of compression on magnetic resonance imaging or neurological outcome 24 hours following surgery [71]. The same group more recently used the same technology to demonstrate that durotomy did not increase spinal cord blood flow following spinal decompression in dogs with intervertebral disc disease [72].

Urine Partial Pressure of Oxygen

Perfusion to the kidney can be determined from PO_2 in the urine, assuming there is no change in arterial oxygen content. This technology was developed in animals and has been used in people. Current technologies rely on either a probe immersed in urine (whether in the bladder or a urine collection system) or optical fiber that can measure urine PO_2 without contact with the urine. Alterations in global hemodynamics translate to changes in urine PO_2 [73]. In sheep, resuscitation from septic shock with fluids and norepinephrine outlined the positive correlation between urine PO_2 and renal medullary blood flow. Studies in rabbits have also suggested the potential for urine PO_2 to predict the risk of acute kidney injury [74]. This technology relies on the absence of oligoanuria.

Microdialysis

Microdialysis is another tool to monitor cell function and changes in perfusion. A microdialysis catheter (outer diameter 0.24–0.5 mm and length 1–10 mm) is inserted in the tissue of interest or inside a blood vessel and then connected to a syringe pump. Similar to hemodialysis, the tip of the microdialysis catheter will allow for solute exchange across a permeable membrane. Upon equilibration, the concentration of markers of cell metabolism and hypoxia (lactate, glutamate, glucose, glycerol, pyruvate, urea) in the dialysate reflect that of the interstitium. Other biomarkers such as cytokines or biomarkers of brain injury can also be retrieved, and this microdialysis sample better reflects their local concentration compared with systemic samples. Analyte concentration can then be measured on a benchtop machine. As for renal replacement therapy, membrane cutoff size impacts the nature of solutes retrieved in the dialysate [75]. This technology is currently being used in both clinical and research settings. The clinical use of microdialysis is especially relevant for neurointensive care patients. A microdialysis probe can be inserted into the spinal cord [76] or the brain [75]. Brain perfusion monitoring via microdialysis has been recommended as part of the management of people with traumatic brain injury and subdural hematoma [77]. In the laboratory, the authors have used microdialysis in various tissues, such as the kidney or liver. Furthermore, placing the microdialysis catheter in a large vessel such as the caudal vena cava allows for frequent evaluation of plasma lactate concentration without the need to remove blood from the animal.

Conclusion

Care providers have a wide range of tools available to guide resuscitation. Optimizing tissue oxygen delivery relies on normalizing arterial oxygen content and tissue perfusion.

Owing to the limitations of markers of macrocirculation, there are ongoing efforts to refine advanced techniques to make them more suitable for clinical practice. Continuing research efforts often focus on perfusion to a specific organ (kidney, brain, heart), which may not always reflect global perfusion. Acute care providers should therefore employ a diverse range of monitoring tools to improve patient care.

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20

Cardiopulmonary Resuscitation

Sean D. Smarick

Cardiopulmonary arrest (CPA) is the sudden cessation of spontaneous and effective circulation and ventilation. It is the common pathway to death from any disease process. Cardiopulmonary resuscitation (CPR) is the treatment to establish effective perfusion to the heart and brain with the ultimate goal of returning the patient to a normal life. Every small-animal veterinary practice, from a vaccination clinic to a multispecialty referral hospital, should have systems in place to address CPA. No practice is exempt from experiencing patients in CPA; however, a recent survey indicates that the awareness and the incorporation of veterinary CPR guidelines is lacking in primary care practices [1]. Considering the expectation and therefore potential fallout to a primary care practice that experiences a CPA for a "routine" procedure and that successful resuscitation are overrepresented in, for example, elective surgeries and allergic reactions, every practice should be prepared to perform CPR [2–5].

Preparing for a Cardiopulmonary Arrest

Studies regarding preparation for CPR have given credence to the rules of the 5 P's – "proper preparation prevents poor performance" – as preparation does affect outcome. Appropriate equipment and drugs must be available, and just as importantly, staff members at all levels must be adequately trained to fulfill their roles during an arrest event.

The Reassessment Campaign on Veterinary Resuscitation (RECOVER) initiative has developed guidelines for veterinary CPR. These guidelines are the collaborative product of over a hundred specialists modeled after human CPR guidelines. The RECOVER Guidelines serve as a primary reference for veterinary CPR; they are available open access on the RECOVER website [6].

Staff Training in Preparation for Cardiopulmonary Arrest

A CPR training program includes both didactic instruction and hands-on skill development. The RECOVER initiative offers training and certification based on the evidence-based guidelines it has developed.

Staff members should understand their potential roles on the CPR team (to perhaps include initial leader), understand closed loop communication, and should be trained appropriately. From initial hire orientation, the individual team member can become familiar with the CPR systems in place. Having a new technician participate in completing the checklist and subsequent supply stocking may help develop familiarity with the location of the practice's drugs and equipment. Without regular training every few months, skills diminish; thus, drills using commercial pet resuscitation mannequins or even simply a toy stuffed animal give the team the opportunity to maintain its resuscitation skills. Debriefing after training or an actual CPR event is very valuable [7].

Notifying the Staff of Cardiopulmonary Arrest

The veterinary care team must be alerted immediately when a CPA occurs. Therefore, each practice should have in place a system that notifies the team of a CPA event. Notification can be in the form of an overhead page or an internal audible alarm. Either or both should be predetermined and universally recognized by the hospital team. Ideally, the signal chosen is one that does not alarm clients; ubiquitous terms such as "code blue" strike fear into every client whose pet is not with them. Once a CPA is suspected, the system is activated, and the resuscitation team reports immediately to the predetermined resuscitation area or the location where the CPA has taken place. The ideal size of the resuscitation team in veterinary

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medicine has yet to be determined as conflicting data from academic institutions exist [4, 5]. While it may impair performance to have too many people present, having too few rescuers results in resuscitator fatigue, lack of efficient execution, and inadequate record keeping and client communication.

Equipment and Drugs Required

Equipment and drugs used in CPR should be readily available. A tackle box with CPR supplies kept in a consistent place, usually near the surgery suites or treatment area, is the minimum recommended for day practices that do not routinely see emergency cases. A list for basic resuscitation supplies can be found in Box 20.1. Emergency hospitals usually designate a central area for all CPR supplies, often with a multidrawer resuscitation (or "crash") cart, oxygen, and suction readily available.

Resuscitation boxes and carts are available from medical suppliers; however, tackle boxes and tool carts can provide alternatives (Figure 20.1). Some practices maintain mechanical means to seal the crash box or cart to ensure its integrity, whereas others incorporate CPR supplies with those used in intravenous (IV) access, treatments, or anesthetic inductions to maintain familiarity and maximize efficiency of space and resources [7, 8].

A checklist is necessary to ensure the crash cart or box and other related equipment are adequately stocked and in working order. The checklist and any necessary restocking should be performed as personnel begin and end shifts, and after each resuscitation (Chapter 4). Specific considerations for CPR-related supplies follow.

Items Used During Cardiac Compressions to Enhance Blood Circulation

While the current paradigm dictates starting compressions as soon as a CPA is suspected, no specialized equipment is needed to provide closed-chest compressions; however, open-chest cardiac massage requires an emergency thoracotomy. Equipment ranging from a pair of mayo scissors to a complete thoracotomy pack can be considered. See Chapter 19 for more information. IV fluids are only warranted in patients suffering a CPA with underlying hypovolemia [7]; nevertheless, having IV fluids and administration set(s) accessible is ideal. Impedance threshold devices continue to be evaluated in veterinary CPR and are a consideration in dogs over 10 kg [9–11].

Items Required to Secure the Airway and Provide Positive Pressure Ventilation

Laryngoscopes, cuffed endotracheal tubes of various sizes, 3- and 6-ml syringes to inflate tube cuffs, ties to secure endotracheal tubes, and a suction system are recommended to gain control of the airway; however, if a practice does not routinely intubate or intubation cannot be performed, tight-fitting face masks are an alternative [12]. Lidocaine in an atomizer or needleless syringe is often needed for cats with laryngospasm, even in CPA situations. The suction system may range from a bulb syringe to a central suction outlet with a collection bottle, tubing, and Yankauer tip. A tracheostomy or cricothyroidotomy pack or kit (Chapter 29) may also be a consideration to address true upper airway obstructions.

Box 20.1 Basic Supplies for Cardiopulmonary Resuscitation

Ventilation:

- Laryngoscope with blades and endotracheal tubes and/or tight-fitting face masks
- Muzzle gauze or tubing to secure endotracheal tubes
- Cuff inflation syringe(s)
- Suction device
- Lidocaine 2% solution in an atomizer or needleless syringe
- Bag–valve (adult, pediatric, neonatal) or other equipment that can provide positive pressure ventilation
- Oxygen source

Monitoring:

- electrocardiograph with leads attached
- Electrode conductive gel
- End-tidal CO_2 monitor (capnometer) with airway adapters

Intravenous (IV) access:

- IV and intraosseous access supplies
- IV fluids and administration sets
- IV flush (0.9% saline)

Drugs:

- Emergency drugs
  - Atropine
  - Epinephrine ± vasopressin
  - Sodium bicarbonate
  - Amiodarone or lidocaine
- Syringes (1–6 ml) and needles
- Anesthetic reversals
  - Naloxone
  - Atipamezole
  - Flumazenil

Defibrillator

Reference and records:

- CPR algorithm chart
- Drug chart
- Post-arrest algorithm chart
- CPR patient record



Figure 20.1 "Crash" or resuscitation cart. Equipment and drugs used in cardiopulmonary resuscitation should be kept together in a readily available, standard place. They can be easily stored in a tool chest or a purpose-specific box.

Positive pressure ventilation is generally performed during in-hospital CPR; it can be provided by an adult or pediatric bag–valve–mask (without the mask), a nonrebreathing circuit, or an anesthetic machine connected to an oxygen source [7, 13].

Monitoring

Beyond basic life support of compressions and ventilation. an electrocardiogram (ECG) is required to assess the cardiac arrest rhythm to determine specific CPR treatments (e.g. drugs, need for defibrillation). With the wide range of ECGs available at many price points (including refurbished ones) and the fact an ECG is an important monitoring tool for anesthesia, it is reasonable that every practice should consider having one. Electrode gel is needed to obtain a diagnostic ECG, while *isopropyl alcohol is avoided* during CPR due to the explosion hazard in the presence of an electric defibrillator.

Post-apneic end-tidal carbon dioxide ($PetCO_2$) measured by a capnometer has proven to correlate with circulation during CPR and there is no substitute to evaluate the effectiveness of chest compressions short of return of spontaneous circulation (ROSC). As with ECGs, capnometers can be purchased for a reasonable price on the used human medical supply market; the fact that capnometers are also valuable monitors of anesthesia makes acquiring one reasonable for most practices [7, 14].

Intravenous Access and Drugs

To administer drugs in CPR, vascular access is achieved through cephalic IV catheter, jugular catheter, or intraosseous (commercial, spinal, or hypodermic) needle placement. Cutdown supplies (i.e. scalpel blade, Kelly or mosquito hemostats, and suture) are considerations for trained providers (Chapter 7).

If vascular access has failed, it is reasonable to deliver drugs via a long, flexible catheter (e.g. red rubber tube, infant feeding tube) threaded down the endotracheal tube past the carina to deliver increased dosages of mediations that are diluted in sterile water for injection. Naloxone, atropine, vasopressin, epinephrine, and lidocaine (referred to by the acronym NAVEL) can all be administered via this route [7, 9]. In general, when drugs are given intratracheally, one may consider the "three Ds": double the IV dose, dilute in a large volume of sterile water, and deliver at the carina.

Appropriately sized syringes, needles, and perhaps sterile water for injection should be stocked with the drugs.

Emergency Drugs Injectable atropine, and a vasopressor (epinephrine and/or vasopressin) are the basic drugs used in asystole and pulseless electrical activity, which are common rhythms encountered in veterinary CPA. A vasopressor, and possibly amiodarone or lidocaine, may be useful in ventricular fibrillation or pulseless ventricular tachycardia; however, they are only adjuvants and not substitutes for electrical defibrillation. Sodium bicarbonate is used in cases of prolonged arrest, pre-existing acidemia, and hyperkalemia [7, 9].

Anesthetic Reversals Anesthetic reversals such as naloxone, atipamezole, and flumazenil are recommended if narcotics, alpha-2 agonists, and benzodiazepines are used in the practice, respectively [7, 9].

Ventricular Fibrillation/Pulseless Ventricular Tachycardia Treatment

The only consistently effective treatment for pulseless ventricular arrythmias is electrical defibrillation; therefore, serious consideration should be made to acquire a defibrillator. There are two basic types of electrical defibrillators: monophasic and biphasic, which refers to the pattern of energy delivery between the paddles. Biphasic defibrillators require less energy than monophasic ones, which means

less myocardial damage during defibrillation. Biphasic defibrillators are available on the veterinary market, and refurbished human models can be purchased online. Monophasic defibrillators continue to be available primarily on the resale market. Defibrillation units usually include an ECG monitor, which helps justify their cost.

The unit should remain connected to an electrical outlet to keep the internal battery charged. Electrode gel is required as a coupling substance between the paddles and the patient; as mentioned previously, isopropyl alcohol and other chemicals are strictly avoided because they can create an explosion hazard [7, 9]. See Chapter 22 for more information.

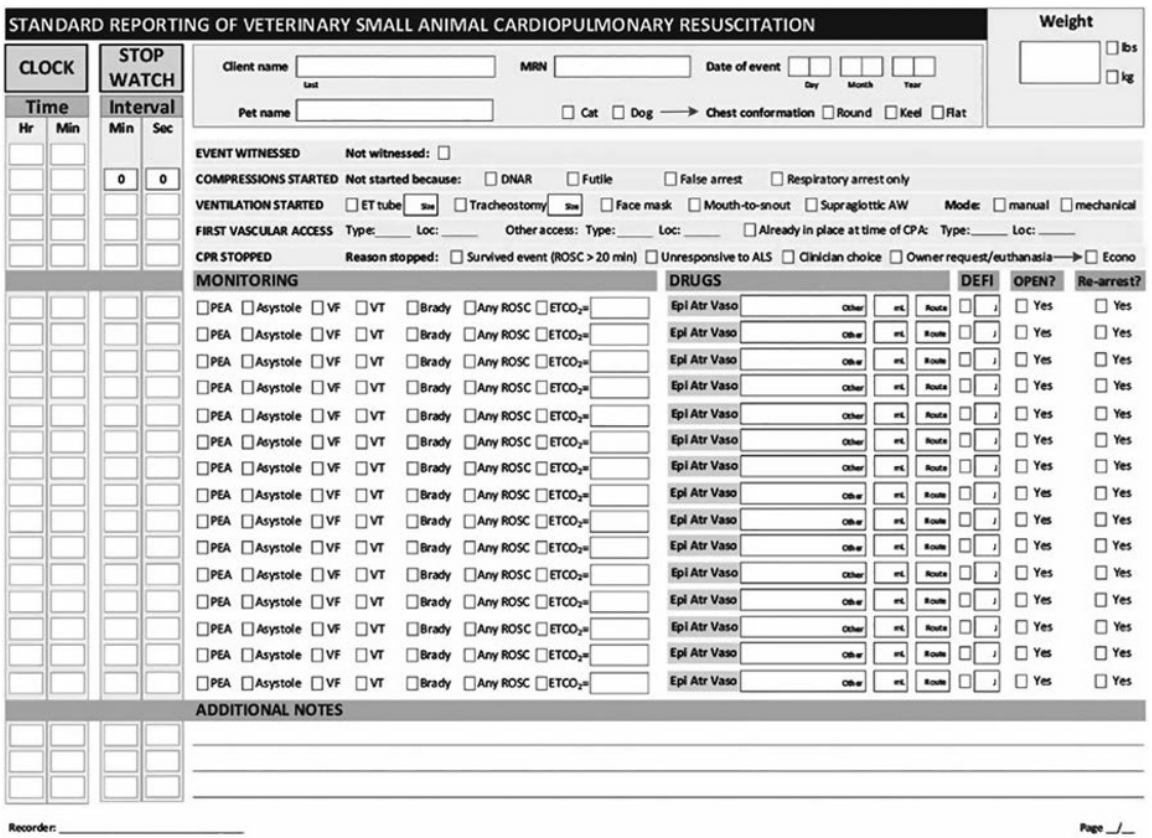
References and Records

Copies of the RECOVER algorithms and drugs charts should be readily available to reference during a CPA. The algorithms act as a checklist even for the most experienced clinician. Drugs charts provide for the efficient and accurate administration of drug and defibrillation dosages. They can be downloaded from the RECOVER website or wall size ones can be purchased at the Veterinary Emergency and Critical Care Society website. A dedicated recording sheet allows for CPR treatment documentation during real time and can also be used to accumulate data for research (Figure 20.2) [7, 15].

Post-Cardiac Arrest Care

Immediate and prolonged critical care of the reanimated patient is crucial to avoid rearrest and to maximize the potential for a good (neurological) outcome. Maintaining adequate ventilation, oxygenation, and blood pressure is paramount. Monitoring blood pressure directly with a transducer system or indirectly with a Doppler or oscillometric monitor allows titration of pressors such as norepinephrine, vasopressin, or epinephrine. Dysrhythmias, which are often encountered after ROSC, can compromise perfusion and further tax the myocardium, so continuous ECG monitoring is warranted, together with appropriate antiarrhythmic use. Oxygen supplementation may be needed to maintain normoxemia, monitored by arterial blood gas analysis or pulse oximetry. Normocapnia, assessed by arterial or central venous blood gas analysis or capnometry (measuring $PetCO_2$), is maintained as needed with PPV. A critical-care ventilator is ideal in this situation; however, anesthetic ventilators and "hand bagging" (manual inflation using a bag–valve, Bain's circuit, or anesthetic machine and circle system) may be adequate.

Lastly, as every organ system will have suffered some degree of ischemia and secondary reperfusion injury, multiple organ dysfunction syndrome should be anticipated


STANDARD REPORTING OF VETERINARY SMALL ANIMAL CARDIOPULMONARY RESUSCITATION

Weight ☐ lbs ☐ kg

CLOCK | STOP WATCH

Time: Hr Min | Interval: Min Sec

Client name (Last) | MRN | Date of event (Day Month Year)

Pet name | ☐ Cat ☐ Dog → Chest conformation ☐ Round ☐ Keel ☐ Flat

EVENT WITNESSED — Not witnessed: ☐

COMPRESSIONS STARTED — Not started because: ☐ DNAR ☐ Futile ☐ False arrest ☐ Respiratory arrest only

VENTILATION STARTED — ☐ ET tube Size ☐ Tracheostomy Size ☐ Face mask ☐ Mouth-to-snout ☐ Supraglottic AW — Mode: ☐ manual ☐ mechanical

FIRST VASCULAR ACCESS — Type:_____ Loc:_____ Other access: Type:_____ Loc:_____ ☐ Already in place at time of CPA: Type:_____ Loc:_____

CPR STOPPED — Reason stopped: ☐ Survived event (ROSC > 20 min) ☐ Unresponsive to ALS ☐ Clinician choice ☐ Owner request/euthanasia → ☐ Econo

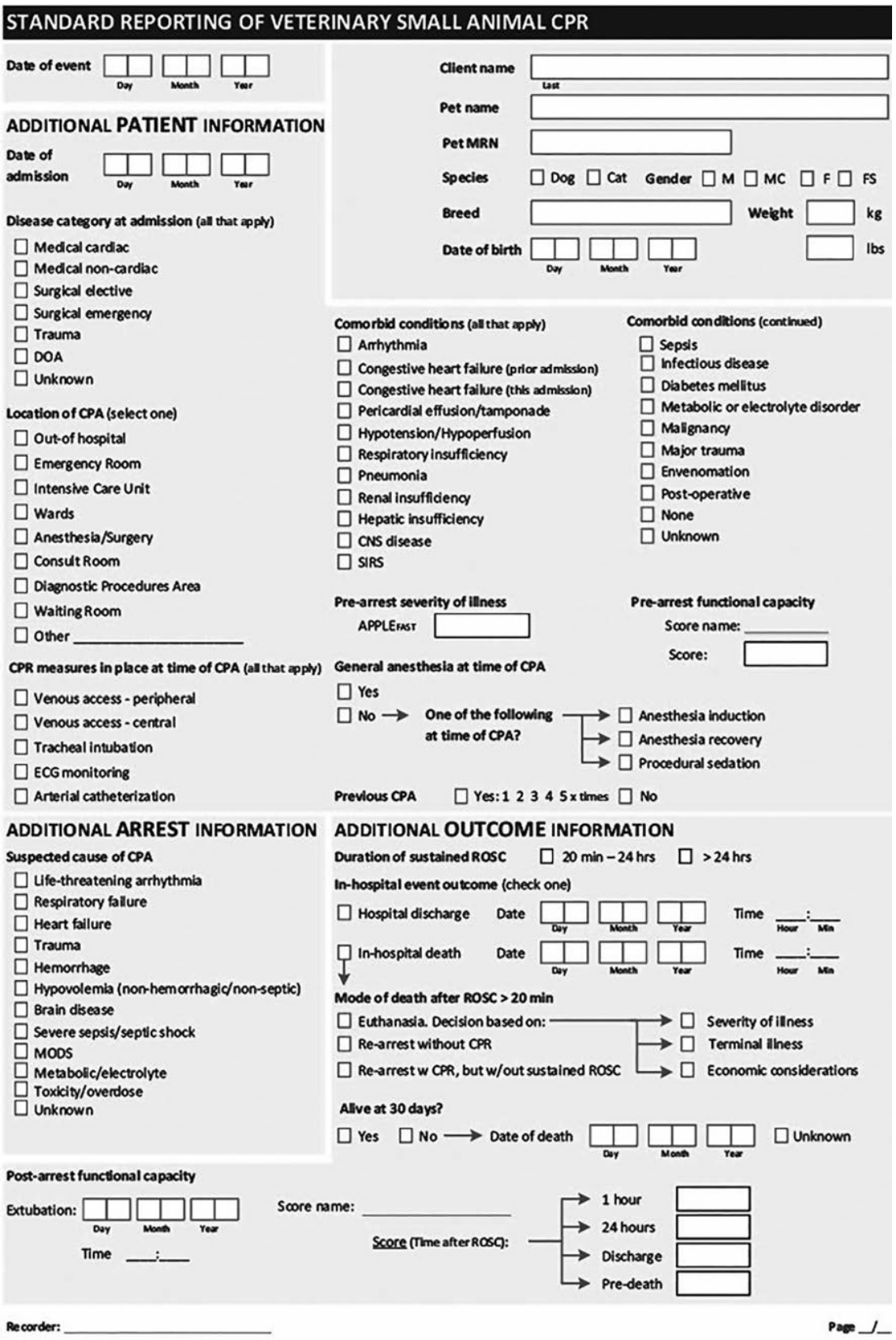
MONITORING	DRUGS	DEFI	OPEN?	Re-arrest?
☐PEA ☐Asystole ☐VF ☐VT ☐Brady ☐Any ROSC ☐$ETCO_2$=	Epi Atr Vaso Other mL Route	☐ J	☐ Yes	☐ Yes

(repeated 20 rows)

ADDITIONAL NOTES

Recorder: _____ Page __/__

Figure 20.2 Standard Reporting Form for cardiopulmonary resuscitation. *Source:* Reprinted with permission from Boller et al. [15].



STANDARD REPORTING OF VETERINARY SMALL ANIMAL CPR

Date of event ☐☐ Day ☐☐ Month ☐☐ Year

Client name ____ Last

Pet name ____

Pet MRN ____

Species ☐ Dog ☐ Cat Gender ☐ M ☐ MC ☐ F ☐ FS

Breed ____ Weight ____ kg ____ lbs

Date of birth ☐☐ Day ☐☐ Month ☐☐ Year

ADDITIONAL PATIENT INFORMATION

Date of admission ☐☐ Day ☐☐ Month ☐☐ Year

Disease category at admission (all that apply)
- ☐ Medical cardiac
- ☐ Medical non-cardiac
- ☐ Surgical elective
- ☐ Surgical emergency
- ☐ Trauma
- ☐ DOA
- ☐ Unknown

Location of CPA (select one)
- ☐ Out-of hospital
- ☐ Emergency Room
- ☐ Intensive Care Unit
- ☐ Wards
- ☐ Anesthesia/Surgery
- ☐ Consult Room
- ☐ Diagnostic Procedures Area
- ☐ Waiting Room
- ☐ Other _____

CPR measures in place at time of CPA (all that apply)
- ☐ Venous access - peripheral
- ☐ Venous access - central
- ☐ Tracheal intubation
- ☐ ECG monitoring
- ☐ Arterial catheterization

Comorbid conditions (all that apply)
- ☐ Arrhythmia
- ☐ Congestive heart failure (prior admission)
- ☐ Congestive heart failure (this admission)
- ☐ Pericardial effusion/tamponade
- ☐ Hypotension/Hypoperfusion
- ☐ Respiratory insufficiency
- ☐ Pneumonia
- ☐ Renal insufficiency
- ☐ Hepatic insufficiency
- ☐ CNS disease
- ☐ SIRS

Comorbid conditions (continued)
- ☐ Sepsis
- ☐ Infectious disease
- ☐ Diabetes mellitus
- ☐ Metabolic or electrolyte disorder
- ☐ Malignancy
- ☐ Major trauma
- ☐ Envenomation
- ☐ Post-operative
- ☐ None
- ☐ Unknown

Pre-arrest severity of illness
APPLE FAST ____

Pre-arrest functional capacity
Score name: _____
Score: ____

General anesthesia at time of CPA
- ☐ Yes
- ☐ No → One of the following at time of CPA?
  - ☐ Anesthesia induction
  - ☐ Anesthesia recovery
  - ☐ Procedural sedation

Previous CPA ☐ Yes: 1 2 3 4 5 x times ☐ No

ADDITIONAL ARREST INFORMATION

Suspected cause of CPA
- ☐ Life-threatening arrhythmia
- ☐ Respiratory failure
- ☐ Heart failure
- ☐ Trauma
- ☐ Hemorrhage
- ☐ Hypovolemia (non-hemorrhagic/non-septic)
- ☐ Brain disease
- ☐ Severe sepsis/septic shock
- ☐ MODS
- ☐ Metabolic/electrolyte
- ☐ Toxicity/overdose
- ☐ Unknown

ADDITIONAL OUTCOME INFORMATION

Duration of sustained ROSC ☐ 20 min – 24 hrs ☐ > 24 hrs

In-hospital event outcome (check one)
- ☐ Hospital discharge Date ☐☐ Day ☐☐ Month ☐☐ Year Time ___:___ Hour Min
- ☐ In-hospital death Date ☐☐ Day ☐☐ Month ☐☐ Year Time ___:___ Hour Min

Mode of death after ROSC > 20 min
- ☐ Euthanasia. Decision based on:
  - ☐ Severity of illness
  - ☐ Terminal illness
  - ☐ Economic considerations
- ☐ Re-arrest without CPR
- ☐ Re-arrest w CPR, but w/out sustained ROSC

Alive at 30 days?
☐ Yes ☐ No → Date of death ☐☐ Day ☐☐ Month ☐☐ Year ☐ Unknown

Post-arrest functional capacity

Extubation: ☐☐ Day ☐☐ Month ☐☐ Year
Time ___:___

Score name: _____

Score (Time after ROSC):
- 1 hour ____
- 24 hours ____
- Discharge ____
- Pre-death ____

Recorder: _____ Page __/__

Figure 20.2 (*Continued*)

and addressed as needed with appropriate monitoring and treatment beyond the scope of this chapter [7, 16]. Because of the frequency and severity of post-resuscitation complications, hospitalization at a 24-hour emergency and specialty facility is recommended.

Recognizing Cardiopulmonary Arrest

Early recognition of a CPA and institution of resuscitative efforts are paramount to a successful outcome. The decision to initiate CPR will often fall on the veterinary technician caring for a pet in the intensive care unit, in the surgical suite area, or during triage. Depending on the individual state board rules, standing orders should be developed by the veterinarians in the practice so that veterinary technicians can initiate CPR and lead the effort until a veterinarian is available to direct the resuscitation effort.

The veterinary technician should be vigilant in monitoring anesthetized patients and those that are critically ill for signs of impending arrest and should be proficient in triage to recognize patients presenting to the practice with immediately life-threatening signs. The best treatment for CPA is not CPR but rather prevention of the CPA in the first place.

Once a CPA is suspected, CPR is instituted immediately by the closest available caregiver unless a do not attempt resuscitation order is in place. The caregivers should be aware of the desires of the pet owner regarding advance resuscitative directives (i.e. "code status") of hospitalized pets. Code status should be communicated in patient rounds and should appear in a predetermined, consistent, and standardized fashion in the patient's record, treatment orders, cage card, and/or identity collar.

Patients that collapse, lose consciousness, or have absent spontaneous respirations (i.e. no chest movement) have signs that suggest CPA. *All patients undergoing CPA experience these signs*, but not all patients with these signs are necessarily dying. That being said, chest compressions are immediately indicated if evaluation over a period of no longer than 10 seconds reveals the animal is unconscious and making no attempts to breathe.

In the anesthetized or critically ill patient, monitoring equipment provides useful information in a nonresponsive, perhaps not spontaneously breathing patient. Esophageal stethoscopes, Doppler blood pressure flow detectors, and multiparameter monitors that measure and report vascular pressures, pulse oximetry, $PetCO_2$, and ECG are invaluable in the recognition of an impending arrest.

The esophageal stethoscope is a cost-effective tool that can alert the anesthetist to real-time changes in the apex beat's rate, rhythm, or intensity; a Doppler blood pressure flow detector does the same for peripheral pulses and allows for the determination of systolic blood pressure. Precipitously decreasing heart rates, pulse intensity, severe tachyarrhythmias or bradyarrhythmias, or hypotension can all lead to CPA. An ECG provides more detailed information regarding the heart's rate and rhythm and is a valuable tool for rhythm evaluation. Pulse oximetry has technical challenges but any changes in the waveform or oxygen saturation should be investigated rather than assumed to be a false alarm. Precipitously decreasing $PetCO_2$ values or a downward stair-stepping capnogram can signal an impending CPA (Chapter 30). While not every alarm or abnormality signals an impending arrest, the integration of available information into an accurate clinical picture can help prevent CPA or give the caregiving team some warning that CPR will be required soon.

Initiating Basic Life Support

Chest compressions should be initiated immediately when any caregiver recognizes unconsciousness combined with apnea; the decision to start compressions based on these clinical findings should be made and compressions initiated within 10 seconds. While assessing and starting compressions, this caregiver signals the team with the predetermined, universally understood signal. The team may include nontraditional caregivers such as receptionists and kennel or maintenance personnel, who can be invaluable by assisting in the resuscitation or record keeping. As stated previously, these individuals should not only be acquainted with their roles prior to being called upon, but also have ample practice and be proficient in the tasks they are being asked to carry out.

Each member of the resuscitation team plays a key role in executing CPR. Many of the skills and much of the knowledge are general nursing skills and not specific to CPR, such as endotracheal intubation; however, there are caveats to those basic skills and specific ones to resuscitation that cumulate into effective CPR.

Ideally, interventions are executed simultaneously but if activities must be prioritized, they are in this order [7, 13].

Compressions

Once the diagnosis of pulselessness is established, circulation must be established and maintained until the underlying cause of the CPA is addressed and ROSC is obtained. The primary goal of this assisted circulation is to support the heart and brain, and external chest compressions are initiated immediately.

Closed-chest CPR is theorized to propel blood forward by the cardiac pump and the thoracic pump models. The cardiac pump theory states that blood circulates during chest

compressions due to direct compression of the ventricles with intact atrioventricular valves preventing retrograde flow. The thoracic pump theory states that blood circulates during external chest compressions because compression increases intrathoracic pressure, which results in a pressure gradient from the thin walled and valved veins to the thick-walled arteries; release of compression leads to venous refilling. Both theories are probably at work, but the cardiac pump is suspected to predominate in smaller animals or in keel shaped chests. In such patients, hand placement is over the heart (fifth–sixth intercostal space, just caudal to the elbow) in lateral recumbency or circumferentially around the thorax. In patients with larger and barrel shaped chests, the hands are probably best placed over the widest portion of the thorax in lateral recumbency or over the caudal sternum in dorsal recumbency (be careful to avoid the xyphoid).

Compressions are performed at a rate of at least 100 compressions/minute, decreasing the thoracic diameter by approximately one-third, and maintaining a ratio ("duty ratio") of 1 : 1 for compression and relaxation. During the non-compression phase, all pressure should be released from the thorax to allow for low intrathoracic pressures and thus venous return to the right heart. It is imperative that chest compressions are not interrupted for greater than 10 seconds as coronary perfusion pressures return to zero and take minutes to return. This lag time from compressions to a perfusing coronary and cerebral perfusion pressure is justification to start CPR with compressions.

If closed-chest CPR is not effective (see below under monitors), several alternatives can be employed. Changing compressors, altering placement of hands, varying the compression rate, and varying compression depth may offer some benefit. Interposed abdominal compressions, which are performed midway between the umbilicus and xyphoid to generate a pressure of 100 mmHg, are reasonable if trained personnel are available. If the patient's size and conformation are amenable, you can place one hand on the chest and one hand on the belly; interposed abdominal compressions are then accomplished by alternating compressions between the left and right arm [7, 13].

In veterinary and human medicine, debate continues regarding the use and timing of open-chest compressions. If there is a chest-wall defect or loss of compliance, penetrating thoracic trauma, cardiac tamponade, or pleural space disease, an emergency thoracotomy is recommended. See Chapter 21 for a discussion of open-chest CPR.

Providing Ventilation

Simultaneous to starting compressions, an airway should be established with a cuffed endotracheal tube (Chapter 28); extending the neck (in the absence of suspected cervical injuries) and pulling the tongue rostrally may open a closed airway and allow for spontaneous breathing. Suction any material from the caudal pharyngeal or laryngeal area. The endotracheal tube must be secured to ensure the airway remains patent and to minimize tracheal trauma.

While the endotracheal tube is in place, any time the patient is moved, and intermittently during the resuscitation, tube placement should be confirmed. This is accomplished by (bilateral) thoracic and stomach auscultation to ensure that breath sounds and not bubbling are heard, by visualization of the tube traveling through the arytenoids, by direct palpation of tissue (larynx) around the tube 360 degrees, and supported by capnometry with measurement of some carbon dioxide.

Once the endotracheal tube is in place, it is connected to a bag–valve or an anesthetic circuit with bag reservoir. The bag is squeezed to provide a breath and is then allowed to recoil completely to avoid positive end-expiratory pressure; expiration occurs passively and should occur without resistance. When using an anesthetic machine, before attaching the system to the patient's endotracheal tube, the circuit must be flushed of any anesthetic gas by depressing the oxygen flush valve and squeezing the reservoir bag into an open pop-off valve. The expiratory pop-off valve is then closed to generate a good breath using the reservoir bag, then opened again to avoid excessive gas accumulation in the system and high pressure in the airways. The opening and closing of the pop-off valve must be repeated to generate breaths. When using a Bain's nonrebreathing circuit, the tip of the reservoir bag must be occluded as with the pop-off valve.

Oxygen flow is required to provide a volume of gas in the reservoir bag. Even the standard "semi-closed" anesthetic circuit, when used as described above, requires relatively high fresh gas flow rates to fill the reservoir bag such that fresh gas is available for each breath. Bain's and other non-rebreathing circuits generally require fresh gas flows twice the minute volume (200–250 ml/kg/minute) to avoid rebreathing.

Bag–valve systems can be used with just room air; while there is some consideration given to detrimental effects of hyperoxia during CPR, the recommendation is to still supplement oxygen as per the manufacturer and models recommendations, especially if hypoxia has contributed to the arrest.

PPV should be performed at a rate of 10 breaths/minute, a tidal volume of 10 ml/kg and a short inspiratory period of one second, as more aggressive ventilation has negative effects on coronary perfusion pressure, cardiac output, and survival. Ventilation should be provided in this pattern without regard for compressions; compressions are not paused for ventilation.

While pharmacologic respiratory stimulants are avoided, the acupuncture GV26 site has been reported to increase respiratory rates in CPA and is stimulated by placing an approximately 25-gauge needle into the nasal philtrum [17].

Cardiopulmonary Resuscitation Basic Life Support Cycle

Two minutes of chest compressions completes a cycle. If a caregiver is a sole rescuer, two breaths are given at the end of the two-minute cycle rather than breaths being delivered every six seconds throughout. At the completion of each two-minute cycle, advanced life support interventions are considered, and chest compressors are exchanged to prevent fatigue. During this exchange between cycles, compressions are interrupted for no more than 10 seconds.

Advanced Life Support

Ideally simultaneous to instituting basic life support, advanced interventions are initiated to include attaching monitors, namely ECG and a capnometer, obtaining vascular access, and administering reversal agents for pertinent anesthetics or sedatives on board. ECG evaluation leads to rhythm-specific interventions consisting of drug administration and/or electrical defibrillations.

Monitors

Pulse checks and pupil size do not offer dependable feedback to the quality or effectiveness of CPR in dogs and cats. While direct arterial pressure monitoring with an indwelling arterial line with a diastolic arterial pressure greater than 30 mmHg is associated with ROSC, having this in place is rare. A capnometer to measure $PetCO_2$ is a simple and invaluable monitor that attaches to the endotracheal tube and provides real-time feedback regarding effectiveness of chest compressions. The presence of CO_2 in exhaled gas requires the delivery of oxygen from the lungs to tissues, respiration at the level of the cells, then return of CO_2 to the lungs that is subsequently ventilated to the detector. Poor chest compression technique leads to poor blood flow to and from tissues, and subsequently lower $PetCO_2$. Thus, when $PetCO_2$ is less than 15 mmHg during a CPR event, every effort should be made to improve chest compression technique by altering hand position, optimizing rate, ensuring adequate compression depth and full chest recoil between compressions, and minimizing hands-off time. End-tidal expired CO_2 tensions exceeding 15–18 mmHg increase the likelihood of ROSC and survival in veterinary and human studies. When using $PetCO_2$to monitor the efficacy of CPR, the team must provide a steady breathing rate because alterations in breathing rate affect PetCO2; be aware that administration of bicarbonate will increase $PetCO_2$; and note that pressor administration will cause the $PetCO_2$ to drop. A dramatic rise in $PetCO_2$ often reflects ROSC as the increased concentrations of carbon dioxide are washed out from the newly perfused venous system [7, 14, 18].

Electrocardiogram evaluation is needed to decide on appropriate interventions based on cardiac rhythm, so ECG leads are connected prior to completion of the first cycle, avoiding alcohol as a conductor in the presence of a defibrillator.

Vascular Access

Vascular access is obtained in anticipation of administering drugs. In CPR, drugs are ideally delivered by the central intravenous route; however, the intraosseous or cranial peripheral intravenous routes (such as the cephalic veins) are acceptable as well. Intraosseous needles can be placed into the proximal humeral condyle, medial proximal tibia, or the proximal lateral femur; the trochanteric fossa of the femur can be used in neonates. If vascular access cannot be obtained, the intratracheal route can be used. The desire for central/cranial vascular access must be weighed against the vascular access present at the time of a CPA and the team's experience in gaining access through cutdown and intraosseous methods. While flushing 5–20 ml of 0.9% saline after an intravenous administration can be helpful in getting the drug distributed, routine fluid administration should only be considered in known cases of hypovolemia as fluid administration in normovolemic CPA is associated with worse outcomes.

Intratracheal administration can be used for NAVEL. For intratracheal administration, drug doses should be at least doubled (and in the case of epinephrine, the "high dose" used); suspended in 5–10 ml of sterile water for injection (preferable) or 0.9% NaCl; injected via long red rubber or polypropylene catheter to the level of the carina; and followed with two breaths [7, 9].

Anesthetic or Sedative Drug Reversal

Anesthetic or sedative drugs that may have contributed to or are in effect during a CPA should be reversed. For example, naloxone should be administered when a narcotic has been used, flumazenil with a benzodiazepine, and atipamezole with an alpha-2 agonist. As discussed under ventilation, inhalant anesthetic gas should have already been flushed from the system if one was in use at the time of CPA [7, 9].

Electrocardiogram and Patient Evaluation

As discussed above, compressors are changed after a two-minute basic life support cycle while the ECG is evaluated for a brief (<10 seconds) period. The protocols for treating the

bradyarrhythmias (pulseless electrical activity or asystole) differ from those used to treat ventricular fibrillation and pulseless ventricular tachycardia. Thus, the ECG is invaluable in distinguishing these two arrest rhythm patient populations. It is important to remember that the CPA patient's ECG rhythm can change through the CPR effort. As an example, in a recent study, 15% of the dogs had a presenting ECG finding of ventricular fibrillation but over more than twice that number experienced ventricular fibrillation during the CPR event [5, 7].

Vasopressors

Compressions alone are unlikely to provide enough circulation to the heart to enable its return to function. A vasopressor is warranted in CPA to help increase coronary perfusion pressures to a level associated with ROSC, no matter the initial ECG rhythm.

Currently, epinephrine is still considered the first-line vasopressor and is administered at a dose of 0.01–0.02 mg/kg, repeated every three to five minutes. "High-dose" (0.1–0.2 mg/kg) epinephrine has not been shown to be superior (except in intratracheal administration or in prolonged CPR) over "low-dose" (0.01–0.02 mg/kg) epinephrine. For endotracheal administration, a higher dose is used to ensure high enough blood concentrations that vasoconstriction is achieved.

Vasopressin is an alternative to epinephrine in CPR, and it may be used once at 0.8 iu/kg as an alternative to epinephrine, either initially or in place of subsequent doses [7, 9].

Parasympatholytics

Atropine has been used regularly in CPR but its usefulness especially beyond cases with high vagal tone continues to be questioned. It is reasonable to administer atropine at a dose of 0.04 mg/kg IV every other cycle in cases of bradyarrhythmias (but not fibrillation) [7, 9].

Buffers

Sodium bicarbonate continues to be the buffer recommended in CPR for cases of pre-existing severe metabolic acidosis or after prolonged (> 10 minutes) of CPA. Additionally, alkalinization is suggested in hyperkalemia, a reversible cause of CPA. Currently, sodium bicarbonate is recommended at 1 mEq/kg slow IV, repeated every five minutes [7, 9].

Fibrillation Treatment

The definitive treatment for ventricular fibrillation is electrical defibrillation. A precordial thump can be attempted in the absence of a defibrillator, but electrical defibrillation is by far the most effective treatment with biphasic (energy flowing in two directions) more effective and less harmful than monophasic. After delivering the appropriate monophasic dose of 4–6 J/kg or biphasic dose of 2–4 J/kg, compressions are immediately resumed to feed the heart much needed oxygen and energy and the ECG is evaluated at the end of a complete two-minute cycle.

Antiarrhythmics have a very limited role during ventricular fibrillation. Amiodarone has replaced lidocaine as adjunctive therapy in people for shock-resistant ventricular fibrillation and the suggested veterinary dose is 5 mg/kg IV. If amiodarone is unavailable and shock-resistant ventricular fibrillation persists despite countershocks, lidocaine at 2 mg/kg IV may be considered.

Caregiver safety is paramount during defibrillation and is ensured by not using isopropyl alcohol for electrode conductivity (either for defibrillator or ECG electrode contact), observing for caregiver-patient contact prior to paddle discharge, and announcing the impending delivery of the shock to ensure all caregivers including the defibrillator is "CLEAR!" [7, 9].

For a more detailed discussion of defibrillation see Chapter 22.

Return of Spontaneous Circulation

ROSC should occur in one-third to one-half of all CPA patients, and while survivors tend to respond sooner, there are documented successful efforts exceeding 20 minutes in reversible causes of death in otherwise healthy patients. Veterinary studies have survival to discharge at 2–5%, so there is a large gap between the percentage of patients undergoing a CPA that experience ROSC and those that are discharged home. This gap represents an opportunity to improve post-cardiac arrest care [5, 7].

Post-Cardiac Arrest Care

Post-cardiac care should be addressed with the same urgency and intensity of CPR for a CPA; an algorithm is available from RECOVER to address this precarious time. Like CPR, ideally several interventions are occurring simultaneously, but if resources are limited, they are prioritized to normocapnia, normoxemia, normotension, and then neuroprotection.

Normocapnia requires evaluation of $PetCO_2$ or arterial blood gases and likely some degree of PPV for some time. Normoxemia can be evaluated with pulse oximetry or arterial blood gases and may require oxygen supplementation and PPV. In the face of a stunned heart and acid–base abnormalities, hypotension is likely to ensue after the CPR vasopressor is metabolized; monitoring blood pressure with vasopressors, fluids and inotropes at the ready is recommended. Pain, residual pressor, or brain dysfunction may result in hypertension and should be addressed if noted. Normalizing serum lactate and keeping the PCV greater than 25% rounds out hemodynamic optimization.

Neuroprotection is an area with ongoing research, but seizures are known to be detrimental and should be treated if observed. Hyperosmotic agents can be used to address known or highly suspected cases of increased intracranial pressure. Targeted temperature management is also a consideration. In a dedicated intensive care unit, induction of mild hypothermia at 32–34°C for 12–24 hours after ROSC is recommended, whereas active rewarming above these temperatures may best be avoided if referral is being considered.

Intensive care is needed to address the complex pathology in the post-resuscitation phase following ROSC. As for any patient with global ischemia and reperfusion, systemic inflammatory response syndrome, multiple organ dysfunction syndrome, or disseminated intravascular coagulation, the caregiver is faced with vigilant monitoring of and comprehensive care for every organ system to affect a successful outcome in providing CPR. If that care cannot be provided, referral should be considered but only after respiratory and hemodynamic optimization as above [7, 16].

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21

Open-Chest Cardiopulmonary Resuscitation

Janelle R. Wierenga and Katherine R. Crosse

Cardiopulmonary arrest (CPA) is the single pathway to death from any underlying disease and is common in small animal emergency and critical care medicine. Some underlying disease conditions are treatable in the acute CPA setting if they can be identified. Other disease conditions are not treatable or not identifiable in the acute setting, making resuscitation efforts from CPA difficult. Immediate cardiopulmonary resuscitation (CPR) is indicated in sudden cardiac arrest. Delay in initiation of cardiac compressions in CPR has been shown to decrease the likelihood of return of spontaneous circulation (ROSC). In human studies evaluating delay of CPR in CPA due to ventricular fibrillation, every minute of untreated ventricular fibrillation decreases survival by 7–10% [1]. Even with immediate CPR the chances of ROSC and survival to discharge are low in veterinary medicine at only 1–16% [2–6]. Survival rates depend on the underlying disease condition, the inciting cause of the arrest, and the resuscitation efforts and timing.

Indications for Open-Chest Cardiopulmonary Resuscitation

"Open-chest" or "internal" CPR (OCCPR) through an emergency thoracotomy was the standard of care for CPA in the early twentieth century [7]. Today, closed-chest CPR is generally instituted first unless specific situations or disease conditions exist that may be indications for immediate OCCPR [7, 8]. A full discussion of closed-chest CPR can be found in Chapter 20. Few veterinary studies have evaluated outcomes of OCCPR, usually due to low case numbers; however, the likelihood of survival to discharge with closed compared with OCCPR does not appear to be clearly different in these reports [2, 4–6, 9].

Any condition that prevents healthcare workers from achieving adequate perfusion to the lungs, heart, and brain through closed-chest CPR is a potential indication for OCCPR with direct cardiac massage. The 2020 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care state that OCCPR is recommended or may be considered for specific indications [10]. The guidelines recommend OCCPR in the case of intraoperative arrest during a laparotomy or thoracotomy procedure, or in the case of CPA shortly following a cardiothoracic surgery (class IIa, level of evidence C), and previous guidelines state that it may be useful in some cases of CPA secondary to penetrating trauma (class IIb, level of evidence C) [11]. The emergency room thoracotomy, also called resuscitative thoracotomy, is recommended in the most recent guidelines for human trauma patients: (i) with signs of life (pupillary response, spontaneous ventilation, presence of a (carotid) pulse, measurable or palpable blood pressure, extremity movement, or cardiac electrical activity) and penetrating trauma (strong recommendation); (ii) with no signs of life and penetrating trauma (conditional recommendation); (iii) with and without signs of life and penetrating trauma in extrathoracic regions (conditional recommendation); (iv) with signs of life and blunt trauma (conditional recommendation) [12]. Similarly, the recommendations from the 2012 Reassessment Campaign on Veterinary Resuscitation guidelines state that OCCPR should be considered for dogs and cats "in cases of significant intrathoracic disease, such as tension pneumothorax or pericardial effusion" (class IIB, level of evidence C) though specific situations such as blunt or penetrating trauma or massive caudal hemorrhage are unknown at this time [13, 14].

After closed-chest CPR has been chosen as the first method of CPR, there is controversy regarding whether and when one should abandon closed-chest efforts and proceed to OCCPR. Some literature recommends initiating OCCPR if there is no response to external chest

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Box 21.1 Possible Indications for Open-Chest Cardiopulmonary Resuscitation

- Failure of closed-chest cardiopulmonary resuscitation efforts
- Pericardial effusion
- Pleural space disease
  - Pneumothorax
  - Moderate to severe pleural effusion
  - Diaphragmatic hernia
- Thoracic wall trauma
  - Rib fractures
  - Penetrating thoracic injuries

compressions within two to five minutes [8, 15–19], while others allow 5–10 minutes of closed-chest CPR before converting to OCCPR [20, 21]. There is a consensus, however, that if the healthcare team is going to perform OCCPR, it should be initiated relatively early in the resuscitation effort. When instituted after 20 minutes of closed-chest compressions, internal cardiac massage does not increase the chances of ROSC [21–23]. When OCCPR is indicated in blunt or penetrating trauma in people, it is recommended that resuscitative thoracotomy be performed within 10 or 15 minutes, respectively [24]. It has been suggested that inadequate forward blood flow during closed-chest efforts is more likely to occur in animals over 20 kg, in which the thoracic pump mechanism probably predominates (see Chapter 20) [25, 26]. Situations in which OCCPR may be indicated in dogs and cats are listed in Box 21.1.

Rationale for Performing Open-Chest Cardiopulmonary Resuscitation

Coronary blood flows from the proximal aorta into the myocardium via the coronary arteries during diastole; most blood returning from the myocardium drains into the right atrium. Thus, coronary perfusion pressure (CoPP) is equal to the difference between diastolic aortic pressure (DAoP) and right-atrial pressure (RAP):

$$\text{CoPP} = \text{DAoP} - \text{RAP} \tag{21.1}$$

Research in animal models and in people with CPA has shown that adequate CoPP is associated with ROSC. A CoPP 15 mmHg or greater during CPR has been associated with increased ROSC and survival to discharge [19, 22, 27, 28]. Clinical studies in people demonstrate an increase in CoPP and increased ROSC with internal cardiac massage compared with external chest compressions [16, 22]. Internal cardiac massage has been shown to produce CoPPs three times greater than external chest compressions [19, 22, 28, 29]. Studies have shown significant increase in cardiac output, arterial blood pressure, forward blood flow, CoPP, and cerebral perfusion pressure with internal cardiac massage, and an increase in ROSC and improvement in neurological outcome in canine models of CPA [19, 23, 27, 28, 30–32]. Nevertheless, the 2020 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care state that OCCPR "can be useful" as recommended in the specific situations mentioned above [10].

There are additional potential benefits of OCCPR. Unlike external chest compressions, OCCPR allows direct visualization of the heart and assessment of ventricular filling. The healthcare worker can directly evaluate for ventricular fibrillation, asystole, or an atonic heart muscle during internal cardiac massage, which can help direct therapy when appropriate treatment is unclear during closed-chest efforts. OCCPR also allows for the occlusion of the descending aorta, which maximizes cardiac output to the most crucial organs: the heart, brain, and lungs. Descending aortic compression is also useful in critical hemorrhage into the abdomen by decreasing further blood loss.

Preparing for Open-Chest Cardiopulmonary Resuscitation

Equipment and Environmental Preparedness

The equipment for open-chest CPR should be ready and immediately available in a crash cart. Emergency thoracotomy equipment should be readily accessible in the crash cart, preferably contained within a single sterilized surgical pack to improve the ease and speed of obtaining all necessary equipment. The sterile surgical pack equipment is listed in Box 21.2 and shown in Figure 21.1. Sterile #10 scalpel blades should be readily available anywhere patient CPA is likely, along with sterile gloves, clippers, aseptic scrub, and isopropyl alcohol. Devices for clamping the

Box 21.2 Equipment in Surgical Pack for Open-Chest Cardiopulmonary Resuscitation

- Scalpel handle
- Mayo and Metzenbaum scissors
- Hemostats
- Needle holder
- Sharp and blunt suture scissors
- Tissue forceps
- Sterile gauze
- Sterile drape
- Finochietto rib retractors if the lateral thoracotomy approach is likely in your environment



Figure 21.1 Surgical pack for emergency thoracotomy for open-chest cardiopulmonary resuscitation.



Figure 21.2 Penrose drain, umbilical tape, and vascular clamps for clamping the descending aorta.

descending aorta should also be aseptically prepared, such as Rumel tourniquet, Penrose drain, umbilical tape, or vascular clamps (Figure 21.2). A device with similar function to a Rumel tourniquet can be created using sterile, flexible tubing, umbilical tape, and hemostats.

Building a Tourniquet for Temporary Aortic Compression

A 12–18 Fr red rubber catheter (or similar sterile tubing) is cut to 3–4 cm in length with openings on both ends. The umbilical tape is passed around the descending aorta with both ends of the tape facing toward the operator (Figure 21.3a). A mosquito hemostat is fed through the sterile tubing and used to grasp and thread the umbilical tape through the tubing (Figure 21.3b). The length of tubing is then moved toward the vessel (Figure 21.3c) and the vessel is compressed by the umbilical tape. The umbilical tape is held in place around the compressed vessel by placing the hemostats perpendicularly on the umbilical tape next to the tubing on the operator's side, as shown in Figure 21.3d.

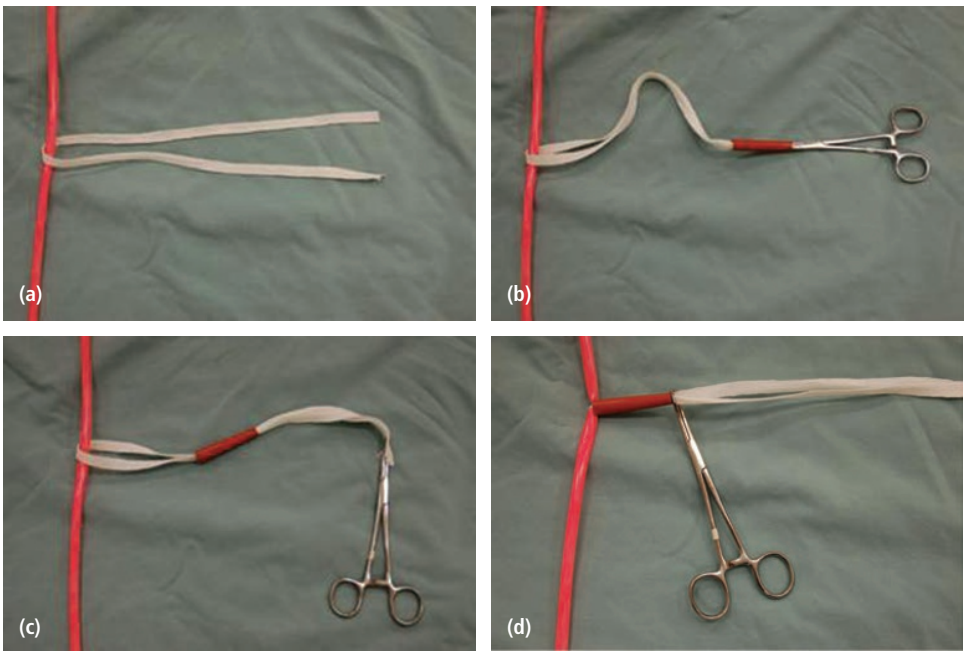


Figure 21.3 Sterile tubing, umbilical tape, and hemostats that can be made (a–c) into a tourniquet for compression or clamping (d) of the descending aorta (pink vertical structure).

Choice of Approach

In dogs and cats, OCCPR is traditionally performed through a lateral thoracotomy at the fourth, fifth, or sixth intercostal space [31, 33]. However a transdiaphragmatic approach via midline celiotomy has also been described, with OCCPR being undertaken either during abdominal surgical procedures [34, 35] or as a stand-alone approach [36]. The transdiaphragmatic approach to the heart is done for other cardiac procedures including epicardial pacemaker placement and pericardiectomy [37–40]. Although a lateral thoracotomy is current standard of care for OCCPR, many practitioners are more experienced and confident with a midline celiotomy versus a lateral thoracotomy, which is a viable alternative.

Performing an Emergency Lateral Thoracotomy

The patient should be placed in right lateral recumbency to prepare for a left lateral thoracotomy at the left fourth, fifth, or sixth intercostal space as shown in Figure 21.4. Throughout, external chest compressions should continue as is possible. The region is prepared quickly with minimal fur clipping at the fourth through sixth intercostal spaces and minimal cleaning with a surgical scrub. Sterile gloves are worn and the patient's skin is incised along the cranial aspect of the chosen rib extending from the dorsal aspect of the scapula to 3–5 cm dorsal to the sternum.

Rapid transection of the latissimus dorsi, serratus ventralis, scalenous, and pectoral muscles will be necessary to reach the external and internal intercostal muscles. The external and internal intercostal muscles are incised along the cranial aspect of the rib with either a scalpel blade or Metzenbaum scissors to the level of the pleura. Care should be taken to avoid lacerating intercostal vessels that run along the caudal aspect of each rib if possible. It is also recommended not to penetrate the pleural space with the scalpel blade or scissors due to the potential for trauma to the lung parenchyma, heart muscle, or coronary vessels; this can be very difficult in the emergency setting.

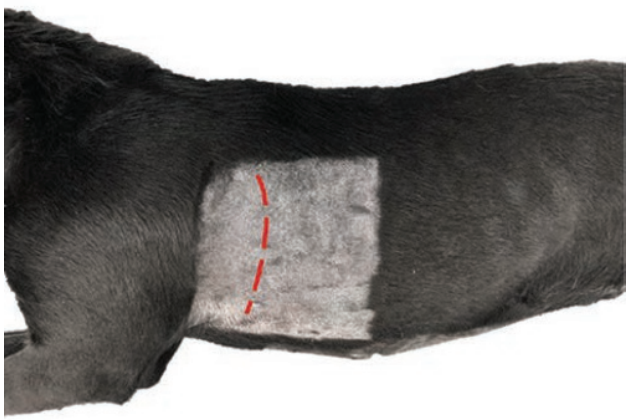


Figure 21.4 Placement of patient and location of incision for emergency thoracotomy to perform internal cardiac massage. Patient is placed in right lateral recumbency for a left lateral thoracotomy at the left fourth to sixth intercostal space. The skin is incised along the cranial aspect of the left fourth to sixth rib extending from the dorsal aspect of the scapula to 3–5 cm dorsal to the sternum.

Manual ventilation of the patient should cease briefly while the pleura is bluntly penetrated with hemostats and the pleural entry extended dorsally and ventrally. Care should be taken to avoid laceration of the relatively large internal thoracic artery running parallel and dorsal to the sternum if possible. Finochietto rib retractors are placed between the ribs to provide intrathoracic exposure with enough space to insert the hand for internal cardiac massage. A rapid, careful partial pericardiectomy should be performed if there is restrictive pericarditis. To achieve a rapid, safe partial pericardiectomy, the pericardium is grasped with tissue forceps and an incision is made with Metzenbaum scissors ventral to the phrenic nerve through the pericardium and extended ventrally. Pericardiotomy or partial pericardiectomy should be adequate to relieve tamponade in the case of effusion. Direct manual cardiac massage is then initiated (see Video 21.1).

Performing an Emergency Transdiaphragmatic Thoracotomy

The patient should be placed in dorsal recumbency. Throughout, external chest compressions should continue in dorsal recumbency as is possible. The abdomen should be prepared with clipping of the fur from the umbilicus to just cranial of the xiphoid in a narrow strip and cleaned with surgical scrub. A skin incision is made starting 5 cm cranial to the xiphoid and extending at least 10 cm caudal to the xiphoid using a #10 scalpel blade to ensure adequate visualization of the fascial layer.

Following exposure of the xiphoid and linea alba, the cranial linea alba is tented and punctured with the scalpel blade, sharp aspect oriented upwards (as in a mid-line celiotomy). The incision in the linea alba is extended cranially along the lateral aspect of the xiphoid and caudally to the extent of the skin incision. This length of incision is to ensure the operator's hand and wrist can easily pass into the incision. The diaphragm is visualized and may already be punctured at the cranial aspect of the linea alba incision. Ventilation should be paused while the diaphragm is incised at the xiphoid. This incision is extended ventrally from the xiphoid with either a scalpel or mayo scissors to end ventral to the central tendon. The incision should be only large enough for the operator to pass their flat hand through to the thorax. If required, a pericardotomy or



partial pericardiectomy can be performed prior to initiation of compressions [37] (see Video 21.2).

Complications of Thoracotomy in the Emergency Setting

Complications associated with emergency thoracotomy include laceration of vessels such as the lateral thoracic vessels along the sternum, intercostal vessels, or coronary vessels; laceration of the lung parenchyma, leading to leakage of air from the lungs and the potential need for suture closure or a partial or complete lung lobectomy; laceration of the liver parenchyma (via transdiaphragmatic approach), leading to hemorrhage; laceration of the heart muscle, leading to profound hemorrhage; penetration into a cardiac chamber, leading to exsanguination; or rib fractures secondary to overzealous retraction with the rib retractor [6, 36]. The complication rate associated with the procedure has been documented to be low in people even with the invasive, urgent nature of OCCPR. Fewer than 2% of people undergoing OCCPR experience iatrogenic cardiac trauma or injury associated with the thoracotomy [41, 42].

Performing Internal Cardiac Massage

Direct compression of the heart has been shown to triple the CoPP compared with external chest compressions [19, 22, 28, 29]. The heart can be massaged in a single hand, between two hands, or between a hand and the internal thoracic wall. Studies have shown that two-handed cardiac massage can be more beneficial than providing cardiac massage with one hand, although this is limited by patient size, heart size, patient conformation, the entry site into the thoracic cavity, and the availability of appropriate instruments such as rib retractors [43]. The heart can be palmed if it is small enough, squeezed between two hands, or pressed up against the inside of the thoracic cavity to compress the chambers (Figure 21.5). Cardiac compression rates should be greater than 100 compressions/minute according to the 2020 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care [10, 44]. Rates of ≥120 compressions/minute and up to 150 compressions/minute have been associated with increased CoPP and perfusion to vital organs in dogs [26]. To allow for adequate refilling of the heart and to account for operator hand fatigue, realistic compression rates for internal cardiac massage of 100–120 compressions/minute are recommended [14, 26, 45, 46].

Sterile gloves should be used to decrease contamination of the thoracic cavity during the emergency thoracotomy and internal cardiac massage. The compressor should be changed no less often than every two minutes with minimal interruption to compression to avoid decreased coronary perfusion that could occur with hand fatigue. Each compressor should wear sterile gloves for internal cardiac massage [10, 11, 44, 47]. Hand placement is important, as inadvertent penetration of the heart muscle with fingers can occur, leading to catastrophic hemorrhage (Figure 21.5d). It is recommended to cup the hand around the heart using either the one- or two-handed technique for internal cardiac massage.

Internal Defibrillation

The heart should be evaluated visually and by gentle palpation for adequate filling, atonic musculature, and ventricular fibrillation during OCCPR. The heart is directly accessible for defibrillation if indicated. In small animals, asystole and pulseless electrical activity are the most common arrest rhythms, although one study found that ventricular fibrillation was nearly as common [5, 6, 9, 14, 48]. Defibrillation in OCCPR can be performed with internal defibrillator paddles wrapped in sterile saline-soaked gauze sponges and placed on either side of the heart. The energy recommended for defibrillation in OCCPR is approximately one-tenth of the energy used for external defibrillation, using 0.5–1 J/kg of body weight [13, 14, 49, 50]. It is recommended to perform internal cardiac massage immediately after defibrillation for one to three minutes before performing further defibrillation [13, 14, 49]. It is recommended to use the least amount of energy required to defibrillate the heart. Ventricular fibrillation is more difficult to convert if the patient has been in ventricular fibrillation for longer than five minutes or is refractory to increasing doses of energy in defibrillation [13, 14, 49, 50]. See Chapter 22 for more information.

Augmentation Techniques

Internal cardiac massage increases the likelihood of ROSC by improving CoPP. Augmentation techniques available during the thoracotomy required for OCCPR may help further increase CoPP. For instance, compression of the descending aorta can increase CoPP and cerebral perfusion pressure by directing the cardiac output to cranial aspects of the body. Descending aortic compression can also help prevent hemorrhage distal to the site of aortic occlusion. The aorta can be compressed using a Rumel tourniquet, sterile tubing, Penrose drain, or umbilical tape that is passed around the major vessel (Figures 21.2 and 21.3). Digital compression of the descending aorta can be performed as an alternative to a tourniquet by gently compressing the vessel dorsally against the ventral aspect of the

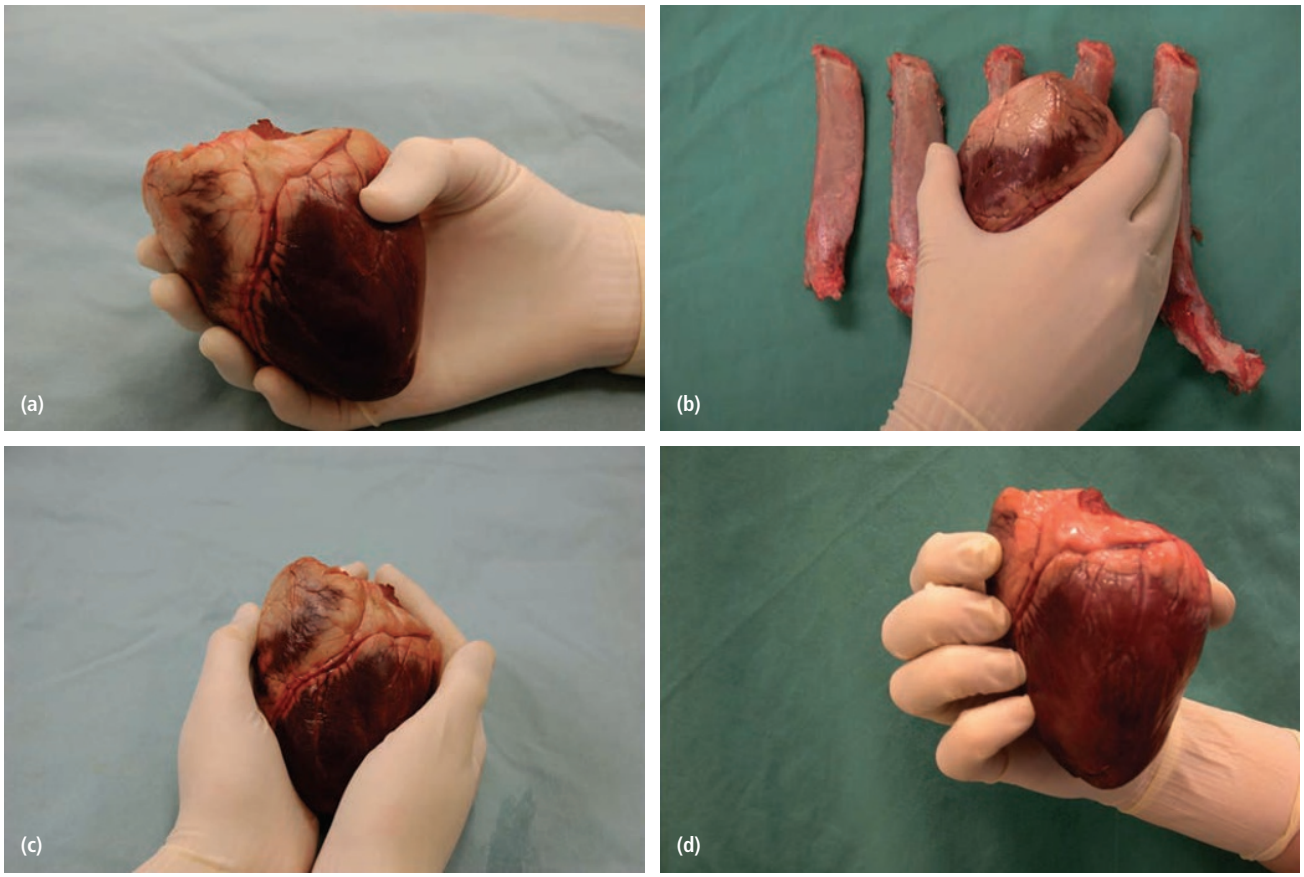


Figure 21.5 Hand placement techniques for internal cardiac massage: (a) a one-handed technique performed by palming the heart. Note the flat orientation of fingers, parallel to the heart muscle; (b) a one-handed technique performed by compressing the heart against the rib cage; (c) a two-handed technique for internal cardiac compression. Pane (d) depicts inappropriate finger orientation to demonstrate how inadvertent penetration of the heart muscle can occur if the fingers point inward toward the heart during internal cardiac massage.

vertebral bodies [8]. After ROSC, aortic compression should be gradually released over 10–15 minutes to help minimize ischemia–reperfusion injury and cardiovascular collapse [15]. Another method used to control massive traumatic hemorrhage in people, which has also been evaluated in dogs, is a technique called resuscitative endovascular balloon occlusion of the aorta (REBOA) [51–53]. REBOA is a less invasive technique using either anatomical landmarks or fluoroscopy to deploy a catheter and balloon device into zones in the aorta to limit massive hemorrhage, thereby salvaging cardiac output. REBOA is not yet used in clinical veterinary medicine.

Post-Resuscitation Care

Surgical Closure

Emergency thoracotomy for OCCPR is invasive, traumatic to the tissues, and is a clean to clean-contaminated procedure that involves unique post-resuscitation care. The opened cavity (or cavities) should be lavaged with sterile saline and intrathoracic samples should be collected for anaerobic and aerobic cultures after lavage, prior to closing the thoracotomy or diaphragm [15, 54]. Closure of the thoracotomy or diaphragm and abdomen should be performed aseptically, usually involving consultation with or closure by an experienced surgeon in a controlled environment [54] (Videos 21.3 and 21.4).

Absorbable monofilament suture (e.g. polydioxanone) or wire is passed around the ribs cranial and caudal to the thoracotomy site, taking care to avoid entrapment of the intercostal vessels on the caudal aspect of the ribs. This can be done by passing the needle backward or using hemostats to bluntly pass suture around the ribs. The sutures are tightened with a square knot closure with a minimum of four throws. After the closure around the ribs, the tissue is sealed and there is no longer communication with the pleural space. Sometimes no fur clipping or surgical scrub is possible prior to emergency thoracotomy; in such cases, fur probably should not be clipped even once the thorax is closed, to prevent excessive intrathoracic fur contamination (see Video 21.3). A thoracostomy tube can be placed aseptically prior to complete closure or thoracocentesis can be performed after closure to evacuate air from the pleural space. Information on thoracostomy tube placement and thoracocentesis is available in Chapter 34.

If a transdiaphragmatic approach has been made for OCCPR, the diaphragmatic incision should be closed with continuous or simple interrupted sutures using an absorbable monofilament material. The suture line should be closed in a dorsal to ventral direction for surgical ease. After the diaphragm and therefore thoracic cavity is closed, pleural drainage via chest drain or thoracocentesis can be performed aseptically to resolve the pneumothorax. The external fascia of the rectus sheath is then closed with a simple continuous suture, or per the surgeon's preference for routine mid-line celiotomy closure [36] (Video 21.4).

Antimicrobials and Analgesia

Owing to the need to enter the thoracic cavity quickly, minimal preparation is performed prior to emergency thoracotomy. Despite this, infection rates following OCCPR are low, reportedly less than 10% in people [8, 41, 42]. Appropriate-spectrum intravenous antimicrobials should be instituted after ROSC; antimicrobials should be continued as indicated based on patient status and degree of contamination during the thoracotomy. It is also important to remember that the patient successfully resuscitated after an OCCPR effort has not usually received anesthesia or analgesia unless the arrest occurred intraoperatively. Therefore, analgesia should be administered at safe doses once ROSC has been established.

Post-Resuscitation Monitoring

Post-resuscitation monitoring and care measures are similar to those for patients resuscitated with external CPR. The cardiovascular system should be evaluated frequently to continuously by monitoring blood pressure and electrocardiogram, and by serially evaluating the patient's perfusion parameters such as mucous membrane color, heart or pulse rate, pulse quality, and capillary refill time. The respiratory system should be monitored with pulse oximetry or arterial blood gases for hypoxemia, end-tidal capnography for hypercapnia secondary to hypoventilation, respiratory rate and effort evaluation, and frequent auscultation of the thorax for decreased lung sounds associated with a persistent or worsening pneumothorax or pleural effusion. Monitoring of the neurologic system is recommended with serial neurological examinations, pupil size and symmetry, pupillary light reflex, palpebral reflex, and changes in mentation to evaluate for injury to the brain secondary to compromised perfusion [48, 55].

The blood glucose and electrolyte concentrations and acid–base status should be monitored frequently and abnormalities corrected as indicated. The patient should be monitored for organ dysfunction such as acute kidney injury; insult to the gastrointestinal tract resulting in hematemesis, hematochezia, or melena; or injury to the liver leading to hepatic insufficiency or failure.

Summary

In summary, OCCPR may be indicated in specific disease conditions such as those associated with pericardial diseases, pleural space diseases, and thoracic wall injury. OCCPR may be indicated when external chest compressions do not result in adequate forward flow of blood during closed-chest CPR. OCCPR has been shown to result in increased coronary and cerebral perfusion pressures, and consequently increased ROSC and survival in certain circumstances. The procedure to enter the thoracic cavity is performed quickly to minimize the time that forward blood flow is compromised. Emergency thoracotomy or transdiaphragmatic approach can lead to traumatic complications such as vessel and intrathoracic or intra-abdominal organ laceration along with infection, though reported complication rates are low. Specific care is taken to decrease the incidence of infection and provide analgesia. Post-resuscitative care and monitoring is like that for any patient that has a sudden cardiac arrest, with monitoring of all organ systems and intensive monitoring of the cardiovascular, respiratory, and neurologic systems.

Video 21.1 Left-lateral thoracotomy for emergency cardiopulmonary resuscitation, fourth or fifth intercostal space.
Video 21.2 Transdiaphragmatic approach for emergency cardiopulmonary resuscitation.
Video 21.3 Left-lateral thoracotomy closure.
Video 21.4 Transdiaphragmatic approach closure.

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22

Defibrillation

Casey J. Kohen

Fibrillation can be defined as a form of cardiac arrhythmia marked by fine, irregular contractions of the cardiac muscle due to rapid, repetitive excitation of myocardial fibers without coordinated contraction of the affected chambers. Fibrillation reflects unsynchronized, random, and continuously changing electrical activity in the myocardium. Fibrillation is typically further categorized by defining the chambers of the heart affected (i.e. atrial fibrillation or ventricular fibrillation). This additional level of categorization is essential because fibrillation of the atria and the ventricles have markedly different effects on cardiac output and usually have different underlying causes.

Atrial Compared with Ventricular Fibrillation

Atrial fibrillation results in the loss of coordinated atrial contraction (the "atrial kick"), which reduces ventricular filling during diastole. Loss of coordinated atrial contraction during ventricular diastole can cause up to a 25% reduction in diastolic filling. This lack of atrial contraction can be overcome if there is adequate time for passive diastolic filling. However, when the atrioventricular (AV) node conducts a high number of atrial impulses, the ventricular rate can be high (e.g. >160 beats/minute in a dog). This form of supraventricular tachycardia compromises passive diastolic filling and, along with the loss of atrial contraction, can result in decreased cardiac preload and subsequently smaller stroke volumes (Starling's law of the heart). Global tissue perfusion can suffer as a result.

Atrial fibrillation is often the result of atrial myocyte injury secondary to structural cardiac disease (e.g. AV valve insufficiencies, primary myocardial dysfunction); however, atrial fibrillation can also occur in the absence of structural heart disease (lone atrial fibrillation), particularly in large and giant breed dogs. Dogs in atrial fibrillation may present in congestive heart failure or for signs associated with decreased forward flow such as weakness, lethargy, or exercise intolerance. Atrial fibrillation in cats is most often associated with myocardial disease such as hypertrophic cardiomyopathy. Atrial fibrillation may be managed medically or through electrical cardioversion, which is a form of defibrillation that is performed in a controlled setting and timed around ventricular conduction (see details later in chapter). Additionally, it is important to determine whether atrial fibrillation is due to structural cardiac disease, as cardioversion in these cases is often unrewarding [1, 2].

Ventricular fibrillation and pulseless ventricular tachycardia are far more acutely life threatening than atrial fibrillation. While atrial fibrillation leads to a loss of atrial contraction, animals can survive without effective atrial contraction if ventricular rates are not excessively high. However, ventricular fibrillation results in ineffective ventricular contraction and cardiac arrest, which is a life-ending event unless a circulating heart rhythm is immediately restored. While atrial fibrillation is often the result of structural heart disease in dogs and cats, ventricular fibrillation has been associated with a wide range of extracardiac causes, including shock, hypoxemia, electrolyte and acid–base disorders, electrical shock, excessive sympathetic tone (or sympathomimetic agents), hypothermia, and drug reactions. Structural heart disease or damage can also lead to ventricular fibrillation; examples include aortic stenosis, primary myocardial disease, and secondary myocardial disease (e.g. contusion, myocarditis, myocardial infarction) [3–7]. Heritable causes of ventricular fibrillation must also be considered. A congenital disease in a group of German Shepherd Dogs has been described in which related dogs are predisposed to sudden death due to ventricular arrhythmias including fibrillation [7]. Boxer dogs with arrhythmogenic right ventricular

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cardiomyopathy are also at risk of ventricular tachycardia and fibrillation [8].

Ventricular fibrillation is a noncirculating rhythm in that stroke volume and thus cardiac output drop to nearly zero. Within seconds of onset, patients with ventricular fibrillation lose consciousness and pulses are absent. This is important to keep in mind when simultaneously evaluating the patient and the electrocardiogram (ECG). If the ECG appears to indicate ventricular fibrillation yet the patient is conscious and has pulses, then ECG artifacts due to such processes as muscle tremors or shivering should be investigated. Conversely, the identification of ventricular fibrillation in an unconscious patient with no palpable pulses should prompt the immediate initiation of cardiopulmonary resuscitation (CPR) unless such resuscitation has been previously identified as inappropriate for the patient (see Chapter 20). Cardiac compressions should be started while the equipment for defibrillation is prepared.

Defibrillation Compared with Cardioversion

Defibrillation is a process by which the entire myocardium is depolarized simultaneously. Fibrillation rhythms depend on sustained activity of re-entry pathways, and the goal of defibrillation is to disrupt the aberrant conduction occurring via these pathways and thus terminate the fibrillatory rhythm. In theory, defibrillation may be achieved via electrical, mechanical (e.g. precordial thump), or chemical means. However, chemical and mechanical defibrillation are rarely successful and should be considered only when electrical defibrillation is unavailable [9, 10]. For the remainder of this chapter the term defibrillation will refer only to the application of electric current to the myocardium and not to chemical or mechanical defibrillation.

Defibrillation is achieved via the delivery of electric current to the myocardium and, as such, success depends partially on how much of the current applied to the patient reaches the myocardium. The current is delivered via the paddles of an electrical defibrillator. Paddles may be applied either externally to the thorax, or internally to the pericardium or epicardium. Impedance to current flow depends on some factors that the clinician controls and some that they do not control. Factors that the caregiver influences include dose of energy administered, paddle size, quality of tissue-paddle contact, and how much gas is in the chest (delivering energy at end-exhalation rather than during inhalation is recommended). Factors outside of the caregiver's control include the absolute amount of gas and tissue in the chest as well as the chest width.

Cardioversion involves the use of defibrillator equipment to convert rhythms other than ventricular fibrillation. Generally, this is done in an attempt to convert an arrhythmia to a sinus rhythm. Although in most patients, arrhythmias are addressed by administration of antiarrhythmic drugs, electrical cardioversion may be attempted in refractory cases. The mechanism is the same as was described previously for defibrillation: the disruption of conduction through aberrant re-entry pathways. Cardioversion is typically reserved for supraventricular or ventricular tachyarrhythmias that are severe enough that cardiovascular performance is compromised, the patient is symptomatic, and pharmacologic approaches have failed.

Equipment

It is strongly recommended that defibrillators be kept in the same area as a "crash cart" (Figure 20.1) or other area where CPR supplies are stored. Defibrillation equipment should be routinely tested and maintained as per the manufacturer's instructions. All personnel who might be called upon to assist in CPR or cardioversion should be instructed in the operation of the equipment and where supplies and accessories for the equipment are stored. Accessories and supplies may include coupling gel, adhesive pads, pediatric external paddles, and a variety of sizes of internal paddles for internal (direct epicardial) defibrillation. A minor surgical pack and rib retractors should also be stored in the same area for times when internal defibrillation is to be attempted.

Until the late 1980s, all defibrillators used monophasic waveforms to deliver defibrillatory energy. Monophasic defibrillators deliver a single pulse of positive current. Monophasic current is unidirectional, traveling from one paddle to the other (hopefully across the myocardium on its way). Defibrillators manufactured after 1990 began using biphasic waveforms; biphasic defibrillators initially deliver a similar (but not identical) positive current followed by a negative current in the opposite direction. The application of biphasic current, when successful, allows for depolarization of the myocardium with the application of lower total energy and thus less risk of myocardial damage. All major manufacturers currently produce and sell biphasic defibrillators.

Another recent development in defibrillators is the degree to which the device can make decisions regarding the diagnosis and treatment of the arrhythmia. Automated external defibrillators (AEDs) are designed to assist rescuers with minimal training to perform CPR, defibrillation, and cardioversion in people. Currently there is no evidence regarding the effectiveness of AEDs in veterinary medicine. Failure of the diagnostic algorithm used in an implantable defibrillator used to treat ventricular tachycardia in a Boxer dog should raise

Table 22.1 Recommended energy doses for defibrillation or synchronized cardioversion using monophasic or biphasic equipment.

	Defibrillation				Synchronized cardioversion			
	Monophasic		Biphasic		Monophasic		Biphasic	
Body weight (kg)	External (J)	Internal (J)	External (J)	Internal (J)	Lower dose (J)	Higher dose (J)	Lower dose (J)	Higher dose (J)
2.5	10	2	6	1	1.25	10	1.25	10
5	20	3	15	2	2.5	20	2.5	20
10	40	5	30	3	5	40	5	40
15	60	8	50	5	7.5	60	7.5	60
20	80	10	75	6	10	80	10	80
30	120	15	100	9	15	120	15	120
40	160	20	150	15	20	160	20	160
50	200	25	150	15	25	200	25	200
60	240	40	150	15	30	240	30	200

concern about the risk of using AEDs designed for people in veterinary medicine [11]. Recommended defibrillation dosage ranges are listed in Table 22.1.

Safety Concerns

Therapeutic application of electrical current can be done safely even in the hectic environment of CPR if specific procedural steps are taken. Attempting defibrillation without having taken such steps runs the risk of significant adverse events occurring such as accidental delivery of current to clinic personnel, burning the patient, or igniting the patient's hair coat. These hazards may be particularly relevant to the veterinary clinical setting wherein patients often have thick, matted hair and may be receiving oxygen (a flammable gas) via unsealed facemasks. These factors may increase the risk of ignition compared with human patients receiving similar care. The accidental delivery of current to clinic personnel can result in painful shocks, burns, and arrhythmias in the person accidentally dosed. To avoid such a scenario, safety measures are imperative; such measures are detailed in Box 22.1.

Accidental patient burns are most likely to occur when skin–paddle contact is poor and arcing of electrical current occurs, or when isopropyl alcohol is used for contact rather than gel. Contact gel intended for defibrillation paddles is preferred over other types of gels and pastes. These gels not only enhance contact between the paddles and patient skin, but they can also prevent arcing and reduce energy impedance. Gel should be applied to the paddle surface liberally while ensuring that an excessive amount has not been applied such that it extends beyond the paddle itself. The application of excessive coupling gel can lead to tracts of gel extending toward (or reaching) the hands of the person holding the paddles, which could lead to inadvertent shock to the operator. Coupling gel is not applied to the surface of internal defibrillator paddles. Instead, internal paddles are wrapped in one or two layers of saline-soaked gauze sponges (4 × 4-inches) prior to application to the pericardium or epicardium (depending on whether the pericardium has been opened).

While small- to medium-sized dogs may be placed in dorsal recumbency for defibrillation, this position may prove unsafe in some larger patients. Stabilizing a large dog in dorsal recumbency using only the defibrillator paddles may place the person holding the paddles in such a position that they are at risk of touching the patient or the table during the delivery of current. Further, other personnel may reflexively reach out to try to stabilize a patient that is tipping over while the energy is being applied. For such large patients, it is advised that a posterior plate or

Box 22.1 Critical Measures to Prevent Accidental Shock in Health Care Workers

- The patient should lie on a nonconducting surface.
- No personnel should touch the patient or any surfaces in contact with the patient at the time of shock delivery.
- Contact gel should not extend beyond the metallic surface of the paddles.
- A warning shout such as "Clear!" should be given prior to shock delivery, with sufficient time for personnel to actually get clear if they are not.
- The operator should visually verify that personnel, including themselves, are clear before delivering the shock.



Figure 22.1 Adhesive electrocardigram/external pacing/defibrillation pads in place on the thorax of an adult dog. Clipping the area prior to placement will reduce thoracic impedance and decrease energy requirements relative to placement on an unclipped site.

adhesive pads (Figure 22.1) be used to enhance patient and personnel safety. Lastly, patients should always be placed on a nonconducting surface rather than directly on a metal examination table prior to defibrillation.

Indications for Defibrillation

The predominant indication for defibrillation is for the treatment of ventricular fibrillation or ventricular flutter (pulseless ventricular tachycardia). In one retrospective study of in-hospital cardiac arrest in dogs and cats, defibrillation was indicated in 28% of dogs and 16% of cats [12]. Ventricular flutter is an unstable rapid ventricular tachyarrhythmia that often converts to ventricular fibrillation in a short time. It should be noted that many patients may develop ventricular fibrillation during CPR even when it is not the initial arrest rhythm. Induced ventricular fibrillation may spontaneously resolve in young, healthy animals in a research laboratory setting, but in clinical patients, time should *never* be wasted waiting to see if ventricular fibrillation is going to resolve on its own [13]. Immediate action is indicated and required. Survival rates decrease by 7–10% for each minute the ventricles fibrillate and thus survival approaches 0% after 10–12 minutes of ventricular fibrillation [14–18].

Defibrillation Procedure and Technique

Defibrillation is nearly always done concurrently with CPR and needs to be properly integrated into the overall resuscitation attempt (Chapter 20). If closed-chest CPR is being performed, external defibrillation will typically be the means initially employed. If ventricular fibrillation cannot be converted with external defibrillation, then internal defibrillation may be more successful and could be attempted. If internal cardiac massage is being attempted, then internal defibrillation is likely to be the first mode attempted although external defibrillation can still be performed even if the chest is open [19–21].

Ideally, defibrillation should be performed as soon as possible once ventricular fibrillation is identified. However, there is evidence that one two-minute cycle of high-quality chest compressions should be performed prior to defibrillation [22]; thus, if an early ECG identifies ventricular fibrillation as an early arrest rhythm, a full two-minute chest compression cycle should be completed prior to attempting defibrillation. If ventricular fibrillation has been identified or is highly suspected, the defibrillator should be prepared during the chest compression cycle. Defibrillation can be achieved in *most* patients without clipping fur; however, in dogs with thick or matted hair, clipping prior to defibrillation may prove more successful. There is evidence from people that human chest hair can significantly increase thoracic impedance and impair current delivery during defibrillation attempts [23]. Standard adult paddles are used for medium- and large-sized dogs (> 13.5 kg); pediatric paddles (Figure 22.2) can be used for cats and small dogs. It is better to have paddles too large than too small, generally. An experimental study using a dog model of ventricular fibrillation has demonstrated that defibrillation is somewhat more effective when larger paddles (12.8 cm diameter) are used rather than smaller paddles (8 cm diameter) in dogs ranging approximately 15–30 kg in body weight [24]. After gel has been applied

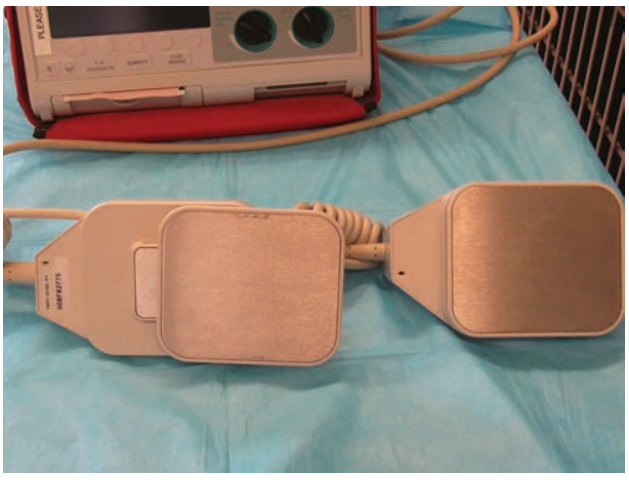


Figure 22.2 Adult and pediatric paddles from a biphasic defibrillator. Some models may have separate pediatric paddles that must be plugged into the base. In the model shown, the pediatric paddles are located beneath the larger paddles (which have been partially removed on the paddle shown to the left).

liberally to the surface of the paddles, they should be held as firmly as possible against each side of the chest wall directly over the area of the heart. If possible, compressing the thorax between the paddles and discharging the energy while the lungs are deflated is advised. The optimal force for the application of the paddles to the thorax has been shown to range from 8 to 12 kgf; however, it has been shown that as few as 14% of humans performing defibrillation are able to generate this much force, nor is force application readily measurable in the clinical setting [25]. As so few human operators are able to generate the optimal force required, the author advises applying the paddles with as much force as can be sustained for the duration of the defibrillation attempt.

Defibrillating the patient with a permanent pacemaker in place bears special mention. If the patient has a pacemaker, the electrodes for the defibrillator *should not* be placed over or close to the pacing generator to minimize the risk of damage during defibrillation [26].

The recommended sequence of events in external and internal defibrillation is described in Protocols 22.1 and 22.2, respectively. For internal defibrillation, the protocol is similar in most regards to that for external defibrillation. However, as noted previously, saline-soaked gauze is substituted for gel or paste. Also, the internal paddles (Figure 22.3) are concave and intended to cradle the heart between them. Firm but not excessive contact should be maintained between the internal paddles and the cardiac structures.

As described previously and in Table 22.1, energy dosage is based on body weight in kilograms and differs for monophasic versus biphasic as well as for internal versus external defibrillation (see Energy Dose Selection, below, for a more detailed discussion of dose determination). It is essential to recall that dosages for internal defibrillation are approximately one-tenth those needed for external defibrillation. Regardless of the initial energy dosage used, it is generally advised that on subsequent attempts the energy delivered be increased by 50–100% after an unsuccessful defibrillation attempt. It should also be noted that the practice of delivering multiple shocks in rapid succession is no longer recommended [22, 26].

Cardioversion of Refractory, Severe Ventricular Tachycardia

Cardioversion involves the use of defibrillator equipment to convert rhythms other than ventricular fibrillation. The major differences between cardioversion and defibrillation involve the setting (i.e. usually performed outside of the emergency room or intensive care unit), circumstances (i.e. nonventricular fibrillatory rhythm), and the timing (i.e. usually a scheduled procedure) of the process. Cardioversion for lone atrial fibrillation is generally a scheduled procedure done under general anesthesia or with significant pre-emptive analgesic administration.

Protocol 22.1 External Defibrillation

Items Required

- Defibrillation dosage chart
- Electrocardiograph
- Defibrillator and paddles of appropriate size for the animal
- Coupling gel
- Clippers, as needed for excessive fur

Procedure

1) Analyze the ECG and confirm ventricular fibrillation is present.
2) Collect necessary supplies.
3) Ensure that isopropyl alcohol or other flammable solvents are not present on the thorax (dilute with water and dry rapidly if needed).
4) Calculate dosage and set defibrillator to appropriate energy setting.
5) Apply coupling gel across the surface of the paddles.
6) Charge paddles.
7) Interrupt chest compressions and apply paddles to each side of the chest at the level of the heart with as much pressure as can be sustained.
8) Temporarily suspend positive-pressure ventilation if it is being provided.
9) Announce to those present that energy is about to be delivered (yell "Clear!") and visually verify that all personnel (including oneself) are clear of the animal, table, and any instruments touching the animal or table.
10) Deliver the energy by discharging the paddles.
11) Evaluate ECG rapidly (< 1 second) while compressor is getting ready to begin again.
12) Restart CPR, including compressions and positive pressure ventilation and perform a complete two-minute cycle.
13) In approximately two minutes, re-evaluate ECG and restart at step 1 if ventricular fibrillation is still present.

Note: In patients with thick or matted hair, it may be necessary to rapidly clip the coat over the thorax to successfully and safely deliver the energy dose.

Protocol 22.2 Internal Defibrillation

Items Required

- Defibrillation dosage chart
- Electrocardiograph
- Defibrillator with internal paddles
- Saline-soaked sterile gauze
- Patient undergoing open-chest CPR

Procedure

1) Analyze the ECG and confirm ventricular fibrillation is present.
2) Direct access to the thoracic cavity may be achieved by one of several means, depending on the setting (see also Chapter 21):
   a) Lateral thoracotomy: If emergency access to the thoracic cavity is needed solely for the purpose of open chest CPR, then a lateral thoracotomy is the preferred approach.
   b) Median sternotomy: This approach is generally reserved for intraoperative CPR when a median sternotomy has already been performed prior to the arrest.
   c) Transdiaphragmatic approach: If open-chest CPR is to be attempted when the abdomen is already open (e.g. an arrest during a laparotomy), then the thorax may be entered via an incision in the diaphragm.
3) Collect necessary supplies.
4) Plug internal paddles into defibrillator base per manufacturer's instructions.
5) Calculate dosage and set defibrillator to appropriate energy setting. (Table 22.1).
6) Cover each paddle with one to two layers of 4 × 4-inch gauze.
7) Moisten each gauze wrapping with sterile 0.9% NaCl.
8) Charge paddles.
9) Interrupt cardiac massage and apply paddles to the heart with one paddle over the region of the right atrium and one over the area of the left ventricle. Apply gentle but firm pressure.
10) Positive pressure ventilation may be briefly suspended if necessary to properly place paddles.
11) Announce that energy is about to be delivered (yell "Clear!") and visually verify that all personnel (including oneself) are clear of the animal, table, and any instruments touching the animal or table.
12) Deliver the energy by discharging the paddles.
13) Evaluate ECG rapidly (< 1 second) while the person performing cardiac massage is getting ready to begin again.
14) Restart CPR, including cardiac massage and positive pressure ventilation and complete a full two-minute cycle.
15) In approximately two minutes, re-evaluate ECG. If ventricular fibrillation is still present, proceed to step 5 (steps 1–4 already having been completed).

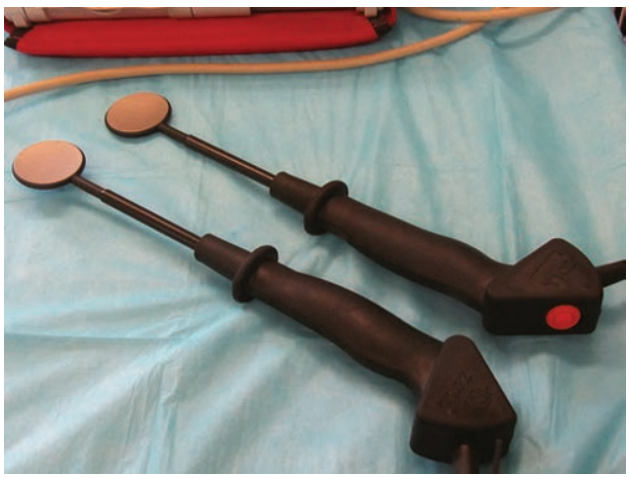


Figure 22.3 Internal paddles are concave with elongated handles to remove the operator's hands from where the current will be discharged. One paddle should be placed over the right atrium and one over the left ventricle.

Prescheduled cardioversion of stable animals is not an emergency procedure and thus will not be covered further herein.

Indications

Cardioversion may be performed in the acute setting for ventricular tachycardia that is life threatening, severe, and/or refractory to pharmacological treatment. This arrhythmia should be distinguished from pulseless ventricular tachycardia (ventricular flutter), which should be considered an "arrest" rhythm and managed with the defibrillation technique described previously. Cardioversion for supraventricular tachycardia (SVT) could also be considered, although these patients are typically not as hemodynamically compromised and thus cardioversion may not be clinically warranted. As patients requiring cardioversion are usually conscious, it is advised that they receive analgesics or be anesthetized prior to attempting cardioversion (which is painful).

Synchronization with the Cardiac Cycle

One major difference between cardioversion in these circumstances compared with defibrillation, as discussed above, is the timing of the energy delivery during the cardiac cycle. Ventricular fibrillation is a chaotic rhythm and timing the delivery to a specific phase of the cardiac cycle is not applicable or possible. However, in the treatment of rapid ventricular tachycardia or SVT, timing becomes relevant. The application of electrical current to the myocardium during the repolarization process (during the T wave on an ECG) can lead to ventricular fibrillation. As such, many modern defibrillators come with a synchronization option that can be activated to help avoid delivering the energy during this most vulnerable period. The synchronization setting will prompt the equipment to deliver the energy during the R or S wave of the QRS complex and avoid the more vulnerable period associated with the T wave. If cardioversion of a refractory ventricular tachycardia or SVT is to be attempted using a defibrillator, it is strongly advised to use equipment in which the synchronization feature is available *and* switched on (Figure 22.4). As a rule, defibrillators should always be used in synchronized mode unless ventricular fibrillation is the arrhythmia being treated. Unfortunately, achieving synchronization is not always possible even with the best equipment. Although limited information is available in veterinary medicine, the American Heart Association guidelines recommend the administration of a high-energy unsynchronized shock in such cases [26].

Procedure

When electrical cardioversion is being attempted, the initial energy setting may be selected at approximately half of the calculated dosage for defibrillation in the same sized patient. The protocol for cardioversion is similar to the protocol described above for defibrillation except that time should be taken to ensure that the synchronization mode is working properly. Many modern defibrillators will give a visual indication of what complexes are being recognized as QRS complexes (Figure 22.4). Additionally, as cardioversion is often attempted in a less acute setting than defibrillation it may be possible to clip fur from the thorax and apply adhesive pads rather than using handheld paddles (as an additional safety measure to reduce risk of shocking personnel). These adhesive pads (Figure 22.5) can also be left on during the initial monitoring period in case fibrillation occurs or cardioversion is again required.

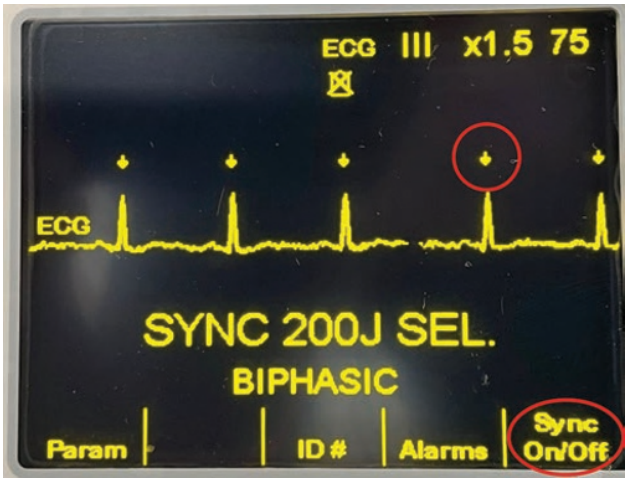


Figure 22.4 A close-up view of a biphasic defibrillator. Note the synchronization option and the indicator arrows marking each detected R-wave (red circles). This synchronized mode is employed when cardioversion is attempted.

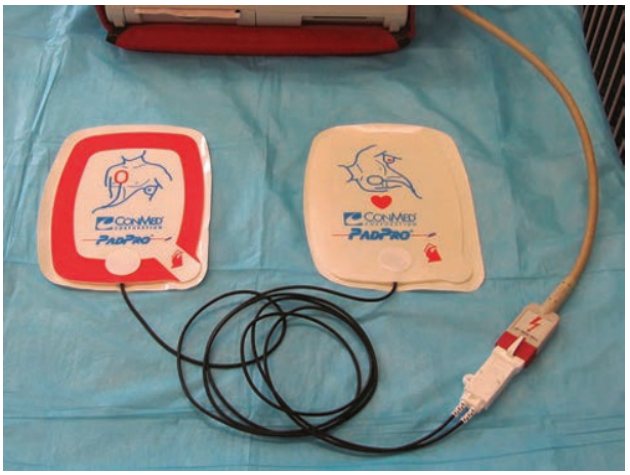


Figure 22.5 Adhesive electrocardiogram/external pacing/defibrillation pads can be placed in advance if a patient is at increased risk of developing ventricular fibrillation or requires external cardiac pacing. These same pad types are shown in place on a patient in Figure 22.1.

Energy Dose Selection

Selecting the proper energy dose (Table 22.1) is important to successfully terminate the arrhythmia while avoiding patient injury. One of the most important determinants of the proper energy dose is body size. Smaller animals can be defibrillated with smaller doses of energy than larger animals. While there is a strong correlation between body size and the energy required for defibrillation, the relationship appears to be nonlinear requiring $0.73 \times BW^{1.52}$ joules (where BW = body weight in kg) using a monophasic defibrillator [27, 28]. The nonlinear relationship between size and dose means that smaller animals would be expected to be defibrillated using fewer joules per kilogram than larger animals. The amplitude, duration, and polarity of electrical current delivered to the heart also

affect the dose required. Defibrillators using biphasic waveforms require approximately 30% less energy to defibrillate than monophasic waveforms [29]. The probability of successful defibrillation increases as the energy dose increases, but so does the risk of injury. In one study, the median effective dose for monophasic defibrillation in dogs weighing an average of 14 kg was 1.5 J/kg. The median dose required to cause injury was 30 J/kg and the median lethal dose was 470 J/kg [30]. Examination of dose–response curves shows that the overlap between effective and injurious doses occurs at approximately 10 J/kg in dogs [30]. Patients can be defibrillated at a lower energy using biphasic waveforms; however, using a biphasic defibrillator at energy doses recommended for monophasic defibrillators does not appear to increase the risk of patient injury [26]. Additionally, it should be noted that the doses needed to defibrillate the heart when paddles are placed directly on the heart (internal defibrillation) are approximately 10% of the doses used for external defibrillation.

Recommended doses of energy for monophasic defibrillators are 4–6 J/kg for external defibrillation and 0.5–1 J/kg for internal defibrillation. For biphasic defibrillators, recommended doses are 2–4 J/kg for external defibrillation and 0.2–0.4 J/kg for internal defibrillation [31].

External cardioversion of arrhythmias is often successful at lower doses of 0.5–2 J/kg, although higher doses may be required, especially for ventricular tachycardia, where energy must travel through a thicker portion of the myocardium. Increasing doses can be attempted until the rhythm is converted or the maximum output of the machine has been reached.

Drug and Defibrillator Interactions

The defibrillation threshold, which is unknown in any individual patient, determines the dose of energy needed to depolarize enough myocytes to terminate the fibrillation. Although the amount of time a patient remains in ventricular fibrillation is the most important determinant of successful defibrillation, certain medications can contribute to the ease (lowered threshold) or difficulty (increased threshold) of converting a fibrillatory rhythm. This threshold can be altered by certain medications commonly used in emergency patients [32]. An increase in this threshold requires increased energy settings for successful defibrillation. Medications that have been shown to increase defibrillation threshold include amiodarone, lidocaine, and mexiletine (Table 22.2). Unfortunately, these medications are used commonly in the emergency setting to treat ventricular tachyarrhythmias. Regardless of concurrent use of these medications, defibrillation should still be attempted as soon as possible. Procainamide has been shown to have little effect on defibrillation threshold.

Table 22.2 Common cardiac drug effects on defibrillation threshold.

Drug	Effect
Amiodarone Lidocaine Mexiletine	Increase (more energy required for defibrillation)
Procainamide	Little effect (no major alteration in energy required)
Sotalol Beta-adrenergic blockers	Decrease (less energy required for defibrillation)

Medications that decrease the defibrillation threshold include sotalol and beta-blockers. In studies that investigated the effects of medications on defibrillation threshold, the effects were more pronounced with monophasic waveform defibrillators than the more modern biphasic defibrillators. This suggests that the contributions of concurrent medications on successful defibrillation may be less clinically important if using a biphasic defibrillator.

Patient Care in the Post-Cardioversion or Post-Defibrillation Period

The care for post-cardioversion patients depends on the severity of illness present prior to cardioversion. If the patient was in congestive heart failure prior to cardioversion, then continued care for this syndrome will likely be required. If the patient was predominantly exhibiting signs of forward failure from poor cardiac output, such signs may abate post-cardioversion; however, significant morbidity secondary to reperfusion injury may necessitate ongoing care for some time. Continued monitoring of perfusion parameters and cardiac rhythm are indicated until the patient is deemed stable.

Most patients having undergone emergency defibrillation (which is only given during CPR for cardiopulmonary arrest) require extensive monitoring and care following a return to spontaneous circulation. Standard post-resuscitation protocols should be followed and are covered elsewhere, as they are beyond the scope of this textbook. Care may be required for surface wounds as a result of the defibrillator use.

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23

Temporary Cardiac Pacing

Anna Grimes and H. Edward Durham, Jr

Cardiac pacemakers maintain cardiac function in patients whose hearts have slowed or stopped beating. Artificial pacemakers were developed by people who observed the tragic and premature deaths of patients whose hearts were capable of contraction but lacked the stimulus to initiate a heartbeat. Techniques for artificially stimulating a heartbeat have been known for hundreds of years. William Harvey used his finger to pace the heart of a dove in the seventeenth century [1], and Galvani used electricity to stimulate muscular contraction in the eighteenth century [2]. It was not until the twentieth century that devices were developed that could stimulate, or pace, the heart in a reliable manner, and it is only in the last 50–60 years that artificial pacemakers have been practical. In the 1950s, transcutaneous cardiac pacing was one of the first types of temporary pacing used clinically [3], though it only became truly practical in the early 1980s after improvements to the design made the associated discomfort more tolerable [4]. Today, epicardial, transvenous (endocardial), percutaneous transmyocardial, and transesophageal pacing techniques are available. Because dogs were used as research subjects, development of techniques for pacing the canine heart generally preceded the introduction of these techniques in people. The first clinical use of an implantable pacemaker in a dog occurred relatively early in the history of artificial pacing, in 1968 [5].

Permanent and temporary pacing techniques are used in different ways. Permanent, implantable pacemakers are used for long-term therapy of bradyarrhythmias. Temporary pacemakers are used for emergency treatment of bradyarrhythmias: as a prophylactic measure in patients that are at risk of developing a serious bradyarrhythmia, maintaining a normal heart rhythm until the patient recovers from a reversible cause of a bradyarrhythmia, and/or until a permanent pacemaker is implanted. In general, temporary pacing techniques are used only for a few hours to a few days.

An artificial electrical pacing system consists of two major components: a pulse generator and an electrode. These may be placed externally or internally, using both external generator and electrode, external generator and internal electrode, or both internal generator and electrode. The generator controls the rate and strength of the electrical stimulus and senses the intrinsic electrical activity of the patient's heart. The electrode is the electrical conduit from the generator to the heart. In temporary pacing, the generator is typically external, and the electrode may be either internal or external. Examples of temporary pacing electrodes are patches placed externally on the thorax, or electrode-tipped pacing catheters placed in the right ventricle via the venous system. Permanent pacemaker electrodes are implantable leads that may be placed endocardially via the venous system, or epicardially through a surgical thoracotomy.

More advanced pacemaker technology allows for dual-chamber pacing, in which separate electrical signals are sent to the atria and the ventricles. The generator also controls the delay between the atrial and ventricular stimuli allowing for an improved physiological pacing profile. The latest advancements in permanent pacemakers include leadless pacemakers, in which there is only a very small generator that is implanted directly into the right ventricular myocardium. This system is limited to ventricular pacing only as no atrial tissue is contacted by the pacemaker, making atrial pacing or sensing impossible. Some devices can be interrogated trans-telephonically without the patient needing to return to the physician's office [6].

All the technology discussed herein is designed for human use and has been adapted to veterinary medicine. To date no specific veterinary temporary or permanent pacing systems have been developed; although one company manufactures pacemakers for the veterinary market, their technology is human medicine-based.

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Indications for Temporary Pacing

Temporary pacing is the therapy of choice for emergency treatment of hemodynamically unstable patients with any of the electrocardiogram (ECG) diagnosed arrhythmias listed in Box 23.1. Temporary pacing is useful as a prophylactic measure for support of asymptomatic bradyarrhythmic patients that require anesthesia since anesthesia can exacerbate bradyarrhythmias in some individuals. Temporary pacing is also widely used as a bridge to permanent pacemaker implantation both during initial hospitalization and the general anesthesia required for permanent pacemaker placement.

Some patients with bradyarrhythmias have concomitant tachyarrhythmias. Since most drugs used to treat tachyarrhythmias can exacerbate bradyarrhythmias, prophylactic temporary pacing can prevent life-threatening bradycardia during early antiarrhythmic therapy for tachyarrhythmia in these brady-tachyarrhythmic animals. When long-term antiarrhythmic therapy is required to control a tachyarrhythmia in these patients, temporary pacing can stabilize the heart rate during implantation of a permanent pacemaker. Temporary pacing may also be indicated in patients undergoing a procedure that risks damage to the sinoatrial (SA) node, atrioventricular (AV) node, bundle of His, or any bundle branches that could lead to complete bundle branch block.

Terminology

The terms used to describe different types of temporary pacing are varied and often confusing. Transthoracic pacing, for example, has been used to describe three different techniques: electrodes placed on the skin of the chest, electrodes passed through the chest wall and sutured to the epicardium, and electrodes inserted through the chest wall and ventricular wall and contacting the endocardium. Temporary transvenous pacing is sometimes referred to as temporary endocardial pacing. Transcutaneous pacing is sometimes known as external pacing, noninvasive pacing, or transthoracic pacing. Temporary epicardial pacing has also been called transthoracic pacing. Thump pacing, percussion pacing, fist pacing, and manual external pacing have all been used by different authors to describe pacing accomplished by striking the chest wall or heart. Transesophageal pacing is often called transesophageal atrial pacing. The technique of using a needle to insert a pacing lead into the ventricular chamber has been called transthoracic pacing, transmyocardial pacing, and percutaneous transthoracic pacing.

In this chapter, temporary pacing techniques are defined as follows: Placing a pacing electrode into the right ventricle by way of a vein is *transvenous pacing* (Figure 23.1a). Pacing using adhesive electrodes placed on the skin on the chest is *transcutaneous pacing* (Figure 23.1b). *Transesophageal pacing* uses an electrode inserted in the esophagus. Striking the

Box 23.1 Arrhythmias for which Temporary Pacing is the Therapy of Choice for Emergency Treatment in Hemodynamically Unstable Patients

- Third-degree atrioventricular block
- Second-degree atrioventricular block
- Persistent atrial standstill
- Symptomatic sinus arrest
- Sinus bradycardia that does not respond to drug therapy

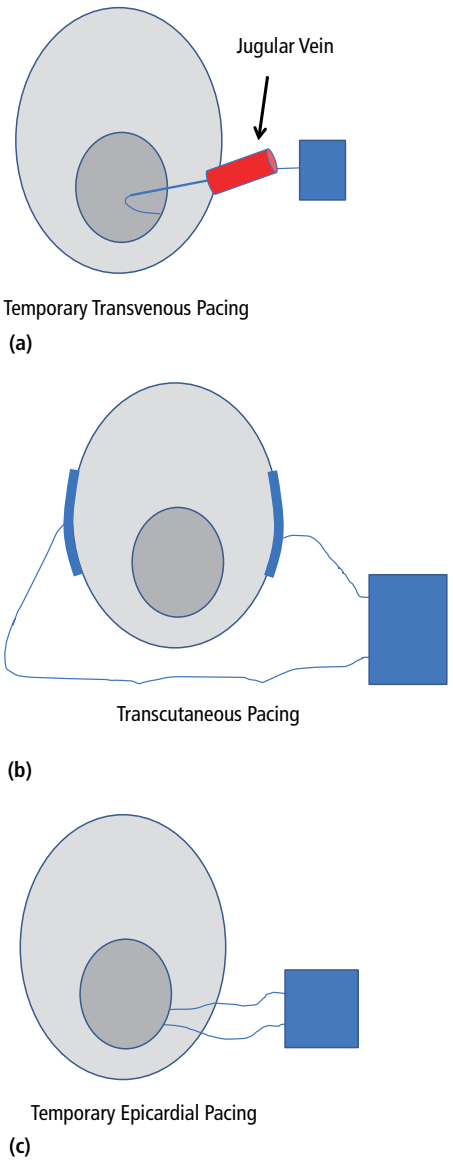


Figure 23.1 Schematics depicting three different methods of temporary cardiac pacing: (a) temporary transvenous pacing; (b) transcutaneous pacing; (c) temporary epicardial pacing.

precordium with the fist is *thump pacing*. A pacing electrode inserted percutaneously into the right or left ventricle is *transthoracic pacing*. *Temporary epicardial pacing* uses detachable electrodes attached to the epicardium and passed through the chest wall during a thoracotomy (Figure 23.1c).

Types of Temporary Pacing

The ideal temporary pacing system is minimally invasive, applied quickly, carries low risk of serious injury, and causes minimal patient discomfort during pacing. While several techniques are available, no single technique meets all these conditions (Table 23.1). Transvenous and transcutaneous pacing techniques are used most frequently, while others are used more often in specific situations such as following cardiac surgery.

Pacing Physiology

Pacemaker technology applies an electrical stimulus to the heart muscle to elicit a contraction from the ventricles. This is generally accomplished by directly stimulating the ventricles. Atrial pacing can be used to create a more physiologic cardiac output profile (providing the "atrial kick" to fill the ventricle prior to ventricular systole) and is more often used in permanent pacing therapy.

Pulse Generators and Their Operation

Pulse generators provide the electrical stimulus that initiates a heartbeat. Some generators can sense the patient's intrinsic heartbeats and are programmed to pause pacing when those occur. This is a safety feature to prevent inappropriate pacing during ventricular repolarization, which could induce fibrillation (which causes cardiopulmonary arrest). Some pacemakers directly pace both the right atrium and ventricle, while some pace only one or the other. Dual-chamber pacing may improve hemodynamic function compared to ventricular pacing alone.

Pulse generators operate in one of two electrical systems: *unipolar* or *bipolar*. Unipolar systems use the electrically conductive properties of the patient's body to complete a circuit; that is, the stimulus is discharged to the heart through a single pacing electrode and returns to the pulse generator via the patient's body. A bipolar pacing system uses a pacing lead with two integrated electrodes so that the stimulus is discharged from one electrode, passes through the myocardium, is then conducted to the second electrode, and finally returned to the pulse generator to complete the circuit.

Temporary pulse generators designed for electrodes in direct contact with the endocardium or epicardium can be used for transvenous, epicardial, or transthoracic pacing. Transesophageal and transcutaneous pacing require much stronger stimuli to depolarize the heart; both the amplitude and the duration of the output pulse are much larger than what is required for techniques in which electrodes directly contact the heart (Figure 23.2). Because they discharge less energy, generators designed for transesophageal or transcutaneous pacing should not be used for any other type of pacing. Some pacemakers allow more sophisticated functions than the basic ones described below.

Pacing Modes

Temporary pacemakers have user-programmed parameters, which vary based on the type of pacemaker lead and patient needs. Transvenous, epicardial, and transthoracic electrodes can pace the atrium, ventricle, or both ("dual" pacing). Transcutaneous electrodes pace the ventricle and do not have dual modality. Transesophageal electrodes, in contrast,

Table 23.1 Characteristics of different modalities of temporary cardiac pacing.

Characteristic	Transvenous	Transcutaneous	Transesophageal	Thump	Transthoracic	Epicardial
General anesthesia required for lead placement	Sedation and local anesthesia	No	Yes	N/A	Yes	Yes
Anesthesia required when pacing	No	Yes	Yes	Yes	No	No
Atrial pacing	Yes	No	Yes	No	No	Yes
Ventricular pacing	Yes	Yes	No	Yes	Yes	Yes
Dual chamber pacing	Yes	No	No	No	No	Yes
Easy to start in an emergency	No	Yes[a]	Yes	Yes	Yes[a]	No

[a] Requirement of fur clipping increases application time.

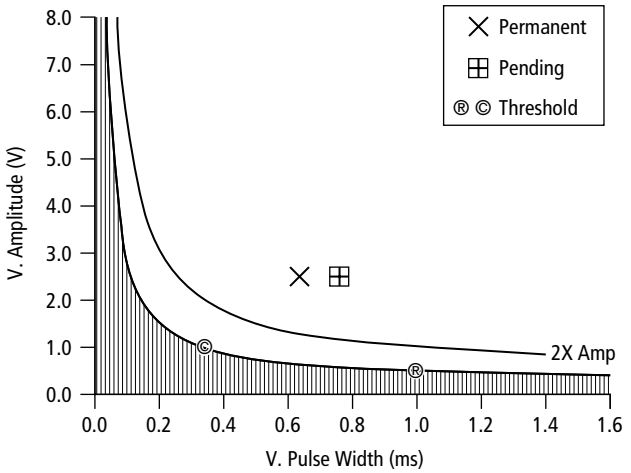


Figure 23.2 A graphic representation of a strength and duration curve with values typical of transvenous pacing. The vertical axis represents the stimulus amplitude. The horizontal axis represents the pulse width or time over which each stimulus is delivered. The output or current is the product of amplitude and pulse width. The shaded area under the curve represents the area of capture loss (threshold). The single line represents an accepted margin of safety at two times the threshold. The "X" indicates the pacemaker's present settings, and the crossed box represents the computer's suggested setting to preserve battery life. *Source:* Durham 2017/with permission of John Wiley & Sons.

can only pace the atrium. Temporary pacemaker modes are referenced using a three-letter system (Table 23.2), which describes the pulse generator's activity and is always listed in the same order: the paced chamber in position 1, the sensed chamber in position 2, and the ability to pause when intrinsic beats arise in position 3. Some permanent pulse generators can change rate (known as rate responsive) depending on the patient's activity level, making them more physiologically appropriate when the patient is active. If rate responsiveness is programmed, there is an additional letter "R" added to the end of the mode, in position 4.

Table 23.2 Modes of temporary pacing leads.

Mode	Paced chamber	Sensed chamber	Inhibited
VOO	Ventricle	None	None
VVI	Ventricle	Ventricle	Inhibited
AOO	Atrium	None	None
AAI	Atrium	Atrium	Inhibited
DDD	Dual	Dual	Dual
VDD	Ventricle	Dual	Dual

V: ventricle; A: atrium; O: off; I: inhibited; D: dual = both atrium and ventricle. The mode describes, in order, which chamber the pulse generator paces, senses, and if the pulse generator inhibits itself when it senses an inherent beat. Note that the sensed chamber and inhibit feature work together, such that if sensing is off, the pulse generator will not inhibit itself and will pace at the prescribed rate regardless of intrinsic heart activity. This is called asynchronous pacing.

Synchronous pacing describes the pacemaker's inhibition ability, which uses sensitivity and refractory settings to allow the pacemaker to pause when it recognizes intrinsic cardiac activity. Synchronous pacing is preferred, as the generator is less likely to send an impulse during the patient's natural refractory period (during the T wave on the ECG), which can cause ventricular fibrillation. *Asynchronous pacing* is when the generator sends an impulse at a programmed rate independent of the patient's inherent cardiac activity.

Advancement in temporary pacemaker technology has made temporary dual pacing possible, which allows the pulse generator to pace and/or sense and inhibit not just the ventricle, but also the atrium. This may be advantageous in certain underlying causes of bradyarrhythmias to optimize patient stability prior to permanent pacemaker implantation or during cardiac surgery.

Output Pulse

Output is the strength of the stimulus delivered to the heart. The term *capture* means that the pacemaker stimulus successfully depolarized the heart and caused myocardial contraction. Output is the combined effect of electrical amplitude and duration in milliseconds (ms) of the stimulus applied and is known as *pulse width*. Amplitude is usually measured in milliamperes for transcutaneous pacing and in volts for transvenous and epicardial pacing. Longer duration stimuli can achieve capture at a lower output than briefer stimuli. The amplitude of the stimulus required varies with the type of pacing: transcutaneous pacing requires the strongest output, while transvenous and epicardial pacing require the least. Thus, transcutaneous pacing devices use a lower amplitude delivered for a longer duration to avoid discomfort and tissue trauma, whereas transvenous or epicardial pacing employs higher voltage for very short durations. This idea is graphically represented with a *strength–duration* curve (Figure 23.2).

On the surface ECG, evidence of pacemaker stimulus is seen as a vertical line, called the pacing spike. If the pacing spike is not followed immediately by a QRS complex, this represents "failure to capture" and the myocardium was neither depolarized nor did it contract. The output required for capture is also affected by the area of the transcutaneous pacing electrode, its location in relation to the heart, drug therapy, and serum electrolyte concentrations.

Pacing spikes vary in amplitude depending on the ECG lead placement, the pacing lead polarity (bipolar vs. unipolar),

and the amplitude of the stimulus. Output settings are determined by the threshold, or the minimum output necessary to achieve capture. When pacing begins, the amplitude, pulse width, or both are increased until capture is achieved. Only the newest transcutaneous pacing systems allow manipulation of the pulse width. Excessive output settings worsen patient discomfort associated with transcutaneous or transesophageal pacing and increase the risk of skeletal muscle contraction, pacing the diaphragm, and causing skin or esophageal burns.

Sensitivity

Currently available pacing generators can detect electrical activity that originates in the heart (intrinsic beats aka native beats) and pause artificial pacing when intrinsic depolarizations occur. The sensitivity function is important because a stimulus delivered during the repolarization phase of the heartbeat may cause ventricular fibrillation. Transvenous pacemaker generators detect intrinsic beats by using the pacing lead, while transcutaneous pacemaker generators use standard limb leads to measure the amplitude of the intrinsic QRS complex. The sensitivity control sets the minimum amplitude that is assumed to identify an intrinsic QRS complex. If, for example, the sensitivity control is set for 4 mV (millivolts), signals that are detected with an amplitude greater than 4 mV are assumed to be intrinsic beats and will cause the pacemaker to pause. Transvenous pacing generators indicate a sensed intrinsic beat with a light on the generator's display (Figure 23.3), while transcutaneous pacing generators insert an indicator mark over the beat on the ECG display (Figure 23.4).

The failure of the generator to detect intrinsic beats is called undersensing. There are other sources of electrical



Figure 23.3 External pulse generator for a transvenous temporary pacemaker. The light at the top indicates that the generator is sensing a patient's intrinsic heartbeat. From the top right, the knobs indicate the following: stimulus measured in volts (V); pacing mode (VVI is ventricular pacing, ventricular sensing, pacemaker inhibition of intrinsic beats); pacing rate per minute (p/min); and sensitivity in millivolts (mV). *Source:* Prague ICU/YouTube, LLC.

signals in the body and the environment that can be detected by the pacemaker. P waves, T waves, and shivering are common sources of other myopotentials in the body. Electrocautery units, clippers, and the electric motors in adjustable-height tables can also produce signals that may be detected by the pacemaker. Since the pacemaker senses

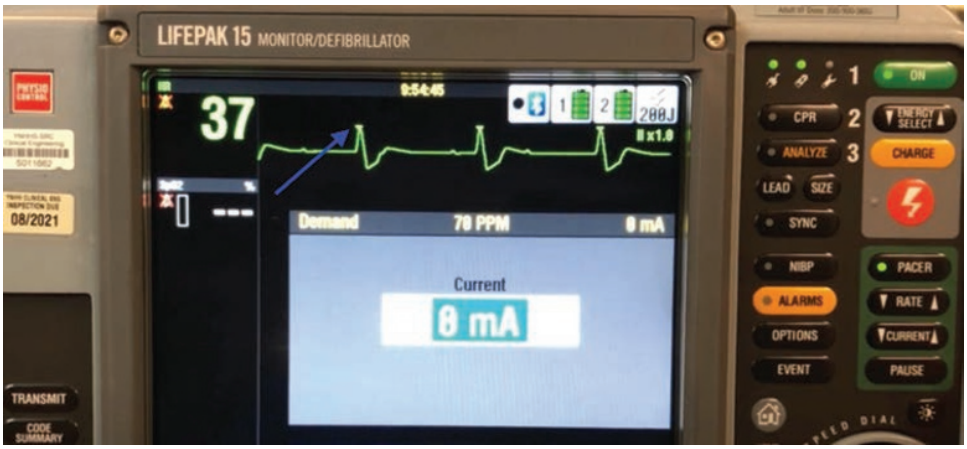


Figure 23.4 The screen display of a LifePak 15 defibrillator on the pacing setting. The small, white notch above the QRS complex indicated by the blue arrow is the generator sensing the patient's intrinsic heart beats. *Source:* Resuscitation Management/YouTube, LLC.

only these signals' amplitudes and is blind to their origins, if these signals exceed the pacemaker's sensitivity setting, they will prevent pacing. This is known as *oversensing*, which could result in periods of ventricular asystole. The objective in adjusting the sensitivity setting is to exceed the amplitude of P waves, T waves, and environmental noise but not exceed the amplitude of the intrinsic QRS. Appropriate sensing may not be possible in every case and if the source of the interference cannot be eliminated, another option is to switch the pacemaker to asynchronous mode in which the pacemaker delivers stimuli regardless of the patient's intrinsic cardiac electrical activity.

Atrioventricular Delay

Transvenous and transthoracic pacing techniques can be used to pace the atrium in addition to the ventricle (termed, dual). During dual-chamber pacing there must be a delay after the atrial stimulus to allow complete atrial contraction before delivering the ventricular stimulus; this pause is called the *A–V delay*. Dual pacing duplicates the delay that normally occurs between atrial and ventricular contraction, which is represented on the ECG as the P–R interval. In some patients A–V synchrony such as by dual pacing can augment stroke volume by >25% [7]. Ideally, the A–V delay should be adjusted to optimize hemodynamic function by monitoring changes in stroke volume or cardiac output in near-real time with the echocardiographic–Doppler velocity time integral, contour analysis of the pressure waveform, change in pulse pressure, or continuous mixed venous oxygen saturation. If this is not possible, a delay of 120 ms may be used [7].

Rate

The rate setting determines the interval between generator output stimuli: it determines the heart rate. Typically, a heart rate between 60 and 100 beats/minute is used in dogs (with smaller dogs receiving the higher rate) and 140–180 beats/minute in cats. During transcutaneous temporary pacing, lower rates may be used during permanent pacemaker implantation to ease the surgical procedure itself, especially if significant muscle contraction occurs. Providing the patient's blood pressure is adequate, the surgeon may opt for a lower paced rate to facilitate rapid placement of a permanent transvenous lead. Long-term rapid pacing should be avoided because it can lead to myocardial failure.

Refractory Period

Advanced temporary pulse generators may have programmable refractory periods. The refractory period is a time when the pulse generator may sense intrinsic cardiac depolarizations but not respond to them. This feature allows for the pulse generator to ignore repolarization of the ventricle (T wave) and not misinterpret it as intrinsic ventricular activity. While the sensitivity setting allows the generator to identify electrical activity, the refractory period inhibits the generator from responding to it. These settings work in conjunction to optimize the patient's physiological profile.

Temporary Transvenous Pacing

Temporary transvenous pacing (endocardial pacing) is one of the most reliable and frequently used types of temporary pacing. The technique is relatively safe and carries the advantage that both electrode insertion and pacing can occur in the conscious patient. Transvenous pacing is also one of a few techniques that can be used to temporarily pace patients for longer periods, such as a day or more. With appropriate venous catheters and pulse generators, transvenous AV sequential pacing (dual-chamber pacing) or physiological ventricular pacing (such as VDD) can be accomplished.

Patient Preparation

Insertion of a transvenous temporary pacing lead often can be performed with heavy sedation and may not require general anesthesia. Continuous monitoring of ECG, pulse quality, and blood pressure is essential to detect hypotension or severe bradycardia during the catheter placement.

Insertion Site Selection

Several factors should be considered when selecting the site for inserting the transvenous pacing lead. Although vascular anomalies are uncommon, they can complicate insertion of the pacing lead [8]. One of the most frequently encountered anomalies is a persistent left cranial vena cava, which has been reported in 5% of dogs with congenital cardiac disease [7, 9, 10]. A persistent left cranial vena cava renders it virtually impossible to insert the pacing lead into the right heart via the left jugular vein. Thus, the right jugular vein is preferred for permanent pacing lead placement since vascular anomaly of the right jugular is less likely and facilitates the use of commonly available 45–58 cm pacemaker leads. If a transvenous temporary pacemaker is used during permanent pacemaker implantation, it is beneficial to save the right jugular vein for the permanent pacing lead and use a peripheral approach for the temporary. The right or left lateral saphenous veins can be used for insertion of a temporary pacing lead, though they may be too small to accommodate an introducer for a 4 or 5-Fr temporary pacing catheter in dogs weighing less than 15 kg. In some large dogs a 110-cm pacing lead may not reach the right ventricle from a saphenous vein. Bulmer [11] described a technique for inserting an

introducer into the femoral vein. Once the temporary insertion site has been selected, the fur should be clipped and the skin aseptically prepared. The patient should be protected from electrical hazards like faulty electrical equipment and static electricity to avoid microshock (see later).

Venous Access and Placement

A percutaneous introducer sheath with a hemostasis valve or a specialized over-the-needle catheter with or without a hemostasis valve is used for venous access (Figure 23.5). The catheter or introducer selected must be large enough to accommodate the temporary pacing lead. The introducer sheath is inserted using a modified Seldinger technique and secured (Chapter 7). The temporary pacing lead is then inserted through the introducer. See Protocol 23.1 for detailed instructions on temporary transvenous pacemaker placement.

Prior to inserting the temporary pacing lead, one should inspect and test the equipment. For instance, some temporary transvenous pacing catheters have a balloon at the tip (Figure 23.6). When inflated, blood flow helps direct the catheter by pulling the balloon across the tricuspid valve into the right ventricular apex. Balloon integrity should be tested prior to catheter insertion. While the easiest, most reliable way to insert a transvenous pacing lead is with fluoroscopic guidance, other methods can be used to determine the location of the pacing lead. An ECG recorded from the catheter tip can be used to determine the location of the temporary pacing lead by observing the polarity and amplitude



Figure 23.5 Individual components of the Avanti+ Sheath Introducer made by Cardinal Health. From left: Introducer catheter with hemostasis port and three-way stopcock; tapered-tip vessel dilator; mini-guidewire; insertion needle. *Source:* Cordis.

Protocol 23.1 Placement of a Temporary Transvenous Pacemaker

Items Required

- Fluoroscope or ECG to determine electrode location
- 2% lidocaine solution in syringe with needle, for subcutaneous infiltration
- Percutaneous introducer kit, or a suitable intravenous (IV) catheter and an access port (Figure 23.5)
- Temporary pacing lead (bipolar or unipolar)
- Pacing generator with fresh batteries
- Sterile #11 scalpel blade on handle, scissors, needle driver, thumb forceps
- Sterile suture
- Sterile surgical drapes
- Sterile sponges
- Sterile gloves, gown
- Surgical caps, masks
- Bandage material
- Assistants
- Patient monitoring equipment (ECG, blood pressure monitor)

Procedure: Introducer Placement Using the Modified Seldinger Technique

1) Collect necessary supplies.
2) Select insertion site, position patient appropriately, and place monitoring equipment.
3) Clip fur at least 2 inches (5 cm) in all directions from planned insertion site and aseptically prepare the skin. Inject local anesthetic agent (2% lidocaine 0.5 mg/kg) [47] subcutaneously around the planned insertion site.
4) Don cap and mask. Perform hand hygiene, don sterile gloves and gown, and drape the prepared insertion field.
5) Use the scalpel blade to create a small skin incision at intended insertion site.
6) Insert an arterial needle (provided with introducer kit) or IV catheter into the vein. This catheter must be large enough to allow a guidewire to pass through it.
7) If using an IV catheter, remove the stylet.

8) Pass the guide wire through the arterial needle/IV catheter and into the vein.
9) Remove the arterial needle/IV catheter while stabilizing the guidewire in the vein.
10) Place the tapered dilator into the introducer sheath and pass the dilator and sheath over the guide wire.
11) Dilate the vein and insert the introducer. Push the dilator and sheath combination over the guide wire through the hole in the skin, through the wall of the vein, and into the lumen of the vein.
12) Remove the dilator and guide wire from the introducer sheath, leaving the introducer in the vein. Flush thoroughly.
13) Suture the introducer to the skin. Check to see that there is no leakage from the port of the hemostasis valve on the introducer.
14) Cover the opening on the hemostasis valve to maintain asepsis.

Procedure: Temporary Pacing Lead Placement Through Introducer

1) If the temporary pacing lead does not have a bend in the tip, form one to facilitate passage into the right atrium. Most catheters are packaged to produce this bend, but the bend may be lost if the catheter has been re-sterilized.
2) Pacing lead introduction:
   a) Use either fluoroscopy or an ECG to determine location of the pacing lead throughout this process, as described in the chapter text.
   b) Pass the end of the pacing lead that will be inserted into the generator to a non-sterile assistant.
   c) If the introducer is in the lateral saphenous vein, insert the lead through the access port, saphenous vein, femoral vein, caudal vena cava, right atrium, tricuspid valve, and right ventricle until it contacts the right ventricular endocardium. If resistance is encountered as the lead reaches the inguinal region, abduct the limb to allow the lead to pass. To avoid venous perforation, DO NOT force the catheter forward.
3) For bipolar leads, with the help of a non-sterile assistant, connect the distal end of the lead to the negative terminal of the generator and the proximal end of the lead to the positive terminal of the generator.
4) Set the rate on the generator and increase the strength of the stimulus until it captures the ventricle, both electrically and mechanically (produces a ventricular complex on the ECG *and a palpable pulse*; Figure 23.10).
5) Adjust the generator sensitivity and/or refractory period to recognize intrinsic beats while ignoring T waves, P waves, shivering, and other artifacts that could inhibit pacing.
6) Secure the lead to the access port.
7) Protect the access port with a bandage.



Figure 23.6 Example of a temporary transvenous pacing catheter with a balloon at the tip. When inflated, blood flow helps direct the catheter by pulling the balloon across the tricuspid valve toward the right ventricular apex.

of the P waves and QRS complexes (Figure 23.7). Bing [12] described this technique in people, and Moïse described it in dogs [13]. Baird [14] described a technique in which the pacing generator is turned on while the temporary lead is inserted blindly. The stimulus from the pacing lead causes muscular contractions to show the location of the electrode. Observation of the ECG, using standard limb leads, will show when the atrium or ventricle is being paced. Echocardiography has also been used to assist in determining the location of the temporary pacing lead [15–17].

Temporary Transcutaneous Pacing

Transcutaneous pacing is particularly well suited for emergency use and for prophylactic use in situations in which there is a risk for life-threatening bradyarrhythmia. Many modern defibrillators include a transcutaneous pacing function. Transcutaneous pacing is commonly used during implantation of a permanent pacemaker system to maintain cardiac output while the permanent pacing lead is implanted. Once the permanent lead is properly placed within the right ventricle, temporary transvenous pacing can be performed through the permanent lead until the permanent external pulse generator is surgically placed and connected to the lead. This allows for the transcutaneous pacing to cease, alleviating the muscle contractions associated with this method of pacing.

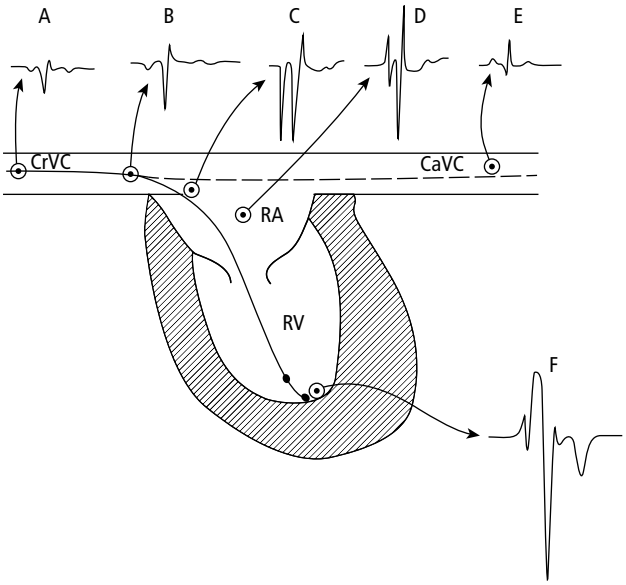


Figure 23.7 Schematic of the electrical activity recorded in lead II ECG as a pacemaker lead enters the right heart. CrVC: cranial vena cava; CaVC: caudal vena cava; RA: right atrium; RV: right ventricle. An ECG can be used to determine the location of the pacing lead by observing the polarity and amplitude of the P waves and QRS complexes (A–F), which change as they move toward the positive pole near the RV. Note that in lead II, as the pacing lead reaches the right ventricular apex, the P wave appears large and positive, and ventricular depolarization appears as a large negative complex (Letter F). Letter E depicts the ECG tracing if the lead bypasses the RA.

The procedure for transcutaneous pacing is relatively simple. Once adhesive electrodes are applied to the appropriate location (discussed in the next section) on the clipped skin of the thorax, pacing can begin. Proper application of the electrodes is important for successful pacing (capture). The size of the electrodes, their contact with the patient's skin and location on the patient, as well as their polarities are important factors that influence capture. The pacing electrodes/patches are sized for human medicine as "adult" or "pediatric" (Figure 23.8).



Figure 23.8 Examples of transcutaneous pacing electrode patches, which come in both adult and pediatric sizes. *Source:* Owens & Minor, Richmond VA, Covidien LLC, Mansfield, MA. Courtesy of HE Durham.

Transcutaneous Pacing Electrode Location

Several studies regarding electrode size, placement, and polarity have been published [18–23]. Falk et al. [23] tested the pacing threshold in people and found that electrode polarity is critical: reversing the polarity resulted in extremely high capture thresholds or failure to capture. Geddes et al. [18] mapped the thoraces of 18 dogs and determined that the specific positive electrode location on the right hemithorax was not critical, but that a distinct window for optimal pacing corresponded with placement of the negative electrode over the apical beat on the left hemithorax. DeFrancesco et al. [20] found that the optimal location for electrode placement was directly over the heart on the left and right hemithoraces. The negative electrode was placed on the left side near the sternum and the positive electrode was placed on the right side more dorsally. Lee et al. [19] tested several different electrode configurations and found that the most effective position was with the electrode centered on the costochondral junction of the sixth rib on the right and left hemithoraces. Unfortunately, the authors did not indicate the polarity of the electrodes. The studies performed by Geddes, DeFrancesco, and Lee

conclude that the location for optimal placement of the electrode on the left hemithorax is sternally over the apical impulse of the heart. Perhaps because placement of the right electrode appears to be less critical, the three studies found slightly different optimal locations for electrode placement on the right hemithorax.

Transcutaneous Pacing Electrode Size

Optimal electrode size appears weakly correlated to body size [18]. DeFrancesco et al. [20] used adult pacing electrodes on dogs 3.8–40 kg of various breeds. Lee [20] found that for Beagles the optimal electrode size was 4 × 5 cm (20 cm^2). Geddes [18] found that there was little advantage in using electrodes greater than 5 cm in diameter in dogs. Both ZOLL Medical and Physio Control recommend using pediatric electrodes for patients less than 15 kg.

Performing Transcutaneous Cardiac Pacing

To ensure that the electrodes make adequate skin contact to allow capture, the skin should be clipped of fur and clean so that the electrode patch adhesive adheres. DeFrancesco et al. [20] recommended using ECG paste and an elastic bandage to hold the electrodes in place. Multifunction electrode patches may be advantageous if future defibrillation is considered likely; fortunately, the optimal pacing and defibrillation windows are nearly identical [24].

All steps to prepare for pacing can be performed with the patient awake; however, before electrical stimulus is delivered the patient should be anesthetized or at least heavily sedated because transcutaneous pacing is painful. The need to clip the thorax may contribute to discomfort associated with transcutaneous pacing, as people report that minor skin nicks or cuts exacerbate the discomfort.

ECG electrodes must be attached to the patient as well as pacing electrodes so that the patient's intrinsic QRS complexes can be monitored by the user. Asynchronous pacing mode, in which the pacemaker does not stop in response to the patient's intrinsic cardiac activity, may be used with little risk to the patient [24, 25] if the pacemaker is inhibited by artifacts or does not sense the QRS complexes. An example of an ECG tracing during transcutaneous pacing is depicted in Figure 23.9.

Pacing is initiated by selecting the desired heart rate and increasing the strength of the stimulus until the ventricle is captured and a pulse detected. A Doppler flow detector secured to the metacarpal or metatarsal is one convenient method of verifying capture, because the user will be able to hear the arterial pulse. Transcutaneous pacing causes contraction of the chest muscles, which causes patient movement and may lighten the plane of anesthesia. See Protocol 23.2 for detailed instructions on transcutaneous cardiac pacing.

Temporary Epicardial Pacing

Temporary epicardial pacing is a specialized technique that is generally used in patients recovering from cardiac surgery. Temporary epicardial pacing allows more flexibility in where the electrode(s) can be located and the mode of pacing than any other temporary pacing technique. The electrodes used for temporary epicardial pacing are thin and resemble wire suture. They have needles on both ends; one end of each electrode is attached to the heart, while the other end passes through the chest wall and skin and is

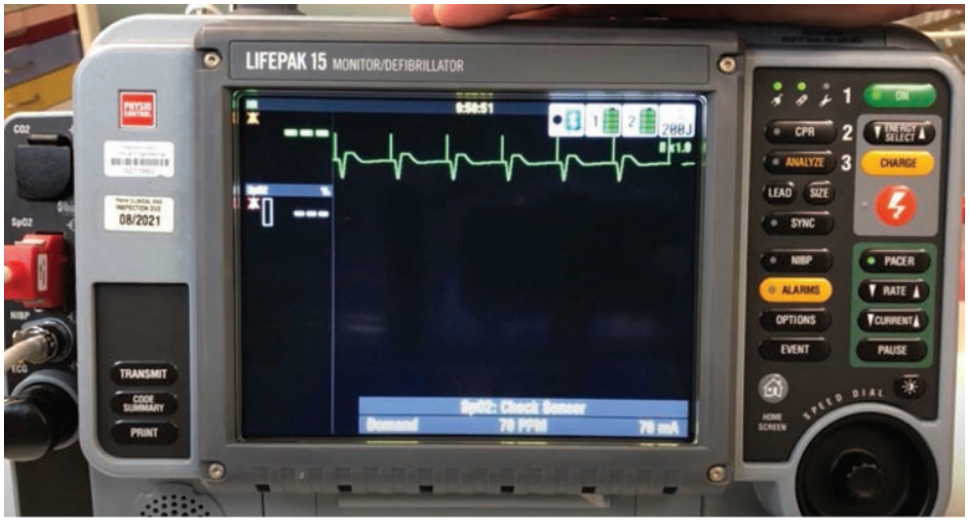


Figure 23.9 ECG tracing of transcutaneous pacing using the LifePak 15 Defibrillator. The ECG shows electrical capture as evidenced by a pacing spike followed by a QRS complex. This machine is using the pacing mode and is set to pace 70 beats/minute.

Protocol 23.2 Transcutaneous Cardiac Pacing

Items Required

- Transcutaneous pacing generator
- Pacing electrode patches of appropriate size, remembering that if the patch is not designed for defibrillation, it may interfere with defibrillation.
- ECG paste for the pacing electrodes
- Clippers
- Saline to clean skin after clipping
- Bandage material to keep pads in place
- ECG electrodes for standard limb lead placement (these often also attach to the pulse generator).
- Doppler blood pressure monitor to verify mechanical capture

Procedure

1) Prepare to monitor the ECG, pulse quality, and blood pressure.
2) Collect necessary supplies.
3) Clip fur from planned electrode site. Avoid abrading or cutting skin.
4) Clean newly shaved area with saline to remove small hairs and oil from skin.
5) Place the electrode patches on the right and left hemithoraces, ideally directly over the palpable precordial impulses.
6) Inspect the electrodes to be sure there is complete contact with the skin.
7) Bandage the electrodes if desired to help them stay in position.
8) Turn on the pulse generator.
9) Connect the standard limb lead ECG electrodes to both the pulse generator and patient. A good ECG tracing is required for demand pacing. The QRS complex should be of greater magnitude compared to the T and P waves; if this is not the case, select a different ECG lead setting on the pulse generator in which the QRS complex is significantly greater in magnitude (positive or negative) than the P or T waves.
10) Anesthetize the patient if not already unconscious.
11) Set the sensitivity and desired pacing rate.
12) Gradually increase the output of pacing generator until the ventricle is captured: ventricular complexes will appear on the ECG after a pacing spike, and pulses should be produced (Figure 23.10).



Figure 23.10 ECG of patient being maintained with thump pacing, courtesy of Craig Cornell. Every QRS complex on this ECG tracing was initiated by a thump delivered to the chest. Each QRS produced a pulse wave that could be seen on a direct arterial pressure monitor.

connected to a pacing generator. The surgeon usually decides where the electrodes will be placed. Since several electrodes may be used, it is important to identify the function of each lead. The pacemaker is operated by inserting the electrode into the appropriate connector on the generator. The pacing leads should be secured to the patient's body so that the leads will not be dislodged if the patient moves or the generator is dropped. When pacing is no longer needed the electrode is designed to be removed by gently pulling on it. A two-part 2007 review [26, 27] provides a more detailed description of the technique.

Thump Pacing

Mechanical force applied to the heart or precordium has a long history of being used to treat bradyarrhythmias. Nevertheless, the technique of thump (aka "percussion" or "fist") pacing, which uses a series of thumps to the precordium to maintain a physiological heart rate, is still not well known. Several authors have described it as an almost forgotten technique [28, 29].

The use of precordial thumps for the treatment of bradyarrhythmias is much safer and more effective than the use of a thump for treating tachyarrhythmias. The 2012 RECOVER (Reassessment Campaign on Veterinary Resuscitation) guidelines by Fletcher et al. [30] mention the use of a single precordial thump as a technique for termination of pulseless ventricular tachycardia or ventricular fibrillation, not for bradyarrhythmias, specifically when means of electrical defibrillation is not possible. However, in human medicine, examination of evidence that supports thump pacing has led to newer resuscitation guidelines of thump pacing for emergency treatment of bradycardia caused by complete AV block when electrical pacing is not available [31, 32]. The 2021 European Resuscitation

Council Guidelines support thump pacing as a bridge to electrical pacing in hemodynamically stable, conscious patients with bradyarrhythmias [33].

Successful thump pacing is as effective as transcutaneous or transvenous pacing [34]. Zoll [35] examined thump pacing in dogs and people and found that the force required to stimulate a heartbeat was 0.04–0.7 J in dogs and 0.04–1.5 J in people. The human subjects were able to tolerate thump pacing at this degree of force and complained of severe discomfort only when the force increased to 2–3 J. This chapter's previous author was able to use thump pacing to maintain normal heart rate and blood pressure in a large dog for 10 minutes while a temporary transvenous pacing lead was inserted. In the ECG shown in Figure 23.10, every QRS complex was initiated by a thump delivered to the chest. Each QRS produced a pulse wave that could be seen on a direct arterial pressure monitor. The heart rate dropped precipitously whenever thump pacing was stopped.

Transesophageal Pacing

Transesophageal pacing has not been widely used or extensively studied in clinical veterinary medicine, but the technique has been used successfully in dogs in research. Transesophageal pacing is minimally invasive and can be initiated quickly in an emergency. The transesophageal method has been shown in dogs to pace the atria, but unfortunately, it does not reliably pace the ventricles [36, 37]. Therefore, if the bradyarrhythmia is due to AV block or complete bundle branch block, another type of pacing method should be used. Once the atria are paced, the depolarization will produce a QRS complex similar to that of an intrinsic complex. Recent studies report that curved electrophysiology catheters with electrodes approximately 4 mm apart provide the best capture of the atria, with the least amount of extraneous muscle contraction [38, 39]. The sensed ECG of the transesophageal catheters can be used to optimally position the pacing catheter [40]. Transesophageal pacing is indicated when drug therapy is not effective in treating sinus bradycardia, sinus block, or sinus arrest, particularly during anesthesia. These bradyarrhythmias are often caused by sick sinus syndrome or by cholinergic or beta-blocking drugs. Generators used for transesophageal pacing now include a sensing function and operate in synchronous mode. Transesophageal pacing can cause patient movement by stimulating muscular contraction. See Protocol 23.3 for guidance on transesophageal cardiac pacing.

Protocol 23.3 Transesophageal Cardiac Pacing

Items Required

- Transesophageal pacing generator
- Transesophageal pacing lead
- Mouth gag
- ECG

Procedure

1) Collect necessary supplies.
2) Anesthetize the patient.
3) Attach the ECG to the pacing electrode catheter.
4) Insert the pacing electrode catheter into the esophagus until it reaches a point close to the atrium. It is assumed that the electrodes are closest to the atrium when the ECG shows maximum amplitude of the P wave.
5) Set the desired pacing rate.
6) Adjust the output of the pacing generator until the atrium is captured electrically and mechanically.

Transthoracic Pacing

The use of percutaneously inserted pacing electrodes dates back to at least 1958; if intracardiac injection of drugs was possible, it was also possible to insert a pacing electrode into the ventricle through a needle. Although the risk of injury associated with transthoracic pacing is significant, it can be started much more quickly in an emergency than transvenous pacing. Gessman [41] demonstrated the use of the technique experimentally in 24 dogs using a subxiphoid and parasternal approach. Although no acute deaths occurred, examination after euthanasia showed significant complications, including hemothorax, hemopericardium without tamponade, and laceration of a coronary vein.

As the reliability and comfort of transcutaneous pacing improved in the 1980s, the transcutaneous method essentially replaced transthoracic pacing. Today, transthoracic pacing is largely obsolete, and the equipment needed to perform the technique is difficult to obtain. However, it is possible to improvise an introducer and electrode. Roberts [42] described a method to make a pacing electrode using a 20-gauge spinal needle and 4–0 stainless steel suture wire. Transthoracic pacing should only be considered in extreme life-threatening situations when safer options have failed or are unavailable. See Protocol 23.4 for instructions on transthoracic pacing.

Protocol 23.4 Transthoracic (Transmyocardial) Cardiac Pacing

Items Required

- Transthoracic pacing kit with adapter or 4–0 stainless steel suture wire (two 40-cm pieces) and two 20-g spinal needles [42].
- Pulse generator
- ECG
- Sterile gloves
- Clippers
- Aseptic preparation of user's choice
- Bandage material to secure leads to body wall

Procedure

1) Transthoracic pacing is generally only performed in extreme life-threatening situations when the patient is unconscious.
2) Collect necessary supplies.
3) Shave and aseptically prepare the insertion site.
4) Perform hand hygiene and don sterile gloves.
5) Insert the needle percutaneously into either the left or right ventricular chamber. Use ultrasound guidance or insert blindly in the fifth intercostal space.
6) If using spinal needles and suture wire, thread a strand of suture through each spinal needle, and follow same instructions as step 5. The second needle is placed subcutaneously (SQ) near the cardiac apex. Remove the needle and skip to step 9.
7) Remove the obturator/stylet from the needle and insert the curved end of the pacing lead.
8) Once the curved portion of the pacing lead is inside the ventricle, withdraw the needle, leaving the pacing lead in place.
9) Use the adapter to connect the lead to the generator. The distal portion of the lead should be connected to the negative pole of the pulse generator. If using suture and spinal needle, attach the suture directly to the generator. Secure SQ lead to body wall with nonconductive bandage material.
10) Set the desired pacing rate on the generator.
11) Adjust the output of the pulse generator until the ventricle is captured both electrically and mechanically.
12) Determine whether the pacemaker is sensing intrinsic beats for demand pacing and adjust the sensitivity accordingly.

Nursing Care of the Patient Undergoing Temporary Pacing

The nursing care required for patients with temporary pacemakers depends on the type of pacing being performed. Patients being paced by invasive pacing techniques such as transvenous, temporary epicardial, or transthoracic pacing should have a dressing applied at the lead insertion site. Whenever the wires or catheters are covered with bandages it is possible to damage or sever them when the bandage is being removed. It is easy to dislodge the electrodes used for invasive pacing, so the cables and generator must be secured to the patient in a way that avoids tension if the patient moves or the generator is dropped. The use of a vest with pockets for the generator helps secure the generator to the patient.

Patients with invasive temporary pacemakers have a direct, low-resistance electrical pathway to the heart, which theoretically puts them at increased risk of ventricular fibrillation or other arrhythmias with only very small electrical shock impulses, called *microshocks*. Death due to electrical shock is uncommon and almost completely preventable. Staff who care for patients with invasive pacemakers should be aware of sources of microshock and how to avoid it. Faulty electrical equipment, current leakage, and static electricity are all potential sources of electrical current that could cause microshock [43]. Many modern medical devices use symbols to indicate when they may be safely used around a patient with a pacemaker (Figure 23.11). Class B equipment is designed to prevent injury if the equipment is attached to the patient's skin. Class C equipment is designed to be safe when equipment breaches the skin, such as pacing leads, central lines, and pulmonary arterial catheters [44]. See Box 23.2 for procedures used to prevent microshock in patients with pacemakers.

Monitoring the Patient with a Temporary Pacemaker

One of the most important parameters to monitor is the patient's pulse. Every stimulus produced by the generator should produce a pulse, and every intrinsic beat should

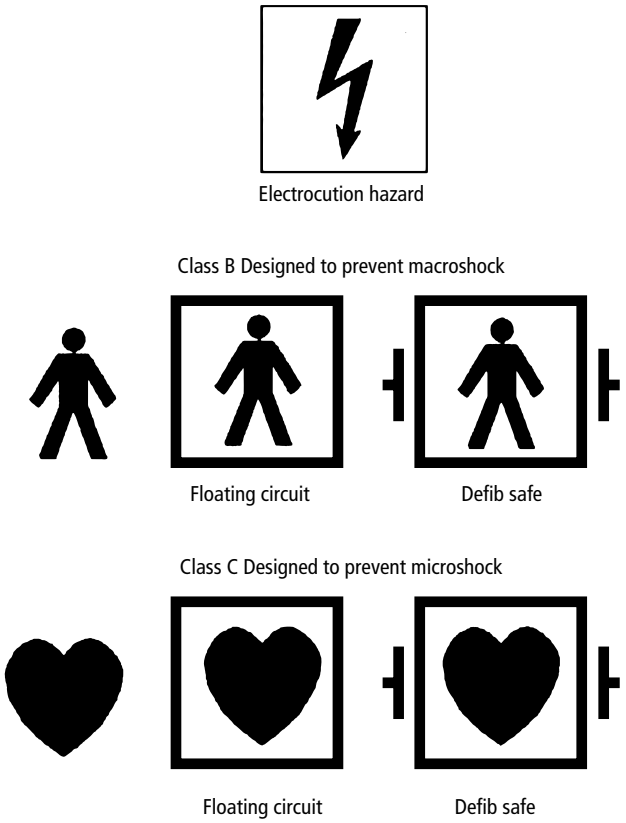


Figure 23.11 Symbols related to electrical safety. Some pieces of medical equipment bear symbols to indicate their safety for use around patients with pacemakers.

produce a pulse as well as be sensed by the pulse generator. Palpation of the apical impulse is ideal because there is minimal delay between mechanical capture and a palpable pulse. Auscultation of heart sounds, Doppler pulse detection, pulse oximetry, and/or direct arterial pressure waveform monitoring are also useful. A surface ECG is required to confirm whether the generator senses correctly, and that the output stimulus correctly captures the ventricle (or atrium). Every pacing spike should be immediately followed by a QRS complex indicating ventricular depolarization that is generally wider in duration than an intrinsic QRS complex.

The objective of pacing is to maintain adequate blood pressure and cardiac output, so it is important to monitor blood pressure, physical perfusion parameters, urine production, and plasma lactate concentration in these cases.

Troubleshooting

Failure to Capture

The pacing indicator shows that the pacemaker has produced a stimulus. The ECG may show a pacing spike, but the stimulus does not produce a ventricular complex (no QRS complex on the ECG) or a contraction (no palpable pulse). See Table 23.3 for recommendations on troubleshooting capture failure.

Undersensing

Undersensing is the pacemaker's failure to detect intrinsic beats: the pacemaker does not recognize or pause when intrinsic beats occur. The ECG may show a pacing spike in the QRS or T wave. See Table 23.4 for troubleshooting tips for undersensing.

Oversensing

Oversensing is used to describe the situation in which the pacemaker interprets non-ventricular sources of electrical signal as a ventricular depolarization, and therefore fails to deliver an output impulse as scheduled. During oversensing, the pulse generator indicates that the patient has produced a heartbeat when it has not. If the pacemaker is in demand mode, pacing will pause. The ECG will not show a QRS complex at the time the generator sensed a beat. Instead, a P wave, T wave, muscle tremor, panting, electrocautery, or some other

Box 23.2 Procedures Used to Prevent Microshock [44–46]

- Identify electrically sensitive patients. Some institutions post a warning sign on the patient's cage.
- Never use damaged or poorly maintained electrical equipment. Avoid using extension cords.
- Wear rubber gloves whenever the leads or terminals of the pacemaker must be touched.
- Pacing leads should always be insulated whenever they are not connected to a pacing generator. They should never be allowed to touch electrically conductive or wet surfaces.
- Water, urine, and other fluids can conduct electricity. Keep the patient and pacing equipment as dry as possible.
- Electrically powered devices that come into contact with patients such as clippers, fans, and warming devices can be dangerous.
- Touching a grounded metal object before touching a patient and touching the patient away from the leads or pacemaker first, reduces the risk of microshock from static electricity.
- Certain modern electrical devices designed to be in contact with a patient are labeled to show whether they are safe to use around electrically sensitive patients and patients that may require defibrillation (Figure 23.11).

Table 23.3 Troubleshooting capture failure. There may be more than one cause of capture failure, and troubleshooting may be trial and error.

Possible cause of capture failure	Troubleshooting method
Transvenous electrode is not in contact with the heart	Reposition the electrode
Transcutaneous electrode is not in contact with the skin	a) Use a bandage to hold electrode against the skin b) Be sure skin has been properly prepared c) Replace electrode patch if necessary
Polarity of the electrode is reversed	Switch the lead's connections to the pulse generator. Confirm that bipolar pacing lead is used with bipolar or unipolar pulse generator (unipolar pacing leads will not pace using a bipolar pulse generator)
Connections are loose	Inspect and tighten connections to the pulse generator
Battery has failed	Replace the battery in the pulse generator
Output is inadequate	Increase the output stimulus from the pulse generator
The heart cannot respond to stimulus	Assess for electrolyte abnormality. Consider reversing any cardiovascularly depressive drugs. If non-responsive and apneic, start CPR
The ECG shows a QRS is produced by pacemaker, but no palpable pulse found.	Increase the output stimulus of the pulse generator. If this does not produce a pulse, treat as if the patient has pulseless electrical activity, and initiate CPR

Table 23.4 Troubleshooting undersensing.

Possible cause of undersensing	Troubleshooting method
Sensitivity control is set too high	Lower the sensitivity setting
Transvenous electrode is not in contact with the heart	Reposition the electrode
Poor-quality ECG or improper lead selection for transcutaneous pacing	Remove sources of interference. Ensure ECG electrodes have adequate patient contact. Select an ECG lead that produces large QRS complexes
Pacemaker is set for asynchronous pacing	Switch to demand mode
Intrinsic activity occurring within the pacemaker refractory period	Reset pulse generator refractory period for a shorter duration

Table 23.5 Troubleshooting oversensing.

Possible causes of oversensing	Troubleshooting method
Electrical interference is present	Identify and eliminate the source of the interference
Sensitivity setting is too low	Increase the sensitivity setting
Low-amplitude QRS complex is lost in noise in the lead selected for transcutaneous pacing	Select an ECG lead with a larger QRS amplitude for sensing
P waves or T waves are larger than QRS in lead selected for transcutaneous pacing	Select an ECG lead with a larger QRS amplitude for sensing
Generator appears to be sensing P waves in transvenous pacing	The pacing lead may not be in the apex of the right ventricle. Check lead position and reposition as necessary

form of electrical interference may be visible on the ECG. See Table 23.5 for methods of troubleshooting oversensing.

Summary

Temporary cardiac pacing can be a life-saving measure for patients with or at risk of severe bradyarrhythmia. There are many different methods by which temporary pacing can be achieved, some of which are simple and relatively noninvasive considering the benefit. Temporary pacing has few complications, most of which can be remedied with basic care and troubleshooting procedures.

Acknowledgment

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Section Three

Respiratory Procedures and Monitoring

24

Oxygen Therapy

Kate Farrell

Respiratory distress is a common presenting complaint to the emergency room and a frequent cause for admission to hospitals and intensive care units. Oxygen therapy plays a crucial role in treatment of patients with hypoxemia and respiratory failure. Responsiveness to oxygen is dependent on the patient's underlying disease process. As oxygen is typically benign in the short term, supplementation is essential for patients exhibiting signs of respiratory distress during triage and stabilization. Oxygen administration can result in complications, and thus judicious use and appropriate concentrations are warranted in the long term. There are numerous techniques for the administration of oxygen, and the best method for supplementation will depend on multiple patient factors and equipment available.

Normal Oxygenation and Hypoxemia

Oxygen is critically important for cellular metabolism and energy production. To reach the cellular level, oxygen must travel down its partial pressure gradient starting from the atmosphere. The atmosphere is composed of 20.9% oxygen. The partial pressure of oxygen (PO_2) in dry air at sea level is 21.2 kPa (159 mmHg), and this partial pressure decreases with elevation [1]. Oxygen and other gases from the atmosphere travel down the airways by bulk flow into the alveoli, where oxygen diffuses across the respiratory membrane into the plasma. Oxygenated blood travels from the arteries to the capillaries, and oxygen diffuses into the tissues to the level of the mitochondria, where it is consumed.

Oxygen is carried in the blood in two forms: (i) as dissolved gas; and (ii) bound to hemoglobin in red blood cells. The dissolved portion is measured as the partial pressure of oxygen in arterial blood (PaO_2) and encompasses only 2–3% of the total oxygen content of arterial blood in health [2]. The bulk of oxygen is carried by hemoglobin, and the saturation of hemoglobin with oxygen in arterial blood (SaO_2) is directly related to PaO_2. The oxygen content of arterial blood (CaO_2) is dependent on PaO_2, SaO_2, and the concentration of hemoglobin. The arterial oxygen content equation is noted in Box 24.1.

The term *hypoxemia* refers to inadequate oxygenation of arterial blood, and this is defined by a PaO_2 of less than 80 mmHg. The term *hypoxia*, on the other hand, refers to decreased oxygen at the level of the tissues. Oxygen delivery to cells is dependent on both CaO_2 and cardiac output; this equation is noted in Box 24.1. While total cardiac output affects global oxygen delivery, transport to individual organs is also affected by distribution of blood flow. Hypoxemia is dangerous because it reduces CaO_2 and can result in hypoxia.

Causes of hypoxemia include three major categories: (i) low partial pressure of inspired oxygen; (ii) hypoventilation; and (iii) venous admixture. Venous admixture encompasses ventilation/perfusion (V/Q) abnormalities, right-to-left anatomic shunts, and diffusion defects. The most common of these causes to result in hypoxemia and respiratory distress in veterinary patients is V/Q mismatch, which can include low V/Q regions in which alveoli are perfused but inadequately ventilated, or it can include no V/Q regions, also known as physiologic or intrapulmonary shunt. Ultimately, the response to oxygen supplementation is dependent on the underlying mechanism of hypoxemia. Most causes of hypoxemia are readily improved with oxygen administration. The exceptions include right-to-left anatomic shunts and diseases resulting in intrapulmonary shunting; with both processes, blood bypasses gas exchange units where oxygenation can occur [1, 3].

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Box 24.1 Equations for Oxygen Delivery and Arterial Oxygen Content

Oxygen Delivery

DO_2 (ml O_2/minute) = Q (ml/minute) × CaO_2 (ml O_2/dl) × 10

- DO_2 = oxygen delivery
- Q = cardiac output
- CaO_2 = arterial oxygen content

Arterial Oxygen Content

CaO_2 (ml O_2/dl) = [1.34 (ml O_2/g) × SaO_2 (%) × Hb (g/dl)] + [PaO_2 (mmHg) × 0.003 (ml O_2/dl/mmHg)]

- SaO_2 = arterial hemoglobin saturation with oxygen
- Hb = hemoglobin
- PaO_2 = partial pressure of oxygen in arterial blood

Indications for Oxygen Supplementation

The aim of oxygen supplementation is to elevate the fraction of inspired oxygen (FiO_2) to cause an increase in PaO_2 and SaO_2, which results in an increase in the content of arterial oxygen and thus increased oxygen delivery to the tissues. The decision to supply oxygen to a patient may be dependent on clinical signs, objective data indicating hypoxemia, or evidence of decreased oxygen delivery to tissues.

Clinical signs of respiratory distress may include, but are not limited to, increased respiratory rate and effort, open-mouth breathing, extended head and neck, abducted elbows, retraction of the lip commissures or "fish-mouth" breathing, nasal flare, severe stertor or stridor, shallow chest movements, recruitment of abdominal muscles for breathing, paradoxical movement of the chest wall and abdomen, abnormal lung sounds (crackles, wheezes, absent lung sounds), or evidence of respiratory fatigue or exhaustion.

When assessing for hypoxemia, supplemental oxygen is often indicated in patients with a PaO_2 less than 70 mmHg, an SaO_2 less than around 93%, or cyanosis [4, 5]. Cyanosis, or the blue discoloration of mucous membranes secondary to significant quantities of deoxygenated hemoglobin, is indicative of severe hypoxemia. Unfortunately, its absence does not rule out hypoxemia. It must be noted that in chronic disease, a patient may tolerate a significantly lower PaO_2 than a patient that is acutely hypoxemic. If there is concern for a disease process that may be contributing to hypoventilation, assessment of the partial pressure of carbon dioxide in the blood (PCO_2) is required, and oxygen supplementation is typically recommended for patients with a PCO_2 greater than 55–60 mmHg.

Decreased oxygen delivery to the tissues can also be caused by severe anemia, dyshemoglobinemias (elevated carboxyhemoglobin or methemoglobin), and poor tissue perfusion. Additionally, increased oxygen demand in the tissues can occur in disease states resulting in elevated metabolic rate or body temperature, such as sepsis, prolonged seizures, and heat stroke [4]. Therefore, under certain circumstances, patients with these abnormalities may also benefit from oxygen supplementation, even if they are not hypoxemic.

Methods of Oxygen Supplementation

Once it has been determined that a patient requires oxygen supplementation, there are multiple methods of delivery that vary in their level of invasiveness. The technique utilized will depend on multiple factors, including length of supplementation needed, patient requirements and tolerance, patient size, severity of hypoxemia, degree of FiO_2 required, equipment available, and experience and skill of the clinical team [6]. The oxygen source for each technique discussed may vary and can include portable oxygen tanks or a central source with wall- or ceiling-mounted outlets. A tank or central oxygen source may also be connected to an anesthesia circuit for delivery of oxygen.

In each of the techniques discussed below, some form of humidification is required to avoid drying and irritation of the respiratory mucosa. This is less critical for short-term therapy, such as during the administration of flow-by or face mask oxygen while a patient is initially being assessed. However, long-term desiccation can result in irritation, mucosal damage, impaired function of the mucociliary apparatus, and increased potential for infection [4, 6, 7]. While some methods may have built-in humidification techniques, such as designed in commercial oxygen cages and in heated humidified high-flow machines, most supplemental oxygen sources will need humidifying by bubbling air through a bottle of sterile water or saline. It is important that the water is sterile to avoid contamination of the respiratory tract with nosocomial infections. Warming delivered oxygen will also help improve humidification. Bubble humidifiers are commercially available (Figure 24.1).

Flow-by Oxygen

Flow-by oxygen is administered by placement of a tube connected to an oxygen source in close proximity to a patient's nose if nasal breathing or mouth if open-mouth breathing (Protocol 24.1; Figure 24.2). The oxygen source



Figure 24.1 A bubble humidifier connected to an oxygen source.

can be a central source, portable tank, or anesthesia machine. Flow-by provides a simple and rapid mechanism for administration of oxygen to a patient in respiratory distress. Most animals will tolerate flow-by tubing, and oxygen can be provided easily while a clinician is initially evaluating and treating a patient. Flow-by oxygen administered to healthy, anesthetized dogs supplied 2 cm from the nose with a flow rate of 2 l/minute was able to achieve a mean FiO_2 of 37.2% (range 25–48%) [8]. However, awake dyspneic dogs may not tolerate consistent flow-by oxygen close to the nose, which can preclude appropriate maintenance of oxygenation. This technique is not practical or economical long-term given the fact that constant supervision is required and significant quantities of oxygen are wasted into the surrounding environment.



Figure 24.2 Oxygen is provided for a trauma patient using a flow-by technique.

Face Mask

Oxygen administration via a face mask is equally simple to flow-by methods and can provide convenient oxygen during triage and initial treatment of a patient (Protocol 24.1). Delivery is similar to flow-by oxygen, but a face mask is placed at the end of the oxygen tubing (Figure 24.3a). Some masks are also designed to function as muzzles (or a muzzle can be secured around a face mask), and these can be attached to a patient to facilitate ease of oxygen administration and to free up an attendant to perform other functions (Figure 24.3b). Higher oxygen concentrations can be achieved compared to the flow-by technique [8, 9]. With a tight-fitting face mask in healthy anesthetized dogs, an oxygen flow rate of 0.5 l/minute provided a mean FiO_2 of 46.5% (range 30–70%) [8]. The FiO_2 provided by a face mask in this study was consistently higher than that produced with flow-by at equivalent oxygen flow rates. In another study, oxygen delivered at 3 l/minute to healthy sedated dogs achieved significantly higher PaO_2 values when provided via face mask compared to flow-by (mean PaO_2 of 371.3 mmHg with a face mask, 182.2 mmHg with

Protocol 24.1 Flow-by or Face-Mask Oxygen Setup and Application

Equipment Required

- Oxygen source with regulator
  - Wall- or ceiling-mounted central source
  - Oxygen tank
  - Anesthetic machine (with central source or oxygen tank)
  - Oxygen tubing or hose
- Face mask ± muzzle (if desired) of appropriate size

Procedure

1) Collect necessary supplies.
2) Hold tubing 2 cm in front of the patient's face and adjust the flowmeter of the oxygen source as desired, with a minimum of 2–3 l/minute.
3) If a mask is used, connect this to the oxygen source and place closely around the patient's muzzle, ensuring some escape of air. A muzzle may be attached to the patient's head to allow hands-free use.



Figure 24.3 (a) Oxygen is administered to a patient using a face mask connected to oxygen tubing. (b) A commercial face mask designed as a muzzle is used to deliver hands-free oxygen, allowing personnel to be freed to perform other tasks. A nylon dog muzzle may also be placed around a face mask and secured to a patient to function similarly.

flow-by, and 82.4 mmHg on room air) [9]. In general, lower flow rates may be adequate for smaller dogs while higher flow rates will be required for larger dogs. With a loose-fitting face mask, higher oxygen flow rates of 2–5 l/minute (or up to 5–10 l/minute in large dogs) have been suggested to meet peak inspiratory needs of patients in respiratory distress and to minimize rebreathing of exhaled carbon dioxide (CO_2) [4, 6]. Unfortunately, not all patients will tolerate a face mask and can become more panicked and tachypneic. Care must be taken to ensure no damage to the eyes with positioning of the face mask, and, importantly, enough venting from the face mask must be provided to prevent rebreathing of CO_2.

Oxygen Hood or Elizabethan Collar

Multiple types of oxygen hoods are manufactured commercially for purchase (Figure 24.4a), but they can also be made cheaply and easily in hospital with the use of a snug-fitting Elizabethan collar (E-collar), cling wrap, and tape (Figure 24.4b). In general, this technique requires minimal equipment, is non-invasive, allows access to the patient, and has the potential to achieve high FiO_2 values. To assemble a hood in hospital, an E-collar can be placed around a patient's neck, and the front of the E-collar can be covered with clear plastic cling wrap. An oxygen line is positioned inside the collar on the neck and adhered to the hood. Very importantly, a small opening must be made in the plastic wrap over an E-collar or the zipper of a commercially available hood must be opened to ensure venting of CO_2 and escape of heated and humidified air (Protocol 24.2). While this is accepted in many patients, not all will tolerate an oxygen hood and there is the potential for overheating and CO_2 rebreathing if a patient is not carefully attended to. The oxygen concentration that can be achieved in the hood is variable and dependent on patient size, minute ventilation, fit of the collar, and size of the vent. An oxygen hood should initially be filled at a higher rate (at least 1–2 l/minute), and then flow rates of 0.75–1 l/minute can be used [4]. In healthy anesthetized dogs, an oxygen flow rate of 1 l/min into an E-collar hood provided a mean tracheal FiO_2 of 40.6% (range 29–56%) [10]. An additional study demonstrated that the E-collar method could achieve an FiO_2 of 95% with a 300 ml/kg/minute flow rate in dogs [11].

Oxygen Cage

Oxygen cages offer multiple advantages, including administration of oxygen in a non-invasive and non-stressful manner with the ability to achieve high FiO_2 values typically up to 60% [4, 6]. Oxygen delivery into a cage is hands-free, convenient for patients presenting in respiratory distress when minimal handling is desired, and also easy for long-term administration of oxygen. Commercial cages allow venting of CO_2 and control of FiO_2, temperature, and



Figure 24.4 (a) Tubing secured at the collar provides oxygen to a commercial hood. Note small holes present in the oxygen hood that provide venting for carbon dioxide and escape of heated and humidified air. (b) An oxygen hood can also be made with an E-collar, clear plastic wrap over the front of the cone, and oxygen tubing adhered within the hood. A sufficient gap in the plastic wrap must also allow heat and CO_2 to escape.

Protocol 24.2 Oxygen Hood or Elizabethan Collar Setup and Application

Equipment Required

- Oxygen source with regulator
  - Wall- or ceiling-mounted central source
  - Oxygen tank
  - Anesthetic machine (with central source or oxygen tank)
- Oxygen tubing or hose
- Commercially available oxygen hood or E-collar
- Clear plastic wrap (if using an E-collar)
- Adhesive tape

Procedure

1) Collect necessary supplies.
2) Place an oxygen hood or E-collar over the patient's head and secure to the neck.
3) Position oxygen tubing/hose under the patient's collar at the neck and secure to the inside of the hood with adhesive tape.
4) If a commercially available hood is used, zip the front most of the way closed, leaving approximately 25% of the zipper open.
5) If an E-collar is used, cover the front of the E-collar with clear plastic wrap, leaving an opening for carbon dioxide, moisture, and heat to escape. Secure the plastic wrap in place with adhesive tape.
6) Fill the hood with oxygen rapidly initially and then use flow rates of 0.75–1.0 l/minute at a minimum depending on patient size and oxygen requirements.
7) An oxygen monitor can be used to determine the FiO_2 within the hood if desired.

humidity (Figure 24.5). Optimal temperature is 70°F (22°C) with a humidity of 40–50% [4, 6]. Ideally, cages have a transparent Plexiglas front to enable visualization of the patient, access ports for intravenous lines and monitoring equipment, and small doors or sleeves to allow manipulation of a patient without significant oxygen loss. An oxygen cage does limit hands-on access to a patient to some degree, but pulse oximetry, electrocardiogram, and blood pressure monitoring can all be added as needed to enhance patient monitoring. Disadvantages include declining FiO_2 when the cage is opened, oxygen waste, expense of a commercial cage, and potential inability to accommodate large patients. If the oxygen cage door requires opening for further triage, stabilization, or completion of a procedure, additional flow-by or face mask oxygen may be required.

Smaller portable and collapsible oxygen cages are available for purchase and can be used for short-term oxygen administration or transport (Figure 24.6). Temporary oxygen cages can also be easily manufactured from an incubator (Figure 24.7) or a pet carrier covered with a plastic bag (Figure 24.8) with placement of an oxygen hose into the cage. However, caution must be exercised to always provide ventilation. If there is no mechanism for outflow of gas, CO_2 can accumulate and result in rebreathing. An additional potential complication is the risk for heat accumulation. Patients should be checked frequently to ensure



Figure 24.5 A commercial cage allows hands-free, non-invasive delivery of oxygen with control of FiO_2, temperature, humidity, and ventilation.



Figure 24.6 Oxygen is administered to a patient in a commercial transportable oxygen cage, and an oxygen sensor is used to monitor the FiO_2 within.

that they have not become hyperthermic and that cage temperature is not excessive. Even in commercial cages with temperature control, large patients can overheat. Ice packs can be placed within a cage as needed for cooling, but care should be taken to avoid direct contact with the patient. Commercial O_2 sensors, CO_2 monitors, and thermometers can all be added to temporary cages to improve monitoring and safety.

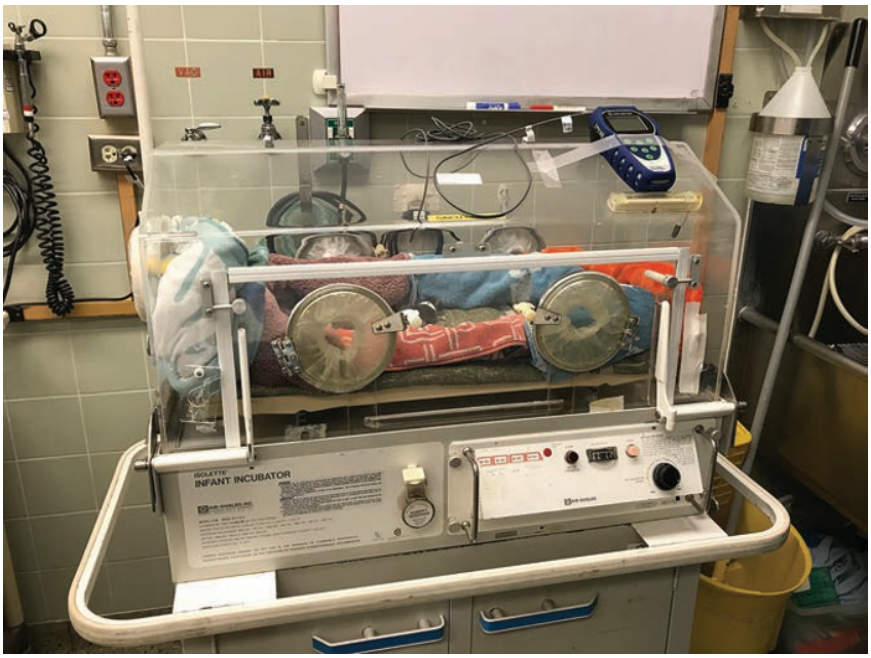


Figure 24.7 A human infant incubator is adapted for oxygen use by delivery of oxygen through tubing into the cage.

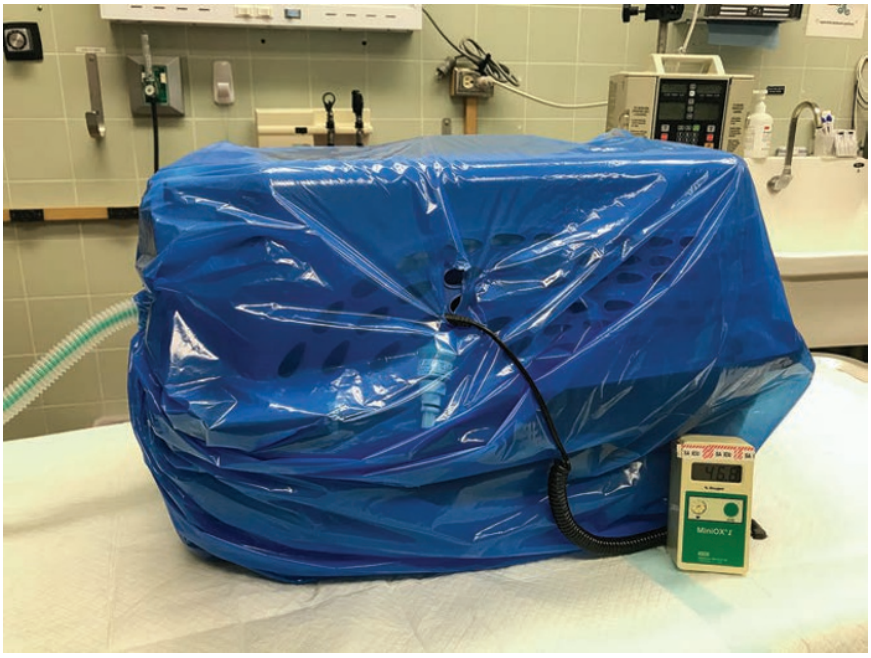


Figure 24.8 A pet carrier or box can be covered with a plastic bag with oxygen delivered via a hose or tube to make a temporary oxygen cage. A hole must be made in the plastic bag to allow for venting of CO_2, and care must be taken to ensure a patient does not overheat. An oxygen sensor here displays FiO_2.

Nasal Oxygen

Nasal prongs or cannulas are also an effective delivery method for oxygen to patients that will need more prolonged oxygen administration. Nasal oxygen is advantageous because it is generally well tolerated, easy to provide, and can allow patients greater mobility and accessibility for treatments and monitoring [7, 12]. It is also typically more economical and less wasteful than flow-by oxygen or oxygen cages. It may provide a good option for patients that are too large for an oxygen cage. This method is not indicated for patients with facial trauma or respiratory signs

attributed to nasal obstruction or other upper airway disease, such as brachycephalic breeds with obstructive airway syndrome. This technique may also be contraindicated in coagulopathic patients that are more prone to mucosal hemorrhage. Adverse effects can include gastric distension at very high flow rates, nasal irritation or inflammation, discharge, sneezing, and epistaxis, which may preclude use in some animals [7, 13]. If local nasal irritation occurs, the catheter can be relocated to the other nare to reduce jet lesions on the mucosa, avoid pressure necrosis, and prevent occlusion of the tube with mucus [13]. Humidification of inspired air is recommended to reduce local irritation and improve patient comfort.

It has been demonstrated that there is a nearly linear relationship between increases in nasal oxygen flow rate and concurrent FiO_2 and PaO_2 [7, 14]. Previous studies in dogs have shown that using unilateral nasal catheters with flow rates of 50–100 ml/kg/minute can increase tracheal FiO_2 up to 50% [7, 10, 15]. By using one or two nasal catheters, flow rates of 50–400 ml/kg/minute can provide tracheal FiO_2 in the range of 30–77% [14]. The advantage to the use of bilateral nasal catheters is the ability to reduce flow rates in each side, as flow rates above 100 ml/kg/minute have been shown to cause discomfort [14]. The nasal passages are unable to adequately heat and humidify incoming air at higher flow rates. Administration of oxygen at a flow rate of 100 ml/kg/minute in bilateral nasal catheters can achieve an FiO_2 of 60% while avoiding patient discomfort and risk of oxygen toxicity [14]. Oxygen concentration will vary if a patient is panting or open-mouth breathing. These studies have evaluated the use of nasal catheters, but unfortunately little data exists in patients with the use of nasal prongs. It is likely that FiO_2 with be lower with nasal prongs than values that can be achieved with nasal catheters, but this could be variable and dependent on patient conformation, compliance, and frequency of dislodgement.

Placing nasal prongs or cannulas can be facilitated by the use of topical anesthetics such as 2% lidocaine or proparacaine in the nares prior to placement. Human nasal prongs, which can be purchased from medical suppliers online, are particularly easy to place and may be less stressful than the placement of cannulas (Protocol 24.3). The prongs are approximately 1 cm in length but can be cut shorter as needed for the patient. The tubing associated with the prongs can be tightened behind the ears to secure them, and tape across the bridge of the nose can help to stabilize them (Figure 24.9). However, depending on the anatomy of the patient, their fit may be variable, they can be dislodged easily, and the FiO_2 they provide is unknown, so close monitoring is warranted. An E-collar may help to prevent patient removal of nasal prongs.

Nasal cannulas or catheters are often placed into the nasal cavity by premeasuring tubing from the nose to the medial canthus of the eye (Figure 24.10), but they can be placed into the nasopharyngeal region by measurement from the nose to the ramus of the mandible. After

Protocol 24.3 Nasal Prong Setup and Application

Equipment Required

- Oxygen source with regulator
  - Wall- or ceiling-mounted central source
  - Oxygen tank
  - Anesthetic machine (with central source or oxygen tank)
  - Heated humidified high-flow nasal oxygen machine
- Nasal prongs set appropriate for the patient size or high-flow nasal cannula (HFNC) set
- 2% lidocaine or proparacaine ophthalmic drops
- ½–1 inch adhesive medical tape
- Nonabsorbable suture material and suturing instruments, or skin staple gun
- Additional oxygen tubing as needed
- Christmas tree adapter, 1-ml cut syringe or other adapter as needed for connection to oxygen source
- Bubble humidifier
- E-collar

Procedure

1) Collect necessary supplies.
2) Place a few drops of 2% lidocaine or proparacaine ophthalmic drops into the nose and wait a few minutes for the local anesthetic to take effect. This step may be less necessary than for placement of a nasal catheter. Administer less than 2 mg/kg lidocaine in dogs and 1 mg/kg lidocaine in cats.
3) Seat the nasal prongs within each nare. The ends of the prongs can be cut as needed for patient conformation. The prongs should not fill the entire opening of the nares.
4) Tighten the tubing associated with the prongs behind the patient's ears to secure them. Connect the tubing across the bridge of the nose with adhesive tape to help stabilize the prongs.
5) The tubing of the prongs can also be positioned and stabilized by suturing or stapling butterfly tabs to the skin on either side of the nares.
6) Attach the tubing to an oxygen source and bubble humidifier. Use additional tubing and connectors as needed.
7) Place an E-collar on the patient.



Figure 24.9 Nasal prongs are secured and stabilized by tightening tubing behind the ears and placing tape across the bridge of the nose. The tubing for the nasal prongs is attached to an oxygen source.



Figure 24.10 A red rubber catheter is measured to the level of the medial canthus of the eye and marked at the tip of the nose prior to placement into the ventral nasal meatus for delivery of nasal oxygen.

treatment with a topical anesthetic, a lubricated rubber catheter (size 3.5–10 Fr depending on the size of the patient) can be placed into the ventral nasal meatus (angling ventromedially) to the premeasured distance (Protocol 24.4). Multiple fenestrations at the end of the catheter will help to avoid jet lesions on the mucosa. The tubing should be secured with suture immediately adjacent to the nostril with a finger-trap or other suture pattern and secured again to the side of the face or forehead with sutures, staples, or an adhesive agent such as cyanoacrylate (Figure 24.11). The patient can then be connected to oxygen via the shortest and widest tubing possible to allow the least resistance to airflow.

Transtracheal Oxygen

Oxygen can be administered directly into the tracheal via a percutaneous catheter, thus bypassing the upper airway. This may be a more effective technique for patients that have an upper airway obstruction, are panting, or do not tolerate other methods. Transtracheal oxygen is reported to be well tolerated in dogs and cats, provides easy access to patients, and can achieve higher FiO_2 and PaO_2 at lower flow rates than nasal insufflation [15]. A flow rate of 50 ml/kg/minute through a transtracheal catheter can provide an FiO_2 of 40–60%, similar to a flow rate of 100 ml/kg/minute through a nasal cannula [15]. As this technique can provide FiO_2 above 60%, close monitoring of PaO_2 and adjustment to the lowest required flow rate is important to reduce the risk of oxygen toxicity. Disadvantages of this technique include difficulty in placement and maintenance and the requirement for constant monitoring and avoidance of kinking or displacement of a catheter. Complications can include tracheal irritation, subcutaneous emphysema, and obstruction of the catheter with mucus or secretions. This method should be avoided in patients with tracheal

Protocol 24.4 Nasal Catheter Setup and Application

Equipment Required

- Oxygen source with regulator
  - Wall- or ceiling-mounted central source
  - Oxygen tank
  - Anesthetic machine (with central source or oxygen tank)
- Red rubber catheter or infant feeding tube (size 3.5–10 Fr depending on patient size)
- 2% lidocaine or proparacaine ophthalmic drops
- Lubricating jelly or lidocaine gel
- ½–1 inch adhesive medical tape
- Non-absorbable suture material and suturing instruments, or skin staple gun
- Additional oxygen tubing as needed
- Christmas tree adapter, 1 ml cut syringe, or other adapter as needed for connection to oxygen source
- Bubble humidifier
- E-collar

Procedure

1) Collect necessary supplies.
2) Place a few drops of 2% lidocaine or proparacaine ophthalmic drops into the nose and wait a few minutes for the local anesthetic to take effect. Administer less than 2 mg/kg lidocaine in dogs and 1 mg/kg lidocaine in cats.

3) Premeasure the catheter to the medial canthus of the eye (if placement in the nasal cavity is desired) or to the ramus of the mandible (if placement in the nasopharynx is desired). A marker or piece of adhesive tape can be used to designate the premeasured distance for placement. If a patient does not tolerate placement in the nasal cavity, attempt advancement into the nasopharynx for improved comfort. The catheter should not fill the entire opening of the nare(s).
4) Catheters with premade fenestrations can be purchased, or additional fenestrations can be made in the distal end of the nasal catheter if desired to help prevent jet lesions on the mucosa. This can be performed if high flow rates are anticipated.
5) Apply lubricating jelly or lidocaine gel to the end of the catheter to facilitate smooth placement into the nasal passage.
6) Restrain the patient with an elevated head. Direct the catheter ventromedially to place it into the ventral nasal meatus of the nasal cavity. This may be facilitated by gently pushing the nasal philtrum dorsally. Advance carefully and expect some nasal irritation. If significant resistance if met, withdraw the catheter and re-direct to avoid causing epistaxis by hitting the ethmoid turbinate if located in the dorsal nasal meatus. Advance to the premeasured location.
7) Secure the tubing with suture immediately adjacent to the nostril with a finger-trap or other suture pattern.
8) Additionally secure the tubing to the side of the face and/or forehead with an adhesive agent (e.g. cyanoacrylate) or sutures/staples applied to butterfly tape tabs on the tubing.
9) Attach the catheter to an oxygen source and bubble humidifier. Use additional tubing and connectors as needed.
10) Start with a flow rate of 50–100 ml/kg/minute and adjust as needed.
11) Place an E-collar on the patient.



Figure 24.11 The bilateral nasal catheter tubing is sutured adjacent to the nares with a finger-trap pattern and sutured to the forehead with the use of adhesive tape butterflies in this patient. Tubing may also be secured to the side of the face.

collapse or coagulopathies. Because this technique bypasses the upper airway, humidification is necessary to reduce tracheal irritation.

To place a transtracheal catheter, a patient's ventral neck is clipped and aseptically prepped (Protocol 24.5). A 2% lidocaine bleb and nick incision through the skin can be performed prior to placement of the catheter. Sedation may be required in some patients to allow placement. A large-gauge over-the-needle or through-the-needle catheter is placed percutaneously between two tracheal rings caudal to the larynx and directly into the trachea, angling caudally toward the carina. The needle is removed and the catheter is secured and attached to a humidified oxygen source with a light wrap around the neck.

Heated Humidified High-Flow Nasal Oxygen

In human medicine, a non-invasive oxygen delivery method known as high-flow nasal cannula (HFNC) has emerged over the past decade as an alternative to conventional oxygen therapy and has been investigated as a method to treat hypoxemic respiratory failure and potentially avoid invasive mechanical ventilation [16–20]. HFNC has more recently been adapted and employed for use in dogs. This method is advantageous because it is able to achieve flow rates up to 40–60 l/minute and can more reliably deliver high FiO_2. HFNC systems use an air-oxygen blender connected to a flow meter that can deliver an FiO_2 of 21–100%. The air is heated and humidified, then delivered to the patient through a heated breathing circuit and specialized nasal prongs sized to occlude about 50% of the nares. The system allows 100% humidification and control of temperature, thereby enhancing patient comfort and tolerance of these high flow rates while preventing airway desiccation, epithelial injury, airway constriction, and impairment of mucociliary function. High flow rates reduce entrainment of

Protocol 24.5 Transtracheal Oxygen Setup and Application

Equipment Required

- Oxygen source with regulator
  - Wall- or ceiling-mounted central source
  - Oxygen tank
  - Anesthetic machine
- Clippers with a clean blade
- Surgical aseptic preparation materials
- Sterile gloves
- 2% lidocaine
- #11 scalpel blade (if desired)
- Large-gauge over-the-needle catheter, through-the-needle catheter, or commercial tracheal catheter
- ½–1 inch adhesive medical tape
- Non-absorbable suture material and suturing instruments, or skin staple gun
- Oxygen tubing
- Christmas tree adapter or other adapter as needed for connection to oxygen source
- Bubble humidifier

Procedure

1) Collect necessary supplies.
2) Palpate the patient's ventral cervical region to locate a proposed insertion site. The transtracheal catheter is typically inserted on ventral midline between tracheal rings, usually between the third and fifth tracheal rings caudal to the cricoid cartilage depending on patient conformation.
3) Measure the distance from the proposed insertion site to the level of the carina, located at the fourth to fifth intercostal space or caudal border of the scapula. The catheter length should not exceed this distance, as passage too far distally could result in airway damage or penetration.
4) Clip the fur from just caudal to the larynx to the thoracic inlet, as well as laterally 3–5 cm off midline on both sides. Aseptically prepare the patient's ventral neck.
5) Place a small bleb of 2% lidocaine subcutaneously at the proposed insertion site and allow time for it to take effect. Administer less than 2 mg/kg lidocaine in dogs and 1 mg/kg lidocaine in cats.
6) A small nick incision with a #11 scalpel blade can be performed to decrease drag and facilitate smooth placement of the catheter.
7) Aseptically prepare the skin again.
8) Put on sterile gloves after appropriate hand hygiene.
9) Relocate the insertion site, ensuring placement on midline between tracheal rings. Using aseptic technique, insert the catheter into the trachea and direct caudally toward the carina. A pop may be felt as the needle enters the tracheal lumen. Once in the trachea, the catheter can be advanced gently without the needle until it is buried to its hub. The needle is then removed and the catheter left in place.
10) Secure the catheter to the neck by applying an adhesive tape butterfly to the hub and attaching it to the patient with stay sutures or staples. Ensure the catheter does not kink at its insertion site.
11) Connect the catheter to a humidified oxygen source, with additional tubing and adapters as needed.
12) Start with a flow rate of 50 ml/kg/minute and adjust as needed.
13) Place a loose wrap around the patient's neck for security and cleanliness.

room air, enable washout of upper airway dead space, and may generate some degree of continuous positive airway pressure (CPAP) with a closed mouth. CPAP can be achieved in healthy awake and sedated dogs using HFNC with flow rates of 1–2 l/kg/minute.[21]

While data are somewhat limited in veterinary species, initial studies in both healthy dogs and hypoxemic dogs assessed to be failing traditional oxygen therapy have shown HFNC to be efficacious for achieving higher PaO_2 compared with traditional oxygen therapy [21–24]. These studies have also demonstrated good tolerance and patient comfort with minimal safety concerns [21–24]. Complications include mild discomfort in patients especially at high flow rates (above 2 l/kg/minute), aerophagia, and gastric distension [21–23]. One patient experienced persistence of a pre-existing traumatic pneumothorax, which resolved upon discontinuation of HFNC; it is unknown whether the pneumothorax could have been exacerbated by HFNC [22]. Caution may be required in patients with hypercapneic respiratory failure, as PCO_2 has been shown to increase slightly, with possible concern for air trapping caused by the CPAP effect [21, 22]. However, decreased work of breathing due to initiation of HFNC cannot be ruled out in these patients.

The specialized nasal prongs are attached to the patient's face as described for standard nasal prongs (Protocol 24.3), with the exception that the oxygen source is a HFNC machine (Figure 24.12). Adequately high rates of inspired oxygen can be achieved that monitoring of PaO_2 and reduction to minimal required FiO_2 is warranted to avoid oxygen toxicity. It may also be justified to monitor PCO_2 in patients with concern for hypercapnea.



Figure 24.12 A patient in the intensive care unit is connected to a high-flow nasal cannula system that is delivering heated (98.6°F; 37°C) and humidified gas at a rate of 40 l/minute with a fraction of inspired oxygen of 100%. This particular model is manufactured by Vapotherm.

Oxygen Delivery via Artificial Airway

While many patients in respiratory distress can be stabilized, diagnosed, and treated with the oxygen supplementation methods discussed above, a subset of these patients have severe enough respiratory failure or hemodynamic instability that they may benefit from intubation and the use of intermittent positive pressure ventilation (IPPV). Indications for immediate intubation include lack of a patent airway (due to airway obstruction), lack of a gag or normal protective airway reflexes, apnea, unconsciousness, and cardiopulmonary arrest [25]. Indications for intubation and long-term mechanical ventilation include severe hypoxemia despite oxygen therapy, severe hypoventilation refractory to intervention, excessive work of breathing with potential for respiratory fatigue, and severe hemodynamic compromise refractory to therapy [26–29]. In subsequent chapters, significantly more detail is discussed regarding tracheal intubation (Chapter 28) and mechanical ventilation (Chapter 31).

Endotracheal Intubation

Endotracheal intubation enables control of a patient's airway, allows provision of an FiO_2 of 100%, requires low oxygen flow rates, and eliminates patient distress. Unless patients have severely altered mentation, they will likely require sedation or anesthesia to achieve and maintain intubation. Initial IPPV may be provided with a bag–valve–mask (or Ambu bag), a nonrebreathing circuit and reservoir bag attached to a central oxygen source or tank, or an anesthesia machine. Short-term intubation and manual ventilation may permit the emergency team to gain sufficient diagnostic information to give owners a better idea of prognosis or to provide stabilizing treatments such as thoracocentesis or a procedure to relieve an upper airway obstruction. For long-term IPPV, most often a mechanical ventilator will be required and intensive 24-hour management is necessary. For hospitals that cannot provide long-term mechanical ventilation, these patients may require stabilization and subsequent transport to another facility.

Tracheostomy Tube Placement

For some patients requiring an artificial airway, oxygen can be delivered via a temporary tracheostomy tube. Indications for a tracheostomy tube include upper airway obstruction (with rare circumstances necessitating emergency tracheostomy tube placement), oral or pharyngeal surgery, and mechanical ventilation for select patients (such as those with respiratory paralysis) [25]. Similar to endotracheal intubation, tracheostomy tube placement allows administration of 100% oxygen and requires anesthesia if a patient is not unconscious. Once placed, tracheostomy tubes may be tolerated in awake patients. See Chapter 29 for further discussion of temporary tracheostomy.

Hyperbaric Oxygen

Hyperbaric oxygen therapy (HBOT) has been used infrequently in veterinary medicine but has become slightly more accessible with the availability of portable hyperbaric chambers designed for veterinary patients. In HBOT, a patient is delivered 100% oxygen into a chamber at an elevated atmospheric pressure (above 760 mmHg or 1 atm). Because hyperbaric oxygen leads to alterations in oxygen pressure and solubility, there is a marked increase in PaO_2 and the amount of oxygen transported to the tissues. At typical working pressures of 2–2.5 atm with an FiO_2 of 100%, the oxygen dissolved in plasma increases by almost 17-fold [30]. Ultimately this results in enhanced diffusion of oxygen into the tissues due to an increase in the diffusion gradient.

Unlike for the majority of uses of oxygen discussed above, HBOT is rarely employed to treat primary respiratory disease. In people, HBOT is used for the treatment of carbon monoxide poisoning, helping to accelerate the dissociation of carbon monoxide from hemoglobin by

increasing PaO_2. It is also used for gas embolism reduction, enhanced oxygen delivery to cells, wound healing, antimicrobial activity, angiogenesis, modulation of inflammation, and vasoconstriction [30, 31]. Data on HBOT in small animal clinical patients are limited mostly to case reports, small populations studies, and anecdotal reports. A prospective clinical trial designed to evaluate safety assessed 230 HBOT treatments in 78 dogs and 12 cats and demonstrated that it was well tolerated with no major adverse effects [32]. A prospective, controlled study evaluating experimentally induced dermal incisions in 10 dogs showed that an HBOT protocol was safe but did not enhance wound healing [33].

Complications of HBOT can include pulmonary oxygen toxicity and oxidative injury, pneumothorax, ruptured tympanic membrane, decompression sickness (gas bubble formation in tissues), and oxygen-induced seizures [31]. There are also practical limitations to the use of HBOT in small animals. When a patient is in an enclosed chamber undergoing therapy, the clinical team does not have access to the patient to allow for monitoring or administration of treatments. In case of emergency, several minutes may be required to decompress the chamber. Because of the 100% oxygen environment, any spark or electrical fire can result in ignition of the pressurized oxygen and patient death. Patient contraindications may include pneumothorax or a predisposing disease (e.g. pulmonary bullae), prior thoracic or ear surgery, upper respiratory infection, uncontrolled seizures, confinement anxiety, and pregnancy [31].

Monitoring the Response to Oxygen Therapy

Once a patient is placed on oxygen supplementation, it is important to monitor clinical signs and objective measures of oxygenation to assess a patient's response to oxygen therapy. Serial assessments of a patient's respiratory rate and effort, mucous membrane color, lung sounds on auscultation, and other signs of respiratory distress are necessary. Additional vital parameters such as temperature, pulse rate and quality, capillary refill time, and mentation are essential in critically ill patients. Arterial blood gas analysis (Chapter 26) and pulse oximetry (Chapter 25) are also helpful for objective assessment of oxygenation and are discussed at more length in their respective chapters.

Ongoing evidence of respiratory distress or hypoxemia warrants re-evaluation of FiO_2, method of oxygen delivery, and other potential medications or techniques to alleviate distress. To avoid complications associated with oxygen therapy, in particular oxygen toxicity, the minimum required FiO_2 to maintain adequate oxygenation and patient comfort should be used at all times. As the patient's underlying disease process improves, room-air trials should be considered to assess a patient's tolerance for discontinuation of oxygen supplementation.

Complications of Oxygen Therapy

Although supplemental oxygen is valuable in many clinical scenarios and necessary for the prevention of potentially life-threatening hypoxia, excessive or inappropriate administration can be deleterious. The term oxygen toxicity is typically reserved for changes caused by oxidative injury to the pulmonary epithelium, but additional gas exchange problems associated with high FiO_2 can include worsening hypoxemia secondary to absorption atelectasis and exacerbation of hypercapnia due to reduced ventilatory drive and worsening V/Q mismatch. While the lungs are normally the initial target of injury with exposure to high FiO_2, excessive oxygen in the blood and tissues (hyperoxemia and hyperoxia, respectively) resulting from high FiO_2 or HBOT can also potentially result in adverse effects on the cardiovascular system, central nervous system, and other organs.

While minimal evidence exists in clinical veterinary patients, there is concern in the human literature for potential increased mortality secondary to hyperoxia in some patient populations [34–36]. Given the associated risks, human guidelines recommend aiming to achieve normal or near-normal oxygenation for most acutely ill patients [37].

Oxygen Toxicity

While short-term oxygen supplementation is rarely problematic, long-term therapy can be deleterious, as oxygen is directly toxic to the pulmonary epithelium. Because pulmonary tissue PO_2 is the highest in the body, and additional oxidizing substances such as air pollutants can be inhaled, the lung is the most vulnerable organ to oxygen toxicity [38]. The main determinants of injury are FiO_2 and length of oxygen therapy. Patients are typically thought to be at risk for oxygen toxicity when oxygen is administered at an FiO_2 greater than 60% for over 24 hours. Dogs exposed to an FiO_2 of 100% developed altered lung function in 24 hours and died within an average of 50–60 hours [39]. Dogs exposed to an FiO_2 of 80% developed lung dysfunction but survived, while dogs exposed to an FiO_2 of 50% did not develop clinical signs of lung dysfunction or lung pathology [40]. Susceptibility to oxygen toxicity varies between species, and cats may be more sensitive than dogs [5]. Additionally, neonates appear more resistant to oxygen toxicity compared to adults [41]. In all animals, it is recommended to titrate FiO_2 to the lowest possible level to achieve acceptable oxygenation. If

titration to an FiO_2 less than 60% is not possible on conventional oxygen, mechanical ventilation may need to be considered.

Diagnosis of oxygen toxicity can be difficult, since many times FiO_2 is unknown, clinical signs may mimic worsening of the underlying disease process, and histopathology is indistinguishable from acute respiratory distress syndrome. Injury occurs in multiple stages [42]. During the initiation phase of oxygen toxicity, which occurs within 24–72 hours of exposure to high oxygen concentration, oxygen-derived free radicals are responsible for direct damage to pulmonary epithelial cells as antioxidant stores are depleted. This leads to the inflammatory phase, during which recruitment of inflammatory cells and release of inflammatory mediators causes increased tissue permeability and pulmonary edema, resulting in a marked destruction phase with potential mortality. In survivors, type II pneumocytes multiply during the proliferative stage, and permanent damage occurs during the fibrotic stage with collagen deposition and interstitial fibrosis.

Absorption Atelectasis

While alveoli are normally replenished with fresh gas during ventilation, if airways become obstructed in a patient on oxygen, the alveoli distal to those airways can experience absorption atelectasis [43]. Normally, nitrogen makes up the majority of inhaled gas that fills the alveoli on room air. However, almost no net nitrogen exchange occurs across the respiratory membrane because the body is already saturated with nitrogen. Thus, nitrogen remains in the alveoli, preventing their collapse and minimizing absorption atelectasis. However, high concentrations of inhaled oxygen displace nitrogen in the alveoli. Oxygen readily travels down its concentration gradient from the alveoli into the pulmonary capillaries. If an airway becomes obstructed and there is no fresh gas flow, the oxygen in the alveoli distal to that airway diffuses into the blood and leaves inadequate gas to hold open the alveoli, causing their collapse. High concentrations of oxygen can therefore accelerate absorption atelectasis.

Hypercapnia

In normal patients, hypercapnia serves as the primary stimulus for respiration. However, in patients with chronic lung diseases resulting in hypercapnia, hypoxia replaces hypercapnia as the primary drive for ventilation. If oxygen is administered to these patients and hypoxemia is relieved, ventilatory drive may diminish markedly, resulting in severe hypercapnia and potentially respiratory failure requiring positive pressure ventilation [44]. If oxygen supplementation is discontinued, severe hypoxemia can occur during the time it takes for CO_2 to be unloaded from the tissues. Although this "blue bloater" syndrome has been well documented in humans, particularly those with chronic obstructive pulmonary disease, it is uncommon in small animal patients and rarely a significant concern.

An exacerbating factor in some of these patients is that oxygen administration can diminish the normally protective hypoxic pulmonary vasoconstriction that occurs in poorly ventilated areas of the lung. As alveolar PO_2 is known to contribute to regulation of hypoxic pulmonary vasoconstriction, elevated alveolar PO_2 can increase blood flow to low V/Q areas, resulting in worsening of V/Q mismatch and CO_2 retention [44].

Extrapulmonary Toxicity

Hyperoxia may result in alterations to multiple organ systems. In regard to cardiovascular function, it has been demonstrated in dogs and humans that hyperoxia can cause systemic arterial vasoconstriction, increased systemic vascular resistance, coronary and cerebral vasoconstriction, bradycardia, and reduced stroke volume and cardiac output [37, 45, 46]. Nonetheless, the clinical significance of these potential hemodynamic changes remains unclear.

Central nervous system complications reported in people primarily include tonic–clonic seizures secondary to the use of HBOT but can include a variety of other neurologic signs [38]. There is conflicting evidence but evolving concern regarding the effects of hyperoxia in various disorders involving the central nervous system, including traumatic brain injury, acute ischemic strokes, and post-cardiopulmonary arrest patients [37]. However, for veterinary patients, the clinical evidence again is lacking.

High inspired oxygen in neonates has also been shown to cause vision-impairing abnormalities in retinal vascular development known as retinopathy of prematurity. While this has mostly been described in premature human infants, it is well demonstrated in animal models, including dogs and cats [47]. As such, oxygen therapy should be minimized in neonates if possible.

Summary

Oxygen supplementation should be considered for all patients presenting to the emergency room in respiratory distress. Conventional oxygen therapy techniques, which are relatively non-invasive for the patient, include flow-by, face mask, oxygen hoods/E-collars, oxygen cages, and nasal oxygen. Intratracheal oxygen supplementation is more difficult but has the potential to achieve higher FiO_2 values. Heated humidified high-flow nasal oxygen systems

have recently been adopted in veterinary medicine and used as an advanced oxygen supplementation technique for patients failing traditional oxygen therapy. HFNC may provide an additional option to prevent some patients from requiring mechanical ventilation. Intubation and IPPV are invasive but can provide the highest level of support for patients in respiratory failure. The choice of technique will depend on patient requirements, response to oxygen, methods available, and owner investment. Patients needing oxygen supplementation must be closely monitored and therapy adjusted as indicated to maximize efficacy and minimize complications.

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25

Pulse Oximetry and Co-Oximetry

Kate Farrell

Pulse oximetry and co-oximetry have become ubiquitous monitoring tools for the assessment of hemoglobin saturation and oxygenation in anesthetized and critically ill human patients, and they have found widespread use in veterinary medicine as well. While a small percentage of oxygen travels in the blood as dissolved gas, the bulk of oxygen is bound to hemoglobin in red blood cells. Oxygen binding changes the light absorption pattern of hemoglobin. Oximetry monitoring takes advantage of this fact and uses light absorbance characteristics to determine the percentage of oxygenated hemoglobin compared with other hemoglobin species (described later). Hemoglobin oxygen saturation of arterial blood (SO_2) can be measured with pulse oximetry (peripheral capillary oxygen saturation, SpO_2) or co-oximetry (SaO_2). Pulse oximetry uses two wavelengths of light (red and infrared) to determine the percentage of oxyhemoglobin compared with deoxyhemoglobin in arterial blood of a patient's tissues. SpO_2 has been used as a surrogate marker for the partial pressure of oxygen in arterial blood (PaO_2), although in veterinary patients there is potential concern for its accuracy. Co-oximetry, on the other hand, uses multiple wavelengths of light to detect numerous hemoglobin forms, including carboxyhemoglobin and methemoglobin, in a blood sample.

History of Oximetry Monitoring

The laboratory use of oximetry originated in the 1930s, while the earliest patient bedside oximeters date to the 1960s. However, these bedside monitors did not become common as they frequently overheated and became uncomfortable for patients. In the 1970s, Takuo Aoyagi invented the pulse oximeter that we use today [1]. In the early 1980s, this technology was integrated into commercial monitors for people and advancements were made in the microprocessing technology to improve pulse oximeter performance [1]. Prior to that time, there was no method of easily and continuously monitoring a patient's arterial hemoglobin oxygen saturation. Instead, evaluation of a patient's oxygenation required blood sampling and ex vivo analysis, which only allowed for intermittent monitoring and could be challenging, invasive, and expensive. Since its inception, pulse oximetry has significantly reduced the incidence of lethal hypoxemia in anesthetized human patients and has become a standard monitoring tool for patients in emergency departments and intensive care units.

Hemoglobin Oxygen Saturation

Oxygen is delivered from the lungs to the tissues via the blood and is carried in two forms. Approximately 2–3% of the total oxygen content of blood in health is dissolved in plasma, and this is measured as the partial pressure of oxygen (PO_2) [2]. Most oxygen in the blood is bound to hemoglobin in red blood cells. Hemoglobin consists of four polypeptide subunits (globins), each of which is associated with an iron-containing heme. Each iron is capable of binding oxygen, and thus hemoglobin can carry four oxygen molecules. Once one oxygen becomes bound, this changes the conformation of hemoglobin, which increases its affinity for additional oxygen molecules; this is known as cooperative binding. When hemoglobin is bound to oxygen, or "saturated," it is called *oxyhemoglobin*, and it imparts a bright red color to arterial blood. Deoxygenated or reduced hemoglobin, known as *deoxyhemoglobin*, gives venous blood its darker color.

Although PO_2 is only a small portion of the content of oxygen in arterial blood, its value determines SO_2. This relationship is described by the *oxyhemoglobin*

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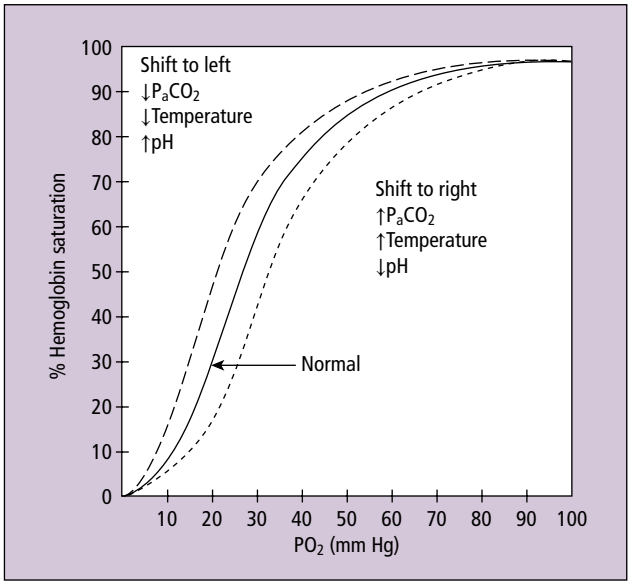


Figure 25.1 The oxyhemoglobin dissociation curve, demonstrating the relationship between the partial pressure of oxygen (PO_2) and the percentage of hemoglobin oxygen saturation. The position of the curve, and therefore hemoglobin's affinity for oxygen, is affected by numerous physiologic factors, including the partial pressure of carbon dioxide (PCO_2), temperature, and pH.

dissociation curve (Figure 25.1), which is sigmoidal in shape given the cooperative binding of oxygen to hemoglobin described above. Accurate determination of PO_2 from SO_2 (or vice versa) depends on a normal position of the curve. However, the position of the curve, and therefore hemoglobin's affinity for oxygen, is affected by multiple physiologic factors, and these may be altered in critical illness [3–5]. An increase in pH and decreases in the partial pressure of carbon dioxide (PCO_2), temperature, and 2,3-diphosphoglycerate shift the curve to the left, increasing hemoglobin's affinity for oxygen (which is beneficial in the lungs where pH is higher and PCO_2 and temperature lower). The opposite changes result in a right-shifted curve, promoting unloading of oxygen in the tissues where pH is lower and PCO_2 and temperature higher.

Approximate corresponding values for PO_2 and SO_2 are presented in Table 25.1 [3, 4]. However, these values are based on a human oxyhemoglobin dissociation curve, and the position of the curve varies between species [6]; *p*50 is a common method of expressing the position of the curve and represents the PO_2 at which 50% of hemoglobin is saturated. The reported *p*50 for human hemoglobin is a PO_2 of approximately 26–27 mmHg, for canine hemoglobin is 28–31 mmHg, and for feline hemoglobin is 34–36 [6–11]. As cats have a significantly right-shifted curve compared with people and dogs, their corresponding SO_2 values (for a given PO_2) are lower.

Table 25.1 Correlation between partial pressure of oxygen in arterial blood (PaO_2) and saturation of hemoglobin with oxygen in arterial blood (SaO_2) based on the human oxyhemoglobin dissociation curve [3, 4].

Oxemia	PaO_2 (mmHg)	SaO_2 (%)
Severe hyperoxemia	500	100
Hyperoxemia	150	99
Normoxemia	80–110	96–98
Hypoxemia	<80	<95
Severe hypoxemia	<60	<90

Dyshemoglobinemias

Oxyhemoglobin and deoxyhemoglobin are known as functional hemoglobin because they can bind, carry, and unload oxygen. Non-functional hemoglobin forms, known as dyshemoglobins, are not capable of binding oxygen and include carboxyhemoglobin, methemoglobin, and sulfhemoglobin.

Carboxyhemoglobin is formed when hemoglobin binds to carbon monoxide rather than oxygen. Because hemoglobin's affinity for carbon monoxide is greater than 200 times its affinity for oxygen, carboxyhemoglobin forms even in the presence of very low concentrations of carbon monoxide [12, 13]. As this prevents the binding and transport of oxygen, and also results in a significant left shift of the oxyhemoglobin dissociation curve, toxic levels can lead to tissue hypoxia and death. The most common reason for excessive exposure to carbon monoxide in small animals is smoke inhalation. In healthy adult dogs, carboxyhemoglobin has been reported to compose a median of 2.3% of total hemoglobin (reference interval 1.3–2.7%) [14]. With carboxyhemoglobin levels exceeding 20%, the brain and heart may become severely affected. Progressive clinical signs described in people include nausea, dizziness, headache, weakness, difficulty breathing, disorientation, seizures, coma, and death [12]. Veterinary literature is more limited, but reported consequences of carbon monoxide intoxication include altered mentation, respiratory changes, hypothermia, acute and delayed neurologic signs, and death [15]. Carboxyhemoglobin can impart a bright cherry-red color to blood, though this is rarely clinically seen [16]. Treatment consists of high concentrations of inspired oxygen to displace carbon monoxide.

Methemoglobin is produced when the ferrous form of iron (Fe^{2+}) in the heme group is oxidized to the ferric form (Fe^{3+}) due to oxidative injury. Because there are multiple protective mechanisms that diminish oxidative injury and reduce methemoglobin to functional hemoglobin, methemoglobin composes a very small proportion of total

hemoglobin in normal dogs and cats. One study reported a median value of 0.2% (reference interval 0.1–0.4%) in healthy adult dogs [14]. However, elevated levels can occur from a congenital abnormality or secondary to drugs or toxins that overwhelm antioxidant defenses. Toxins known to cause methemoglobinemia include acetaminophen, nitrites, nitrates, topical benzocaine, phenazopyridine, hydroxycarbamide, and skunk musk. At levels greater than 10%, methemoglobin can impart a brown discoloration to blood, and chocolate-brown mucous membranes and cyanosis can be noted [17]. Clinical signs are typically seen with methemoglobin levels above 20% and can include tachycardia, tachypnea, weakness, anorexia, vomiting, ataxia, hypothermia, ptyalism, and seizures. Coma and death can be seen at methemoglobin levels of 80% [17].

Sulfhemoglobin is another non-functional hemoglobin that occurs rarely when hemoglobin reacts with sulfide and oxygen. However, unlike carboxyhemoglobin and methemoglobin, sulfhemoglobin is not typically detected by co-oximeters.

Functional Compared with Fractional Hemoglobin Saturation

Oxyhemoglobin and deoxyhemoglobin, as discussed above, can bind and unbind oxygen, and for this reason they are called functional hemoglobin species. The percentage of oxyhemoglobin compared with all functional hemoglobin is known as *functional hemoglobin saturation*. It is calculated as follows:

$$\text{Functional } SO_2 = \left[\left(HbO_2\right) / \left(HbO_2 + HHb\right)\right] \times 100 \qquad (25.1)$$

where HbO_2 is oxyhemoglobin and HHb is deoxyhemoglobin. Because pulse oximeters only use two wavelengths of light to detect oxyhemoglobin and deoxyhemoglobin (discussed in more detail below), they report functional hemoglobin saturation.

In contrast, *fractional hemoglobin saturation* refers to the percentage of oxyhemoglobin compared with all measured hemoglobin forms, including dyshemoglobins. The equation to calculate fractional hemoglobin saturation is as follows:

$$\text{Fractional } SO_2 = \left[\left(HbO_2\right) / \left(HbO_2 + HHb + COHb + MetHb\right)\right] \times 100 \qquad (25.2)$$

where COHb is carboxyhemoglobin and MetHb is methemoglobin. Co-oximeters can measure multiple hemoglobin species, and thus they report fractional hemoglobin saturation. Typically, this calculation includes carboxyhemoglobin and methemoglobin; in the less common case that the co-oximeter measures sulfhemoglobin, that species is also included in the calculation.

Types and Technology of Oximeters

Spectrophotometry is a technology that uses light reflection and absorption properties of substances to determine their concentration in liquid or gaseous environments. When applied to determine the relative percentage of oxygenated hemoglobin in blood, this is called oximetry. This technology uses the Beer–Lambert law, which relates the concentration of a substance to the intensity of light transmitted through a solution [18]. These principles can be applied to the detection of hemoglobin species given the fact that binding of oxygen and other substances to hemoglobin alter its light absorbance properties.

Pulse oximeters use two wavelengths of light to detect relative concentrations of oxyhemoglobin and deoxyhemoglobin. Alternatively, *co-oximeters* use several wavelengths of light to distinguish multiple hemoglobin species, including dyshemoglobins discussed above. Hemoglobin oxygen saturation measured by pulse oximeter is typically referred to as SpO_2, while measurement by co-oximeter is typically referred to as SaO_2. *Pulse co-oximeters* use the technology of co-oximeters but in a continuous, non-invasive bedside format. They have been employed in people for noninvasive testing for carbon monoxide poisoning and methemoglobinemia resulting from drug overdoses, but they are not generally used in clinical veterinary medicine.

Pulse Oximetry

Understanding the Technology

Pulse oximeters are non-invasive bedside monitors that determine arterial functional hemoglobin saturation in a patient's tissue. To determine SpO_2, a pulse oximeter emits two wavelengths of light, red light at 660 nm and infrared light at 940 nm, from the probe's light-emitting diodes (LED) into a patient's tissue. Depending on the probe's style, transmitted light either reflects off tissue or passes through to a receiving probe, where a photodetector collects a signal to be analyzed by the machine's microprocessing unit. Oxyhemoglobin absorbs more infrared light, while deoxyhemoglobin absorbs more red light. Because of their differing light absorption characteristics, the ratio of the two can be determined to calculate the percentage of oxyhemoglobin based on total functional hemoglobin.

Pulse oximetry relies on the detection of light absorption through tissue, and the technology was developed based on



Figure 25.2 A multiparameter monitor displaying a pulse oximeter's hemoglobin oxygen saturation (SpO_2) reading of 92% and accompanying high-quality plethysmograph (blue waveform).

the absorption spectrum of human oxyhemoglobin. It has been demonstrated, however, that hemoglobin isolated from multiple species, including people, dogs, cats, horses, cows, and pigs, showed only minor and statistically insignificant differences in absorbance spectra of oxyhemoglobin and deoxyhemoglobin [19]. Therefore, it is likely that the same pulse oximetry technology can be applied across these species.

To differentiate arterial blood from venous or capillary blood and surrounding tissue, pulse oximeters use an optical technique known as photoplethysmography to evaluate only pulsatile wavelengths. For this reason, pulse oximeters function best when placed over tissue with good arterial blood flow, and the pulse rate read by the machine should match the patient's. To enhance evaluation of the pulse oximeter's accuracy, many machines display a plethysmograph, or peripheral pulse waveform, the amplitude of which corresponds to pulse strength (Figure 25.2). A normal waveform should have a dicrotic notched appearance similar to that of an arterial waveform.

Indications for Pulse Oximetry

Because SpO_2 correlates with PaO_2 according to the oxyhemoglobin dissociation curve, pulse oximetry is used as a non-invasive surrogate for PaO_2. Arterial blood gas analysis, which is necessary to obtain a PaO_2, requires arterial sampling; however, this can be challenging, invasive, and costly. Additionally, not all patients in respiratory distress tolerate the handling required for arterial sampling, and some patients are too small for easy sampling. Pulse oximetry, on the other hand, is easy to perform, can provide continuous monitoring, and carries minimal cost.

Given these advantages, in human medicine pulse oximetry is standard of care for monitoring anesthetic patients and is used for a wide variety of monitoring needs for patients in emergency departments and intensive care units. It has also become a common tool in veterinary medicine to monitor anesthetic patients, evaluate and triage patients in respiratory distress in the emergency room, and to assess oxygenation in patients in intensive care. In some hospital and in out-of-hospital settings, SpO_2 may be the only objective measurement available to a veterinarian.

Equipment Options and Care

When electing to purchase a pulse oximeter, considerations may include brand/model, convenience or ease of use, available probes, presence of a plethysmograph, battery life, and cost. Because of their utility in human medicine and now extensive use in veterinary medicine, pulse oximeter models and probes have been adapted and marketed for veterinary use, and their costs have been adjusted for veterinary audiences [5]. Models are available from Nellcor, Masimo, Apexx, Respironics, and multiple other companies. Because the algorithms built into each model vary, investigating any data related to accuracy of an individual unit may be worthwhile. Many manufacturers and distributors will also allow for trial periods within a facility prior to purchase.

In regard to probe selection, a clip-style sensor is the most commonly available and provides versatile options for use. Some pulse oximeters offer other choices, including reflectance probes for rectal or esophageal monitoring. While the LED on rectal sensors may become obscured with feces, they are useful for acquiring a reading from the ventral tail base.

Pulse oximeters that function as hand-held mobile units provide the greatest flexibility, as opposed to those that are part of multiparameter monitors. These units allow for convenient bedside monitoring and may have longer battery life. Additional considerations may include ease of alarm setting, company warranties, and costs of probes, batteries, and chargers.

For long-term care of pulse oximeters, they should avoid contact with excessive water, chemicals, and direct sunlight. The wires for the probes should be looped loosely around or next to the machine during storage, as tight winding of the wires may damage them. In between use, probes should be cleaned according to the manufacturer's instructions. For many, mild soap and water will be sufficient. Disinfection with isopropyl alcohol or chlorhexidine may be appropriate, though some diodes can be damaged by chemicals.

Performing Pulse Oximetry

When acquiring an SpO_2, multiple steps should be considered to maximize the accuracy of the measurement (Protocol 25.1). First, it should be ensured that the machine

Protocol 25.1 Obtaining a Pulse Oximetry Measurement

Equipment Required

- Pulse oximeter, charged with a clean probe
- Moistened gauze squares, as needed
- Clippers, if fur removal is needed

Procedure

1) Collect necessary supplies.
2) Turn monitor on and ensure that it is functioning and has battery life.
3) Select and prepare a site. Choose a location that is well perfused, warm, thin, and has minimal pigmentation; unpigmented mucous membranes are ideal.
4) Place the probe sensor on the selected site.
5) Wait for the monitor to obtain a pulse rate and confirm that this matches the patient's pulse.
6) If a plethysmograph is available, check to ensure a strong signal and adequate waveform.
7) If the pulse rate does not match or plethysmograph waveform is poor, attempt another site.
8) Wait until a stable SpO_2 is obtained, then record.
9) If the SpO_2 is dangerously low (<90%) or unexpected, notify the doctor immediately. Attempt to confirm at another site.
10) If an SpO_2 is obtained, consider whether it matches the patient's clinical condition. If not or if further assessment is required, obtain another reading or consider an arterial blood gas.

is charged and the probe is clean and undamaged. Testing the pulse oximeter on one's own finger can help to ensure it is functioning accurately. Choosing an appropriate site, monitoring plethysmograph waveform and matching pulse rate, and comparing the acquired SpO_2 to patient status are important steps.

When selecting a site for probe placement, the location should be well perfused, warm, thin enough for probe clip application, and have minimal pigmentation (Figure 25.3). Mucous membranes are typically ideal; potential sites are listed in Table 25.2. The site should be cleaned and clipped of fur as needed. If a patient is vasoconstricted or hypothermic, locations such as the ears, distal tail, or digits may not provide accurate or any readings. In these conditions, sites should be chosen as close to a central vessel as possible, such as the tongue or lips, to provide the best results [20]. Site selection in awake animals may depend on accessible areas. For instance, in aggressive or non-compliant animals, application to the lips or tongue may be impossible. The pinna may provide an accessible site but in one study was shown to give erroneous readings [20].

In anesthetized animals, common sites include the tongue and lips. However, when probes are left in place for a prolonged time, the SpO_2 reading may decrease over time due to pressure on the arterioles and reduced blood flow in the underlying tissue [5]. Gradual drying of the mucous membranes may also lead to false results. These factors can be corrected with repositioning of the probe and repeated moistening of the tissue. With protracted pulse oximetry monitoring, for instance in cases of mechanical ventilation, the slight heat and pressure from the probe can lead to injury and necrosis of underlying tissue in extreme circumstances. This is easily avoided by relocating the probe every two to four hours during careful routine ventilator patient management [21]. Reflectance probes can be secured in place on the ventral tail base in anesthetized or poorly mobile animals but should be checked for cleanliness and tissue damage (burns).

Particular problems can be encountered with obtaining readings from small animals, especially cats. The size and shape of available probes are not always appropriate for very small animals. While data are limited, it may be more difficult to achieve reliable readings in cats [5]. However, as it is also more difficult to acquire arterial blood gases in cats given their size, obtaining objective measures of oxygenation in cats can be challenging. Cats in general may be more resistant to handling and easily stressed with both types of monitoring.

Once a probe has been placed at the designated site, it may require at least several seconds to provide an SpO_2 reading. If the measured value is variable or inconsistent, allowing more time or switching to another site may be necessary. To ensure accuracy, the pulse rate noted on the machine should match the patient's, and the plethysmograph (if available) should display an adequate waveform. Some pulse oximeters designed for people have neonatal, pediatric, and adult settings, and this should be set to reflect the patient's pulse rate rather than its size (neonatal for high heart rates, adult for lower heart rates). It may be helpful to trend values over time or to monitor changes in the SpO_2 reading with oxygen delivery.

Interpreting the SpO_2 Reading

Once an SpO_2 measurement has been achieved, careful interpretation of the value and the patient's circumstances is required. A normal PaO_2 for an animal breathing room air at sea level is 80–110 mmHg, and this corresponds to an SpO_2 value of approximately 96–98%. Values less than this indicate hypoxemia and warrant investigation. If a dog curve, as opposed to a human curve, is used for



Figure 25.3 Probe placement for pulse oximeter measurements. (a) Probe placement on the pinna of an awake dog. The pulse oximeter provides a hemoglobin oxygen saturation (SpO_2) value of 96%, which is appropriate for a dog on room air. (b) Placement of a pulse oximeter probe interdigitally in a cat receiving supplemental oxygen. Awake cats will not typically tolerate lip or tongue measurements. (c) A probe placed on the tongue of an anesthetized dog receiving supplemental oxygen. A matching pulse rate (82) and indicator of a strong pulse signal (full-sized gray bar, which pulsates with the pulse rate) help confirm the accuracy of the reading. An SpO_2 value of 90% on supplemental oxygen indicates marked lung dysfunction. (d) A probe placed on pigmented mucous membranes is unlikely to give a reliable reading.

Table 25.2 Locations for pulse oximeter probe placement.

Canine	Feline
Tongue	Tongue
Lips	Lips
Pinna	Pinna
Vulva/prepuce	Interdigital
Interdigital	Ventral skin
Ventral skin	Ventral tail base
Ventral tail base	
Gastrocnemius tendon	

interpretation of corresponding PaO_2 and SpO_2 values, a PaO_2 of 80 mmHg actually equates to an SpO_2 of approximately 93%, while a PaO_2 of 60 mmHg corresponds to an SpO_2 of around 86% [8]. Although theoretically altered compared with people, the SpO_2 cutoff values of 95% and 90% may be more reliable for the identification of hypoxemia and severe hypoxemia, respectively, in clinical dogs given limitations of the technology (discussed below) [22]. Because the cat oxyhemoglobin dissociation curve is further right-shifted, corresponding SpO_2 values could be expected to be lower.

Further complicating interpretation of pulse oximeter readings is the fact that SpO_2 and PaO_2 are not linearly

related. The oxyhemoglobin dissociation curve is sigmoidal in shape, which means that very small changes in SpO_2 indicate substantial changes in PaO_2. For instance, if an SpO_2 measurement decreases from 95% to 90%, this correlates to a decline in PaO_2 of approximately 20 mmHg. This patient has gone from having close to normal lung function (if on room air) to being markedly hypoxemic. Therefore, slight errors in the pulse oximeter reading in this range could result in dramatically different and incorrect interpretations of patient status, which can be problematic for clinical decision making.

Given the shape of the oxyhemoglobin dissociation curve, pulse oximetry is very sensitive (when accurate) for detecting changes in PaO_2 when SpO_2 is in the 80–97% range. However, above this range, only minuscule changes in hemoglobin saturation can occur even with large increases in PaO_2. This flattening of the curve limits the ability of SpO_2 to predict PaO_2, particularly in patients receiving supplemental oxygen. For example, a patient with normal lung function breathing 100% oxygen should have a PaO_2 of 500 mmHg; at this value, an SpO_2 would read 100%. However, if the animal's PaO_2 on 100% oxygen dropped from 500 mmHg (normal lung function) to 175 mmHg (severely abnormal lung function), the SpO_2 would remain 100% and the operator would be unable to detect the compromised lung function. Thus, a pulse oximeter is an insensitive indicator of changing lung function when the patient is receiving supplemental oxygen. On the other hand, an SpO_2 less than 99–100% on supplemental oxygen clearly indicates a problem.

While SpO_2 can estimate oxygenation, it does not assess ventilation, which requires a PCO_2 value on blood gas analysis or end-tidal monitoring. SpO_2 also gives no indication of a total hemoglobin value (just its saturation), and therefore anemia as a cause of decreased tissue oxygen delivery could be missed.

Factors Affecting Pulse Oximetry Measurements

Though pulse oximetry is a popular and convenient monitoring tool, unfortunately many factors affect its accuracy or the ability to acquire a reading, including patient features and technologic limitations. Factors known to affect the accuracy of pulse oximetry include motion, hypoperfusion, hypothermia, pigmentation at the probe site, ambient light, severe anemia, and dyshemoglobinemias [3, 23–25]. See Table 25.3 for a summarized list of these factors.

Movement artifact, such as that seen with tachypnea, tremoring, and shivering, can be common in veterinary patients. With excessive motion, the monitor can mistake venous blood for arterial blood and SpO_2 values may be falsely low [26]. Poor perfusion, peripheral vasoconstriction, or hypotension may affect the ability of the pulse oximeter to evaluate pulsatile blood flow as well. Anemia can also result in inaccurate readings, as pulse oximetry is dependent on light absorption by hemoglobin within red blood cells. One veterinary study revealed that severe anemia affected bias and precision of the pulse oximeter, finding unacceptable accuracy with a hematocrit less than 10% [27].

Table 25.3 Factors affecting the accuracy of pulse oximetry.

Patient factors	Machine factors
Movement artifact (tachypnea, tremoring, shivering)	Monitor broken
Hypoperfusion (vasoconstriction, poor cardiac output, hypotension)	Monitor not charged
Hypothermia	Probe damaged
Severe anemia	Improper probe placement
Dyshemoglobinemias	Dirty probe
Skin pigmentation	Ambient light
Excessive hair	
Dry mucous membranes	

Additionally, variations in probe location and instrument selection have been demonstrated to alter SpO_2 readings [20, 28, 29]. Placement of probes on haired or pigmented tissue may result in erroneously low readings or no readings at all [30]. Fiberoptic and fluorescent lighting can also interfere with pulse oximeter readings, and care should be taken to cover the probe from this lighting. Methylene blue has been reported to interfere with SpO_2 measurements as well [31].

Dyshemoglobinemias are another factor that can result in false SpO_2 readings. As discussed previously, pulse oximeters typically emit only two wavelengths of light and are meant to detect exclusively functional hemoglobin species (oxyhemoglobin and deoxyhemoglobin). Unfortunately, carboxyhemoglobin and methemoglobin can confuse this technology. Carboxyhemoglobin absorbs red light like oxyhemoglobin, and thus elevated levels can falsely increase SpO_2 [32, 33]. Methemoglobin, on the other hand, absorbs both red and infrared light. Concentrations above 30% will cause the SpO_2 to plateau at ~85% [34, 35]. If both an SpO_2 and SaO_2 are acquired, a gap between these values should raise suspicion for dyshemoglobinemia.

See Table 25.4 for methods of troubleshooting pulse oximetry problems.

Accuracy Concerns in Pulse Oximetry

While pulse oximetry carries no direct risk for a patient, its danger lies in the potential for inadequate understanding of the equipment's limitations and excessive

Table 25.4 Guidelines for troubleshooting pulse oximeter readings[a].

Potential problems	Solutions
1) No reading or poor signal/waveform:	
Monitor off/out of battery	Charge and turn on monitor
Probe off patient	Place probe on patient
Site is dirty, haired, dry	Prepare site – clean, shave, moisten
Site is pigmented	Reposition probe to unpigmented site
Hypothermia	Warm patient if stable
Hypoperfusion	Correct underlying problem
2) Pulse rate is inaccurate:	
Tachypnea	Reposition probe to minimize effects of respiration
Other motion artifact	Reposition probe to less affected site
Dysrhythmia	May not read; consider arterial blood gas analysis
Poor signal/waveform	Consider solutions for Problem 1
3) SpO_2 is unexpectedly/falsely high	
Carboxyhemoglobin	Perform co-oximetry
Methemoglobin (if PaO_2 severely low)	Perform co-oximetry
Ambient light	Remove ambient light source or cover probe site
4) SpO_2 is unexpectedly/falsely low:	
Poor signal/waveform	Consider solutions for Problem 1
Motion artifact	Reposition probe to less affected site
Methemoglobin	Perform co-oximetry
Severe anemia	Transfuse as needed; consider arterial blood gas analysis
Ambient light	Remove ambient light source or cover probe site
Intravenous pigmented dyes	Typically transient; consider arterial blood gas analysis
5) SpO_2 obtained is variable:	
Poor signal/waveform	Consider solutions for Problem 1; attempt other probe locations
Dysrhythmia or variable pulse pressure	May not read; consider arterial blood gas analysis

[a] If patient appears to be in respiratory distress and user is unable to obtain an SpO_2 that fits the clinical picture, provide oxygen and attempt later or consider other monitoring options.

confidence in the values obtained. As discussed above, many factors affect the accuracy of pulse oximetry and research confirming its validity in small animal veterinary patients is lacking. Many human and veterinary studies have shown good correlation between SpO_2 and SaO_2; however, these correlations deteriorate when SO_2 is less than 70% or less than 80% [20, 30, 36–38]. Unfortunately, in clinical small-animal patients, there has been little direct comparison of SpO_2 and PaO_2. One study recently demonstrated that in awake dogs breathing room air, SpO_2 was not a clinically suitable surrogate for PaO_2 [22]. Pulse oximetry did perform better in a population of mechanically ventilated dogs on supplemental oxygen [22]. In both groups, pulse oximetry performed poorly at detecting hypoxemia, which is concerning in a screening test.

For these reasons, clinical judgments based exclusively on SpO_2 could be misguided and inappropriate. Because of these concerns, clinicians should always rely on multiple factors, including clinical assessment of the patient, when determining a patient's pulmonary function and oxygen requirements. If possible, pulse oximetry should be correlated with arterial blood gas findings, as PaO_2 remains the gold standard for assessment of oxygenation.

Co-Oximetry

Understanding the Technology

Co-oximeters are benchtop analyzers that use spectrophotometry to distinguish hemoglobin species in a blood sample rather than through tissue. Whereas pulse oximeters use two wavelengths of light and are thus limited to identifying oxyhemoglobin and deoxyhemoglobin, co-oximeters often use four or more wavelengths of light to differentiate and quantify multiple hemoglobin species. In addition to oxyhemoglobin and deoxyhemoglobin, most co-oximeters also report carboxyhemoglobin and methemoglobin, though rarely sulfhemoglobin.

Once concentrations of individual hemoglobin species are known, they are typically reported as a percentage of total hemoglobin (tHb). For instance, the equation for carboxyhemoglobin (COHb) percentage would be as follows:

$$\mathrm{COHb}(\%) = (\mathrm{CoHb} / \mathrm{tHb}) \times 100 \qquad (25.3)$$

In addition to percentages of hemoglobin species, many analyzers also provide blood gas values, acid–base parameters, electrolytes, glucose, lactate, and potentially other values. These machines can thus provide much useful information for emergency or ICU patients (Figure 25.4).

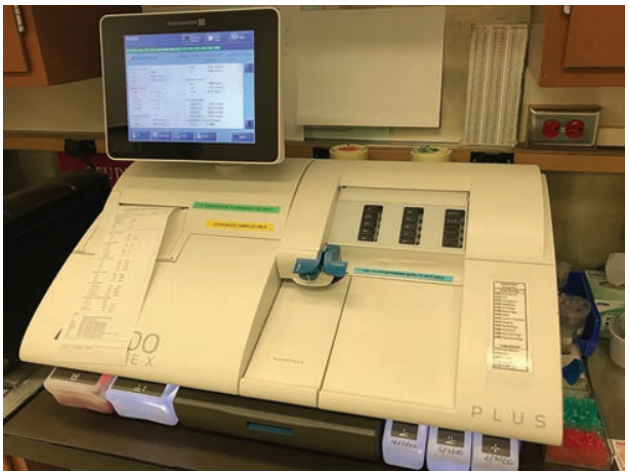


Figure 25.4 A multiparameter benchtop analyzer that includes a co-oximeter.

Indications for Co-Oximetry

Co-oximetry, like pulse oximetry, can detect oxyhemoglobin and deoxyhemoglobin. However, because co-oximetry can quantify all types of hemoglobin, its use is typically indicated when a dyshemoglobinemia is suspected. A dyshemoglobinemia may be suspected with a history of appropriate toxin or environmental exposure, when clinical signs are consistent (e.g. bright red, brown, or cyanotic mucous membranes), when evidence of unexplained respiratory distress or hypoxia persists despite oxygen supplementation, or when there is a discrepancy between a pulse oximetry reading and a PaO_2 or SaO_2 obtained from an arterial blood gas (performed on an analyzer without co-oximetry).

Because co-oximeters detect multiple hemoglobin species, they report fractional SO_2. If a dyshemoglobinemia is present, only fractional SO_2 accurately reflects the blood's oxyhemoglobin content. This value (compared with functional SO_2) will be a better indicator of blood oxygen content and thus oxygen delivery to the tissues. Despite the advantages of fractional SO_2, functional SO_2 more closely corresponds with PaO_2 and thus provides a better assessment of pulmonary function [39].

Equipment Options and Care

Many co-oximeters are available from human biomechanical companies and some are marketed to the veterinary field, but currently there are no veterinary-specific models. Because of this, co-oximeters available for use in veterinary species still base their calculations on human algorithms, and thus the accuracy in dogs and cats is unknown.

Large multi-parameter analyzers with co-oximeters can be preset or manually designated to undergo quality control monitoring and calibrations. Maintenance of these machines requires replacement of consumables required for running samples, calibrations, and quality control. Errors noted during calibrations and quality control will alert users and some machines will not report the failing parameter until the problem is fixed to minimize inaccurate readings.

Performing Co-Oximetry

Co-oximetry analyzers are typically able to produce rapid results with a small volume of blood. If assessment of arterial oxygen content is desired, an arterial blood sample is necessary. This can be obtained via direct arterial puncture with a needle and syringe or by placement of an indwelling arterial catheter (see Chapter 8). The most commonly sampled arterial sites are the dorsal pedal and femoral arteries, although brachial, radial, auricular, and sublingual sites can also be considered depending on the size of the patient and whether it is anesthetized. If the diagnosis of a dyshemoglobinemia is desired, a venous blood sample is sufficient. In hypoperfused patients, a more central vein is ideal for the most accurate values. Follow appropriate scavenging techniques if a sample is taken from an arterial or venous catheter (Chapter 53).

Careful sampling and handling of blood samples is essential to minimize in vitro alterations of blood gases and other values [4]. Samples must be anticoagulated prior to testing, and this is usually performed with sodium or lithium heparin. Dilution of samples with excessive heparin has been shown to significantly alter PO_2 and other values, so this must be performed carefully [40]. Because blood cells continue to be metabolically active, samples are most accurate when analyzed as soon as possible, but they can be held anaerobically in an ice bath for up to one hour if necessary. Attention is warranted to prevent exposure of the sample contents to air or bubbles, as this will alter PaO_2 and SaO_2 due to gas diffusion. Follow all manufacturer guidelines and see Chapter 53 for additional recommendations.

Factors Affecting Co-Oximetry Measurements

Most problems related to co-oximetry measurements are due to sample handling errors. These include inadequate sample mixing, air exposure or air bubbles, excessive dilution or incorrect anticoagulant use, clotting of the sample, delay in testing leading to erroneous results, or interference of particular substances in the blood with spectrophotometric testing. Case reports, animal studies, and laboratory studies have demonstrated that treatment with hydroxocobalamin or cyanocobalamin, which are used as antidotes for cyanide poisoning, can result in measurement interference due to their dark red color [41–43]. Interference

from high serum lipids has also been described to cause falsely high carboxyhemoglobin levels [44]. Sulfhemoglobin can cause falsely elevated methemoglobin and decreased carboxyhemoglobin [45].

Pulse Co-Oximetry

In 2005, a lightweight point-of-care pulse co-oximeter (RAD-57™, Masimo Corporation, Irvine, CA) was approved for use in clinical practice in people. It uses multiple wavelengths of light to noninvasively measure carboxyhemoglobin and methemoglobin. There has been particular interest in employment of the technology as a first-line screening tool to enable rapid identification of patients with carbon monoxide poisoning presenting to human emergency departments. However, clinical data regarding its accuracy are still sparse and have shown somewhat conflicting results [46–48]. Thus far, this technology has been minimally used or studied in veterinary patients. For the diagnosis of dyshemoglobinemias in small-animal patients, co-oximetry will continue to be required until further data arises.

Summary

Pulse oximetry has become an important tool in human and veterinary medicine for monitoring the oxygenation status of anesthetized and critically ill patients. While pulse oximetry is easy to perform, is non-invasive, and can provide potentially valuable information, users must understand the limitations of the technology and thus the risks in over-reliance on SpO_2 measurements. Ultimately, pulse oximetry cannot replace the information gained from an arterial blood gas analysis. Co-oximetry, although generally less available to veterinarians, is also a helpful monitoring tool and enables diagnosis of dyshemoglobinemias. The strengths and limitations of these technologies must be fully understood to provide the best patient care.

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26

Blood Gas Analysis

Sarah Gray

Abnormalities in Oxygenation and Ventilation

Hypoxemia is defined as a partial pressure of oxygen (also called oxygen tension) in the arterial blood (PaO_2) of less than 80 mmHg. There are five main causes of hypoxemia: decreased inspired oxygen content; hypoventilation; ventilation/perfusion (V/Q) mismatch, including intrapulmonary shunt and anatomic right-to-left shunt; and diffusion impairment (Box 26.1). Decreased inspired oxygen content is associated with altitude or from anesthetic machine errors. Hypoventilation (increased partial pressure of carbon dioxide, $PaCO_2$, in arterial blood) can be caused by central nervous system disease, medications that suppress ventilation (opioids, other anesthetic agents), neuromuscular disease (tick paralysis, botulism), upper airway obstruction, severe pleural space disease, chest wall injury (flail chest, pain), or respiratory muscle fatigue.

V/Q mismatch is the most common cause of hypoxemia in dogs and cats. It is a state in which fresh gas (oxygen) delivery to the alveolus is inadequate despite continued perfusion of the pulmonary capillary serving that alveolus. Thus, blood coming from the right side of the heart passes through the gas exchange region of the lung without being properly oxygenated. "Low V/Q" is a state in which narrowed airways such as from inflammatory lower airway conditions (asthma, chronic bronchitis), interstitial lung disease like neoplasia, inflammatory disease like pneumonia or pneumonitis, or other conditions limit oxygen entry into the alveolus. When conditions that cause low V/Q become severe and fresh gas flow cannot reach the alveoli, or when fluid or cell accumulation in the airways lead to alveolar collapse, a "no V/Q" state occurs; this phenomenon can also be called intrapulmonary right-to-left shunting or a "zero V/Q" state. In the no V/Q state, collapsed alveoli or alveoli filled with nongas substance are perfused but (due to the collapse or nongas substance) are not ventilated at all (zero ventilation) – thus all blood passing through no V/Q lung regions returns to the left atrium as venous (nonarterialized) blood. Diseases such as aspiration pneumonia and pulmonary edema commonly cause this intrapulmonary shunting, though any lung disease that becomes adequately severe to block or flood airways and alveoli can lead to "no V/Q." Congenital vascular anomalies such as patent ductus arteriosus or septal defects can also cause blood to flow from the right side of the heart to the left side of the heart without being oxygenated (shunting), although these conditions are less common than the V/Q mismatch caused by lung disease. Diffusion impairment results in incomplete arterialization of the pulmonary capillary blood due to thickening of the gas exchange layer in the alveoli, which is typically the result of type II (thick) pneumocyte proliferation following severe pulmonary inflammation. Under normal conditions the equilibration of oxygen occurs rapidly, but with a thickened gas exchange layer more time is needed, resulting in hypoxemia. Diffusion impairment is believed to be an uncommon cause of hypoxemia in small animals.

When evaluating a patient's oxygenation and ventilation status, the gold standard is to obtain an arterial blood sample for direct analysis of the partial pressures of dissolved oxygen and carbon dioxide in the blood: a blood gas analysis. Blood gases analyzers, results, and interpretation are the topics of this chapter.

Blood Gas Analyzers

Benchtop and portable blood gas analyzer models are available. Dedicated blood gas benchtop analyzers are generally only used in academic facilities and large institutions due to their maintenance requirements and expense. Benchtop

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Box 26.1 Causes of Hypoxemia and Hypoventilation

Hypoxemia

- Decreased inspired oxygen content
- Hypoventilation
- Ventilation/perfusion (V/Q) mismatch
  - Low V/Q
  - No V/Q (intrapulmonary shunt)
- Diffusion impairment

Hypoventilation

- Central nervous system disease
- Medications (opioids, other anesthetic agents)
- Neuromuscular disease
- Upper airway obstruction
- Pleural space disease (severe)
- Respiratory muscle fatigue

analyzers use a polarographic oxygen electrode and the Severinghaus carbon dioxide (CO_2) electrode to analyze the partial pressures of gases in the blood sample. The amount of CO_2 in the sample is evaluated based on comparison to a known amount of CO_2, which is supplied by conventional gas tanks. This severely limits the portability of the benchtop analyzers.

Portable (or "bedside") blood gas analyzers accurately measure oxygen and CO_2 using a small foil-wrapped cartridge containing a pH electrode, a polarographic oxygen (O_2) electrode, and a CO_2 electrode. When the cartridge is sealed, the CO_2 equilibrates with bicarbonate within the cartridge. Upon removal of the foil package, the gas surrounding the CO_2 electrode is the known partial pressure of CO_2 in the ambient air, and the electrode calibrates to this pressure upon insertion into the machine.

There are many advantages to the use of portable blood gas analyzers. These machines are usually less expensive than the benchtop devices, can analyze full blood gases on a very small blood sample, and are portable. A 2016 study compared four commonly used point-of-care analyzers and found differences between the analyzers for PO_2, PCO_2, and pH measurements from both healthy dogs and those with respiratory disease [1]. The availability of bedside analyzers has enhanced the care of critically ill veterinary patients, but since there is variation in measured values from different bedside analyzers, obtained values should be interpreted considering the analyzer's own reference interval. Bedside blood gas analyzers allow for frequent patient monitoring, diagnosis of ventilation and oxygenation problems, and tailored therapy and prognostication for patients with respiratory disease.

Transcutaneous Blood Gas Monitoring

Transcutaneous (tc) monitoring allows continuous measurement of $PtcO_2$ and $PtcCO_2$ by placement of a probe on the patient's skin. It is less invasive and allows the clinician to monitor the patient's response to therapy on an almost interactive basis. These monitors are widely used in human medicine.

Traditional arterial blood gas analysis requires challenging arterial sample collection, appropriate sample handling, and clinically appropriate interpretation on serial samples to best manage critical patients. Thus, the use of transcutaneous monitoring in critically ill veterinary patients would be helpful. One study evaluated the use of transcutaneous blood gas monitoring in dogs to determine its agreement with arterial samples [2]. Unfortunately, the authors concluded that agreement was inferior to that reported for people as the transcutaneous monitor consistently overestimated PaO_2 and $PaCO_2$ in critically ill dogs [2]. While it may play a role in veterinary patients in the future, currently the technology would have to be paired with intermittent arterial blood gas analysis.

Measuring Arterial Blood Gases

To be clinically useful, the arterial blood sample must provide an accurate reflection of the patient's status. This requires appropriate blood sampling and handling (see Chapters 8 and 53 for more information). Accurately determining arterial blood gas content involves obtaining an arterial blood sample and inserting it into the blood gas analyzer within a short period of time, to minimize sample gas equilibration with the atmosphere.

Arterial Blood Sampling

The most commonly sampled arterial sites are the dorsal pedal (dorsal metatarsal) and femoral arteries. Less commonly sampled sites are the brachial, radial, or auricular arteries, or sublingual vessels. Sampling is generally done using palpation and knowledge of anatomy. The dorsal pedal artery can be palpated over the dorsomedial aspect of the proximal metatarsal region; the femoral can be palpated on the medial aspect of the thigh. A single sample can be drawn using a needle and heparinized syringe, or a catheter can be placed for repeated blood gas sampling and for direct blood pressure monitoring. The catheter material can affect catheter handling during placement and the likelihood of thrombophlebitis [3]. Monitoring for evidence of thrombosis is important, especially in peripherally placed anatomic sites because the catheter and associated

thrombosis may compromise blood flow to distal tissues. Refer to Chapter 8 for more information.

Arterial Blood Sample Handling

To ensure accuracy of blood gas results, knowledge of appropriate sample handling is important. Exposure of the sample to room air alters results, so sampling should be anaerobic if possible (disallowing a sample/room air interface). Air bubbles in the sample syringe should be expelled immediately. Owing to gas diffusion, exposure to room air causes a drop in the sample $PaCO_2$ (PCO_2 of room air is approximately zero) and change in the sample PaO_2 (PO_2 of room air is approximately 150 mmHg at sea level).

Analysis delay also alters the accuracy of the blood gas results. Blood cells remain metabolically active for some time ex vivo, consuming O_2 and producing CO_2. Holding the sample on ice decreases the cells' metabolic rate and preserves the sample's true gas levels. If sample analysis must be delayed, the sample should be stored anaerobically in an ice water bath at 4°C; this allows up to a one-hour delay in analysis without significantly affecting results [4].

Blood gas analysis requires whole blood samples, and as such anticoagulants must be used to prevent clot formation. Unfortunately, dilutional errors associated with anticoagulants can affect results. Heparin is commonly used as the anticoagulant for arterial blood. Dilution of the sample with liquid heparin can lower the $PaCO_2$, affects the measure of oxygen, and can affect electrolytes, lactate, and bicarbonate results. A less than 10% dilution with heparin minimizes effects on the analytes; this dilution can be achieved with the evacuated syringe method using a 3-cc syringe and 22-gauge needle. Liquid sodium heparin (1000 iu/ml) 0.5 ml is drawn into the syringe, and then the plunger is drawn to the 3-cc mark to allow the heparin to coat the interior of the entire syringe. Then the contents of the syringe are removed by expelling all the air and any heparin forcibly. The expulsion procedure (pulling the plunger back to the 3-cc mark followed by forceful expulsion of contents) is repeated three times. Finally, the syringe is filled with at least 1 cc of blood, which represents a 3.9% dilution [5]. See Chapter 8 for further details about this technique.

Measurement with a Blood Gas Analyzer

The blood gas analyzer measures the pH, the PO_2, and the PCO_2 of the sample; the bicarbonate concentration and base deficit are calculated. The pH is measured using a pH-sensitive membrane. The pH is determined by measuring the voltage difference across this membrane.

The PCO_2 measurement is made by measuring the hydrogen ion difference between two solutions after allowing a chemical reaction that results in hydrogen ion production. Resultant hydrogen ion production is determined using a pH-sensitive membrane similar to that used for the pH measurement. The hydrogen ion production is directly proportional to the PCO_2 of the blood sample.

The PO_2 is measured using a Clark electrode, which uses oxidation and reduction reactions involving the oxygen in the sample to quantify the partial pressure of oxygen present. The change in current as a result of the oxidation–reduction reactions is proportional to the PO_2 of the blood gas sample. Once pH, PCO_2, and PO_2 are measured, a microprocessor then calculates the bicarbonate, base deficit, and temperature-corrected gas values (see later) [6].

Temperature Correction

Blood gas analysis is performed at 37°C, which may or may not reflect patient body temperature and thus in vivo blood gas values. When the patient's body temperature is entered into the blood gas analyzer, the machine calculates the blood gas results accurate for the individual. However, changes in temperature alter gas solubility (Charles law) and alter hemoglobin's affinity for oxygen. Taken together with the fact that the metabolic, vascular, and respiratory effects of hyper- and hypothermia are not fully understood, the value of temperature correcting blood gases remains controversial [7].

Evaluating Arterial Blood Gas Results

Normal arterial blood gas values for dogs and cats are listed in Box 26.2 [8].

Box 26.2 Normal Arterial Blood Gas Values for Dogs and Cats Breathing Room Air at Sea Level

Dog [8]

PaO_2: 92 mmHg (80–105)
$PaCO_2$: 37 mmHg (32–43)
SaO_2: > 95%

Cat [8]

PaO_2: 105 mmHg (95–115)
$PaCO_2$: 31 mmHg (26–36)
SaO_2: > 95%

Assessment of the Partial Pressure of Carbon Dioxide

The partial pressure of carbon dioxide (PCO_2) is the amount of CO_2 dissolved in blood and is determined by the balance between production in the tissues and elimination from the body; inhaled carbon dioxide from environmental air is negligible.

$PaCO_2$ is determined primarily by ventilation because when tissue CO_2 production increases, increases in respiratory rate and/or tidal volume (i.e. minute ventilation) normalize $PaCO_2$ in a healthy animal. Alterations in $PaCO_2$ are usually associated with neurologic or neuromuscular disease, respiratory depressant medications, or severe pathology of the upper or lower airways, pleural space, or chest wall.

Control of ventilation is primarily driven by the medulla. Increased PCO_2 diffuses into the cerebrospinal fluid and lowers its pH, which stimulates local chemoreceptors and triggers increased ventilation. Hypoxemia can also trigger central chemoreceptors to increase ventilation when the PaO_2 is less than 60 mmHg. The carotid and aortic bodies are peripheral chemoreceptors that can stimulate ventilation secondary to decreased PaO_2, increased $PaCO_2$, or a drop in pH [9].

Although this chapter focuses on arterial blood gas analysis, note that given adequate cardiovascular performance, PCO_2 (unlike PO_2) is similar in the venous and arterial blood. Therefore, venous blood can and is used to evaluate PCO_2 in cardiovascularly stable animals, with normal values expected to be only a few millimeters of mercury higher in venous than in arterial samples. The same is not true for evaluation of PO_2, which must be performed on arterial blood to provide information about the lung's ability to oxygenate the blood; venous PO_2 cannot provide this information.

Increased PCO_2 (Hypercapnia)

Respiratory acidosis (also called hypercapnia) is an increase in the partial pressure of carbon dioxide above the analyzer's reference interval for the species. This most commonly occurs due to decreased alveolar ventilation, which can occur for several reasons. Central nervous system dysfunction, as with anesthetics, traumatic brain injury, neoplasia, infections, or cerebral edema, can affect ventilatory drive. Diseases affecting the cranial cervical spinal cord such as intervertebral disk disease, neoplasia, infection, or inflammation can also lead to hypoventilation due to effects on the upper and lower (phrenic nerve) motor neurons responsible for diaphragmatic innervation. Peripherally mediated neuromuscular disease such as tetanus, botulism, and tick paralysis can also lead to a respiratory acidosis due to hypoventilation. Diseases of the pleural space, such as severe pleural effusion or pneumothorax, or diaphragmatic hernia can also result in hypoventilation but are more likely to cause severe hypoxemia before hypoventilation is clinically relevant. Obstructive diseases of the upper and lower airway alter airway resistance and can lead to hypercapnia. Tracheal collapse, an upper airway mass, or a foreign body can lead to upper airway obstruction and hypercapnia. Lower airway diseases such as asthma or bronchitis can also cause hypercapnia due to gas trapping in lower airways and decreased fresh gas introduction. Respiratory acidosis will result when there is increased CO_2 production in the tissues without the appropriate compensation in ventilation, as in heat stroke and malignant hyperthermia; however, such situations are much less common than inadequate CO_2 elimination as the cause for hypercapnia.

Depending on the cause, interventions may vary significantly, so the primary goal is to determine the underlying cause and attempt to fix it (e.g., reverse respiratory depressant medications, decrease anesthetic depth, relieve pleural space disorders). A $PaCO_2$ greater than 60 mmHg is a significant problem and warrants immediate correction of the underlying problem.

Decreased PCO_2 (Hypocapnia)

Respiratory alkalosis, or hypocapnia, is a decrease in the partial pressure of carbon dioxide below the reference interval of your analyzer for the species. Hypocapnia can occur secondary to lung disease, which stimulates pulmonary afferents to drive ventilation. Inadequate cellular energy production also triggers both central and peripheral chemoreceptors and increases ventilation. Examples of non-pulmonary conditions that cause inadequate cellular energy production include anemia, decreased cardiac output (shock, poor cardiac contractility), and mitochondrial dysfunction (as with sepsis or cyanide intoxication).

Animals with metabolic acidosis also may develop compensatory respiratory alkalosis in an attempt to normalize blood pH. See Chapter 57 for more information about acid–base status. A drop in the $PaCO_2$ below 25 mmHg can result in compromised cerebral blood flow [10] and should be addressed immediately by determining the underlying cause and addressing it.

Assessment of the Partial Pressure of Oxygen in Arterial Blood

Oxygen is carried in the blood in only two forms. One form is the portion of gas dissolved in the blood and measured as a partial pressure (PO_2). This represents only 2–3% of the total arterial blood oxygen content [11]. The other form is

the oxygen bound to hemoglobin in the red blood cells. In arterial blood gas analysis, the PaO_2 is measured to evaluate the lung's ability to oxygenate blood, often called simply "lung function." The movement of oxygen from the alveoli into the pulmonary capillaries occurs via diffusion down a concentration gradient. Alveolar partial pressure of oxygen (PAO_2) is higher than the partial pressure of oxygen in the pulmonary capillary as it leaves the pulmonary arteriole and approaches the alveolus. At the alveolar–pulmonary capillary junction, oxygen passively diffuses across the alveolar membrane into the pulmonary capillaries. The rate of diffusion is driven by Fick's law, which states that the diffusion of a gas across a membrane is directly proportional to the area of the tissue membrane and the pressure gradient, and inversely proportional to the thickness of the membrane [12]. Clinically, this means that diffusion will be altered if the surface area for gas exchange is decreased or if the respiratory membrane is thickened (which is uncommon in small animals). Decreased surface area for gas exchange occurs secondary to airway constriction (low V/Q) or lung unit collapse or consolidation (no V/Q: intrapulmonary shunting). PaO_2 is essentially a measure of the proportion of perfused lung units that are ventilated adequately to allow oxygen to diffuse into the pulmonary capillaries as they head to the left heart for systemic circulation. Hence PaO_2 is used as a primary measure of pulmonary function.

A PaO_2 less than 80 mmHg is considered hypoxemia, and oxygen supplementation should be considered. A PaO_2 less than 60 mmHg is considered severe hypoxemia, and immediate intervention is necessary.

Pulmonary Function Assessment Using Arterial Blood Gas Data

Once arterial blood gas values are attained, further analysis may be helpful to determine the severity of the pulmonary dysfunction and to rule out hypoventilation as the cause of hypoxemia. A summary of these analyses is available in Box 26.3.

Alveolar to Arterial Oxygen Gradient

After gas exchange occurs, the dissolved oxygen content in the pulmonary capillary is normally the same as the oxygen content in the alveolus ($PaO_2 \sim 105$ mmHg at sea level on room air) because diffusion is complete. Oxygenated

Box 26.3 Pulmonary Function Assessment Using Arterial Blood Gas Data

Alveolar–Arterial Oxygen Gradient

$$\text{A–a gradient} = PAO_2 - PaO_2$$
$$= \left[FiO_2\left(P_B - P_{H_2O}\right) - PaCO_2 / RQ\right] - PaO_2 \quad (26.1)$$

- Normal ≤ 15 mmHg at FiO_2 0.21 (room air), normal ≤ 150 mmHg at FiO_2 1.0 (100% oxygen); values in excess of 15 mmHg on room air indicate pulmonary dysfunction.
- Use: At any P_B, at any $PaCO_2$.
- Reliable only at room air or 100% oxygen.

The "120" Rule

$PaCO_2 + PaO_2 \geq 120$: adequate lung function
$PaCO_2 + PaO_2 < 120$: abnormal lung function (26.2)

- Use: Only at sea level on room air.

PaO_2/FiO_2

$$PaO_2/FiO_2 = \text{"P : F ratio"} \quad (26.3)$$

- Normal ≥ 500 mmHg; mild pulmonary dysfunction 300–500 mmHg; moderate pulmonary dysfunction consistent with acute lung injury 200–300 mmHg; severe pulmonary dysfunction consistent with acute respiratory distress syndrome < 200 mmHg.
- Use: At $FiO_2 > 0.21$ (ideally used when oxygen is supplemented, as $PaCO_2$ is not taken into account).
- Expected values apply only to sea level; extrapolated, reliable values would be available at other P_B.

$FiO_2 \times 5$ Rule

$$\text{Normal } PaO_2 \geq (FiO_2, \%) \times 5 \quad (26.4)$$

- Use: At $FiO_2 > 0.21$ (ideally used when oxygen is supplemented, as $PaCO_2$ is not taken into account)
- Expected value of $PaO_2 \geq 5 \times FiO_2$ applies only to sea level; extrapolated, reliable values would be available at other P_B

Total Arterial Oxygen Content

$$CaO_2 = \left(SaO_2 \times Hgb \times 1.34\right) + \left(0.003 \times PaO_2\right) \quad (26.5)$$

- Normal in dogs: 16.9–18.0 ml/dl
- Relevant at all P_B, PCO_2, and FiO_2

pulmonary capillary blood then flows to the left heart for systemic circulation. However, a small amount of blood (from bronchial and Thebesian circulations) normally returns to the left heart deoxygenated; this small amount of deoxygenated blood mixes with the arterialized blood returning from the pulmonary capillaries, which drops the PaO_2 below the PAO_2. The normal difference between PAO_2 and PaO_2 (the "A–a gradient" or "A–a difference") should be less than 15 mmHg and is generally considered "physiologic shunting." However, when an increased amount of blood enters the left atrium without being oxygenated (for instance, because it perfused lung units that were not well ventilated due to a low V/Q or no V/Q scenario), the excessive deoxygenated blood further dilutes the properly arterialized blood coming from functional lung units. This situation is commonly referred to as venous admixture. To determine whether there is pathologic venous admixture, the A–a gradient can be calculated (see Box 26.3, equation 26.2); increased gradients are associated with underlying pathology and help direct diagnostic tests and intervention.

Where the FiO_2 is the fraction of inspired oxygen (0.21 or 21% on room air), P_B is the barometric pressure (~ 760 mmHg at sea level), and P_{H20} is the vapor pressure of water. P_{H2O} does vary with temperature, but generally 47 mmHg is used because this is the vapor pressure of water at 37°C (human body temperature). The $PaCO_2$ is measured from the arterial blood gas sample, and RQ is the respiratory quotient, which is approximately 0.8. This assessment also works when the patient is receiving 100% oxygen, in which case the expected gradient is less than 150 mmHg. For fractional inspired oxygen levels between room air and pure oxygen, the expected gradient has not been established and must be extrapolated [13].

The "120" Rule

Because the PCO_2 affects PAO_2 (see Box 26.3, expanded Eq. 26.3), when an animal is at sea level breathing room air, one can estimate what PaO_2 to expect when one knows the $PaCO_2$ by using the "120 rule." When an animal is breathing room air at sea level, the sum of $PaCO_2$ and PaO_2 is generally 120–160 mmHg. When the sum of $PaCO_2$ and PaO_2 is less than 120, pulmonary dysfunction is present [13]. The major inherent limitation to this method is the requirement for room air, sea level conditions. The major advantage is its ease of use.

PaO_2/FiO_2 ("The P : F Ratio")

Equation Eq. (26.4) can be used to evaluate pulmonary function at any FiO_2, which provides an advantage over the A-a gradient and the 120 rule. The value is acquired by dividing the PaO_2 by the FiO_2:

$$P:F\,\text{ratio} = PaO_2 / FiO_2$$

where the FiO_2 is expressed as a decimal. With normal pulmonary function, the P : F ratio should exceed 500 mmHg. The P : F ratio can be used to approximate the severity of pulmonary dysfunction. Animals with P : F between 300 and 500 mmHg have mild lung dysfunction; those with P : F between 300 and 200 mmHg have moderate dysfunction, and those with P : F less than 200 mmHg are considered to have severe dysfunction. This calculated ratio, along with several other criteria such as acute onset of respiratory distress, bilateral dorsocaudal pulmonary infiltrates, absence of fluid overload or congestive heart failure, and an appropriate underlying disease process, is used to identify veterinary patients with acute lung injury and acute respiratory distress syndrome [14]. Values less than 500 mmHg indicate that there is compromised pulmonary function and diagnostics are indicated. The advantage of this method is that it is simple, quick, and can be used at any FiO_2. The main disadvantage of the method is the disregard for the effect of ventilation (PCO_2), although this is only really an issue when making the calculation on room air. The P : F is thus most appropriately used in animals receiving supplemental oxygen.

$FiO_2 \times 5$

Another way to approximate the expected PaO_2 is to multiply the FiO_2 (as a whole number percentage) by five. With normal pulmonary function the PaO_2 on room air at sea level is approximately 100 mmHg, which is about five times the FiO_2 expressed as a whole number (21). This can then be extrapolated out to estimate what the PaO_2 should be with normal pulmonary function for any given FiO_2. Values attained that are less than 5 indicate lung dysfunction [13]. Advantages and disadvantages are identical to those described for the P : F ratio.

Calculation of the Total Oxygen Content

As stated earlier, O_2 is carried in the blood two ways: dissolved in plasma and attached to hemoglobin. Total blood O_2 content can be calculated easily using the blood gas analyzer results if a hemoglobin (Hgb) concentration is known (or estimated). For arterial blood,

$$CaO_2, \text{ml / dl} = \left(\left[\text{Hgb}\right] \times 1.34 \times SaO_2\right) + \left(PaO_2 \times 0.003\right)$$

where CaO_2 is the total arterial blood oxygen content, SaO_2 is the saturation of hemoglobin with oxygen expressed as a decimal (see later), and 1.34 and 0.003 are constants.

Normal O_2 content in dogs is reported to be 16.9–18.0 ml/dl [15], although an idealized canine value is closer to 20 ml/dl. Note that red blood cell mass has a far more profound effect on total blood oxygen content than does PO_2 within the survivable range.

Venous Samples

Although arterial samples are preferable for assessment of both oxygenation and ventilation, a venous sample can also help evaluate the respiratory system.

Venous Partial Pressure of Carbon Dioxide

There is an expected arterial–venous gradient for venous PCO_2 ($PvCO_2$). Venous blood contains CO_2 from the metabolically active tissue bed(s) upstream from where the sample was acquired, and as such, it generally has a PCO_2 approximately 5 mmHg higher than arterial blood. CO_2 produced in the tissues is carried in several forms by the blood to the lungs for removal. The dissolved PCO_2 represents approximately 10% of the total CO_2 [13]. Most of the CO_2 is buffered within the red blood cell and then transported as bicarbonate to the lungs where this process is reversed to facilitate removal of CO_2 by the lungs. The disadvantage of venous samples is that in certain disease states, the arterial-to-venous PCO_2 gradient may increase. Such disease states include anemia, where there is a decrease in the ability to buffer the CO_2. Venous stasis (as in cardiovascular instability from hypovolemia or cardiogenic shock) also increases the gradient. As a general guide, a $PvCO_2$ greater than 48 mmHg indicates hypoventilation assuming no concurrent perfusion compromise.

Venous Partial Pressure of Oxygen

The venous PO_2 (PvO_2) cannot be used to determine lung function; however, it can be used for other purposes. For instance, PvO_2 can be used to determine the oxygen extraction ratio (OER), a ratio that sheds some light on the adequacy of oxygen delivery in comparison with the patient's oxygen consumption. Traditionally, calculation of the OER requires a mixed venous sample collected from the distal port of a pulmonary arterial catheter; however, blood from a central venous line (a catheter with a distal port near the right atrium) is probably adequate for most purposes. See Chapter 19 for more information regarding the OER. Normal PvO_2 values range from 40 to 50 mmHg. Generally, when PvO_2 is less than 30 mmHg, this may be an indication of inadequate oxygen delivery (via any of the mechanisms mentioned above), and diagnostics to determine the cause should be considered if the patient's clinical condition supports this finding.

Saturation of Hemoglobin with Oxygen

Blood gas analyzers often report the saturation of oxygen (SO_2). This value represents the amount of oxygen bound to the hemoglobin molecule and is reported as a percentage of hemoglobin saturation with oxygen. Blood in which all hemoglobin oxygen sites are bound with oxygen is 100% saturated; blood in which 75% of hemoglobin oxygen sites are bound with oxygen is 75% saturated. The arterial hemoglobin oxygen saturation (SaO_2) is calculated in most blood gas analyzers from the pO_2, pH, and bicarbonate values; the analyzer assumes normal conditions for the calculation [13]. Some blood gas analyzers may have co-oximetry functionality that allows direct measurement of SO_2 from the blood sample, but this is uncommon in veterinary medicine. The oxyhemoglobin equilibrium curve (also called the oxyhemoglobin dissociation curve) shows the relationship between the PO_2 and the SO_2 graphically; it is a sigmoid curve where initially there is rapid binding of oxygen to the hemoglobin molecule. Pulse oximetry is a noninvasive method of estimating the PaO_2 using the oxyhemoglobin dissociation curve. See Chapter 25 for more information about the oxyhemoglobin equilibrium curve and these monitoring tools.

Summary

Blood gas analysis allows detailed assessment of a patient's respiratory function. Arterial blood gases provide the opportunity to monitor a patient's pulmonary function in response to time and therapy, particularly when tools like the A–a gradient and the P : F ratio are used. Arterial and venous blood gases can be used to inform the clinician and technician about the patient's ventilatory adequacy. Venous partial pressure of oxygen can be used as a marker of adequacy of tissue perfusion. Blood gas analysis is readily available and relatively inexpensive with the use of bedside monitoring equipment.

Acknowledgment

This chapter was originally co-authored by Drs. Sarah Gray and Lisa Powell for the previous edition, and some material from that chapter appears in this one. The author and editors thank Dr. Powell for her contributions.

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27

Point-of-Care Lung and Pleural Space Ultrasound

Søren Boysen and Valerie Madden

Two major structures assessed during thoracic veterinary point-of-care ultrasound (VPOCUS) include the pleural space and lungs, both of which are commonly associated with pathology in companion animals presenting in respiratory distress. Historically, pleural space pathology has been evaluated with thoracic focused assessment with sonography for trauma (TFAST), while lung pathology has been evaluated using regional lung scanning protocols such as Vet BLUE® or more comprehensive protocols that scan a larger lung surface area [1–8]. As it is not possible to assess the lungs without concurrently visualizing the pleural space, the two have been assessed concurrently in human medicine as pleural and lung ultrasound (PLUS) [9]. PLUS has been described in companion animals and has the advantage of incorporating both TFAST and regional lung ultrasound principles into a single rapid protocol that takes advantage of sonographically identifiable borders (Figure 27.1) to orientate operators and assesses the most sensitive sites for pathology, which vary based on patient positioning [2]. The other advantage of PLUS is that it scans more regions of the thorax than some regional lung ultrasound protocols and takes advantage of different transducer orientations, both of which have been shown to increase the chances of finding pleural and lung pathology [2, 8, 10–13].

Like other VPOCUS protocols, PLUS can be applied at triage, keeping the examination focused to the most likely cause of dyspnea as suggested by signalment, triage evaluation, history, and/or clinical examination. It can be used serially to monitor progression or resolution of identified pathology (e.g. severity of B-lines following furosemide therapy for congestive heart failure). In addition, it can be used to guide interventions (e.g. ultrasound-guided thoracocentesis). Finally, PLUS can be applied systemically as part of routine patient assessment (Chapter 6).

Indications for Pleural and Lung Ultrasound

There are a variety of indications for PLUS in the emergency and critical care setting. Primary indications include assessment of the pulmonary parenchyma for evidence of pathology and/or volume overload as well as assessment for pleural space disease [1–7]. This, in conjunction with point-of-care abdominal and cardiac ultrasound performed in the clinical setting, provides additional relevant information to patient assessment and management (Chapter 6).

Patient Positioning and Machine Settings

PLUS is ideally performed with the patient in a standing position or sternal recumbency, especially those presenting in respiratory distress [2]. This minimizes restraint, limits stress (work of breathing), and avoids potential respiratory decompensation in dyspneic patients. Patients with respiratory distress should be provided oxygen therapy, anxiolytics, and other stabilization measures performed concurrently with PLUS, to ensure patient safety. Do not compromise patient safety to complete any VPOCUS evaluation. An exception to standing/sternal positioning is a patient with a flail chest, as these patients ideally should be placed in lateral recumbency with the flail side down as a stabilization measure. If necessary, lung ultrasound may be performed in lateral recumbency in more stable patients that tolerate lateral restraint or in patients with concurrent injuries that may preclude sternal positioning (e.g. spinal fracture) [2].

Patient positioning has a significant effect on where pleural space pathology accumulates; fluid falls to gravity-dependent

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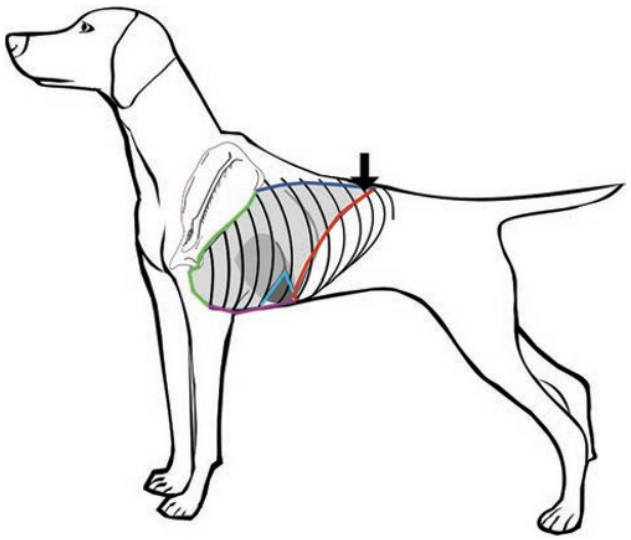


Figure 27.1 Identifying the sonographically identifiable pleural and lung ultrasound (PLUS) borders is essential to orientate the operator, and ensures assessment of the most sensitive areas pleural space pathology accumulates. The sonographically identifiable PLUS borders include the caudal border (abdominal curtain sign, outlined in red), dorsal border (hypaxial/lumbar muscles, outlined in dark blue), cranial border (flexor muscles of the shoulder, thoracic inlet, outlined in green) and ventral border (pectoral/sternal muscles, outlined in pink). Pleural space pathology often accumulates at these borders and regional lung scanning occurs within the borders. It should be noted that the ventral border of the lung deviates dorsally at the cardiac notch, which is important if the goal is to look for ventral lung pathology or pleural space pathology (ventral border of the lung at the cardiac notch is shown in light blue). The black arrow indicates the most caudal dorsal location in the standing patient, which will be the most sensitive site for detection of the pneumothorax in the standing or sternal patient. The nongravity-dependent widest part of the chest will be the most sensitive location for detection of pneumothorax in the laterally recumbent patient (not shown in the image).

regions while air rises to non-gravity-dependent areas [2]. Therefore, the most sensitive sites to identify pleural effusion and pneumothorax are not the same for patients in lateral compared with standing/sternal (i.e. in standing/sternal air with pneumothorax will accumulate caudodorsally while fluid with pleural effusion accumulates ventrally; in lateral recumbency, air accumulates at the widest gravity independent point, fluid at the widest most gravity-dependent point). PLUS should be modified based on the specific questions asked and patient positioning [2].

Recognizing the sonographically identifiable PLUS borders helps to orientate the sonographer where the transducer is positioned on the thorax and ensures assessment of the most sensitive areas where pleural space pathology accumulates [2, 10]. The thoracic borders that can be identified during PLUS include the caudal border (abdominal curtain sign, see below), dorsal border (hypaxial/lumbar muscles), cranial border (flexor muscles of the shoulder, thoracic inlet) and ventral border (pectoral/sternal muscles). Pleural space pathology often is localized at these borders and regional lung scanning occurs within the borders (Figure 27.1) [2, 10].

Depth is determined by the body condition score of the patient and is typically set at 4–6 cm for PLUS. Generally, the depth is adjusted until the pleural line occupies the proximal third of the image.

Lung sliding, also known as the "glide sign" is often easier to see with shallow depths (2–3 cm), but the depth should be increased again after assessing lung sliding because other pathology (B-lines, Figure 27.2) and landmarks such as the abdominal curtain sign (Figure 27.3) can be missed with shallow depth settings.

Decreasing the gain often makes the pleural line appear grainy, subsequently allowing lung sliding to become more visible; however, if gain is reduced to assess lung sliding it should be increased again to allow B-lines and other findings to be more easily identified.

Placing the transducer over a single rib (centered in the ultrasound image) may allow lung sliding to be more easily visualized. This ultrasound image is referred to as the "dead bat" [14] (Figure 27.4) or "one-eyed gator" sign [15].

If the transducer is oriented across intercostal spaces (perpendicular or transverse to the ribs), the "bat sign" is

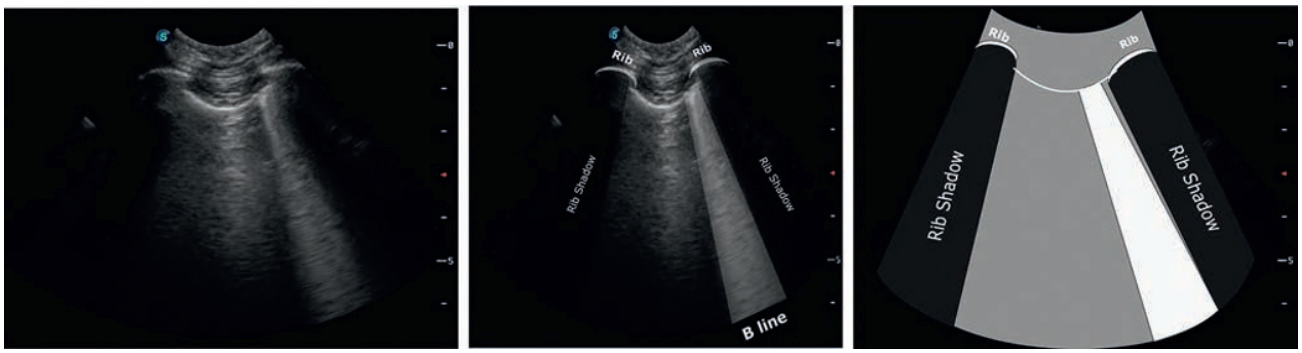


Figure 27.2 Ultrasound still and schematic images of a single B-line. B-lines appear as white vertical narrow (individual) to broad (coalescing) bands that extend vertically from the lung surface that move with lung sliding during respiration. B-lines often obliterate A-lines and usually extend to the far field of the ultrasound image (see text for more detail on B-lines).

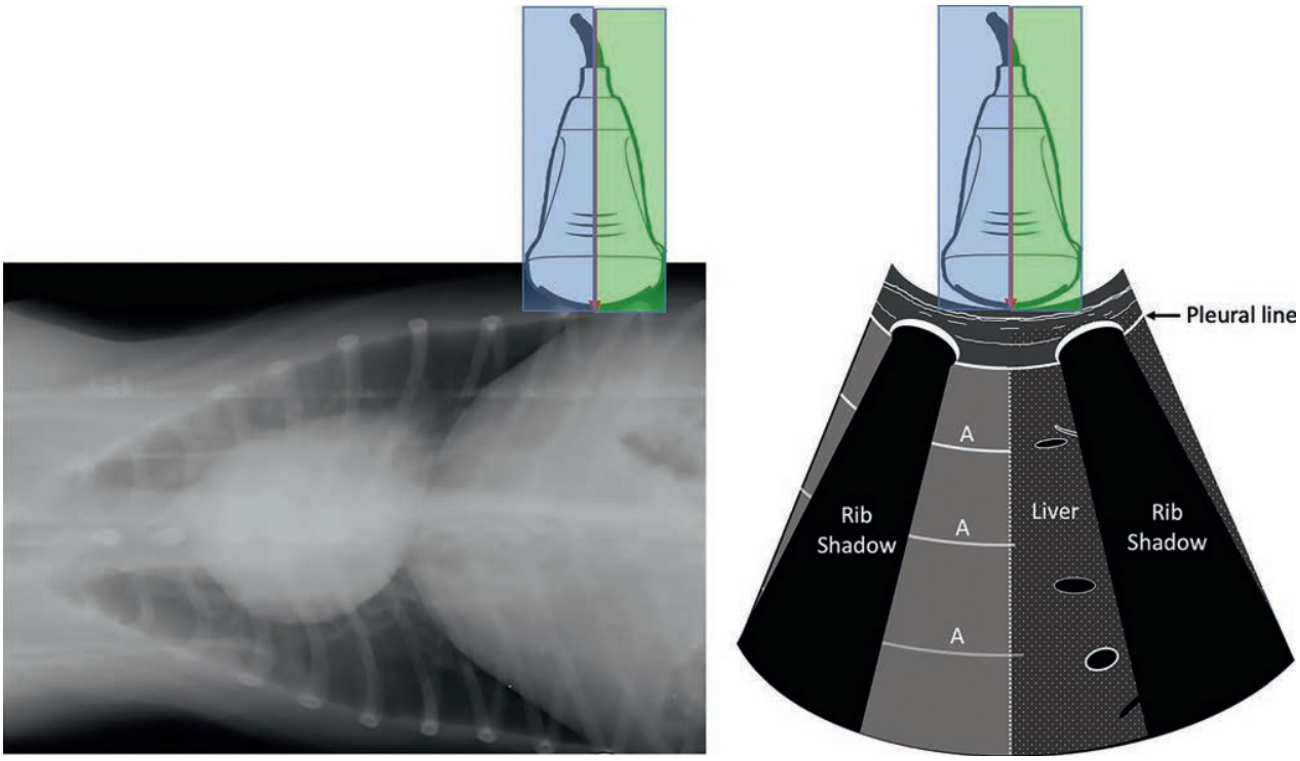


Figure 27.3 Chest radiograph demonstrating the location, and schematic image demonstrating the appearance, of the abdominal curtain sign in a healthy animal. When the transducer is situated such that it is partially over aerated lung (blue shaded half of the ultrasound transducer) and partially over the soft tissue structures of the abdomen (green shaded half of the transducer), a sharply demarcated vertical artifact (vertical dotted white line) becomes sonographically visible at the transition of aerated lung to soft tissue abdominal structures (marked by the red arrow). In this example the transducer is positioned over the liver, which makes up the soft tissue structures caudal to the curtain sign. The abdominal curtain sign helps with directing the transducer to the most gravity independent (pneumothorax caudodorsally) and dependent (pleural effusion caudoventrally) sites with the patient in standing/sternal recumbency and may help differentiate pleural from pericardial effusion and/or confirm the presence of pneumothorax. A, A-lines.

created (Figure 27.5), which provides orientation by identifying proximal rib surfaces, rib shadows, and the pleural line. The bat sign thus allows lung sliding to be assessed, and orientation is achieved [14].

Placing the transducer parallel to the intercostal space allows an extended view of the pleural surface and may be preferred in some situations (see below) [2, 10, 11]. There is also evidence in both the human and veterinary literature that a parallel transducer orientation relative to the ribs improves detection of some pleural space and lung pathology [2, 10–12].

The appearance of pathology is different when using a phased array transducer, although evidence suggests linear and microconvex transducers can be used interchangeably [16, 17].

Curvilinear Transducer

The curvilinear transducer is most commonly used and is the authors' preference for PLUS scanning. Its larger footprint enables examination of a wider field of the pleural surface. This transducer may operate at low frequencies, and achieves good depth for identification of translobar lung consolidation (see below).

Linear Transducer

If a higher resolution transducer is required once pathology is found, a change to the linear transducer is reasonable. The linear transducer operates at a higher frequency and is good at interrogating finer or more shallow structures such as the pleural surface. This is the transducer of choice for examining the ribs for fractures (not covered here).

Phased Array Transducer

The advantage of the phased array transducer is that the small transducer footprint means that it is easier to achieve a view in a small intercostal space. The limited field of view thus increases the risk of missing pathology that is localized distant from the transducer. It is reasonable to use when diffuse pathology is suspected (pleural effusion, pulmonary edema). Note that the appearance of pathology, such as B-lines, differs based on the transducer used;

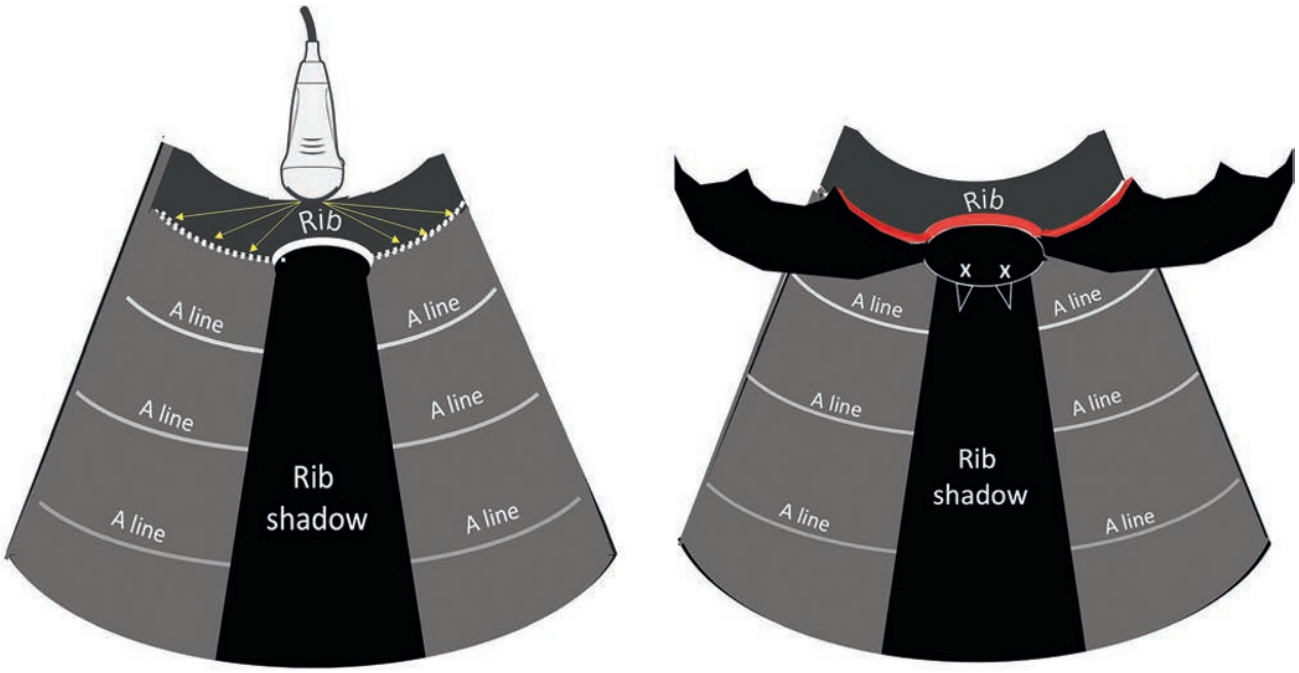


Figure 27.4 Schematic images of the "dead bat sign" formed when a rib is centered within the ultrasound image. When a single rib is visible it creates the image of an upside down "dead bat" where the body of the bad forms the proximal rib and the wings of the bat form the pleural line. Centering the transducer over a single rib reflects more of the ultrasound beam away from the transducer, which makes the pleural line appear "grainier" and lung sliding (aka the glide sign) easier to visualize (the curved dotted white lines represent the pleural line).

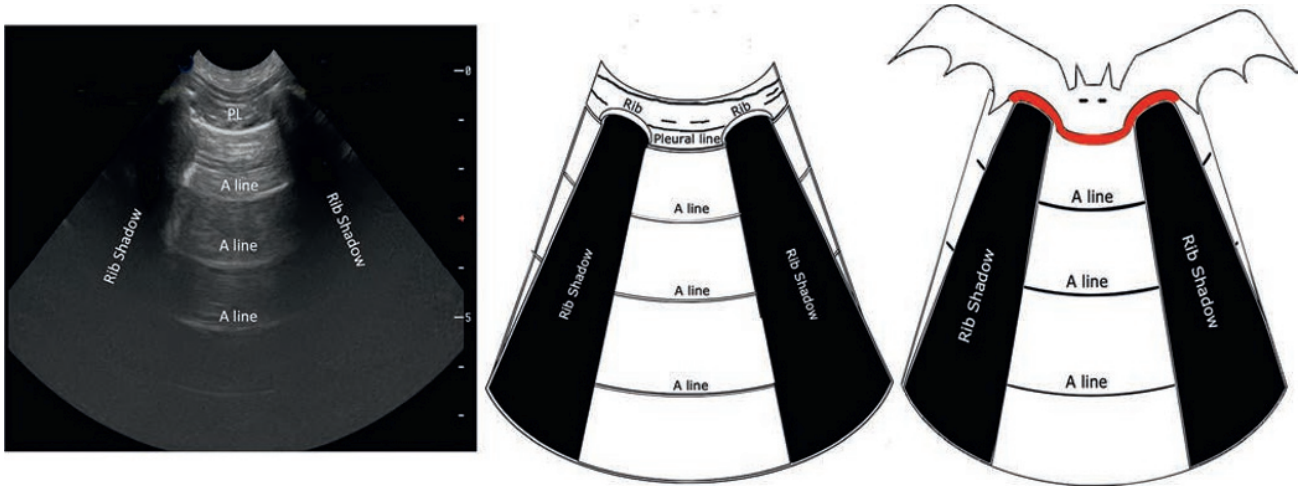


Figure 27.5 Still image and schematic representations of the BAT sign when the transducer is orientated perpendicular to the ribs. BAT is used as it makes the mnemonic "bone and air with the transducer transverse to the ribs," which helps novice operators know they should be looking for bone (ribs) and the soft tissue air interface (air filled lung in healthy animals) when the transducer is transverse (perpendicular) to the ribs. Two ribs are identified in the image as downward facing white curvilinear structures (bat wings), both of which cast an acoustic shadow (black lines extending through the far field of the image) obstructing the view of anything deep to them. Between the two ribs is a thin white line approximately 0.5 cm deep to the proximal rib surface (varies by patient size and body condition score), joining the rib shadows, which is the pleural line. The pleural line demarks the soft tissue air interface and the "body" of the bat. A-lines are visualized as horizontal white lines that appear parallel to the pleural line and extend throughout the depth of the ultrasound image at regular intervals. The intensity of the A-lines decreases with depth as some of the ultrasound beam is absorbed each time the beam returns to the transducer. PL, Pleural line.

therefore, operators should only use a transducer with which they are comfortable, which will be the microconvex curvilinear transducer in many cases [17].

Technique and Identification of Normal Lung Ultrasound Images Compared With Pathology

Pleural and Lung Ultrasound Scanning Technique

The most sensitive thoracic location to sonographically identify pathology varies depending on the clinical scenario, patient position, and specific question asked [2]. Therefore, the choice of the first point of investigation of PLUS often depends on patient assessment and suspicion of type of pathology present. For example, the pleural space may be evaluated first if pleural space disease is anticipated compared with the lungs in patients with suspected primary parenchymal disease.

The transducer may be oriented either perpendicular or parallel to the ribs [2]. A parallel orientation allows more lung surface to be visualized and may be more sensitive for detection of pathology [11, 12]. For novice sonographers, perpendicular orientation allows for easier recognition of normal structures via identification of the "bat sign" (Figure 27.5) [14].

The transducer orientation should remain consistent. The indicator marker on the transducer should be oriented toward the patient's head when scanning perpendicular to the ribs and oriented dorsally (toward the spine) when scanning parallel to the ribs [14].

Technique for Pleural Effusion

When scanning the pleural space for effusion, moderate to large volumes can easily be identified in the ventral third of the thorax at the level of the costochondral junction with the patient in sternal recumbency. For patients in lateral recumbency, the widest, most gravity-dependent area should be scanned. With large volume pleural effusion, the accuracy of detecting fluid in the ventral third of the thorax is high regardless of ultrasound transducer orientation [10].

To identify smaller volumes of pleural effusion, the most ventral borders of the pleural space should be scanned with the patient in a standing or sternal position, with the transducer parallel to the ribs; this creates the "ski jump" sign (Figure 27.6) [2, 10, 14].

To identify the most ventral pleural borders of the thorax, a change in orientation of the transducer and the window into the thorax is required. The transducer is slid caudally from the mid-thoracic region until the abdominal curtain sign is identified, and then ventrally along the abdominal curtain sign until the heart becomes visible or the costochondral junctions are reached (Figure 27.7). This is the pericardiodiaphragmatic window (Figure 27.8). If obvious pleural fluid is not seen with the transducer perpendicular to the ribs, the transducer can be rotated until it is parallel to the ribs with the marker directed dorsally. Slide the transducer ventrally, maintaining a parallel orientation, until the pectoral/sternal muscles fill one-third of the sonographic image, which the authors refer to as the "ski jump" sign (Figure 27.6) [2, 14].

Technique for Pneumothorax

To scan the pleural space for pneumothorax, the most caudodorsal regions should be scanned with the patient in a standing/sternal position, or the widest, non-gravity-dependent point of the thorax for patients in lateral recumbency [2]. To locate the most caudodorsal site in the standing or sternal patient, the transducer is slid caudally from the mid-thoracic region until the abdominal curtain sign is identified, and then slid dorsally along the abdominal curtain sign until the pleural line disappears in the hypaxial muscles (Figure 27.9). Slide the transducer ventrally until the pleural line is again visible: this is the most caudodorsal location [2]. The appearance of lung sliding at this location rules out pneumothorax for the pertinent hemithorax in the sternal/standing patient; lung sliding need not be assessed elsewhere on this hemithorax. The other hemithorax should be similarly interrogated. The objective of the remaining PLUS exam should focus on pathology that arises from the pleural line (e.g. B-lines, pleural effusion, subpleural consolidation).

Regional Lung Scanning

Much of lung ultrasound is based on the interpretation of artifacts produced at the lung surface and relates to the way ultrasound and air interact with each other at this location. Ultrasound can only be used to interpret the characteristics of the surface of a soft tissue–air interface because the ultrasound beam cannot penetrate gas. Since pathology lying deep to a soft tissue to gas interface cannot be visualized, lung ultrasound is a surface imaging modality. Fortunately, most pulmonary pathology involves the pleural surface to some extent.

There are several lung ultrasound protocols published in veterinary medicine, all of which have similar findings with regards to lung pathology, and no studies have compared the accuracy of one lung protocol over another [2–7]. A canine study published as an abstract in 2014 [13], and a feline study (in press at time of writing) [8] suggest that lung ultrasound protocols that evaluate more lung surface area can detect more pathology. Regardless of the actual lung ultrasound protocol chosen, they all tend to have two things in common: (i) they scan multiple lung regions

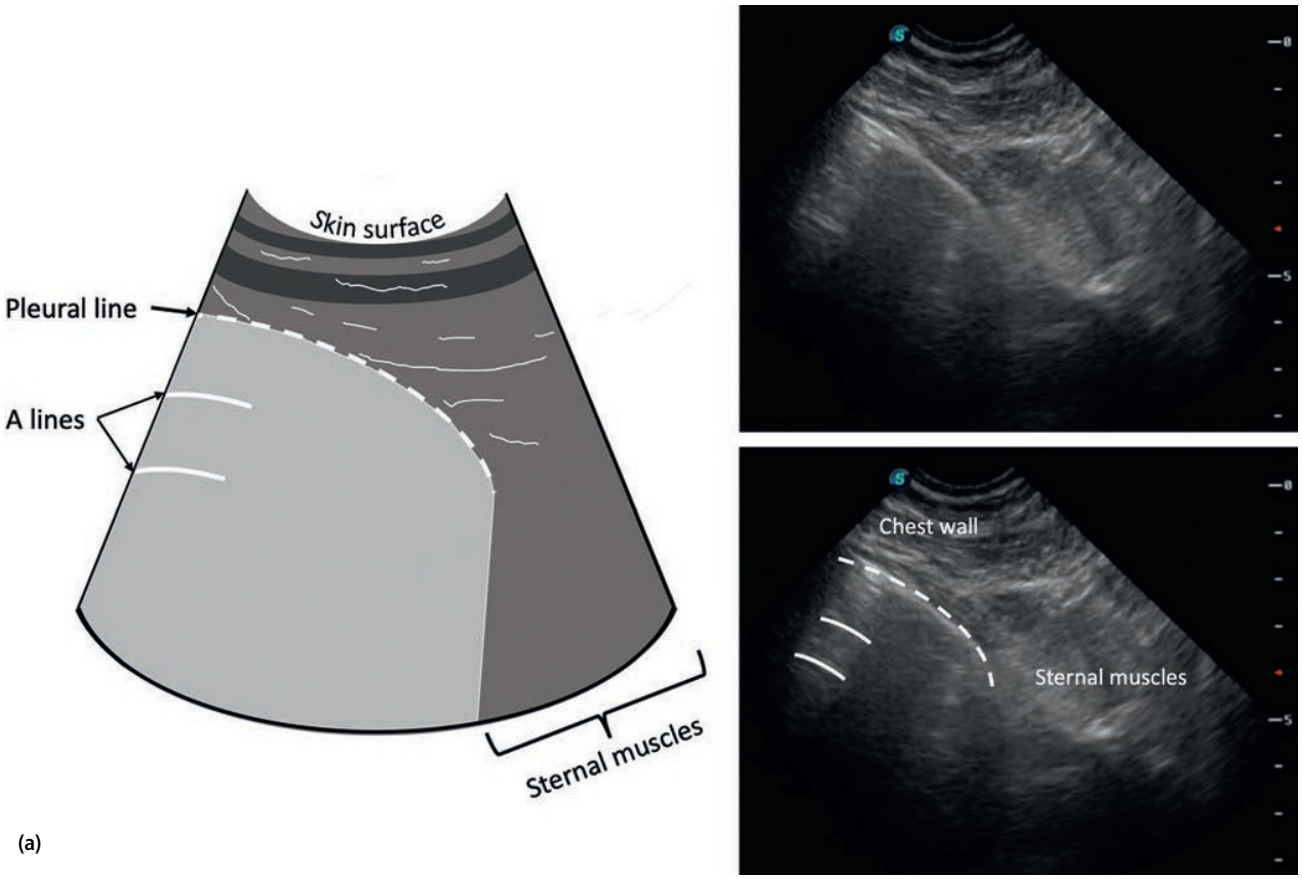


Figure 27.6 Schematic and still images depicting the "ski jump" sign with the transducer oriented parallel to the ribs at the most ventral regions of the thorax. The proper image is obtained by first finding the pericardiodiaphragmatic window (or costochondral junction), turning the transducer parallel to the ribs with the marker directed dorsally and sliding the transducer ventrally until pectoral/sternal muscles fill one-third to half of the sonographic image. The transducer angle may need to be rocked slightly to maintain contact in round chested patients. (a) Still ultrasound image obtained when the transducer is situated over lung and slid ventrally until the pectoral/sternal muscles fill one-third of the sonographic field on the right side of the thorax. The curved white dotted line represents the interface between the pleura, which resembles an Olympic ski jump. (b) Still ultrasound image obtained when the transducer is situated over the heart at the cardiac notch and slid ventrally until the pectoral/sternal muscles fill one-third of the sonographic field on the right side of the thorax. The curved white dotted line represents the interface between the pericardium and the parietal pleura, which resembles an Olympic ski jump. (c) Each intercostal space is assessed by sweeping the transducer cranial a rib space at a time, while also sliding ventrally and dorsally within each intercostal space to assess the ventral lung and ventral pleural space borders (blue double headed arrows represent sliding the transducer parallel to the ribs between the ventral sternal and ventral lung regions). LV, left ventricle; LVFW, left ventricular free wall; RV, right ventricle; RVFW, right ventricular free wall.

bilaterally, and (ii) they often start at the most caudodorsal site in a sternal/standing patient (Figure 27.10) [2–7].

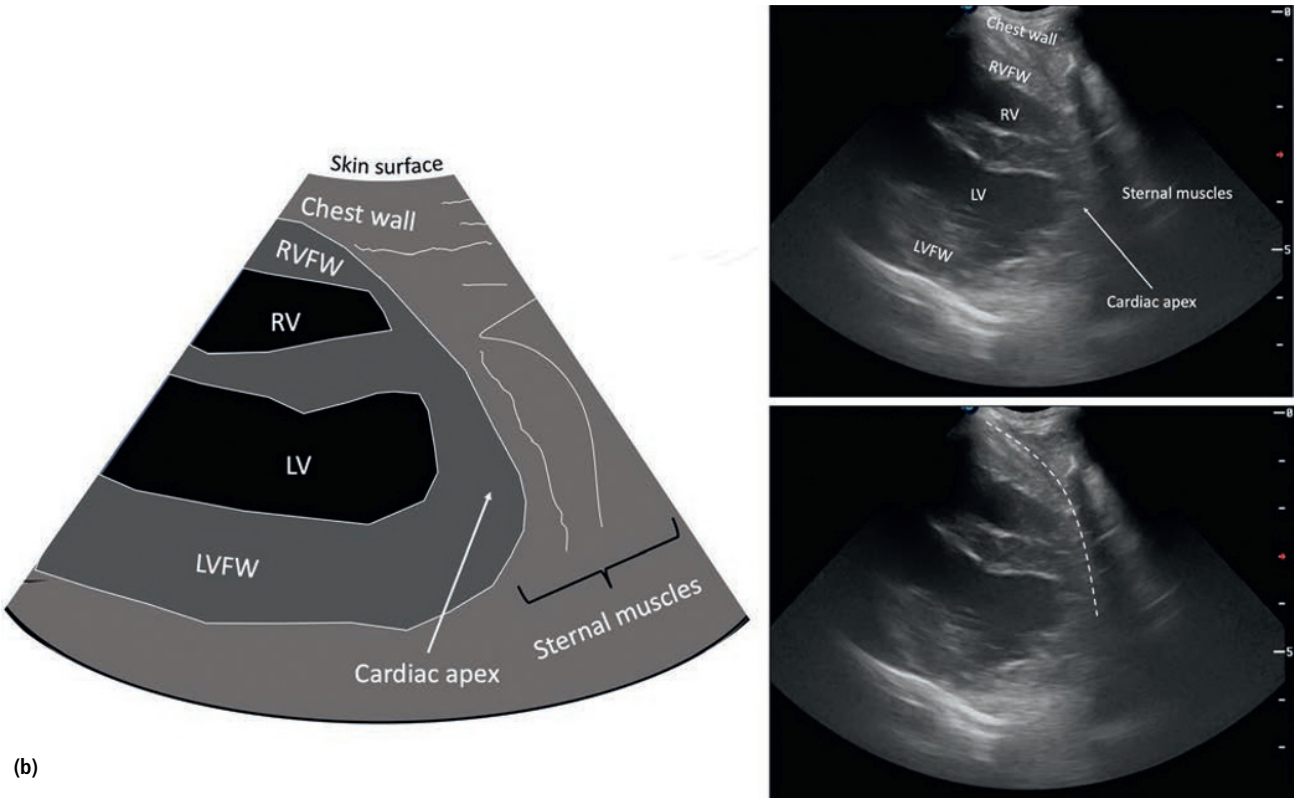
To assess the lungs, novice sonographers should begin by placing the transducer caudal to the cranial lung border (thoracic limb; between the fifth and sixth intercostal spaces), at the level of the middle to upper 2/3 of the thorax (Figure 27.11) [2, 18]. This will allow identification of the pulmonary parenchyma and avoid inadvertent identification of the "abdominal curtain sign," cardiac notch, abdominal cavity, or musculature (the transducer should be over only ribs and lung to start). From this point, depending on the clinical scenario and specific clinical question to be answered, different pleural space and lung pathologies can be assessed (Figure 27.12): (i) multiple lung regions can be scanned for lung surface pathology; (ii) the ventral pleural space can be assessed as described above; or (iii) the dorsal pleural space can be assessed as described above. With experience, structures are more easily identified and the operator can begin closer to the sonographic thoracic borders.

Many protocols also include the subxiphoid site for evaluation of the lungs using the liver as an acoustic window into the thorax, which is also the authors' preference (Figure 27.13) [2].

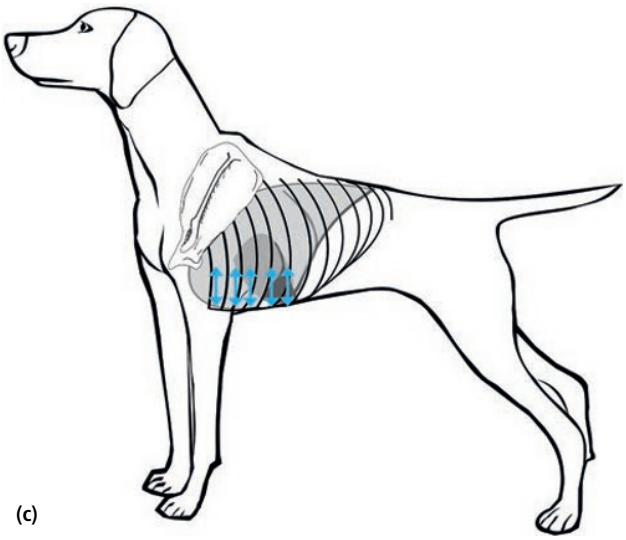
Normal Structures and Signs

The BAT or Gator Sign

The BAT sign is the term preferred by the authors, as it creates the mnemonic "bone and air with the transducer transverse to the ribs," which helps novice operators know



(b)



(c)

Figure 27.6 (Continued)

that they should be looking for bone (ribs) and the soft tissue–air interface (air-filled lung in healthy animals) when the transducer is transverse (perpendicular) to the ribs [14]. Two ribs are identified in the image as downward-facing white curvilinear structures (bat wings), both of which cast an acoustic shadow (black lines extending through the far field of the image) and obstruct the view of anything deep to them. It is often easiest for novices to identify the rib shadows in the far field of the ultrasound image and follow them toward the near field until the rib surface is located (i.e. where the rib shadow ends in the near field of the ultrasound image). Between the two ribs is a thin white line approximately 0.5 cm deep to the proximal rib surface (varies by patient size and body condition score), joining the rib shadows, which is the *pleural line*. The pleural line demarcates the soft tissue–air interface

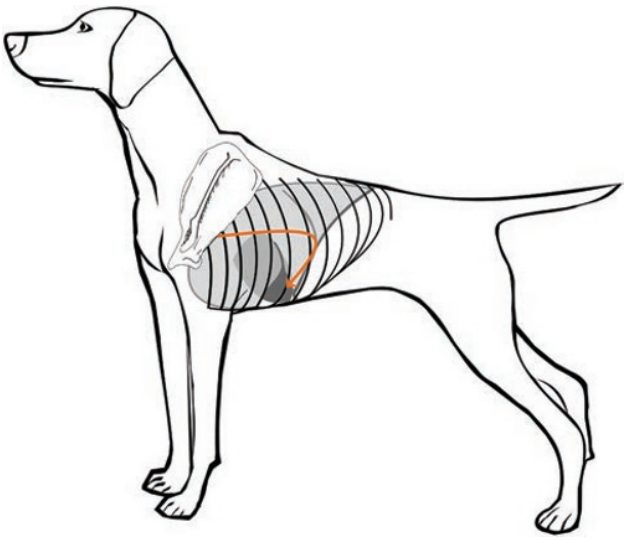


Figure 27.7 To identify the most ventral pleural borders of the thorax, the transducer is slid caudally from the mid-thoracic region until the curtain sign is identified, and then ventrally along the curtain sign until the heart becomes visible or the costochondral junctions are reached (orange curved arrow). This is the pericardiodiaphragmatic window. Moderate to large volumes of pleural effusion can be identified at this location.

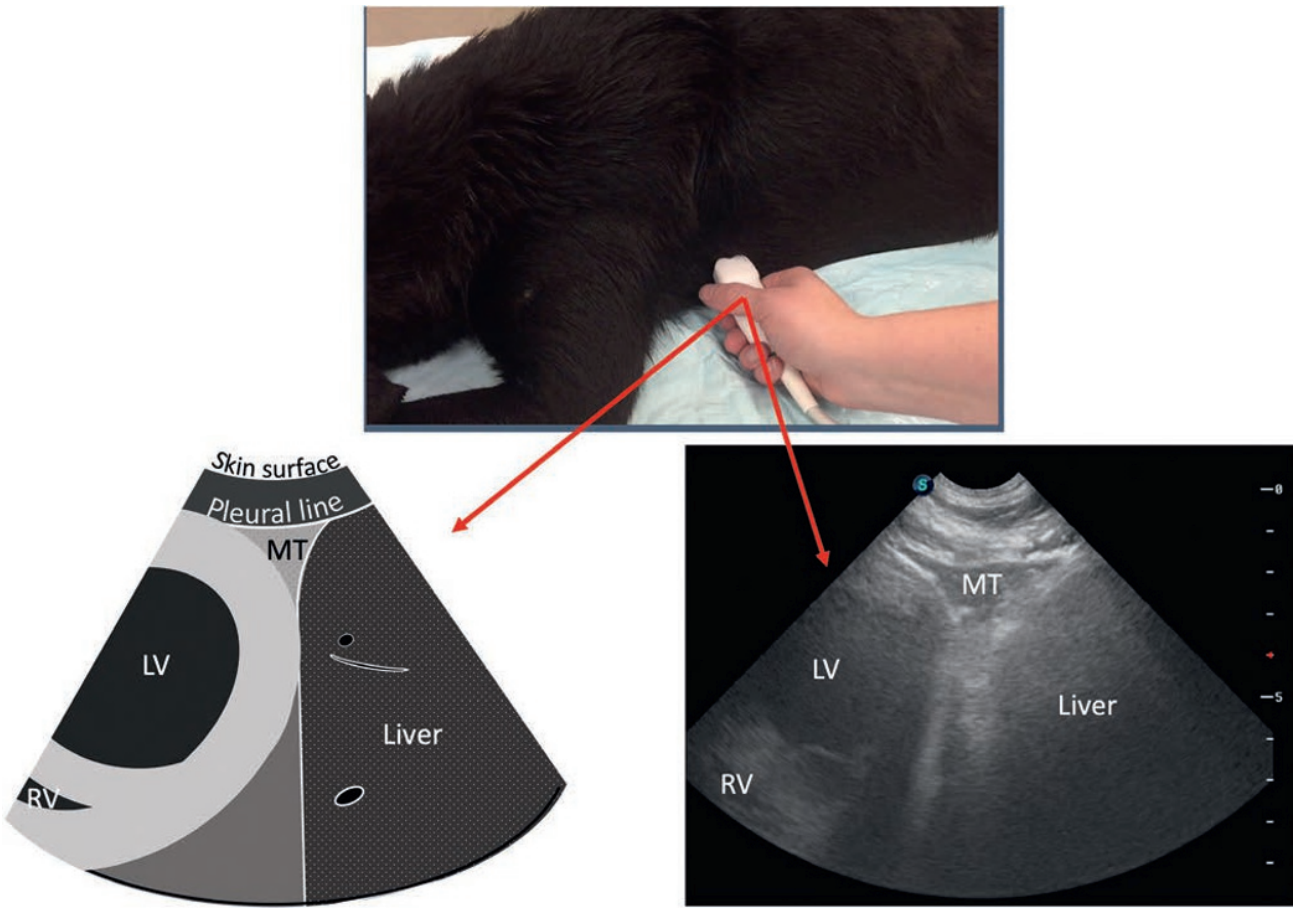


Figure 27.8 Photograph, schematic and still image from a dog depicting the pericardio-diaphragmatic window ventrally where the heart and the diaphragm can be visualized within the same sonographic image. The dog (upper image) is positioned in sternal recumbency (frog legged, head to the left of the photo) with the traducer placed perpendicular to the ribs on the left side of the chest, and the indicator marker directed cranially. The transducer will be situated close to the costochondral junction at roughly the sixth intercostal space, although the exact location of the transducer will vary slightly between patients as sonographic landmarks are used to find the correct window. LV, left ventricle; MT, mediastinal triangle; RV, right ventricle.

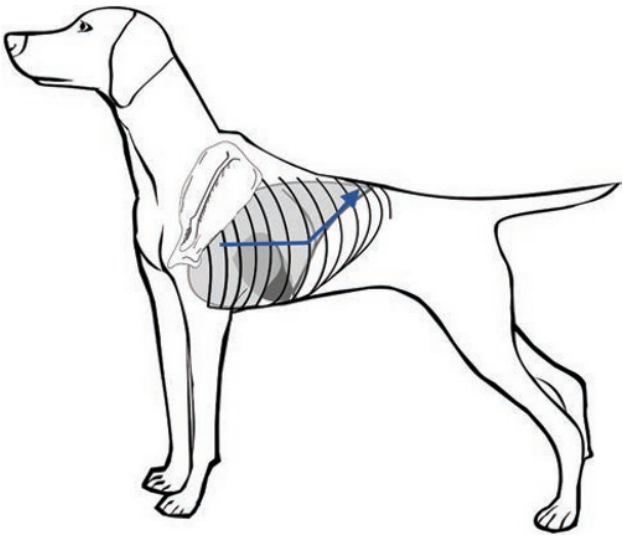


Figure 27.9 To locate the most caudal–dorsal site the transducer is slid caudally from the cranial–mid-thoracic region until the abdominal curtain sign is identified, and then slid dorsally along the curtain sign until the pleural line disappears in the hypaxial muscles (blue arrow). Slide the transducer ventrally from the hypaxial muscles until the pleural line is again visible; this is the most caudal–dorsal location.

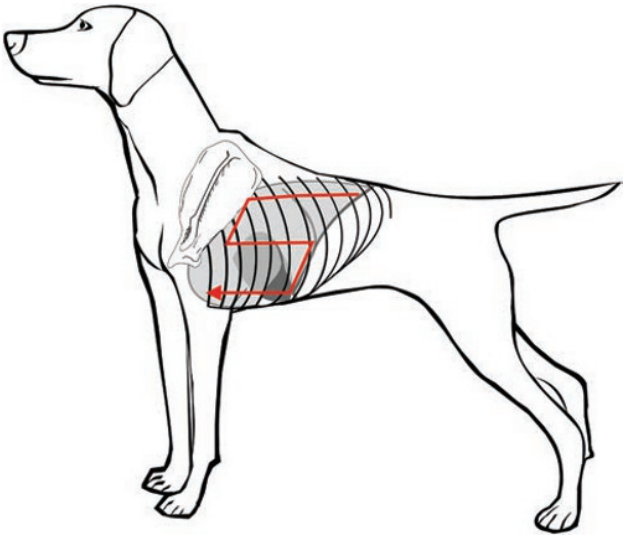


Figure 27.10 The sliding pleural and lung ultrasound protocol begins at the most caudodorsal site and scans the dorsal third, middle third, and ventral third of the thorax bilaterally between sonographically defined lung borders to ensure multiple lung regions are assessed in a systematic "S" shaped fashion (red arrow). Pulling the skin toward the starting location (like when placing a chest tube) will significantly reduce the quantity of alcohol required to scan the lungs sites as the transducer and skin can be moved together.

and the "body" of the bat. With the gator sign, the wings of the bat are exchanged for the "eyes" of an alligator and the body of the bat is exchanged for the "bridge of the nose" of an alligator [15].

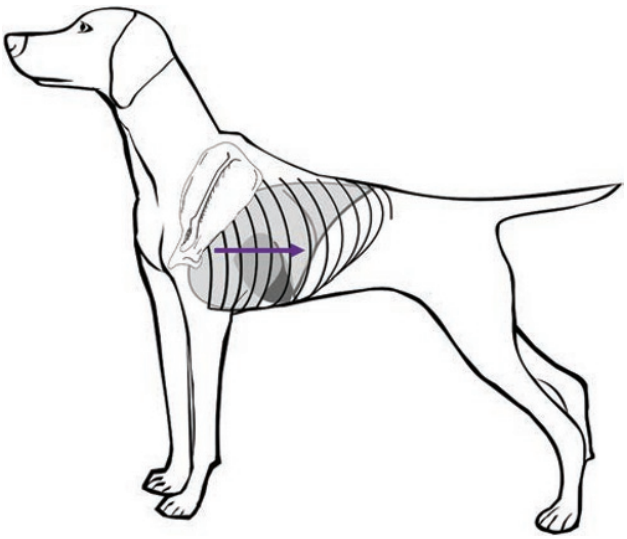


Figure 27.11 To assess the lungs or to consistently find the abdominal curtain sign, novice sonographers should begin by placing the transducer just behind the cranial lung border (thoracic limb; between the fifth and sixth intercostal spaces), at the level of the middle to upper two-thirds of the thorax (purple arrow). This allows identification of the pulmonary parenchyma and avoids inadvertent identification of the "abdominal curtain sign," cardiac notch, abdominal cavity, or musculature (the transducer should be over only ribs and lung to start).

"Glide Sign" or Lung Sliding

The visceral and parietal pleura are usually closely apposed with a minute volume of fluid between them, sliding over one another with each respiration. Unfortunately, the two pleura and scant pleural fluid are not normally identifiable with ultrasound. Instead, a sliding/shimmering of a single thin white pleural line can be seen, which indicates the two pleural surfaces are in contact. Two criteria are needed to see lung sliding: (i) the visceral (lung) and parietal (chest wall) pleura must be in contact; and (ii) the pleura must move or "slide" along each other. Identification of lung sliding rules out the presence of a pneumothorax at that transducer location. The appearance of the glide sign or lung sliding is best appreciated with experience. Use of a high frequency transducer may enhance ability to identify lung sliding in some instances.

A-Lines

A-lines are a horizontal reverberation artifact created by the reflection of the ultrasound beam back and forth between two highly reflective surfaces: (i) the ultrasound transducer; and (ii) a soft tissue–air interface. With lung ultrasound, the normal soft tissue–air interface is comprised of the two pleura and the air-filled lung below the pleural line. A-lines are visualized as horizontal white lines that appear parallel to the pleural line and extend throughout the depth of the ultrasound image at regular intervals (Figure 27.14). The intensity

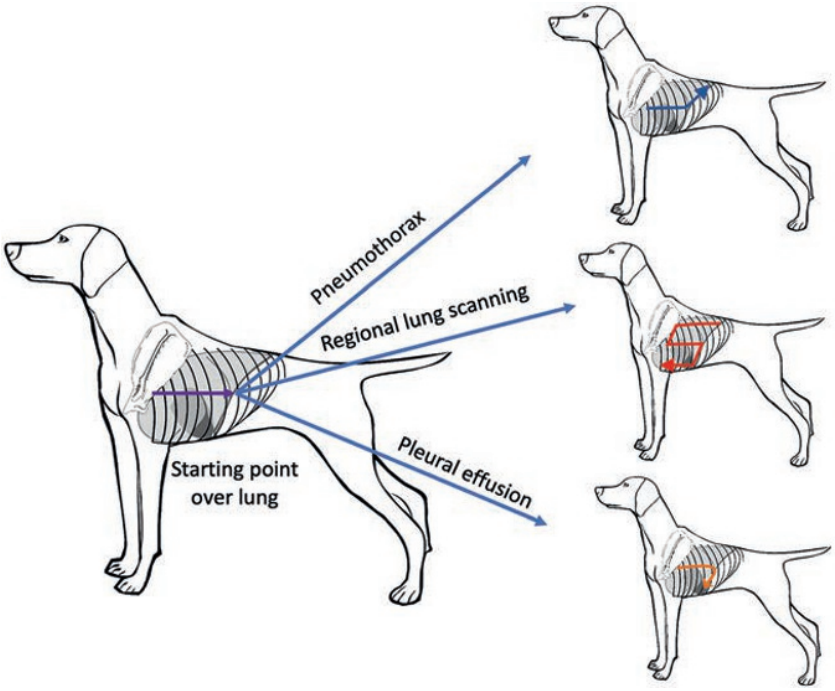


Figure 27.12 Depending on the clinical presentation and triage findings different clinically relevant questions may need to be ruled out with greater urgency than others. The advantage of pleural space and lung ultrasound is that it allows the operator to rule in/out the most urgent life-threatening conditions first. Novices should start at the purple arrow and identify the curtain sign and then tailor the protocol depending on the most probable life-threatening condition; (i) pneumothorax, blue arrow; (ii) alveolar interstitial disease or lung consolidations, red arrow; (iii) pleural effusion, orange arrow. With experience structures are more easily identified and operators can begin closer to the sonographically defined PLUS borders.

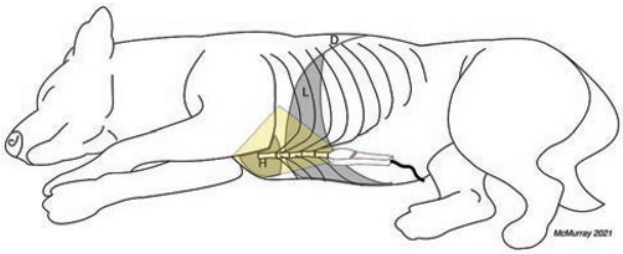


Figure 27.13 Schematic image of the subxiphoid site, which is a great location to identify pericardial, lung and pleural pathology and may allow some degree of cardiac function to be estimated. The depth setting must be increased at this location to be able to sonographically visualize regions beyond the diaphragm. The transducer may also need to be rocked almost parallel to the spine to assess the ventral pleural and lung regions, as well as the apex of the heart. D, diaphragm; H, heart; L, liver.

of the A-lines tends to decrease with depth because some of the ultrasound beam is absorbed each time the beam returns to the transducer. A-lines can be seen with any thoracic soft tissue air interface (i.e. they are present with aerated lung and with pneumothorax). A-lines are considered a "normal finding." Also, because only a 2–3 mm depth of aerated lung need be present to create reverberation artifact, lung pathology that is separated from the pleural surface by as little as 2–3 mm of aerated lung cannot be seen, and only reverberation artifact and A-lines will be visualized [19].

B-Lines

B-lines (also called "comet tails" or "lung rockets") occur when there is decreased aerated lung at the lung periphery, most often secondary to increased extravascular lung water (i.e. pulmonary edema) [20–22]. If lung is in contact with the chest wall, B-lines originate at the pleural line and extend perpendicularly into the lung. As many as three B-lines per sonographic window may be normal, with one to three B-lines occurring in 10–30% of healthy dogs and cats [6–8]. It is more common to see only one B-line at one to two locations on either hemithorax, or two B-lines at a single location when scanning the lung surfaces. Although three B-lines at a single location has been described in healthy cats and dogs, this number of B-lines in a single sonographic window is rare and should not occur at multiple lung locations over the same hemithorax [7, 8].

There are five criteria used to define a B-line (Figure 27.2):

1) They are white vertical narrow (individual) to broad (coalescing) bands.
2) They extend vertically (perpendicularly) from the lung surface.
3) They move in sync with lung sliding during respiration.
4) They often obliterate A-lines.
5) They usually extend to the far field of the ultrasound image.

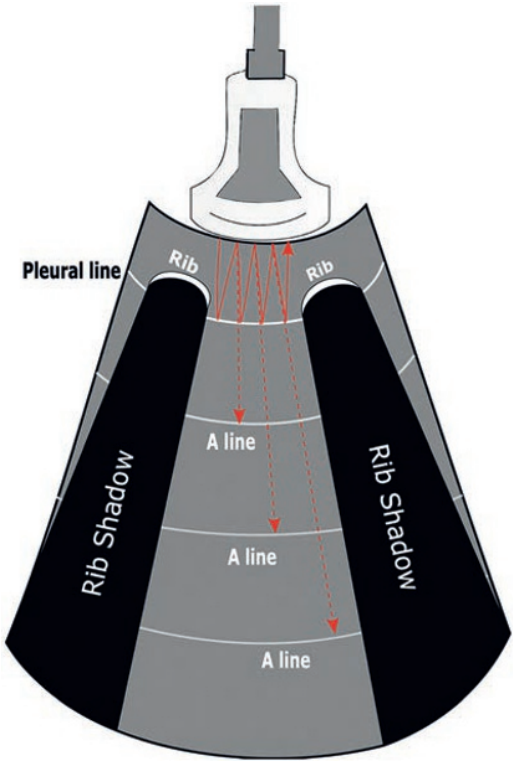


Figure 27.14 Schematic image depicting the creation of A-lines caused by the reflection of the ultrasound beam between two highly reflective surfaces (transducer surface and the soft tissue air interface). Red zigzag arrow represents the beam being reflected between the pleural line and the transducer surface. Dotted red arrows represent the "perceived distance" the ultrasound machine detects A-lines each time the beam is reflected back to the transducer surface.

Multiple individual to coalescing B-lines (more than three per sonographic window) indicate the presence of decreased aerated lung at the lung periphery, often referred to as "wet lung" [7, 23]. However, it should be kept in mind that increased B-lines can result from any decrease in aerated lung, including atelectasis [21, 22], and therefore, although the most common cause is increased extravascular lung water (hence the term "wet lung"), there are other causes of increased B-lines (Table 27.1).

Table 27.1 Causes of diffuse and localized B-lines, with common history and clinical findings.

Multiple diffuse bilateral B-lines	Focal/unilateral/localized B-lines
Interstitial pneumonia or pneumonitis (e.g. lymphocytic interstitial pneumonitis, eosinophilic pneumonia, drug reactions)	Focal pneumonia/ pneumonitis, particularly aspiration: often a history of vomiting
Pulmonary edema – cardiogenic or non-cardiogenic: heart murmur common with cardiogenic causes, may have history of seizures, chewing an electrical cord, upper airway obstruction or smoke inhalation	Pulmonary contusions: often a history of trauma
Diffuse parenchymal lung disease (e.g. pulmonary fibrosis)	Atelectasis: often a history of prolonged recumbency and/or anesthesia
Acute respiratory distress syndrome: may have spared lung regions	Pleural disease: interpret B-lines cautiously when pleural effusion is present as compression atelectasis can cause B-lines
	Neoplasia: often associated with sonographic areas of focal lung consolidation, particularly nodules

Abdominal Curtain Sign

Once lung sliding is identified, the transducer is moved caudally along the thorax to achieve identification of the abdominal curtain sign. In healthy animals, the abdominal curtain sign is sonographically identified as a sharply demarcated vertical artifact that occurs at the transition of aerated lung to soft tissue abdominal structures and demarcates the caudal border of PLUS (Figure 27.3) [2]. The curtain sign helps with directing the transducer to the most gravity independent (caudodorsally to find pneumothorax) and dependent (caudoventrally to find pleural effusion) spots with the patient in standing/sternal recumbency and may help differentiate pleural from pericardial effusion or confirm the presence of pneumothorax (see pleural effusion and abnormal abdominal curtain signs below).

The abdominal curtain sign is created by the combination of two factors:

1) The acoustic impedance mismatch between air (air-filled lung in healthy animals) and soft tissue (abdominal structures) that casts the characteristic sharp vertical artifact at the interface between the two; and
2) The anatomical relationship of the thorax with the abdomen, resulting in the costophrenic recess covering parts of the cranial abdomen and the diaphragm, which abuts against air-filled lung cranially in healthy patients.

The overlap of the costophrenic recess onto the abdomen creates a sharp, vertically demarcated lung–air–soft-tissue artifact. In essence, the caudal aerated lung border acts similarly to a "curtain"; when it fills with air, it expands sliding caudally into the costophrenic recess and over visible abdominal organs, momentarily obscuring them from view, like a curtain is drawn across a window obscuring what is visible through the window. The underlying organs disappear as the lung expands across them on inspiration and reappear as the lung deflates on exhalation.

Ventral Pleural Border and the "Ski Jump" Sign

The ski jump sign is used to describe the normal appearance of the sonographically defined ventral pleural border in the absence of pleural space pathology (Figure 27.6) [14]. When the transducer is oriented parallel to the ribs at the sonographically defined ventral pleural border either lung or heart can be seen curving away from the chest wall along the pectoral sternal muscles. This creates an ultrasound image with a similar appearance to an Olympic ski jump. The ski jump sign comprises either sternal/pectoral muscles interfacing with lung (Figure 27.6a) or sternal/pectoral muscles interfacing with cardiac muscle (Figure 27.6b) depending on whether the transducer is situated over the lung or the cardiac notch, respectively. The ski jump sign is used to rule out smaller volumes of pleural effusion that might otherwise be missed with the transducer oriented transverse to the ribs (see pleural effusion below) [2, 10].

Abnormal Findings in Patients with Pleural Space and Pulmonary Pathology

Pleural Effusion

Pleural effusion appears as an anechoic/hypoechoic accumulation of a fluid medium between the body wall and the visceral pleura/lung surface. Note that lung sliding is lost when pleural effusion is present because the fluid separates the visceral from the parietal pleural surfaces. However, because the ultrasound beam can transverse fluid, the lung surface (the visceral pleura) is still visible distal to the fluid. The appearance of the effusion varies depending on volume, location, cellularity, and presence of fibrin within the pleural space.

Locating the pericardiodiaphragmatic window (Figures 27.7 and 27.8) helps to differentiate pleural effusion from pericardial effusion (Figure 27.15). While pericardial effusion is contained within the pericardial space and curves away from the diaphragm and around the heart, pleural effusion is not contained and may be identified as triangular hypoechoic to anechoic structures along the borders of the diaphragm filling the costophrenic recess and outlining lung lobes.

In the absence of pericardial effusion, the pericardium should appear as a bright white line surrounding the border of the myocardium. Pleural effusion may be located adjacent to the pericardium in addition to surrounding other normal thoracic structures (lungs, diaphragm, mediastinum, vessels) or thoracic pathology (e.g. intrathoracic mass).

The "sail sign" is most readily identified by placing the transducer parallel to the ribs. It appears as an accumulation of fluid between the lung and ventral sternal muscles, which creates a triangular shape of anechoic fluid like that of a sail (Figure 27.16) [14]

The "jellyfish sign" shows consolidated lung floating in a volume of pleural effusion, so named because the lung takes on the shape of a jellyfish suspended in water (Figure 27.17) [24].

Pneumothorax

Pneumothorax is defined by the interposition of gas between visceral and parietal pleural layers. Thoracic ultrasound has been shown to be a rapid, noninvasive, sensitive,

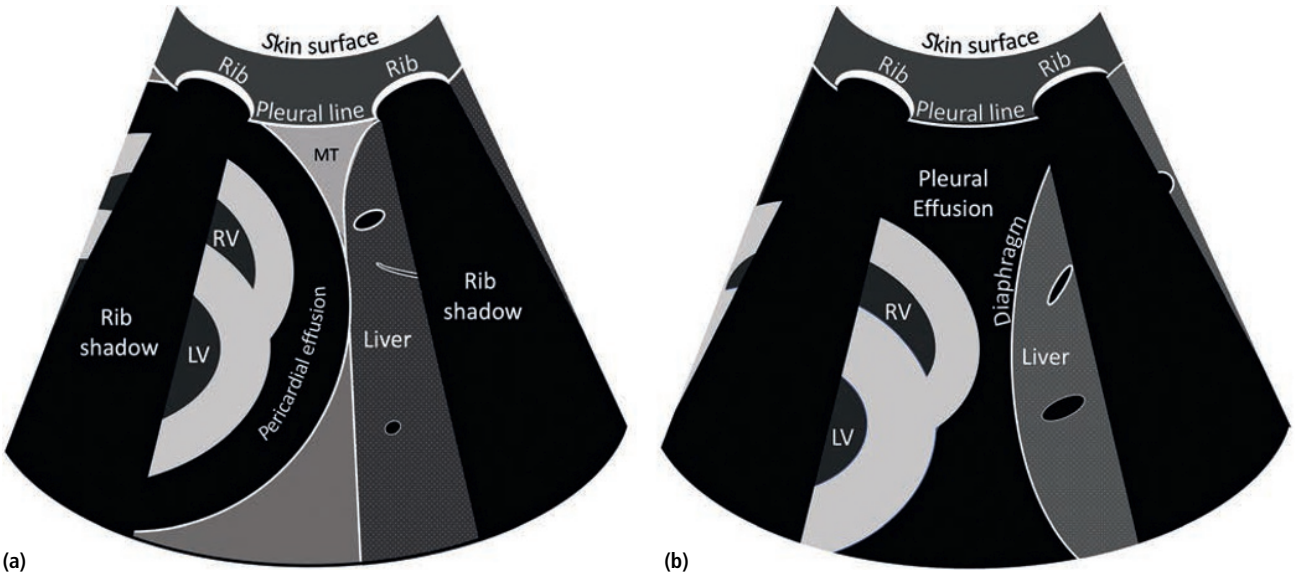


Figure 27.15 Schematic images of pericardial and pleural effusion at the right pericardiodiaphragmatic window. (a) Pericardial effusion is contained within the pericardial sac and curves away from the diaphragm. (b) Pleural effusion is uncontained and tends to track along the diaphragm, filling the costophrenic recess which allows the diaphragm to be seen curving away from the chest wall. LV, left ventricle; RV, right ventricle.

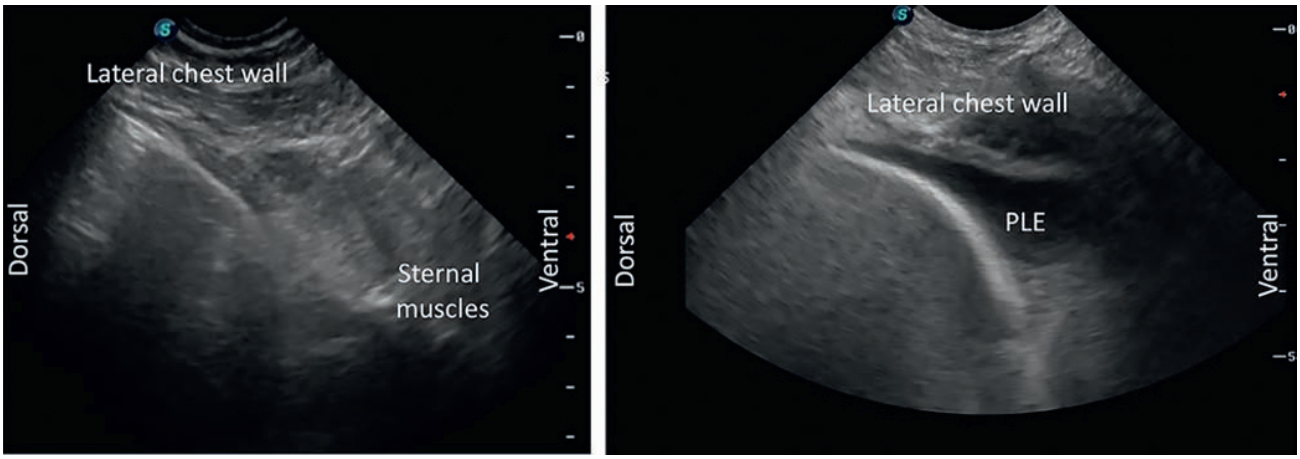


Figure 27.16 Still ultrasound images of the ventral pleural border with the transducer oriented parallel to the ribs and the indicator marker directed dorsally. The image on the left is referred to as the "ski jump" sign and rules out significant pleural effusion. The image on the right is referred to as the "pleural sail sign" and describes the elevation of the lung away from the ventral chest wall and sternal muscles which creates a fluid silhouette like a billowing spinnaker sail from a boat. PLE, pleural effusion.



Figure 27.17 The finding of consolidated lung floating within pleural effusion is referred to as the "jellyfish" sign.

and specific diagnostic modality for ruling out or confirming the presence of a pneumothorax.

PLUS is often preferable to standard survey radiography in the emergency clinical setting because it requires minimal patient manipulation and may limit transport and restraint of critical patients. In addition, PLUS allows rapid identification of patients with pneumothorax with limited operator skill necessary.

Timely identification of a pneumothorax with limited restraint may provide the safest means of identifying this issue and allows for rapid institution of life saving interventions, such as thoracocentesis.

Regarding sensitivity and specificity of lung ultrasound compared with standard survey thoracic radiography, human studies demonstrate a sensitivity and specificity of 87% and 97% for lung ultrasound and 28% and 100% for thoracic radiography, respectively [25]. Thus, thoracic ultrasound is a more sensitive test for pneumothorax than radiography and an excellent early diagnostic screening tool for this condition. The sensitivity and specificity of ultrasound to diagnose pneumothorax in companion animals is not well established.

Means of Assessing for a Pneumothorax

When sonographically assessing pneumothorax, the patient is often maintained in a standing position or in sternal recumbency to limit restraint and respiratory compromise. The transducer is placed in the intercostal space either parallel or perpendicular to the ribs, using a high frequency setting with the depth decreased to maximize evaluation of the body wall and parietal–visceral pleural interface.

There are four criteria that are used to help rule out or confirm the presence of a pneumothorax: lung sliding and B-lines rule out the presence of a pneumothorax at the location at which they are identified, while the lung point and the double/asynchronous abdominal curtain signs rule in pneumothorax (see below) on that side of the thorax [2].

Lung Sliding/Glide Sign Rules Out Pneumothorax Where Sliding is Identified

As previously discussed, as a standard rule, identification of lung sliding or the glide sign rules out the presence of a pneumothorax at the transducer location [26]. Look for the classic "shimmering" of the pleural line as the lung slides along the thoracic wall with respiration. If lung sliding is detected in the dorsal one-third of the thorax, then a significant pneumothorax is unlikely on that side of the chest.

Absence of lung sliding in a patient with a history or clinical signs consistent with a pneumothorax should alert the clinician to the potential presence of this condition.

One challenge is that lung sliding, even without pneumothorax, can be difficult to identify, which is particularly true when the patient has a rapid shallow breathing pattern [25]. Therefore, the absence of lung sliding should prompt consideration of a pneumothorax. If the history and signalment are supportive, along with other clinical findings (e.g. absence of breath sounds), thoracocentesis is indicated.

If the patient is sufficiently stable and the operator wishes to further rule in pneumothorax, a search for the lung point and/or abnormal curtain signs should be undertaken (see below) [2]. If the clinician is uncertain, diagnostic thoracentesis may be performed to confirm or rule out this condition or a single thoracic radiograph may be performed if the patient is adequately stable to tolerate transport and restraint for this diagnostic.

B-Lines Rule Out Pneumothorax at the Location Where They are Identified

Like lung sliding, B-lines rule out the presence of a pneumothorax as they should move with the pleural line and should be identified with lung sliding [27]. They appear as bright white lines originating at the pleural line, extending vertically downwards and should move with the lung during respirations (see B-lines, above). Note that A-lines can still be present with a pneumothorax (see A-lines, above).

Lung Point Confirms Pneumothorax

Lung point is the point over the thorax where the return of lung sliding (with or without underlying B-lines) is identified in patients with pneumothorax (Figure 27.18) [27]. Note that where the lung regains contact with the thoracic wall, it creates lung sliding within only a portion of the ultrasound image when the patient breathes.

The lung point can be identified by sliding the transducer toward the hilus of the lung. Given air rises to the highest nongravity sites, in the standing or sternal patient this will often involve sliding the transducer from the caudodorsal regions toward the elbow, from an area where no lung sliding was detected. Using the lung point the degree of pneumothorax is estimated by dividing the chest into thirds.

If lung sliding is detected in the dorsal one-third of the thorax, pneumothorax is likely ruled out or very mild on that side of the chest. To increase the sensitivity of detecting small volume pneumothorax that might get larger over time, the sonographically identifiable caudodorsal border can be identified by first locating the abdominal curtain sign and then following it dorsally until the pleural line disappears in the hypaxial muscles. Slide the transducer ventrally again until the pleural line reappears. This is the most caudodorsal location of the lung within the thorax.

If a lung point is detected in the middle third of the thorax (one-third down the thorax from the spine, again moving down the thorax toward the elbow), a moderate pneumothorax is likely present. Thoracocentesis may be indicated depending on the patient's clinical signs.

If a lung point is only detected in the lower third of the height of the thorax and glide sign/lung sliding is absent in the dorsal two-thirds, a severe pneumothorax is likely present. These patients tend to demonstrate marked respiratory distress warranting immediate thoracocentesis to evacuate gas and stabilize the patient.

The lung point will not be seen if the lung fails to recontact the chest wall in cases of massive/complete pneumothorax. These patients are in severe respiratory distress and can usually be diagnosed based on history and clinical findings, which justifies thoracocentesis in the absence of performing PLUS.

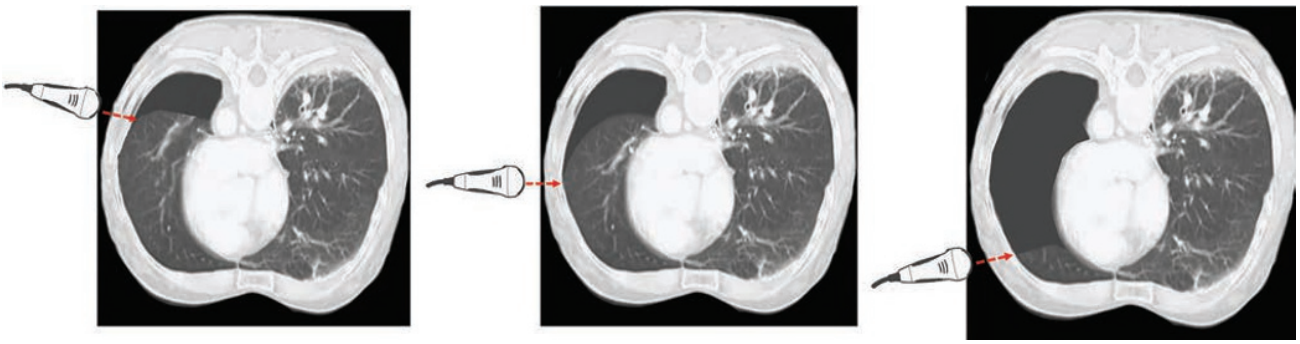


Figure 27.18 Computed tomography images and schematic representation of the lung point. The lung point is the location over the thorax where the return of lung sliding (with or without underlying B-lines) is identified in patients with pneumothorax. The lung point can be identified by moving the transducer toward the lung hilus. As lung sliding is most often assessed in the caudodorsal regions, this typically involves moving the transducer from the caudodorsal regions toward the elbow. The more severe the pneumothorax the lower on the chest wall the lung point will be located.

Abnormal Abdominal Curtain Signs Rule in Pneumothorax

A 2019 case series described abnormal abdominal curtain signs in dogs with pneumothorax, referred to as asynchronous curtain signs and double curtain signs [2]. Asynchronous curtain signs occur when movement of the vertical edge of the curtain sign moves in the opposite direction to the abdominal contents, or minimal movement of the vertical edge is seen while abdominal contents move caudally. The double curtain sign occurs when two vertical edges (two soft tissue–air interfaces) are visible in the same sonographic window, disappearing and reappearing at a single focal point without sliding out of the sonographically visible ultrasound image. The sensitivity and specificity of these signs in relation to pneumothorax have not been assessed, and it is unknown what role they will play in diagnosing pneumothorax compared with current sonographic criteria used to make the diagnosis. Readers are referred elsewhere for more detail regarding abnormal curtain signs [2].

Alveolar Interstitial Syndrome (Wet Lung)

An increase in the number of B-lines may be identified in cases of alveolar interstitial diseases such as pulmonary edema, pulmonary hemorrhage, pneumonia, atelectasis, infiltrative neoplasia, and in certain cases of chronic interstitial disease (e.g. pulmonary fibrosis, asthma). Therefore, when three or more B-lines are present in a single window (Figure 27.19), or more than two sites are positive on a single hemithorax, they should prompt a similar set of differential diagnosis as the radiographic presence of alveolar interstitial disease [27].

B-lines may be used as part of patient volume assessment as they correlate with extravascular lung water. An increasing number of B-lines in a patient with other signs of volume overload lends additional confirmation of interstitial overhydration [27].

Identification of B-lines rules out the presence of a pneumothorax at that transducer location [2, 27].

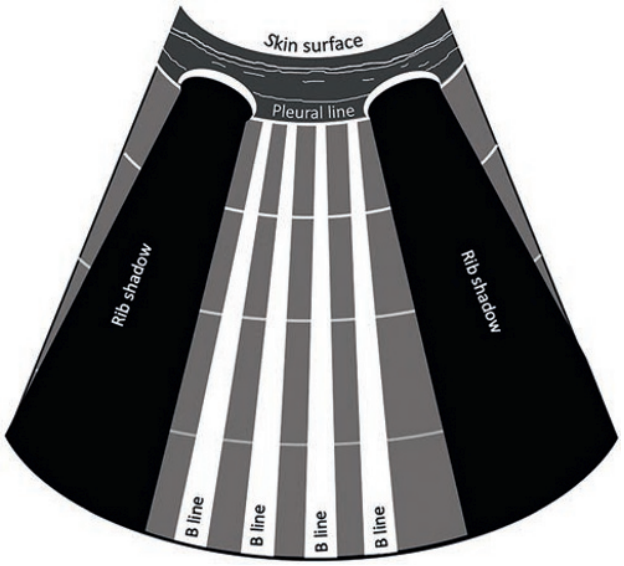


Figure 27.19 Schematic representation of increased B-lines. In this example, four B-lines can be seen within the intercostal space. An increase in the number of B-lines may be identified in cases of alveolar interstitial disease, including pulmonary edema, pulmonary hemorrhage, pneumonia, atelectasis, infiltrative neoplasia, and in certain cases of chronic interstitial disease (e.g. pulmonary fibrosis, asthma).

Alveolar Consolidation

Lung consolidation can occur as a result atelectasis, hemorrhage, bronchopneumonia, thromboembolism, lung lobe torsion, neoplasia, and inflammatory conditions such as pulmonary contusions and acute lung injury. Consolidation occurs when lung pathology is severe enough that insufficient air is present (< 10% air) to reflect the ultrasound beam back to the transducer, allowing it to pass through the lung as it would through a soft tissue structure [27]. Consolidation can be translobar (traversing the entire lung lobe from one surface to the other), or partial (partial thickness where the consolidated region within the lobe encounters air-filled lung distal to the consolidated region) [28]. Although many pathologies can result in consolidation, most will result in three common ultrasonographic appearances:

The "shred sign" represents a form of partial lung consolidation. The appearance occurs when there is a focal area of consolidated lung that abuts air-filled lung deep to it. The deep border (fractal/"shredded" border) of the consolidated lung appears shredded at the boundary of consolidated and aerated lung (Figure 27.20).

"Hepatization" of the lung lobe is when the alveoli of an entire lobe are fluid-filled and, together with the intralobular septa, create a macroscopic image pattern referred to as the tissue-like sign (Figure 27.21). This is a translobar form of consolidation. The term hepatization equates this pathology with the echogenic appearance of liver on ultrasound. The lung dimensions appear relatively conserved and the lung parenchyma appears similar to liver parenchyma. Air bronchograms, if present, can be seen within lung consolidation as small white hyperechoic pointed foci or lines.

The "Nodule sign" is a specific type of partial lung-lobe consolidation that differs from the shred sign in that the

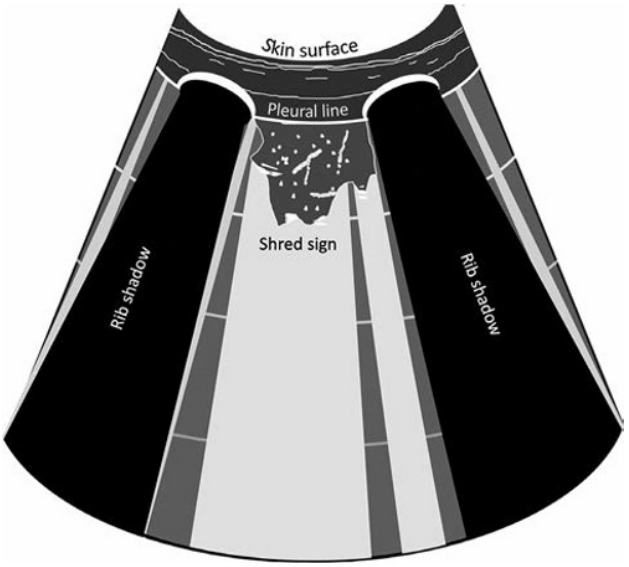


Figure 27.20 Schematic image depicting the "shred sign." The shred sign represents a form of partial lung consolidation (does not traverse the entire lung lobe). The appearance occurs when there is a focal area of consolidated lung that abuts air-filled lung deep to it. The deep border (fractal/shredded border) of the consolidated lung appears shredded at the boundary of consolidated and aerated lung. Air bronchograms (white punctate and short lines) may be visible within consolidated lung that creates a shred sign. Ring down artifact is often visible deep to and arising from the distal border of a shred sign.

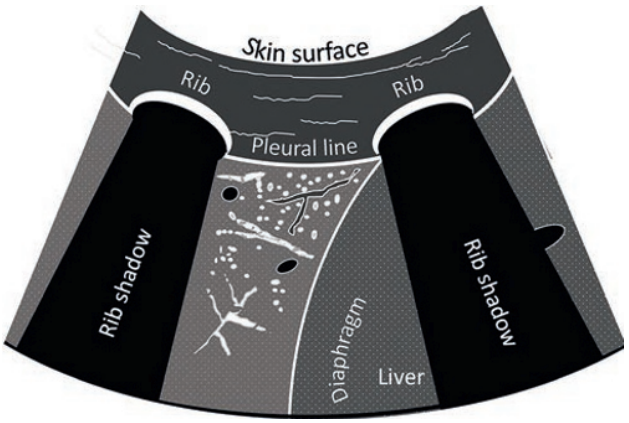


Figure 27.21 Schematic image of translobar "hepatization." Translobar hepatization occurs when alveoli of an entire lung lobe (or region of a lung lobe from one surface to the other) contain so little gas (e.g. atelectasis, fluid, pus, blood) that, together with the intralobular septa, they create a macroscopic image pattern resembling a tissue. This is a translobar form of consolidation. The term hepatization equates this pathology with the echogenic appearance of liver on ultrasound. Unlike a shred sign the lung consolidation is continuous from one surface to the other with no aerated lung below the consolidated region. The lung dimensions appear relatively conserved and the lung parenchyma appears similar to liver parenchyma. Air bronchograms, if present, can be seen within lung consolidation as small white hyperechoic pointed foci or lines.

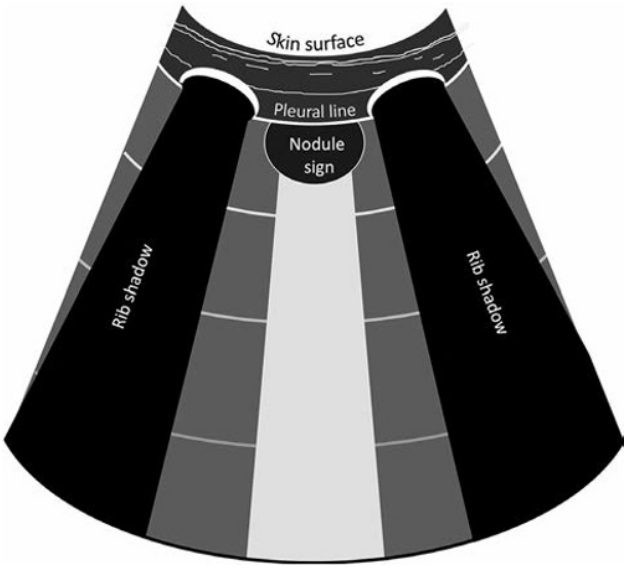


Figure 27.22 Schematic representation of a "nodule sign." A nodule sign is a specific type of partial lung lobe consolidation that differs from the shred sign in that the distal border between the consolidation and aerated lung is usually smooth and circular. Ring down artifact is often visible deep to and arising from the distal border of a nodule.

distal border between the consolidation and aerated lung is smoother and usually circular. The differential diagnosis for a sonographic nodule sign is similar to radiographic nodules, with neoplasia and fungal disease being two of the more common causes of nodule signs (Figure 27.22).

In human studies, lung ultrasound has been shown to be more sensitive and specific than thoracic radiography at identifying consolidation because pathology is often evident at earlier stages [26–28]. A "wedge sign" has also been described in human medicine, which appears as a partial consolidation that is triangular shaped. The wedge sign is often associated with pulmonary thromboembolism and is often seen in people that also have deep vein thrombosis. The significance of the "wedge sign" requires further investigation in veterinary medicine.

Limitations of Pleural and Lung Ultrasound

Lung ultrasound can be routinely performed as a point-of-care test by non-specialist clinicians and may provide accurate information on lung status with diagnostic and therapeutic relevance. Limitations for lung ultrasound do exist, however, and may include operator skill and equipment characteristics. The diagnosis of pneumothorax can be challenging because lung sliding can be difficult to identify even in healthy animals. Panting and rapid shallow breathing also make it difficult to identify lung sliding,

which emphasizes the importance of identifying the lung point or abnormal curtain sign, particularly in patients with smaller volume pneumothorax that may not show signs of marked respiratory distress.

If several lung ultrasound examinations are performed daily, the learning curve for acquiring skills for diagnosing pleural effusion, lung consolidation and alveolar interstitial syndrome is short, and proficiency is reported to be achievable in less than six weeks. Patient size and body condition score may affect the ability to obtain relevant information with PLUS, in that very large patients are difficult to image. Obese patients are frequently difficult to examine with PLUS as excess fat thickens their thoracic wall and impedes image acquisition. Chest-wall thickness may require alterations in transducer selection and ultrasound settings to acquire a good diagnostic image. The presence of subcutaneous emphysema, subcutaneous fluid accumulation (e.g. hemorrhage, edema), or body-wall disruption may alter or preclude the propagation of ultrasound beams to the pleural line. Finally, it should be noted that PLUS cannot detect lung overinflation resulting from an increase in airway pressures.

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28

Tracheal Intubation

Marc Raffe and Rachel Bassett

The ability to establish airway support through endotracheal (ET) intubation is a skill that all members of the veterinary team should master. Team members should be comfortable with ET tube (ETT) types and be familiar with ancillary materials needed to perform ET intubation. Understanding airway anatomy and oropharyngeal conformation are critical factors in selecting the most appropriate intubation technique when airway access is required (Figure 28.1). If initial attempts to gain airway access are unsuccessful, all team members should know advanced techniques that can be used to quickly provide a secure airway.

Indications

Tracheal intubation is indicated for a variety of clinical scenarios (Table 28.1). In the emergency and critical care setting, intubation may be required to facilitate treatment of upper airway obstruction, anaphylaxis, trauma, oropharyngeal tumors, laryngeal paralysis, brachycephalic obstructive airway syndrome, and tracheal collapse. Intubation is indicated in any patient with decreased or absent gag reflex (including sedated animals) to maintain a functional and protected airway. In these cases, the primary purpose of intubation is to protect the airway from regurgitated gastric contents and oral secretions. In certain cases, ETT placement cannot be performed due to the location of the obstruction; in these cases, alternative intubation techniques or tracheostomy tube placement (discussed in detail in Chapter 29) are indicated.

Severe head trauma or other intracranial disease (e.g. brain tumor, meningitis) may cause increased intracranial pressure and altered level of consciousness (semi-coma, coma). Neurologic dysfunction carries a risk of hypoventilation and carbon dioxide (CO_2) retention, which can further increase intracranial pressure. Intubation and positive pressure ventilation may be required to remove excess CO_2.

Neuromuscular weakness of respiratory muscles can result in inadequate respiratory excursion producing hypoventilation, hypercapnia, respiratory acidosis, and hypoxemia. Neuromuscular weakness may be a result of cervical injury (damage to tracts of the phrenic nerve, which serves the diaphragm), thoracic spinal cord damage (intercostal muscles), or peripheral neuromuscular disease (myasthenia gravis, tick paralysis, acute polyradiculoneuritis, botulism). Electrolyte disturbance (hypokalemia) can also produce marked muscular weakness. Care should be taken to monitor PCO_2, ideally arterial, or at a minimum, diaphragmatic movement (diaphragm moving caudally and causing expansion of the abdomen during inhalation) and chest wall expansion in these patients due to increased risk of hypoventilation. Severe cases may require ventilatory support (see Chapter 31). Megaesophagus may require airway protection to prevent aspiration secondary to regurgitation.

Severe hypoxemia (partial pressure of oxygen in arterial blood, PaO_2, < 60 mmHg) that persists despite oxygen supplementation is an indication for intubation and mechanical ventilation. In these cases, sterile equipment and intubation technique are important because bacterial contamination can increase the risk of ventilator-associated pneumonia and sepsis. Sterile ETT placement requires the use of sterile gloves, a disinfected laryngoscope, and a sterile ETT. Placement of a sterile ETT is also important for diagnostic airway washes.

Equipment Needed for Intubation

Intubation requires equipment beyond the ETT itself; necessary equipment and supplies vary slightly by species (see Protocols 28.1 and 28.2). It is recommended that a dedicated area with readily accessible space for all equipment and supplies needed for intubation be

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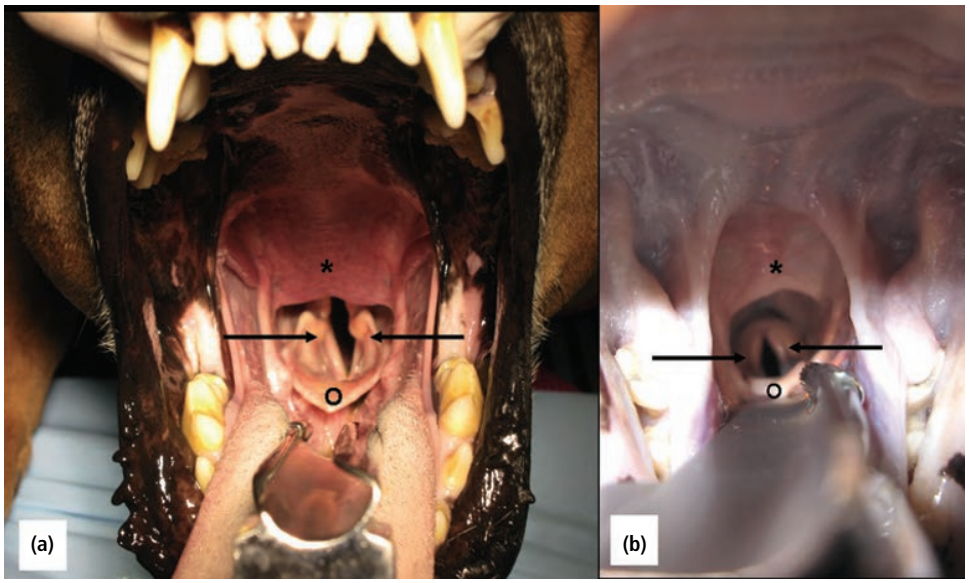


Figure 28.1 Anatomy of the oropharynx of the (a) dog and (b) cat. A laryngoscope is placing pressure on the base of the tongue to expose the larynx and surrounding anatomy. The arrows are pointing to the arytenoids (vocal folds). The asterisk indicates the soft palate. The O indicates the epiglottis.

Table 28.1 Indications for tracheal intubation.

Indication	Possible cause
Cardiopulmonary resuscitation	Upper airway obstruction
	Foreign body
	Soft tissue swelling
	Anatomical defect
	Trauma
	Laryngeal paralysis
	Masses
	Tracheal collapse
Decreased level of consciousness	Sedation
	General anesthesia
	Head trauma
	Intracranial disease
Decreased ability to ventilate	Neuromuscular disease:
	Tick paralysis
	Polyradiculoneuritis (including coonhound paralysis)
	Botulism
	Myasthenia gravis
	Neuromuscular blocking agent use
	Epidural complication
	Phrenic nerve damage
	Cranial cervical spinal cord lesion
	Head trauma
	Hypokalemia
Mechanical ventilation	
Endotracheal wash	

identified. This may be the same space that is dedicated to cardiopulmonary resuscitation (CPR). This designated space should be inventoried and restocked regularly to assure that all supplies are present and in proper working order. Having dedicated space set aside will increase efficiency and minimize stress in an emergency scenario.

Endotracheal Tubes

Cuffed Endotracheal Tubes

ETTs are manufactured using silicone (Figure 28.3a), rubber (Figure 28.3b), or polyvinyl chloride (PVC; Figure 28.3c). Both silicone and PVC have greater flexibility compared with red rubber tubes; silicone tubes are the most flexible. PVC and silicone tubes are clear to semiopaque, which permits visualization of airflow or airway secretions within the tube. These materials are less irritating to airway epithelium than red rubber tubes. PVC tubes have a preformed curve; silicone tubes are straight (the authors curved the ETT pictured in Figure 28.3 with a piece of purple suture to fit it in the image; without the suture, it is straight).

Rubber ETTs are less desirable for routine use. Rubber is prone to drying out and cracking with repeated use, which results in air leaks. Rubber is opaque, which makes it difficult to assess air movement within the tube and cleanliness of the tube's interior surface. Rubber is porous and more likely to absorb cleaning agents that can cause tracheal irritation. A Murphy tip is the most common style of cuffed ETT (Figures 28.2 and 28.3). This tube style has a beveled tip with an eye hole ("Murphy eye") in the wall opposite the bevel to ensure airflow if the end hole becomes occluded.

Protocol 28.1 Endotracheal Intubation in a Dog

Items Required

- 3 × endotracheal tubes, cuffs previously checked if applicable:
  - 1 × estimated size
  - 1 each of size smaller and larger
- Laryngoscope handle and straight Miller blade
- Sterile aqueous lubricant
- Gauze squares for grasping the tongue
- Length of roll bandage gauze for securing ETT
- Appropriately sized cuff inflation syringe:
  - 3–5 ml (small dog)
  - 5–10 ml (large dog)
- Atraumatic clamp if using rubber ETT
- Cotton-tipped applicators
- Oxygen, mask
- Assistant

Procedure

1) Collect necessary supplies.
2) For conscious animals, heavy sedation to light anesthesia will be required.
3) Position the dog in sternal recumbency (lateral recumbency can be used if needed in large dogs, and during CPR).
4) Provide preoxygenation if possible.
5) Assistant: Open the mouth by using thumb and one finger of the dominant hand to grasp caudal to the maxillary canine teeth and the nondominant hand to grasp the distal portion of the tongue using a dry gauze square. Pull the tongue out over the lower incisors and ventrally, opening the oral cavity widely. Pull the head rostrally and dorsally, to facilitate intubator's visualization of the pharynx and larynx.
6) Intubator: Place the laryngoscope blade into the oral cavity. Depress the tongue just rostral to, but not touching, the base of the epiglottis. The epiglottis should flip ventrally to reveal the arytenoids.
7) Select the appropriately sized ETT and apply sterile aqueous lubricant to the distal end, avoiding the Murphy eye.
8) Advance the ETT over the epiglottis without contact and through the arytenoids. Orient the ETT with the bevel oriented to the right of the person inserting the tube (in tubes with Murphy eyes, the Murphy eye would be on the left).
9) The adapter (Figure 28.2) should remain just rostral to the incisors on midline. Secure the tube with a length of roll bandage gauze or similar material. Tie the gauze around the ETT near the junction of the adapter. In large dogs, the gauze should be secured around the muzzle caudal to the canines. In small dogs, the gauze can be secured around the head behind the ears.
10) With an appropriately sized syringe, inject air into the pilot balloon to inflate the cuff to a maximum pressure of 20–25 cm H_2O. If the ability to measure pressure is unavailable, use the smallest amount needed to maintain a slight leak when breaths are manually delivered. If using a red rubber ETT, occlude the pilot balloon tubing with an atraumatic clamp.
11) While the ETT is being secured and the cuff inflated, a manual resuscitator can be connected to the adapter to provide breaths. If general anesthesia is being performed, the breathing circuit is connected to the ETT adapter.

The ETT cuff serves two purposes: (i) it prevents gas leaks around the ETT; and (ii) it reduces gastric and oral secretion aspiration risk (Figure 28.2). There are two ETT cuff styles: a high-pressure, low-volume cuff which is a thicker material that conforms to the ETT when deflated (Figure 28.3a,b), and a low-pressure, high-volume cuff made of thinner material that is not tightly adherent to the ETT when deflated (Figure 28.3c). Low-pressure, high-volume cuffs are preferred because they generate less surface contact pressure for a given inflation volume compared with high-pressure, low-volume cuffs. Low-contact pressure between the cuff and tracheal mucosa reduces tracheal mucosal injury. High-contact pressure between the cuff and tracheal mucosa can produce mucosal injury, mucosal necrosis, cartilaginous ring injury, and rupture of the dorsal tracheal membrane. PVC and silicone ETTs are commercially available with both cuff styles. If possible, select a low-pressure, high-volume cuff type to reduce injury. Rubber tubes are only available in high-pressure, low-volume cuff design. PVC and silicone ETTs have a self-sealing valve built into the cuff pilot tube to retain cuff volume following inflation. Rubber ETTs do not have a valve on the pilot tube and thus require use of an external clamp (Figure 28.4).

Uncuffed Endotracheal Tubes

When smaller animals (small cats, pediatric patients, avian and exotic species) require intubation, an uncuffed ETT may be the only option. It is difficult to find cuffed ETTs with an internal diameter less than 2.5 mm. Uncuffed ETTs are commercially available in several styles. Murphy style tubes similar in appearance to cuffed tubes are available from 1.0 to 3.0 mm internal diameter. Cole tubes are

Protocol 28.2 Endotracheal Intubation in a Cat

Items Required

- 3 × endotracheal tubes, cuffs previously checked if applicable:
  - 1 × estimated size
  - 1 each of size smaller and larger
- Laryngoscope and curved Macintosh blade, if available
- Sterile aqueous lubricant
- Gauze squares for grasping the tongue
- Length of roll bandage gauze for securing ETT
- Appropriately sized cuff inflation syringe (3 ml usually sufficient)
- Atraumatic clamp if using red rubber ETT
- 2% lidocaine injectable solution to prevent laryngospasm, in either needleless 1-ml syringe or in an atomizer
- Stylet
- Cotton-tipped applicators
- Oxygen, mask
- Assistant

Procedure

1) Collect necessary supplies.
2) For conscious animals, heavy sedation to light anesthesia will be required.
3) Position the cat in sternal recumbency. Lateral recumbency is preferred in CPR.
4) Provide preoxygenation if possible: more important than in most dogs due to potential for laryngospasm.
5) Assistant: Open the mouth by using thumb and one finger of the dominant hand to grasp caudal to the maxillary canine teeth and the nondominant hand to grasp the distal portion of the tongue using a dry gauze square. Pull the tongue out over the lower incisors and ventrally, opening the oral cavity widely. Pull the head rostrally and dorsally, to facilitate visualization of the pharynx and larynx.
6) Intubator: Place the laryngoscope blade into the oral cavity. Depress the tongue ventrally just rostral to, but not touching, the base of the epiglottis. The epiglottis should flip ventrally to reveal the arytenoids.
7) Apply 2% lidocaine injectable solution to the arytenoids (vocal cords): either one drop applied to each vocal cord with a needleless tuberculin syringe or sprayed using an atomizer. Do not contact the arytenoids. Usually, 30–60 seconds are required for the lidocaine to take effect, during which time flow-by or mask oxygen should be supplied to the cat.
8) Select the appropriately sized ETT and apply sterile aqueous lubricant to the distal end, avoiding the Murphy eye. If a stylet is used, it must not protrude past the end of the ETT.
9) Advance the ETT over the epiglottis and through the arytenoids. Orient the ETT with the bevel facing the right of the person inserting the tube (in tubes with Murphy eyes, the Murphy eye would be on the left).
10) The adapter should remain just rostral to the incisors on midline. Secure the tube with a length of roll bandage gauze or similar material. First tie the gauze around the ETT near the junction of the adapter, and then tie the gauze around the head caudal to the ears.
11) With a 3-ml syringe, inject air to inflate the cuff to a maximum pressure of 20–25 cm H_2O. If the ability to measure pressure is unavailable, use the smallest amount needed to maintain a slight leak when breaths are manually delivered. If using a red rubber ETT, occlude the pilot balloon tubing with an atraumatic clamp.
12) While the ETT is being secured and the cuff inflated, a manual resuscitator can be connected to the adapter to provide breaths. If general anesthesia is being performed, the breathing system would then be connected to the patient.

commonly used in veterinary medicine in this size range. The Cole tube is designed with a narrow tube diameter at the patient end and a widening of the tube diameter approximately 10–15 mm proximal to the tip; the area where the tube widens is called the shoulder. Cole tubes are designed so the narrow portion of the tube is inserted into the larynx and trachea while the shoulder portion of the tube is seated at the level of the arytenoids to form a seal (Figure 28.5a). When using uncuffed tubes, it is important to use the largest size that can be fit into the trachea without causing injury; this helps to create the best seal for providing positive pressure breaths during anesthesia, limiting air/gas leaks, and minimizing the risk for aspiration.

Oropharyngeal Airway (Laryngeal Mask)

An alternative device for feline and lagomorph airway support is an oropharyngeal airway (OA). An OA incorporates a tight-fitting laryngeal mask embedded in a silicone cast of the feline and lagomorph oral cavity. By placing the device in the oral cavity, the airway mask is seated on the laryngeal opening. In felines, it is then sealed by cuff inflation to provide airway support. The OA is an adaptation of

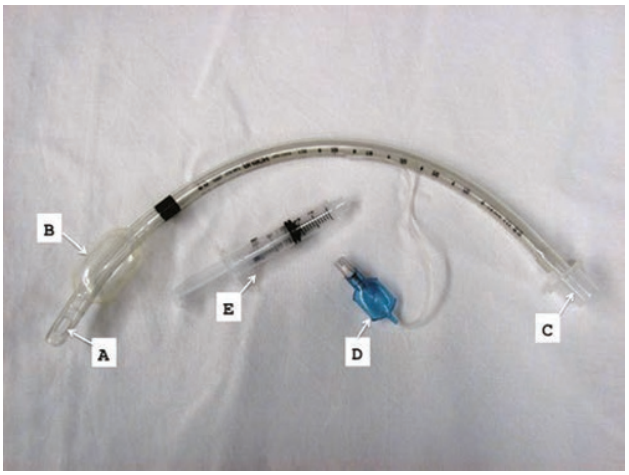


Figure 28.2 Anatomy of an endotracheal tube. (A) Murphy eye; (B) cuff; (C) adapter; (D) pilot balloon; (E) cuff inflation syringe.

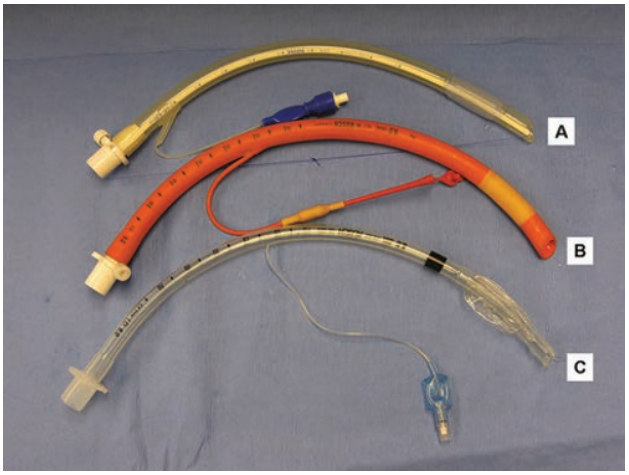


Figure 28.3 Three different types of large-cuffed endotracheal tubes. (A) silicone; (B) rubber; (C) polyvinyl chloride (PVC). Note the differences in the cuff between (A) and (B) (low volume, high pressure) and (C) (high volume, low pressure). Note that the silicone tube has been curved for the purpose of this image with a length of suture; silicone endotracheal tubes are straight.

the laryngeal mask airway (LMA) that has been popular for airway management in human anesthesia. The advantage of the LMA or OA is airway support without tracheal mucosal damage, thus avoiding post-intubation tracheitis and cough. The disadvantage of an OA is that the airway support may not be as secure as ET intubation.

Laryngoscope with Illumination

Laryngoscope use during intubation dramatically improves airway visualization and facilitates timely and accurate intubation. Laryngoscopes are particularly valuable in facilitating intubation when abnormal or distorted upper airway and oropharyngeal anatomy is present. They are also essential in species that have long oropharynx and



Figure 28.4 Because the pilot balloon (asterisk) on a rubber tube does not have an incorporated one-way valve, an atraumatic clamp should be applied to the cuff tubing to prevent accidental deflation of the cuff.

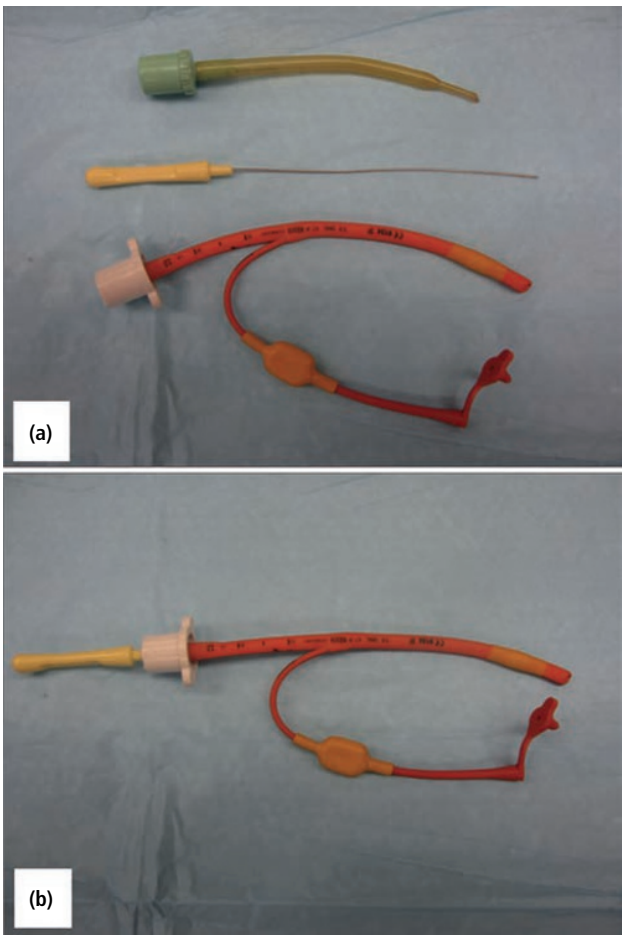


Figure 28.5 (a) Options for intubation of smaller animals such as cats and pediatric patients: an uncuffed Cole endotracheal tube (ETT; top) and a smaller cuffed ETT and metal stylet. (b) The metal stylet inserted into the smaller cuffed ETT, with care that the tip of the stylet does not protrude from the distal end of the tube. The stylet provides support to the more flexible tube to facilitate intubation.

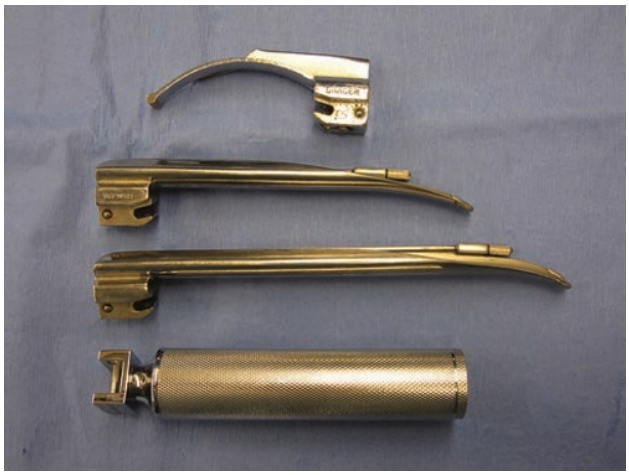


Figure 28.6 Illuminated laryngoscope. The top blade, a pediatric Macintosh blade, is most useful in cats. The straight Miller blades (middle and bottom) are most useful for dogs.



Figure 28.7 An atomizer used to distill lidocaine onto the arytenoids to facilitate intubation.

limitations in oral cavity opening (rabbits, rodents, guinea pigs). Today, several laryngoscope styles are available. The "traditional" style uses a power source in the laryngoscope handle and a detachable blade/light unit. Numerous blade styles are commercially available. Laryngoscope blade styles commonly used in veterinary medicine include the Miller and Macintosh blades. Each type comes in a variety of lengths to match oropharyngeal and laryngeal depth. In general, short, curved blades (Macintosh) work best in cats, whereas straight blades (Miller) work best for dogs (Figure 28.6). The light source may be a local bulb or via fiberoptic transmission from a remote bulb location. Either style works well in most cases. Laryngoscope blades should be correctly attached to the laryngoscope handle and tested to ensure that the light source is operational. Light sources should be routinely checked and repaired to ensure the light bulb is in working order. Laryngoscopes should be disinfected prior to use.

In recent years, fiberoptic laryngoscopes have been commercialized. Fiberoptic laryngoscopes are either flexible or rigid in design; in both styles, the fiberoptic shaft is attached to a light source, viewing controls, and an optical port or camera interface. The laryngoscope performs two functions: airway visualization and guide for ETT placement. To accomplish these goals, the ETT is placed over the shaft and positioned for placement. The scope is used to visualize, and in some cases enter, the laryngeal orifice. Once positioned, the tube is advanced into the airway and secured. Fiberoptic laryngoscopes are sterilized after each use to reduce risk of cross-contamination and infection.

Stylets

Stylets are used to facilitate ETT placement. Stylets are made of malleable material; they are generally either coated wire or a small diameter aluminum rod. Their purpose is to facilitate intubation when small or extremely flexible ETTs are used. Preplacement of a stylet into the lumen of the tube aids in tube placement by helping to direct the tube tip to the laryngeal opening. Care should be taken to ensure that the tip of the stylet does not protrude beyond the tip of the ETT to minimize injury to the larynx and trachea (Figure 28.5b).

Lidocaine Injectable Solution (2%)

Lidocaine can be topically applied to the arytenoid folds prior to intubation. Local desensitization helps minimize laryngospasm and facilitate intubation. In addition, a lighter plane of anesthesia can be maintained while intubation occurs because airway reflexes are blunted. Lidocaine can be delivered via a needleless syringe or an atomizer (Figure 28.7). If syringe delivery is used, a standard intravenous (IV) catheter can be attached to the syringe to direct topical lidocaine delivery. Cats are more sensitive to the toxic effects of lidocaine; a dose of 0.5 ml or less of 2% lidocaine should provide laryngeal desensitization without intoxication.

Securing the Endotracheal Tube

Once the ETT is introduced into the airway, securing the tube in place is essential to stabilize location, limit airway trauma, and prevent accidental extubation. Several methods for securing the ETT have been used in dogs and cats. A common method is to use standard 1-inch bandage gauze that is loop tied around the ETT caudal to the adapter but rostral to the pilot balloon tubing, positioned just behind the canine teeth and secured to the upper or lower jaw or behind the ears. Alternatively, a section of IV fluid tubing can be used in place of gauze. One advantage of IV fluid tubing is that it can be washed and reused between

patients, limiting waste. However, it provides a less secure grip on the ETT. A second advantage is the ability to withstand moisture and ease of removal when wet, especially during dental prophylaxis or oral and maxillofacial surgery. Care should be taken so that excessive tightening of the tie does not compromise the lumen of the tube with the above methods. A third option is use of wide rubber bands. A rubber band is loop secured to the ETT and positioned as described above. Care should be taken so that excessive tightening does not create a tourniquet effect.

Choosing Appropriate Endotracheal Tube Size

Selecting an appropriate ETT size is important to facilitate rapid intubation, minimize tracheal trauma, and ensure airway protection. Too large a tube may cause airway trauma; too small a tube will result in leaks around the cuff and incomplete airway protection. Cuff overinflation to compensate for an undersized ETT may cause tracheal necrosis and rupture [1]. In addition, increased resistance to gas flow through the tube may be seen when the ETT is too small.

Various methods for choosing an appropriately sized ETT have been described. Unfortunately, no single method is routinely reliable due to variation in airway anatomy. Time and experience will allow one to confidently select the correct ET size based on patient size and head anatomy. In general, it is prudent to have available an ETT that is one size larger and one size smaller than the tube you initially choose in case visual inspection reveals an airway size that is different than expected.

Choosing the Endotracheal Tube Width

One method of selecting an ETT is by correlating body weight to tracheal size [2–4]. Size charts may not be accurate in certain animals such as brachycephalic breeds and those animals with abnormal body condition score (those that are excessively thin or overweight) [2].

Measuring the width of the nasal septum is another method for selecting ETT size. An ETT is held up to the narrowest point between the nares (Figure 28.8a). An ETT with an outer diameter that best matches the width of the nasal septum should be selected. Unfortunately, one study found that this technique was accurate only 21% of the time [2].

Palpation of tracheal width just above the thoracic inlet can estimate ETT size (Figure 28.8b). This technique was found to be more accurate than measuring nasal septum width, but it still had a wide margin of error [2]. Palpation of the trachea can be difficult in brachycephalic breeds and in obese pets.

Choosing the Endotracheal Tube Length

ETT length measurement is much easier to perform. Following intubation, the ETT adapter should be positioned just cranial to the incisor teeth while the tube tip should end at the thoracic inlet. This length can be easily measured by holding an ETT up next the patient's neck and confirming tube length is the distance from the tip of the nose to the thoracic inlet. This length is important because placement of too long an ETT contributes to mechanical dead space and increases work of breathing. Too long a tube also carries the risk of extending beyond the tracheal bifurcation into a bronchus, resulting in ventilating only one portion of the lung (single bronchus or "one-lung" intubation).

If the tube is too long, it can easily be cut to the appropriate length. Remove the adapter at the rostral end, cut to size, and reattach the adapter to the tube. Be careful not to accidentally cut into the cuff inflation tube or the molded channel in the ETT wall below the cuff inflation tube.

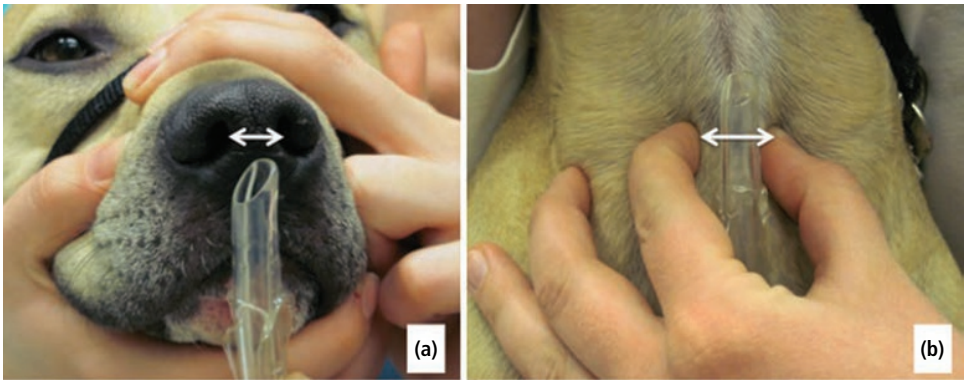


Figure 28.8 Techniques for estimating endotracheal tube (ETT) size. (a) The width of the nasal septum is measured and used as a guide for selection of ETT size. (b) The extra thoracic trachea is palpated and used as a guide for selection of ETT size.

Intubation Techniques

Intubating Dogs

Sternal recumbency is the preferred intubation posture; lateral recumbency can be used in large dogs and should be used during CPR (Chapter 20). Lateral recumbency intubation should be avoided in patients with increased risk of regurgitation. If the dog is spontaneously breathing, preoxygenation is ideal. An assistant opens the mouth by placing the thumb and index finger (thumb and middle finger in large dogs) of one hand on the maxilla just caudal to the canine teeth (Figure 28.9a). The other hand grasps the tip of the tongue using a dry gauze square. The tongue is extended over the lower incisors and moved ventrally, opening the oral cavity for easier visualization. The head should be pulled rostrally and dorsally, which straightens the neck and facilitates full view of the pharynx and larynx for the person performing intubation (Figure 28.9b). Caution must be exercised if cervical or cranial injury, disease, or malformation is suspected. In these cases, the head–neck angle should not be hyperextended beyond what is natural.

The person performing intubation should insert the laryngoscope blade into the caudal part of the oral cavity. The tongue should be depressed in a ventral direction just rostral to, but not touching, the base of the epiglottis. The epiglottis, which normally sits covering the laryngeal opening, should fall ventrally to reveal the arytenoids (vocal cords; Figure 28.9c). At times, the epiglottis is caught behind the soft palate; the laryngoscope can be used to gently release entrapment. Care should be taken not to apply pressure to the epiglottis because edema can result.

The appropriately sized ETT is selected and sterile aqueous lubricant applied to the distal end. Care is taken not to occlude the Murphy eye (if applicable) with sterile lubricant. The ETT is advanced over the epiglottis and between the vocal cords (Figure 28.9d). The ETT should be oriented with the bevel facing the right of the person inserting the tube (in tubes with Murphy eyes, the Murphy eye is oriented to the left). In this orientation, the curve of the ETT follows the curve of the trachea. The ETT may be rotated so that its curvature is parallel with the tracheal lumen as it is advanced into the airway. The tube adapter should be seated on the midline just rostral to the incisors. The tube is secured with roll bandage gauze or similar material as described above.

While the ETT is being secured, a manual resuscitator or anesthesia breathing circuit can be attached to provide breathing support. See Protocol 28.1 for concise step-by-step instructions for intubating dogs.

Intubating Cats

The preferred posture for feline intubation is sternal recumbency. Cats may be positioned in lateral or dorsal recumbency if required. Lateral recumbency is preferred during CPR (Chapter 20). Dorsal recumbency is used in cats to facilitate visualization of airway in brachycephalic breeds. If the cat is breathing spontaneously, preoxygenation is ideal. An assistant opens the mouth by placing one hand just caudal to the maxillary canine teeth with the thumb and index finger. The other hand should grasp the tip of the tongue using a dry gauze square. The tongue should be pulled rostrally over the lower incisors and ventrally, thus opening the oral cavity for easier visualization.

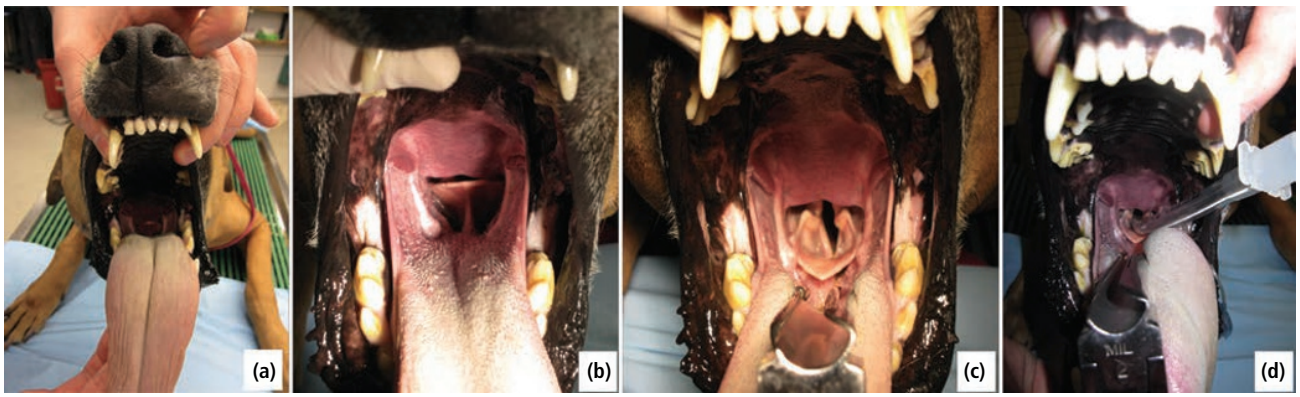


Figure 28.9 Images of the intubation sequence of a dog. (a) The dog is placed in sternal recumbency and the upper jaw should be grasped caudal to the canine teeth while the tongue is pulled rostrally between the canine teeth and the lower jaw is opened. (b) View of the caudal oropharynx when the mouth is properly opened for intubation. Note that the epiglottis is elevated, obscuring view of the glottis and arytenoids. (c) The laryngoscope is inserted into the mouth and the tip is used to place ventral pressure on the base of the tongue. Care should be taken to avoid touching the epiglottis with the laryngoscope blade to minimize airway trauma. When properly positioned, a good view of the glottis and arytenoids is achieved. (d) Following intubation, direct visualization of the endotracheal tube between the arytenoids is the most accurate way to ensure proper intubation.

The head should be pulled rostrally and dorsally, which straightens the neck and facilitates full view of the pharynx and larynx. Caution must be exercised if cervical or cranial injury, disease, or malformation is suspected. In these cases, the head–neck angle should not be hyperextended beyond what is natural.

Short, curved laryngoscope blades (Macintosh style) tend to work better in cats. The operator introduces the laryngoscope blade into the oral cavity. The tongue should be depressed rostral to, but not touching, the base of the epiglottis. The epiglottis should drop down to reveal the arytenoids (vocal cords). Care should be taken when intubating a cat because they are prone to ETT-induced laryngeal edema and inflammation secondary to intubation trauma.

The feline larynx is prone to laryngospasm from mechanical stimulation. To avoid laryngospasm, 2% lidocaine injectable solution is used to desensitize the vocal cords to facilitate intubation. A needleless tuberculin syringe is used to deliver the lidocaine. One drop of lidocaine applied to each vocal cord is adequate to produce desensitization. The vocal cords should not be touched by the syringe or laryngospasm may result. Alternatively, an atomizer may be used to spray each vocal cord. Commercially available 10% lidocaine dental spray is also safe and effective in vocal cord desensitization. Following application, 30 seconds to one minute is needed for the lidocaine to take effect. During this time, preoxygenation is suggested to avoid hypoxemia secondary to hypoventilation.

The appropriately sized ETT is selected and sterile aqueous lubricant applied to the distal end. Care should be taken not to occlude the Murphy eye (if applicable) with the aqueous lubricant. Because ETT sizes used in cats are small, the tube itself is more flexible. A stylet may be used allow better directional control and easier passage between the vocal cords. Care must be taken to ensure that the stylet tip does not protrude past the end of the ETT because laryngotracheal injury can result. The ETT is advanced over the epiglottis and between the vocal cords. The ETT should be oriented with the bevel oriented to the right of the person inserting the tube (in tubes with Murphy eyes, the eye would be on the left). In this orientation, the curve of the ETT follows the curve of the trachea.

The tube adapter should be seated just cranial to the incisors on midline with the nasal septum. The tube should be tied in with a length of roll bandage gauze or similar material. The gauze is first tied around the ETT caudal to the adapter and can be secured around the head caudal to the ears.

While the ETT is being secured, a manual resuscitator or anesthesia breathing circuit can be attached to provide breathing support. See Protocol 28.2 for concise, step-by-step instructions for intubating cats.

Cuff Inflation

The ETT cuff is intended to fill the space between the ETT and the trachea. It is used to "seal" the airway to protect against introduction of blood, gastric secretions, and saliva into the airway. It is also required to provide positive pressure ventilation to the patient. Cuff inflation volume is critical to ensure good ETT sealing while minimizing tracheal mucosa damage due to excessive cuff pressure on the tracheal mucosa. To minimize mucosal injury, the cuff should be inflated to the lowest pressure that achieves airway sealing. Cuff inflation pressure should not exceed 25 cmH_2O. Cuff pressure can be measured with commercial devices that measure and/or limit cuff inflation pressure. These devices attach to the cuff pilot tube for measurement. If a device is not available, either the minimal occlusive volume technique or the minimal leak technique should be used to inflate the cuff.

The minimal occlusive volume technique is performed by injecting air into the cuff pilot tube while tracheal auscultation during breath delivery is performed. Air is slowly injected until no air leak can be heard. Small aliquots of air are then removed until a leak can be auscultated. Small air volumes (0.1 ml) are instilled until the leak can no longer be heard. This technique minimizes risk of aspiration but may result in a higher incidence of tracheal injury compared with the minimal leak technique.

The minimal leak technique is performed by injecting air into the cuff pilot tube during a support breath while auscultating the trachea. Air is injected until no leak is noted when the breathing circuit or resuscitation bag is inflated to 20 cm water pressure. At that point, air is removed from the cuff in 0.1 ml aliquots until a small leak can be detected. This technique results in less injury to the tracheal wall but may increase the risk for aspiration. It can only be performed with ETTs that have a pilot balloon valve.

While the ETT is being secured and the cuff inflated, a manual resuscitator can be connected to the adapter to provide breaths. For general anesthesia the breathing system would be connected to the patient. Cuff inflation pressure should be re-evaluated after desired anesthetic depth is achieved and when changing patient recumbency.

Intubation of Difficult Airways

Intubation challenges should be expected and planned for in animals with oropharyngeal trauma, upper airway obstruction, and brachycephalic obstructive airway syndrome. In many situations, difficult intubation can be remedied by providing a deeper level of sedation with injectable

anesthetic agents, optimizing patient positioning, using a laryngoscope, and illuminating the oral cavity. If excessive oropharyngeal secretions are present, they should be removed with gauze, cotton-tipped applicators, or suction to improve airway visualization. If intubation cannot be accomplished, a smaller size ETT than expected may be used to establish airway control. Once the patient is stabilized, the tube can be replaced with a larger tube if a leak is present.

If the ETT is flexible, a stylet can be inserted into the tube to stabilize it. As noted above, a stylet is frequently used with small size ETTs to facilitate placement. It is also helpful in cases of partial airway obstruction to direct the tube around the obstruction point. Care should be taken to ensure that the stylet does not extend beyond the tip of the tube to prevent airway injury.

A quality light source is helpful to facilitate intubation. Use a rigid or flexible fiberoptic device for directing additional light into the caudal pharynx. These devices are also used as an ETT stylet to assist in directing the ETT into the laryngeal opening. A headlamp positioned to increase light in the caudal pharynx may also be helpful.

A guidewire, such as a 5 or 8 Fr polyurethane or red rubber catheter, can be used to facilitate difficult intubations. The catheter can be inserted into the airway and then the ETT directed over the catheter into the airway. The catheter is then removed once an airway has been established. Care should be taken not to drop any wire or catheter into the airway. An oxygen source may be attached to catheter to provide insufflation during intubation.

Retrograde intubation is a minimally invasive technique that can be used to facilitate ET intubation when standard techniques are unsuccessful. There are two options to perform retrograde intubation: a flexible guidewire or long through-the-needle catheter. Either method requires the guide to be introduced into the airway through the cricothyroid ligament. If the long catheter option is used, the catheter bevel is directed cranially when inserted into the airway. Once introduction is made, the needle hub is lowered toward the thorax so that the needle tip and catheter point toward the head; the catheter is then advanced in an orad direction up through the larynx. Once visualized in the oral cavity using a laryngoscope, it is grasped with forceps and advanced to exit the oral cavity. The catheter is then used as a guide over which to pass the ETT into the airway. The catheter is removed when the ETT enters the larynx.

If a vascular guidewire is used, the same principles apply. The difference is that a short, large bore catheter is inserted into the cricothyroid membrane and cranially directed. Following catheter insertion, a vascular guidewire is introduced into the catheter lumen and advanced cranially until it exits into the oral cavity. It is then grasped with forceps under laryngoscope visualization to exit the mouth. An ETT is placed over the wire and advanced into the airway. Once the ETT is engaged into the larynx, the guidewire is removed and tube advanced to its final location.

If ET intubation cannot be accomplished, transtracheal oxygen delivery can be used as an emergency support procedure. A 16-gauge × 2-inch catheter (Venocath-16, Venisystems, Abbott Ireland, Sligo, Ireland) is percutaneously inserted into the trachea through the cricothyroid ligament or between two tracheal rings and directed distally. It is important to secure this catheter with sutures to the outside of the neck to prevent catheter migration producing subcutaneous emphysema. The catheter can be connected to the anesthesia machine via an adapter (i.e. connect a 1-ml syringe to the catheter, and then cut off the end and attach that to noncompliant oxygen tubing). Constant flow oxygen is delivered by using the oxygen flowmeter to determine the best flow rate to support oxygen without causing airway pressurization. The flush valve on the anesthesia machine is used to provide breaths to the animal [5].

In most instances, airway access can be obtained via the one of the techniques described here. If unsuccessful, tracheostomy can be performed in a controlled manner (see Chapter 29).

Techniques to Confirm Endotracheal Tube Location

ETT placement into the trachea should always be verified for correct location. Accidental esophageal placement is a possible risk, due to close proximity of the esophagus and trachea in the caudal pharynx. Verification of ETT location is performed by visually confirming its passage between vocal cords. Other methods to confirm placement have been described; the following techniques should be used in addition to direct visualization:

- The animal coughs when the ETT is advanced into the trachea. This usually occurs if the animal is not heavily sedated.
- Condensation is seen through the tube on exhalation.
- Only one tubular structure is palpable in the ventral cervical region. The presence of two tubular structures indicates placement of the ETT in the esophagus.
- Bilateral auscultation of the lungs for respiratory sounds when an assisted breath is delivered.
- Chest wall expansion during an assisted breath. The abdomen should not be distended.
- Capnometry confirmation. If the tube is correctly placed in the trachea, CO_2 should be measured and a capnograph waveform should be associated with individual breaths. If the ETT is in the esophagus, no CO_2 value is reported or waveform detected (see Chapter 30). Note that this technique is not useful in cardiac arrest due to the absence of circulation and CO_2 presence in the lungs.

Extubation

Extubation should proceed only after airway reflexes have returned. Prior to extubation, the oral cavity should be examined for saliva, blood, or regurgitant. All secretions should be suctioned or swabbed from the oral cavity prior to ETT removal to minimize the chance of aspiration into the upper airway.

The cuff should be fully deflated before extubation except in cases with concern for airway protection, such as in animals with megaesophagus or brachycephalic breeds. In these cases, a small amount of residual air volume in the cuff may be beneficial to reduce risk of aspiration. If airway protection is a concern, delayed extubation until the animal is actively coughing and attempts self-extubation is preferred. It is important to keep the ETT on midline with the nasal septum so that shearing of tracheal mucosa does not occur.

Once extubated, the animal should be closely monitored for hypoxemia or respiratory distress. These scenarios may warrant re-intubation. Sternal posture with head elevation is preferred to minimize risk of saliva or gastric content aspiration. Flow-by oxygen should be administered to assist in transition from high oxygen support to room air oxygen concentration. Respiration and oxygenation should be monitored until the animal is fully awake.

Risks and Complications of Tracheal Intubation

ETT placement has many benefits; however, complications can occur if proper technique is not used. Cats have a greater risk for intubation-associated complications compared with dogs [6–8]. Cats are prone to laryngospasm, which can make tracheal intubation a challenge. Excessive pressure on the ETT to overcome laryngospasm carries an associated risk of tissue injury. Dogs are susceptible to vagal stimulation during intubation, which can cause bradycardia and bradyarrhythmias. Dogs with elongated soft palate have an increased risk for epiglottic entrapment and airway occlusion. Brachycephalic breeds have redundant perilaryngeal tissue and hypoplastic tracheal lumen size, making intubation more difficult. Multiple intubation attempts in brachycephalic breeds can result in pharyngeal and laryngeal edema.

Choosing the correct diameter and length ETT can make the difference between rapid, successful intubation and multiple failed attempts with increased risk of oropharyngeal and laryngeal trauma. Forceful intubation and excessive laryngoscope blade pressure can produce perilaryngeal tissue edema and iatrogenic arytenoid tears [8]. Perilaryngeal tissue edema can lead to upper airway obstruction and the need for aggressive airway support (tracheostomy) to maintain airway patency.

When patient repositioning is required, the ETT should be disconnected from the breathing circuit or oxygen support device to prevent excessive traction or torsion on the ETT. Mucosal injury from ETT traction or torsion can result in mucosal injury or necrosis.

ETT cuff overinflation is a major cause of tracheal injury. Cuff overinflation is more common in cats due to the small inflation volume required for cuff sealing. Inflated cuff contact with the tracheal wall produces increased pressure and decreased mucosal blood flow at the point of contact. Prolonged high-pressure contact can result in tracheal necrosis, tearing, and stenosis. When long-term cuff inflation (longer than two hours) is required (mechanical ventilation, longer general anesthesia), it is recommended that the cuff be deflated, repositioned, and reinflated every two hours. This will allow blood flow to be periodically restored to cuff contact areas on tracheal mucosa. Cuff overinflation can also produce tracheal wall or dorsal tracheal membrane tears. This is more commonly seen in cats than dogs [6, 7]. Tracheal tears result in subcutaneous emphysema and pneumomediastinum. In severe cases, secondary pneumothorax, pneumoretroperitoneum, and pneumoperitoneum have been reported. Most tracheal tear cases do not require surgery but can take several weeks before the air leak site heals and subcutaneous emphysema reabsorbs [6, 7].

Excessive pressure created by too tight a tie securing the ETT to the muzzle or face can produce tissue edema and injury due to vascular occlusion and lymphatic stasis (tourniquet effect). Irrespective of material, all tube securing methods should be tied to allow a finger to be inserted between the tie and skin.

Summary

ET intubation is an essential skill for all veterinary team members to master. It is a lifesaving measure in patients with cardiopulmonary arrest or severe respiratory distress. It is required during most general anesthetic procedures. Proper equipment and technique help minimize trauma and complications associated with intubation. Special techniques exist to help facilitate difficult intubation. The veterinary team should practice these skills frequently to be prepared in case of an emergency.

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29

Temporary Tracheostomy

F. A. (Tony) Mann

Indications

The primary indication for temporary tracheostomy tube placement is to relieve life-threatening upper airway obstruction due to trauma, foreign body, laryngeal paralysis, or neoplasia. Other indications include facilitating removal of lower airway secretions when there is compromised cough reflex, allowing manual or mechanical ventilation when orotracheal intubation is not practical, reducing airway resistance in patients where increased intracranial pressure is a concern, and permitting inhalant anesthesia during certain intraoral surgical procedures.

Equipment

Most commercially available tracheostomy tubes are made of plastic or silicone, but reusable metal tracheostomy tubes are also available. The most versatile tubes contain an inflatable cuff and inner cannula as well as the standard obturator and tube tie (Figures 29.1 and 29.2). Some tube sizes are too small in diameter to include a cuff and inner cannula, so many small dogs and cats do not benefit from those two accessories. The cuff is only needed when positive-pressure ventilation is necessary, such as with patients requiring general anesthesia or ventilator therapy. In fact, the cuff may be disadvantageous in management of airway obstruction in awake patients because of the possibility of secretions accumulating around the deflated cuff.

High-volume, low-pressure cuffs are preferred for patients requiring ventilation, and when these cuffs are deflated there is significant surface area of the wrinkled cuff to harbor accumulations of secretions. The inner cannula is helpful for tube hygiene and maintenance because the cannula can be easily removed and replaced. Without the inner cannula, there is more emphasis on tracheal suctioning, and more frequent tube changes may be necessary. Single-lumen tubes must be removed and replaced with a new tube or reinserted into the stoma site after each cleaning.

When an appropriate commercial tracheostomy tube is not available, a tracheostomy tube may be fashioned from a standard endotracheal tube (ETT; Figure 29.3). Choose an ETT that is approximately one size smaller than would be chosen for orotracheal intubation. Remove the ETT connector and bisect the tube along its length until the uncut portion of the tube is the necessary tracheostomy tube length. If the need to inflate the cuff is anticipated, the cuts can be carefully made to preserve the inflation channel. The two halves of the bisected portion of the tube may be used as flanges to which umbilical tape ties can be attached. Reattach the ETT connector at the flange–tube junction. The flanges may be shortened to a convenient length prior to attaching the ties. One disadvantage of this modified tube when the inflation channel is preserved is that the natural curve of the tube is about 90 degrees rotated; however, no untoward patient consequences with this deviation have yet to be reported.

The size of tracheostomy tube is important to a successful procedure and good outcome. Ideally, the tracheostomy tube should be one-third to one-half of the tracheal diameter to minimize iatrogenic tracheal trauma and decrease the incidence of postintubation stenosis. Also, an appropriately sized tube that allows some airflow around it may prevent respiratory arrest should tube occlusion occur. However, a tube that is too small may cause resistance to airflow if the patient must breathe entirely through the tube with no airflow around it. Tube size should allow air passage both around and through the tube for uncuffed tubes and for tubes with deflated cuffs. Available tracheostomy tube sizes that may be applied to dogs and cats range from 00 to 10.

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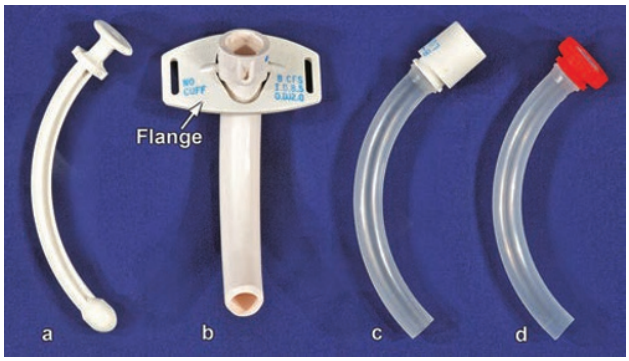


Figure 29.1 Commercially available uncuffed tracheostomy tube with disassembled components: (a) obturator, which is inserted into the tracheostomy tube immediately before placement and removed as soon as the tube is in the trachea; (b) tracheostomy tube, which is secured with umbilical tape attached to eyelets in the flange and tied behind the neck; (c) inner cannula, which is placed in the tracheostomy tube and replaced at regular intervals, and (d) closed-end cannula, which can be used to temporarily occlude the tracheostomy tube to see if the patient can breathe around the tube.

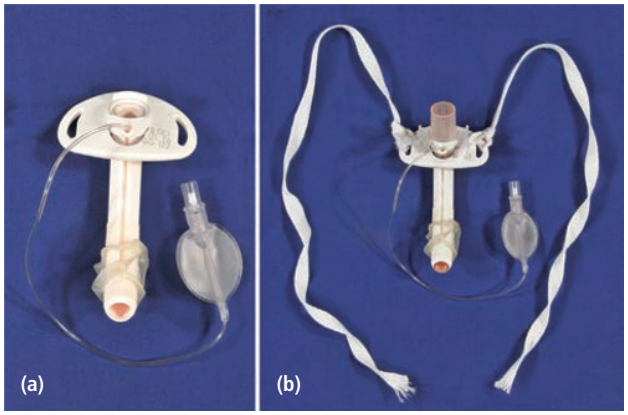


Figure 29.2 Commercially available cuffed tracheostomy tube (a) without inner cannula and (b) with inner cannula locked in place and umbilical tape tied to eyelets in the flange. Note that the cuff would only be inflated when positive-pressure ventilation is necessary, and the deflated cuff would increase the surface area on which secretions could accumulate, thereby complicating tracheostomy hygiene.

Sizes 00–5.5 are neonatal/pediatric sizes and are too small to be available with inner cannulas, whereas sizes 6.0 or larger may come with removable inner cannulas. Tracheostomy tube sizes are equivalent to endotracheal tube sizes but can be misleading because they do not necessarily correlate to the millimeter diameters of the tubes. For example, a size 6.0 tracheostomy tube may have an inner diameter of 6.4 mm but an outer diameter of 10.8 mm. Therefore, one should always check the inner and outer diameter dimensions typically located on the tube flange to choose the tube that will best fit the trachea in question. The largest possible inner diameter is always desired, but the outer diameter will

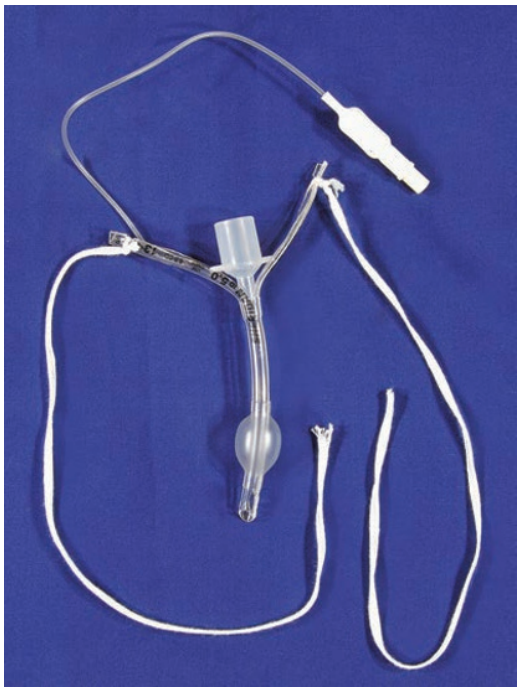


Figure 29.3 Tracheostomy tube fashioned from a standard endotracheal tube. Note that the inflation channel can be preserved if the cuff is needed. Preserving the inflation channel results in a curve to the tube that is rotated about 90 degrees to the desired curve.

determine whether the tube will fit appropriately into the trachea.

Tube length should extend six to seven tracheal rings down from the placement site. Ideal tube length may not be achieved in all veterinary patients because of the limits of what is commercially available, but tailormade tubes fashioned from ETTs can be used when the length of commercially available tubes is too far from appropriate.

Other equipment needed for tracheostomy tube placement includes an ETT for orotracheal intubation, standard anesthetic supplies, clippers, prepping scrub and solution, barrier drape, sterile surgical gloves, sterile gauze, surgical instruments, suture material, and, if not supplied with the tracheostomy tube, umbilical tape for securing the tube after it is in place. The minimally suggested surgical instruments, which may be packaged together in a tracheostomy pack, are two towel forceps, a scalpel handle to accommodate a #10 or #15 scalpel blade, thumb forceps (Adson–Brown or DeBakey tissue forceps), Metzenbaum scissors, needle holders, two mosquito hemostatic forceps, and suture scissors. It is also handy to have some self-retaining retractors, such as a Gelpi perineal retractor (preferably two) or a Weitlaner retractor, which may be included in the tracheostomy pack or packaged separately. Suction capability and an oxygen source should be readily available during the procedure (Box 29.1).

Box 29.1 Suggested Minimum Surgical Instruments Required for Tracheostomy

- 2 × towel forceps
- Scalpel handle to accommodate a #10 or #15 blade
- Thumb forceps (Adson–Brown or DeBakey tissue forceps)
- Metzenbaum scissors
- Needle holders
- 2 × mosquito hemostatic forceps
- Suture scissors
- Ideally, self-retaining retractors, such as Gelpi perineal retractors (preferably × 2) or a Weitlaner retractor

Positioning and Aseptic Preparation

The emergent "slash" tracheostomy should be a rare occurrence; there is usually time to capture the airway with an endotracheal tube and prepare the patient for a controlled surgical approach (Chapter 28). Once intubated and in an appropriate plane of general anesthesia, the patient is placed in dorsal recumbence (Figure 29.4). The neck is extended and the thoracic limbs are pulled caudally and secured to the table with ties or tape. A positioning aid, such as a thoracic positioning trough, may be used to keep the patient from leaning to one side, and a rolled towel dorsal to the neck is essential to keep the trachea elevated toward the surgeon.



Figure 29.4 Positioning and preparation for tracheostomy tube placement. The dog is in dorsal recumbence with the thoracic limbs pulled caudally. A rolled towel (not shown here) placed dorsal to the neck is helpful to keep the trachea elevated toward the surgeon. The airway is captured with an endotracheal tube which is attached to anesthetic tubing. The curved mark on the skin represents the area of the caudal aspect of the thyroid cartilage, the straight mark immediately caudal to the curved mark represents the cricoid cartilage, and the midline straight mark indicates the proposed skin incision. Note the widely clipped and prepared surgical field. The syringe is present as a reminder that the endotracheal tube will need to be deflated and the endotracheal tube removed as the tracheostomy tube is inserted.

An area large enough for a ventral midline skin incision made longitudinally from the cricoid cartilage to the fourth or fifth tracheal ring is clipped and prepared; this area should extend sufficiently far laterally to create an area on each side wide enough to allow easy skin cleaning and tube maintenance. In most cases, a sufficient surgical field can be achieved by clipping the ventral neck from the angle of the mandibles to the manubrium and laterally past the level of the jugular veins (Figure 29.4). For longhaired dogs and cats, overhanging fur should be trimmed. Breeds with excessive skin folds may require skin to be deflected dorsally and taped or temporarily tacked with sutures to prevent the skin folds from contaminating or occluding the tracheostomy area when the animal is no longer in dorsal recumbence.

After clipping and vacuuming, a rolled towel should be placed underneath (dorsal to) the neck to stabilize the neck and prevent the surgical site from sinking away during surgical manipulations, and a standard aseptic surgical skin preparation is performed.

Procedure

The prepared surgical site is isolated with a barrier drape secured with two towel forceps (Figure 29.5). A single drape fenestrated over the proposed skin incision is usually sufficient, but quarter draping may be performed if desired. Quarter draping requires at least four towel forceps. Make a ventral midline cervical skin incision just caudal to the larynx for a distance of approximately 3–4 cm, depending on the size of the patient (Figure 29.5a). Apply a self-retaining retractor to hold open the skin edges and clear just enough subcutaneous tissue to identify the midline division of the sternohyoideus muscles (Figure 29.5b). Using Metzenbaum scissors, bluntly separate the sternohyoideus muscles on the midline (Figure 29.5c), taking care to avoid the thyroidea caudalis vein on the midline between these two muscles (Figure 29.5d). Retract the thyroidea caudalis vein to one side along with one of the sternohyoideus muscles (Figure 29.6a). Reposition the self-retaining retractors on the sternohyoideus muscles to expose the trachea, then clear the loose fascia off the ventral trachea at the proposed tracheotomy site (Figure 29.6b). Application of a second self-retaining retractor at a right angle to the original retractor to retract the skin in a craniocaudal direction enhances exposure of the tracheal rings and interannular ligaments (Figure 29.6c).

Using a scalpel blade, incise the interannular ligament between the appropriate tracheal rings, in this case the second and third rings (Figure 29.7). This tracheal location is

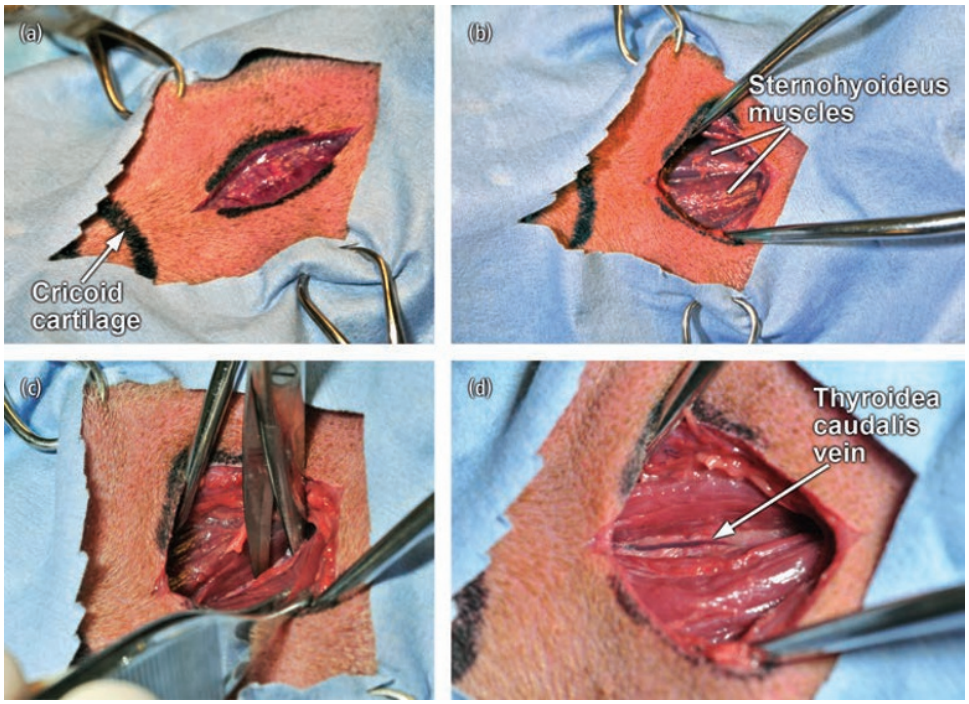


Figure 29.5 Surgical approach to the trachea for tracheostomy tube placement. Cranial is to the left in all frames. (a) The skin incision is made immediately caudal to the cricoid cartilage over approximately the first through fourth tracheal rings. (b) The skin edges are retracted with a Gelpi perineal retractor to facilitate identification of the sternohyoideus muscles and their midline division. (c) Metzenbaum scissors are used to bluntly separate the sternohyoideus muscles on the midline. (d) The thyroidea caudalis vein located on the midline on the dorsal aspect of the sternohyoideus muscles is avoided to minimize bleeding.

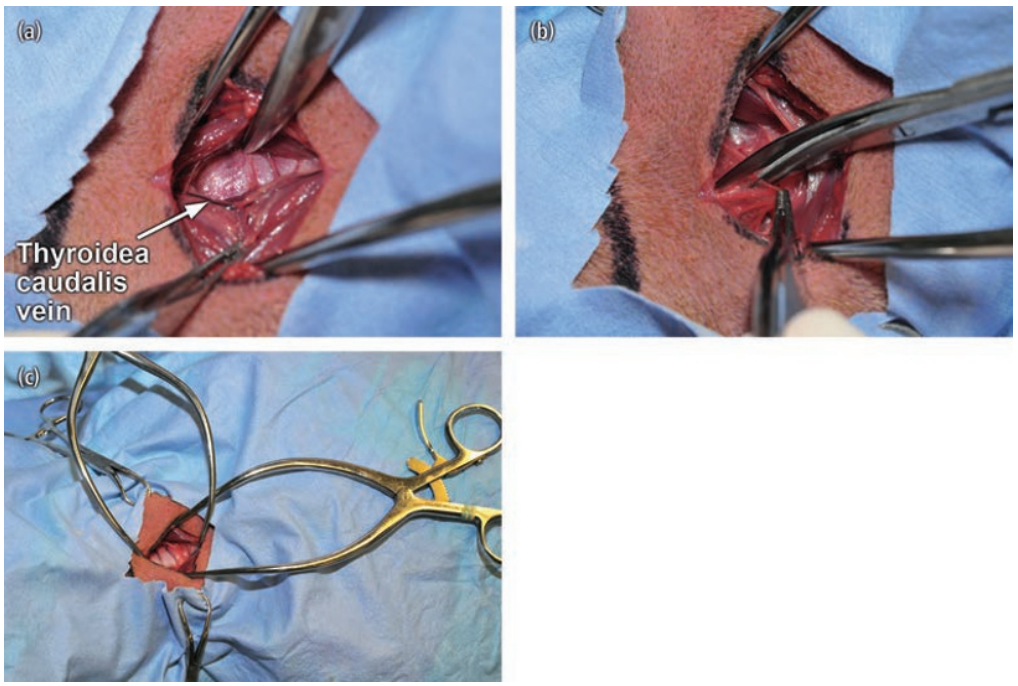


Figure 29.6 Isolating the trachea prior to tracheotomy for tracheostomy tube placement. Cranial is to the left in all frames. (a) The thyroidea caudalis vein is retracted laterally with one of the sternohyoideus muscles, the right sternohyoideus muscle in this case. (b) The Gelpi perineal retractor is repositioned to retract the sternohyoideus muscles, and the loose fascia covering the ventral surface of the trachea is incised with Metzenbaum scissors. (c) A second Gelpi perineal retractor is placed for craniocaudal retraction of skin and loose fascia exposing the tracheal rings.

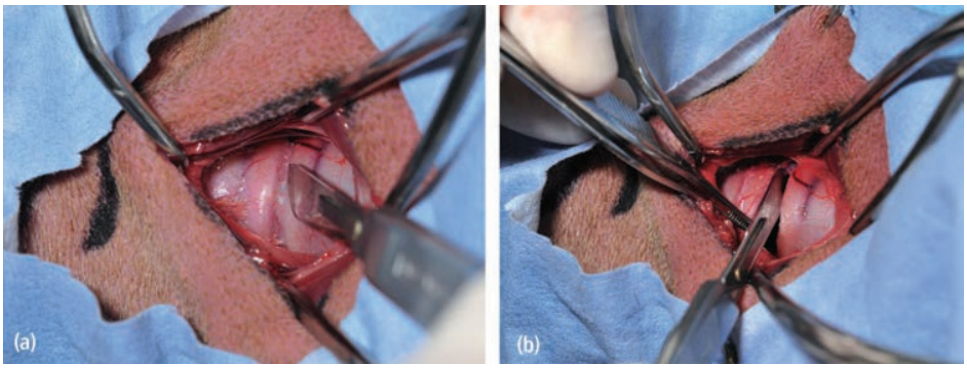


Figure 29.7 Incising the interannular ligament for tracheostomy tube placement. Cranial is to the left in both frames. (a) The interannular ligament between the second and third tracheal rings is isolated. (b) After the initial interannular incision, the scalpel blade is turned upward to extend the incision, taking care to not damage the underlying endotracheal tube or its cuff. The tracheotomy is limited to 50% or less of the tracheal circumference.

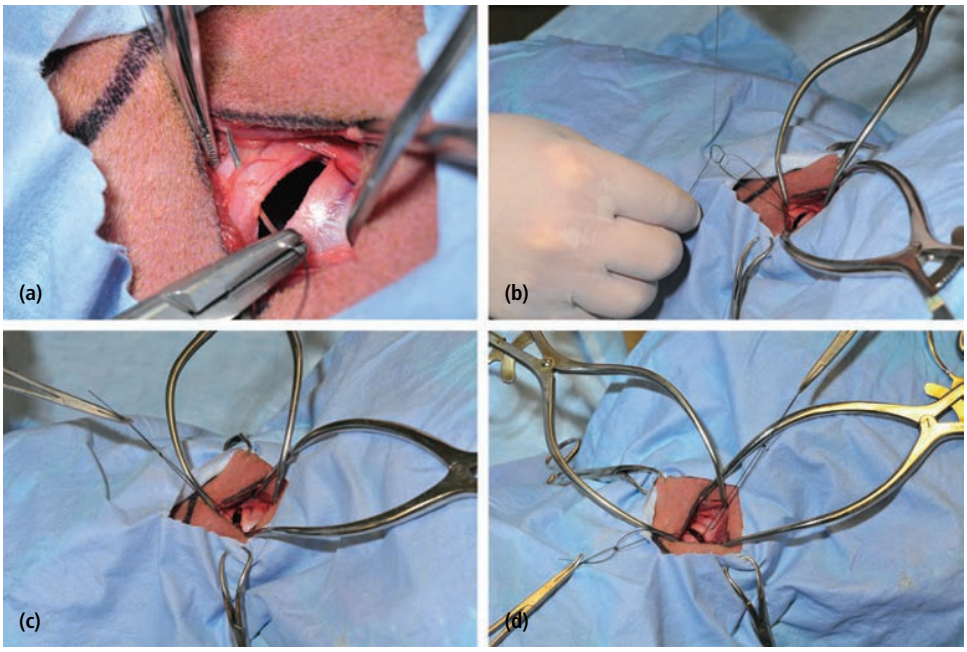


Figure 29.8 Placing tracheal stay sutures immediately before tracheostomy tube placement. Cranial is to the left in all frames. (a) The suture needle is placed around the second tracheal ring, the ring just cranial to the tracheotomy. (b) The suture is knotted such that a long loop will be retained. (c) The free ends of the stay suture are tagged temporarily with a mosquito hemostatic forceps distal to the knot. (d) A second identical stay suture is placed around the third tracheal ring, the ring just caudal to the tracheotomy.

chosen because the area of the second through fourth tracheal rings is the preferred stomal site for permanent tracheostomy should it be required later. Do not incise the interannular ligament beyond 50% of the tracheal circumference. Place stay sutures (3-0 or 2-0 nylon for cats/small dogs or medium/large dogs, respectively) around the second and third tracheal rings, knot the sutures to create large suture loops, and tag the suture strands with mosquito hemostatic forceps distal to the knot (Figure 29.8). Use the stay sutures to manipulate the interannular opening while the endotracheal tube is removed and the tracheostomy tube is inserted (Figure 29.9). During the postoperative course, the stay sutures can be used for manipulation during reinsertion of a tube that has been inadvertently dislodged or requires changing; therefore, it is recommended that they not be removed intraoperatively. Insert the tracheostomy tube with the obturator in place (Figure 29.9b), and quickly remove the obturator and replace with an inner cannula as soon as the tracheostomy tube is positioned in the trachea (Figure 29.10; note that removing the self-retaining retractors before inserting the tracheostomy tube is recommended because it is awkward to remove them once the tube is in place). Secure the tracheostomy tube by attaching umbilical tape to the flange

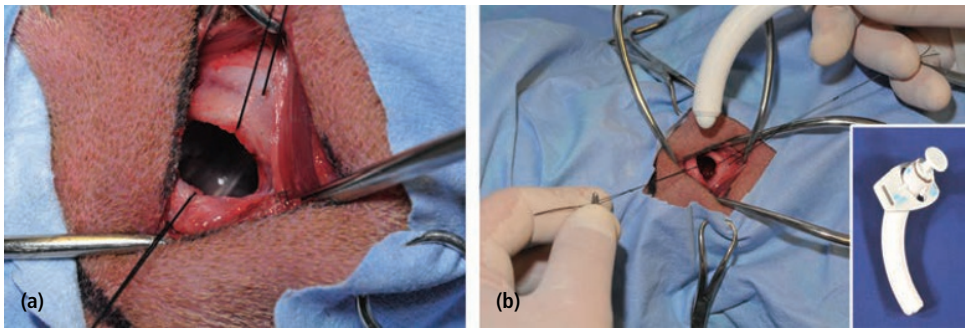


Figure 29.9 Preparing to insert a tracheostomy tube. (a) Cranial is to the lower left. The cranial and caudal stay sutures are retracted to pull open the tracheotomy. Note the endotracheal tube within the tracheal lumen. The cuff will now be deflated and the endotracheal tube removed as the tracheostomy tube is inserted. (b) Cranial is to the left. The obturator is inserted into the tracheostomy tube immediately before placing the tube into the trachea. The purpose of the obturator is to keep blood and other secretions from being scraped into the tracheostomy tube lumen during placement.

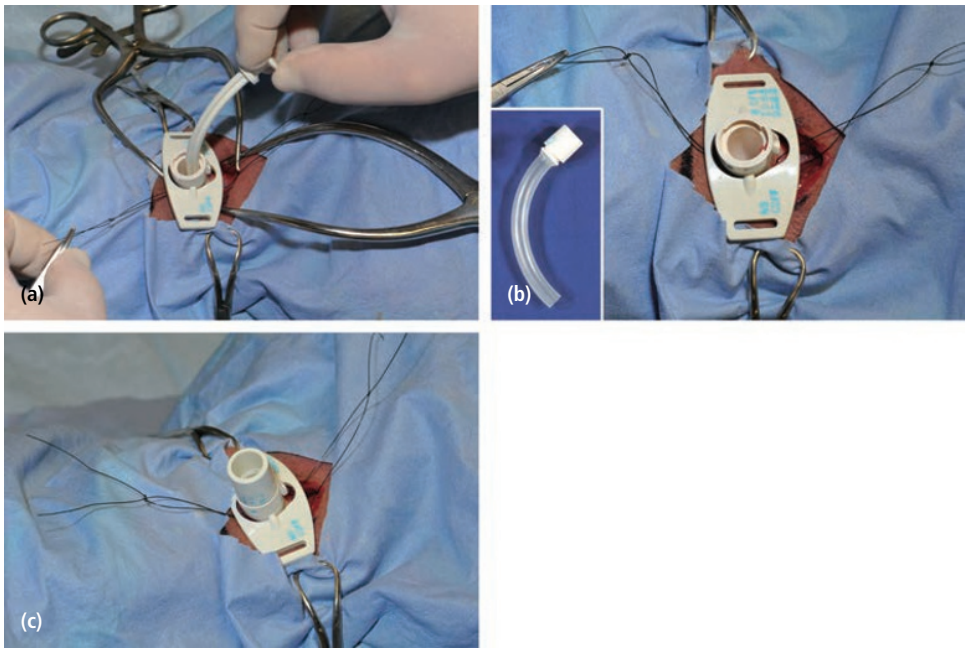


Figure 29.10 Insertion of a tracheostomy tube. Cranial is to the left in all frames. Contrary to what is illustrated, the Gelpi perineal retractors should be removed prior to tube insertion because removing them is awkward once the tube is in place. (a) The obturator is removed as soon as the tracheostomy tube is in place. (b) After removing the obturator, the inner cannula (inset) is placed into the tracheostomy tube. (c) The inner cannula has been inserted into the tracheostomy tube and locked in place. The hemostatic forceps are removed from the stay sutures and the stay sutures are left in place.

eyelets and tying the tapes behind the neck (Figure 29.11). Secure ties firmly behind the animal's neck, leaving enough room for two fingers to be placed between the neck and ties. There should be no need to suture the tracheostomy wound, unless the incision was made too large. In that case, a few interrupted sutures may be placed in the subcutaneous tissue and/or skin to decrease the size of the wound; however, care should be taken not to make the wound too small because the wound is contaminated and must be able to drain. See Protocol 29.1 for concise step-by-step instructions.

Once the surgical procedure is completed, the area is gently cleaned. Tape tabs are applied to the two stay sutures (in place of the hemostatic forceps), one tab bearing the word "UP" on one side and "CRANIAL" on the opposite side, and the other tab bearing the word "DOWN" on one side and "CAUDAL" on the opposite side, to clearly indicate which way one should hold the stay sutures in the event of a tube dislodgement emergency. Duct tape is preferred over medical tape for these tabs for more effective cleaning and disinfection. It is usually best to leave the incision area uncovered for easy observation of swelling,

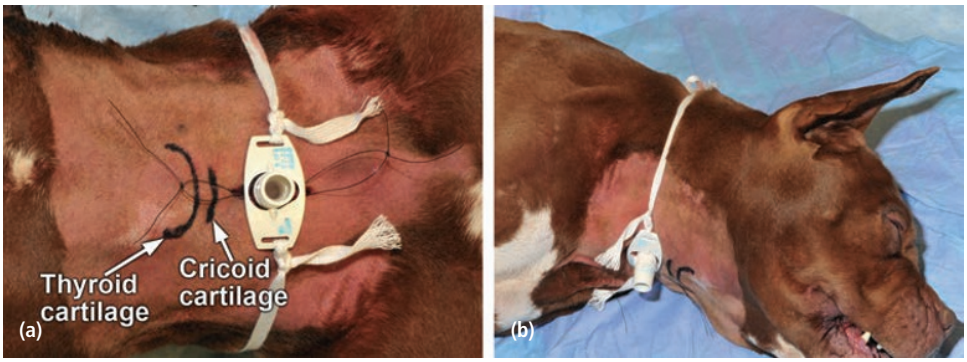


Figure 29.11 Completed tracheostomy tube placement. (a) Umbilical tapes are tied to the eyelets in the tracheostomy tube flange. The knotted stay sutures are left in place. Labeled tape tabs will be placed where the hemostatic forceps were. The stay sutures are used to facilitate replacement of the tracheostomy tube in the case of inadvertent or planned removal. (b) The umbilical tapes are tied on the back of the neck.

Protocol 29.1 Temporary Tracheostomy Tube Placement

Items Required

- Appropriate type and size tracheostomy tube
  - Alternatively, standard endotracheal tube (ETT) fashioned into tracheostomy tube, one size smaller than for endotracheal intubation
- Appropriately sized ETT for endotracheal intubation, with cuff syringe
- Clippers with clean blade
- Surgical scrub supplies
- Barrier drapes
- Sterile surgical gloves
- Sterile gauze
- Surgical instruments (Box 29.1)
- Suture material for stay sutures (3-0 or 2-0 nylon)
- Umbilical tape
- Assistant
- Suction capability
- Oxygen/standard anesthetic supplies and equipment

Procedure

1) Collect necessary supplies.
2) Anesthetize and orotracheally intubate the patient with a cuffed ETT.
3) Position the animal in dorsal recumbence with a towel rolled under the neck. Clip and aseptically prepare a large surgical field on the ventral cervical surface.
4) Perform hand hygiene, and don cap, mask, and sterile gloves.
5) Isolate the prepared surgical site with a barrier drape.
6) Make a ventral midline cervical skin incision just caudal to the larynx for a distance of approximately 3–4 cm.
7) Apply a self-retaining retractor to hold open the skin edges and clear just enough subcutaneous tissue to identify the midline division of the sternohyoideus muscles.
8) Using Metzenbaum scissors, bluntly separate the sternohyoideus muscles on the midline, taking care to avoid the thyroidea caudalis vein on the midline between these two muscles. Retract the thyroidea caudalis vein to one side, together with one of the sternohyoideus muscles.
9) Reposition the self-retaining retractors on the sternohyoideus muscles to expose the trachea and clear the loose fascia away at the proposed tracheotomy site. Application of a second self-retaining retractor at a right angle to the original retractor to retract the skin in a craniocaudal direction enhances exposure.
10) Using a scalpel blade, incise the interannular ligament between the appropriate tracheal rings. Do not incise the interannular ligament beyond 50% of the tracheal circumference.
11) Place stay sutures around the tracheal rings on the cranial and caudal borders of the interannular ligament incision, knot the sutures to create large suture loops, and tag the suture strands with mosquito hemostatic forceps.
12) Use the stay sutures to manipulate the interannular opening while the orotracheal tube is removed. Insert the tracheostomy tube with the obturator in place, and then quickly remove the obturator and replace it with an inner cannula.
13) Leave the stay sutures in place for postoperative nursing care manipulations. Apply labeled tape tabs to the stay sutures.
14) Secure the tracheostomy tube by attaching umbilical tape to the flange eyelets and tying the tapes behind the neck.
15) Do not suture the tracheostomy wound unless the incision was made too large. In that case, place a few interrupted sutures in the subcutaneous tissue and/or skin to decrease the size of the wound, taking care not to make the wound too small.
16) Once the surgical procedure is completed, the area is gently cleaned and left uncovered for easy observation.

bleeding, accumulated secretions, and tube position, and for prompt intervention as needed. If a bandage is applied, wait until the animal is awake and standing or in ventral recumbence. A bandage applied to the neck in a recumbent patient could change position when the patient recovers and becomes more active. The altered bandage position could cause patient discomfort or interfere with tube positioning and thus breathing. The bandage must be changed at least once daily to observe for tube site complications. Simply peeking under the bandage is insufficient.

Contraindications

There is no absolute contraindication for placing a tracheostomy tube when upper airway obstruction is causing respiratory distress. However, endotracheal intubation is always the preferred method of capturing an airway in an animal with airway distress. Once the airway is controlled, tracheostomy may be performed under controlled conditions. The quick "slash" tracheostomy is rarely needed but may be necessary if endotracheal intubation is not possible.

Relative contraindications for tracheostomy tube placement include uncorrected coagulopathy, symptomatic thrombocytopenia, unstable cervical spine, increased intracranial pressure, presence of a cervical mass that would interfere with the surgical approach, and recent cervical surgery. If tube tracheostomy is indicated in patients with any of these conditions, the condition is brought under control or preparations are undertaken to deal with the consequences prior to placement of the tracheostomy tube. Plasma may be given to coagulopathic patients, and meticulous hemostasis should be exercised during surgical placement to avoid excessive blood loss in patients with coagulopathy and thrombocytopenia. Careful positioning and cautious manipulation must be performed in patients at risk for cervical and intracranial neurologic complications. The consequences of invading a cervical mass or previous surgical site in the neck must be weighed against the need for tracheostomy tube placement. Tracheostomy tube placement may be lifesaving in the face of relative contraindications, as long as proper preparations and precautions are used.

Possible Complications During the Procedure

Intraoperative complications with tracheostomy tube placement can be avoided by diligent attention to surgical technique. Hemorrhage can be minimized with meticulous attention to hemostasis and avoidance of inadvertent vessel damage. The thyroidea caudalis vein (Figure 29.5d) is a single midline vein on the dorsal aspect of the sternohyoideus muscles, which can be avoided by careful separation of these muscles, but care must also be taken not to tear or puncture the vessel with self-retaining retractors. Recurrent laryngeal nerve damage can be prevented by limiting the clearing of peritracheal fascia to the ventral aspect of the trachea over the intended interannular ligament and associated tracheal rings. Incising an interannular ligament that is too close to the larynx or too far caudal can be avoided by precise attention to the regional anatomy. Inadvertent puncture of the endotracheal tube cuff during interannular ligament incision or puncturing the cuff during stay suture placement can be avoided by palpation of the inflated cuff and close communication with the anesthetist to deflate and reposition the ETT and cuff before making the tracheal incision and placing sutures.

Nursing Care Considerations for Patients with Temporary Tracheostomy Tubes

Patient management and monitoring begin immediately after the procedure to minimize the risk of airway obstruction from dislodgment or occlusion of the tube. Regular care may be scheduled every 2–3 hours but may be needed as often as every 15 minutes if a patient's condition warrants. The objectives of regular management are to prevent buildup of secretions that may block the tube, provide aseptic wound care, and humidify inspired air. Frequent observation is the best way to gauge the need for increased tube care. Abnormal respiratory pattern, coughing, or pawing at the tube or face should signal the need to check for occlusions and increase the frequency of tube care.

Airway Humidification

One may humidify inspired air by instilling sterile isotonic saline (small patients, 0.5 ml; large dogs, up to 3 ml) into the tube hourly. Aseptic instillation is achieved by cleansing the external portions of the tracheostomy tube and surrounding skin with 0.05% chlorhexidine solution, drawing sterile saline into a sterile syringe, removing the hypodermic needle used to aspirate the saline, and quickly injecting the saline into the tracheostomy tube without coming in contact with surroundings. Alternatively, the airway can be humidified with a humidity exchange filter (also called a heat moisture exchanger or "artificial nose") if the tube is attached to a rebreathing circuit, or a nebulizer can be used. Humidity exchange filters are disposable devices that interface between the tracheostomy tube and breathing circuit of a ventilator or anesthetic machine and trap exhaled moisture, which then humidifies the inhaled gas. Aerosol therapy (nebulization) can be used as a means of airway

humidification. Nebulization with sterile isotonic saline can be used in sessions of 10–15 minutes every 4–6 hours. Sterile water can be nebulized if humidification is the sole purpose, but saline is better for loosening thick airway secretions to facilitate their removal. Nebulization produces particles small enough to oversaturate the lungs if used excessively.

Tracheostomy Site Hygiene

The tracheostomy site and surrounding skin should be examined frequently, at least whenever the tube is cleaned. Good wound care decreases bacterial growth and facilitates patient comfort. Prior to tending to the tracheostomy wound, the caregiver should disinfect hands and don gloves. Sterile gloves are preferred, but clean examination gloves suffice when sterile gloves are not practical. Skin surrounding the incision, especially under the flange of the tube, should be gently cleansed with sterile cotton-tipped applicators or gauze sponges soaked in dilute (0.05%) chlorhexidine solution. Begin at the wound edges and work away from the incision. Squeeze excess solution from the cleaning materials before the skin is cleaned. Keep antiseptic soaps and antibacterial ointments away from the incision and wound area because they can irritate exposed tracheal mucosa. Remove exudate from the incision promptly to prevent abscess formation and skin maceration. If gauze pads or fenestrated tracheostomy sponges are used, apply fresh ones after each wound care session. The area of cleaned skin must be completely dry before new sponges are placed. Gauze pads should never be trimmed or cut because fibers could embed in secretions from the surgical incision or be inhaled into the airway. Fold sponges in a triangle pattern and place them on each side of the site under the flange and ties. Triangular folding allows the long base of the triangle to contact the wound, leaving a pointed apex more externally located for ease of grasping during dressing changes. Special fenestrated sponges are placed above the tube, with the side panels extending down under the flange and ties. Wound areas can also be maintained without pad materials to allow easier visualization of the tissue around the tube and incision area.

Tube Tie Inspection

The wound maintenance session is an excellent time to inspect the ties. Ties should be checked regularly for security. Umbilical tape tied with a double bow will hold firmly but can be easily loosened. Ties secured with a knot may be difficult to remove, and thus scissors should be hanging on the cage or readily available near the patient. A tube that is untied even briefly is in danger of being dislodged. If not secured, a tube can be expelled in an instant with unexpected force. Ties that are too tight will cause discomfort, whereas ties that are too loose will allow the tube to slide freely within the trachea and can cause mucosal damage. When changing soiled ties, secure the clean ties before removing the old ones.

Tracheostomy Tube Suctioning

Regular suctioning of the tracheostomy tube helps prevent accumulation of secretions that may cause occlusions but must be done gently to minimize patient discomfort and to avoid adverse effects such as tracheal mucosal irritation, gagging, and bradycardia due to vagal stimulation. Patients should be preoxygenated for several breaths from an oxygen source held at the tracheostomy tube opening. Using aseptic technique, a small sterile suction catheter made of pliable tubing with side fenestrations is then introduced. Suctioning is not begun until the suction catheter is in place; intermittent light suction is then applied as the catheter is withdrawn with a circular motion. Most suction catheters are controlled by a thumb port to allow adjustment of the amount of suction. The entire suctioning procedure should be completed in less than 15 seconds. The patient is allowed a few moments to "catch its breath," supplemental oxygen is again held near the tracheostomy tube opening, and suctioning is repeated if necessary. If power-driven suction is unavailable, a handheld suction unit (available at automotive stores) fits medical suction tubing and can be used for patient care. Because the action of suctioning can initiate gagging or vomiting, patients should not be suctioned immediately after eating. Stop suctioning immediately if respiratory or cardiac changes or excessive patient discomfort occur.

Tracheostomy Tube Cleaning

For cannulated tubes, the inner cannula is removed as often as the patient's condition warrants but no less often than every four to six hours. The inner cannula is removed and immediately replaced with a second sterile (or adequately sanitized) inner cannula, making sure that the locking mechanism is secured. When copious secretions are noted, it may be necessary to instill saline into the outer lumen tube and suction that tube before placing the new inner cannula. The removed cannula is meticulously cleaned and placed in a soak solution of 0.05% chlorhexidine until the next tube change. When the inner cannula is next changed, the sanitized cannula is removed from the antiseptic solution and thoroughly rinsed with sterile water or saline before being reused.

If an uncannulated tube is used, periodically replace the tube with a fresh one. However, if frequent tube changes are needed, less damage and irritation to the tissue occurs if saline is instilled and suction applied while the tube remains in place. The frequency of suctioning depends on

the patient. Patients with conditions that produce excessive secretions may need frequent suctioning or more frequent humidification than those with minimal secretions. Patients with conditions that produce moderate secretions may be suctioned every four to six hours. Cats seem to need more frequent maintenance than dogs because they have a tendency to produce thick mucus, especially on the second and third day after tube placement. Blockage of the tube by secretions is the most common life-threatening occurrence in patients with a temporary tracheostomy tube. Increasing amounts or changes in consistency of secretions can be a warning of greater risk of tracheal occlusion and warrant more frequent suctioning, humidification, or nebulization. If an occlusion is suspected, remove the inner cannula or replace the tube. Attempts to force a suction catheter tip down the tube or to clear the tube by running any form of stylet through it will force material into the patient's airway and should thus be avoided.

Replacement of Tracheostomy Tubes

Tracheostomy tubes (cannulated or not) should be replaced no less often than every 24 hours. All materials should be prepared and available, and an assistant should restrain the patient. After untying the tube ties, pull the tape tabs opposite each other so that the stay sutures open the tracheostomy, and remove the tube. The new tube is then inserted, ensuring that it enters the trachea and not the subcutaneous tissue, and new neck ties are secured. This procedure should be done as quickly as possible after preoxygenation, and oxygen should remain available in case the patient becomes distressed. A mask and eye protection are recommended for the caregiver when the patient's condition produces excessive secretions to keep expelled secretions out of the caregiver's face during inspection of the incision and tube care.

General Patient Observations

When respiratory distress is relieved, patients often relax and are able to rest. Many dogs and most cats assume a position with the head down or neck bent that will seem to block the free flow of air to and from the tracheostomy tube; however, the flexed neck rarely causes a problem. The rate and character of respiration should be closely observed rather than constantly waking, disturbing, or repositioning the patient. Any abnormal breathing pattern, such as dyspnea, tachypnea, or hyperpnea, warrants intervention.

Environmental Considerations and Patient Hygiene

Cages must be kept clean and free from materials that could be inhaled, such as lint from bedding and excess hair shed by the patient. Fur inevitably accumulates in the cage and creates a hazard. For patients that seem to be at greater risk for aspiration of such debris, a gauze shield can be placed over the tube opening; human stoma shields might also be adapted for this purpose. Cats should be provided with long strips of cut paper for litter rather than clay or any material that could produce dust or small particles that could adhere to the incision or be inhaled. For cats that refuse to void except in their customary litter materials, the litter box should be offered regularly and then removed from the cage, especially because many hospitalized cats seem to prefer to sleep in their litter boxes. Tracheostomy patients require general hygiene care as with any hospitalized patient, but common sense dictates modification of certain nursing procedures. For example, tub bathing could result in aspiration of soap and water into the tracheostomy tube or the wound around the tube; animals with tracheostomies should never be bathed.

Attention to Hydration Status

Close monitoring of hydration is important in patients with a temporary tracheostomy tube in place. Normally, inhaled air is heated and humidified by the nasopharynx and tracheobronchial tree so that alveolar air is 100% humidified at body temperature and contains four to six times the water vapor content of room air. Inspired air that bypasses the upper airways increases humidification requirements and desiccates the respiratory mucosa, resulting in viscous secretions, impaired mucociliary transport, inflammation, small airway collapse, decreased functional residual capacity, reduced pulmonary compliance, and increased risk of infection. The flow of un-humidified inspired air causes evaporation on the surface of the respiratory mucosa, resulting in a loss of heat from the epithelial surface and, ultimately, patient hypothermia. Therefore, monitoring body temperature goes hand in hand with attention to hydration status. Animals on diuretic therapy and small, hypermetabolic patients must be encouraged to drink or be given continuous parenteral or enteral fluid support. Patients should be weighed no less often than once daily to aid in the evaluation of patient hydration. Most patients capable of food and water intake seem to have little trouble eating and drinking with a tracheostomy tube in place. However, it is still good nursing practice to observe animals while they are eating and drinking. Food and water bowls may need to be adjusted to avoid possible aspiration, especially in short-necked or brachycephalic animals. If problems are noted with bowls or the acts of eating and drinking, food and water should be kept out of the cage and offered intermittently while the patient is supervised.

Tracheostomy Tube Removal

The longer a tracheostomy tube must remain in place, the more likely intubation complications and stenosis become. The tube should be removed as soon as assisted ventilation is no longer needed, the patient can move a normal volume of air around or without the tube, or the condition that required the procedure is resolved. When a patient is to be weaned from the tube, replace the existing tube with a tube of the next smaller size and closely observe the patient for 10–15 minutes for signs of dyspnea. If no distress is noted, then a smaller diameter tube is placed at the next scheduled maintenance session. If a patient shows signs of respiratory distress, the tube that was just removed (i.e. the larger tube) is replaced and tube removal attempted again in 12–24 hours. If breathing is adequate with the smaller tube in place, this tube is occluded with a cap or occlusion cannula and the patient is observed for 30 minutes. If no dyspnea is noted, the tube is removed. If a patient already has the smallest available tube in place, then weaning must be attempted by complete removal of the tube and close observation of the patient for 15 minutes. If distress occurs, the tube must be immediately replaced. Do not remove the tape-tabbed stay sutures until it is certain that the tracheostomy tube will no longer be needed. These sutures are easily removed even if still in place 48 or more hours after tube removal; therefore, leave the stay sutures in place until there is no doubt that the tube will no longer be needed. Tracheostomy wounds are allowed to heal by second intention. The area is cleaned daily of any drainage with sterile saline-moistened gauze, avoiding excess liquid near the stoma. Patients with temporary tracheostomy tubes require 24-hour observation and are almost never released to home care. Patients with tracheostomy wounds in the process of healing may be returned to an owner's care with strict instructions that the animal must not be bathed, cannot be allowed to swim, cannot wear collars or neck leads, and should be returned for reexamination and further directions.

Summary

Temporary tracheostomy tube placement is a skill all emergency and intensive care veterinarians should master. A well-prepared technical staff with a good nursing plan in place makes care of patients with temporary tracheostomies less stressful for all concerned and helps to ensure a satisfactory outcome.

Acknowledgement

The author and editors would like to acknowledge the contribution of Mary M. Flanders (deceased) to the chapter that appeared in the first edition of *Advanced Monitoring and Procedures for Small Animal Emergency and Critical Care*, upon which this chapter is based.

30

Capnography

Linda S. Barter and Alessia Cenani

Terminology

A continuous plot of carbon dioxide (CO_2) in the respired gas against time is called a capnogram and is recorded by an instrument known as a capnograph. A capnometer detects the highest and lowest values for CO_2 in the respired gas and reports them as inspired and end-expired (also known as end-tidal) partial pressures or concentrations. The practices of measuring and recording CO_2 are called capnometry and capnography, respectively. Many monitors function as both capnometers and capnographs, displaying both numerical and graphical information about CO_2 in the respired gas. As with all monitoring tools, a capnogram is only a snapshot in time of one aspect of the patient's respiratory system function and should be evaluated in light of the patient's clinical condition.

Physiology

Aerobic metabolism in tissues consumes oxygen, glucose, and other substrates and eventually produces energy, CO_2, and water. CO_2 produced in cells easily diffuses into the surrounding interstitial fluid raising local partial pressure of carbon dioxide (PCO_2). Arterial blood entering the tissues has a lower PCO_2 than the interstitial tissue and thus CO_2 diffuses from the interstitial fluid into blood. Venous blood leaves the tissues with a PCO_2 higher than that of arterial blood but equal to that of interstitial fluid. Venous blood carries the CO_2 produced by metabolism to the lungs to be removed from the body by ventilation. The process of ventilation replaces CO_2-rich gas from the alveoli with CO_2-free gas from the atmosphere or breathing circuit.

Alveolar partial pressure of CO_2 reflects a balance between CO_2 delivery to the alveoli by the cardiovascular system and its removal by ventilation. In the steady state, alveolar partial pressure of CO_2 is directly related to the metabolic production of CO_2 and inversely related to alveolar ventilation. CO_2 is highly diffusible such that in perfused alveoli, alveolar and arterial partial pressures of CO_2 are considered equivalent. Gas sampled at the end of expiration (end-expired gas) is representative of alveolar gas; thus, the end-expired CO_2 measured by a capnograph is used as an estimate of arterial PCO_2.

Under the control of both the central and peripheral chemoreceptors, the body normally maintains arterial PCO_2 within a tight range by adjusting ventilation to the amount of CO_2 produced. Normal range for arterial PCO_2 is 35–45 mmHg, with some minor variations between species [1, 2].

Hyperventilation describes the situation where alveolar ventilation exceeds metabolic CO_2 production resulting in alveolar (and thus arterial) PCO_2 levels below the normal range (hypocapnia). Low values for PCO_2 in arterial blood ($PaCO_2$) are associated with respiratory alkalosis and reduced cerebral blood flow. Hypoventilation describes the opposite situation in which alveolar ventilation is insufficient to remove the metabolically produced CO_2 causing alveolar (and arterial) PCO_2 to rise above the normal range (hypercapnia). Most anesthetic and sedative drugs result in dose-dependent respiratory depression and respiratory acidosis. A $PaCO_2$ greater than 60 mmHg is generally considered respiratory depression significant enough to warrant positive pressure ventilation in small animal patients.

Types of Carbon Dioxide Analyzers

There are two types of CO_2 analyzers: mainstream and sidestream.

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Companion website: www.wiley.com/go/burkitt/monitoring

Mainstream

In mainstream analyzers, a sample cell or cuvette is inserted directly in the artificial airway between the tracheal tube and the breathing circuit. An infrared sensor fits over the sample cell and emits light through windows in the sample cell (Figure 30.1). Light reaching the photodetector on the opposing side of the sensor measures PCO_2. Because the measurements are made directly in the airway, this technology eliminates the need for sampling tubes and scavenging but limits its use to the intubated patient. The capnographic waveforms generated by mainstream analyzers are crisper than those from sidestream analyzers because they reflect real-time CO_2 measurements and suffer no deformity due to dispersion of gases in a sample line.

To prevent condensation on the sample cell windows, which can cause falsely high CO_2 readings, the mainstream sensor is heated. Thermal injury to the patient is possible; however, newer analyzers now have limited upper temperatures to avoid such problems. Disadvantages of mainstream analyzers are that they can be bulky and can have relatively large internal volume. This bulk puts traction on the endotracheal tube, which may increase the risk of inadvertent extubation, and their internal volume adds to apparatus dead space. These factors are more troublesome in smaller patients. The sensor unit in older models was fragile and easily broken; however, newer models are of simpler design and are lightweight, increasing their durability and suitability to daily use in veterinary practice. Many models use disposable sensor windows, available in standard and pediatric sizes (Figure 30.1), which can be changed between patients, to prevent contamination and minimize apparatus dead space.

Sidestream

In sidestream capnography, the CO_2 measuring unit (monitor) is remote from the patient. A small pump within the monitor aspirates gas from the patient's airway through a long sampling tube. Ports onto which a sample line can be attached can be found on some endotracheal tube adapters, some breathing circuit Y-pieces, or most commonly, lightweight connectors designed to be inserted between the endotracheal tube or mask and the breathing circuit (Figure 30.2).

With sidestream capnography, gas analysis is delayed because it takes time for the gas to reach the monitor and some time to make the measurement (depending on the technology used). The effect of this delay is that capnographic waveforms generated by sidestream analyzers do not appear synchronously with each breath as is the case with mainstream capnography. Additionally, capnographic waveforms produced by sidestream monitors tend to be more rounded than those produced by mainstream devices in the same situations (Figure 30.3). The delay due to transit time depends on the length and diameter of the tubing and the rate at which gas is aspirated (this can vary from 50 to 250 ml/minute depending on the monitor). As gas aspirated from the airway travels through the tubing to the monitor, the gas molecules can move around and start to mix (i.e. the CO_2-containing gas starts to mix with non-CO_2-containing gas). The faster the transit time between the airway and the monitor, the less mixing will occur and the more representative will be the capnogram of actual changes in respiratory gas composition. Slow rates of aspiration, long sample lines, and large-bore sample lines result in capnogram waveforms with slurred up- and downstrokes (see section on capnographic interpretation).

Sidestream analyzers remove gas from the patient's breathing circuit. This must be accounted for when calculating fresh gas flow rates and means that if patients are anesthetized with an inhaled anesthetic agent, then the gas must be appropriately scavenged or returned to the patient's breathing system. Gas may pass through conduits within the analyzer that cannot be cleaned or sterilized. This may pose an infectious disease risk to subsequent patients if analyzed gas is returned to the patient. The

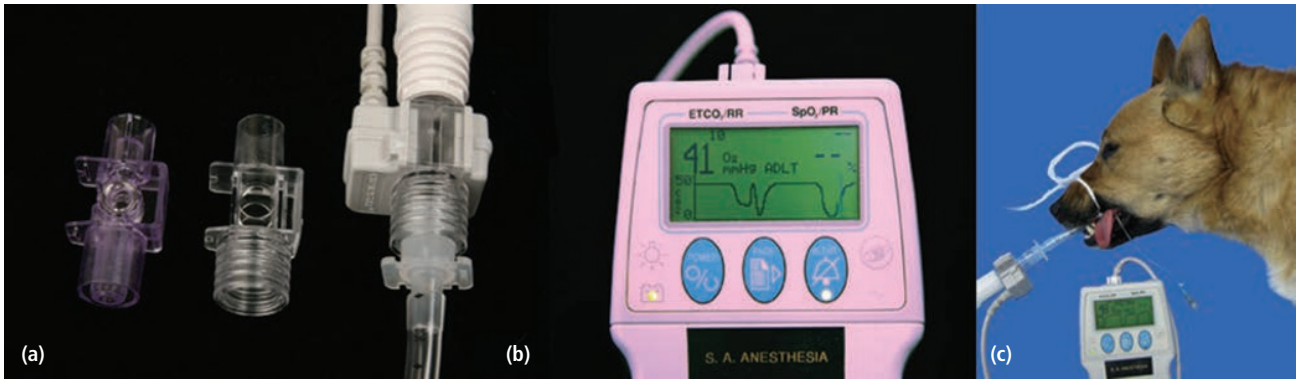


Figure 30.1 Mainstream capnography. (a) From left to right are micro cuvette, standard cuvette, and standard cuvette inside the gray infrared sensor, connected between an endotracheal tube and the white patient breathing circuit. (b) The display screen of a portable mainstream capnograph. (c) Mainstream capnograph in use on an anesthetized dog.

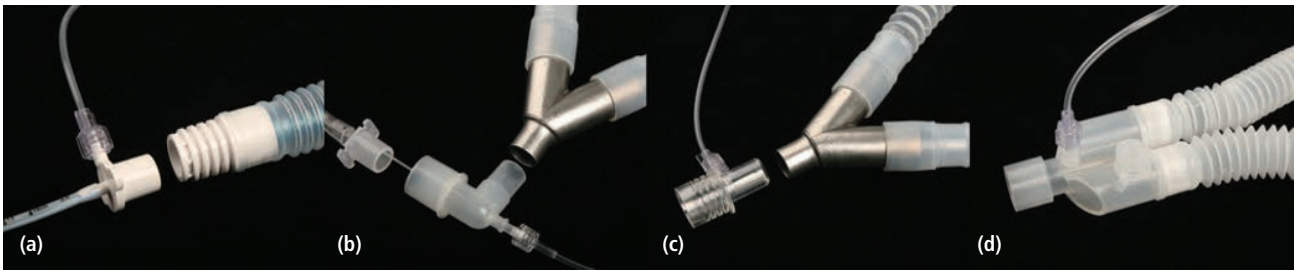


Figure 30.2 Different connection options for a sidestream capnograph gas sampling line. (a) Endotracheal tube adaptor with sample line adaptor. No additional apparatus dead space is added by attaching the capnograph in this way. (b) An elbow connector with adaptor port through which a small-bore catheter has been placed, which will be situated in the distal third of the endotracheal tube lumen to improve accuracy of sidestream gas sampling. (c) Short in-line connector between endotracheal tube and breathing circuit to which gas sampling line can be attached. (d) Some circuit Y-pieces have built-in sampling line connection ports.

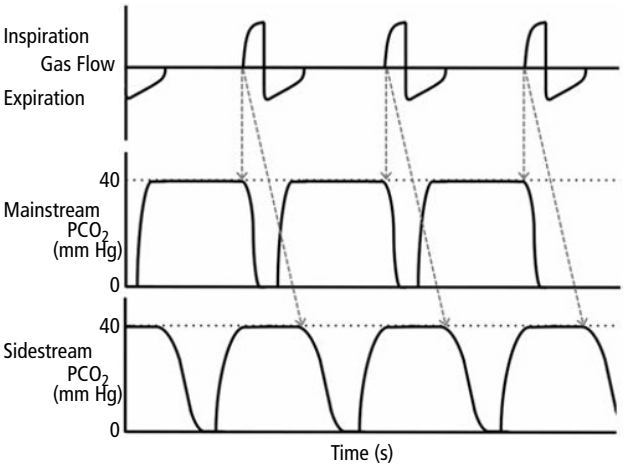


Figure 30.3 Comparison of capnograms obtained from mainstream and sidestream capnographs. The upper tracing depicts gas flow during the respiratory cycle, with gas flow above the line representing inspiration and gas flow below the line representing expiration. The middle tracing is a mainstream capnogram and the lower a sidestream capnogram recorded from the same patient at the same time. The dashed lines connecting the flow tracing with the capnograms illustrate the delay in registering changes in partial pressure of carbon dioxide (PCO_2) by capnography. The magnitude of this delay with a sidestream analyzer varies with the monitor settings and equipment (see text for details). A major difference between the two types of analyzers can be seen in the shape of the capnograms. Mainstream capnograms tend to record sharper changes in PCO_2, creating more vertical up-and-down strokes on the capnogram when compared with sidestream capnograms (see text for more details).

small-bore sample lines used by sidestream analyzers can easily become obstructed with moisture or aspirated secretions, and methods must be instituted to collect moisture, such as the use of Nafion tubing (Nafion, E. I. du Pont de Nemours and Company) or water traps.

Sidestream CO_2 analyzers may be single-purpose monitors or part of a larger monitoring unit with capabilities to analyze other respiratory gases as well as inhaled anesthetic agents. A unique advantage of sidestream capnographs is the ability to use them to monitor non-intubated patients. For example, expired gases may be sampled from the nasal cavity using nasal cannulas, or these monitors may be connected to a feeding tube to obtain information to ensure correct placement (see indications section for more detail).

Equipment Setup

In smaller patients mainstream CO_2 analyzers are technically superior to sidestream analyzers [3, 4]. Owing to their faster response time, PCO_2 measurements are more reliable, especially when tidal volumes are small and respiratory rates are high. Mainstream analyzers are less susceptible than sidestream ones to artifacts caused by the small tidal volumes and high fresh gas flows encountered when using non-rebreathing systems in small patients. The main disadvantage of mainstream measurements is the addition of apparatus dead space. This can lead to rebreathing of CO_2 and either elevation in $PaCO_2$ or increased work of breathing and altered ventilatory patterns to maintain a normal $PaCO_2$ [5]. The size of the sampling cuvette relative to the tidal volume of the patient should be considered when choosing the type of analyzer to use on an individual (mainstream vs. sidestream). Other considerations in making that decision would include whether there is any additional apparatus dead space as well as the length of time you intend to use the monitor. Routine mainstream capnography would not be recommended for long-term use in small patients [6]. It is always good to minimize apparatus dead space; however, small internal volume connectors or cuvettes have a small internal diameter. As such a compromise is made to avoid unnecessary increases in airway resistance due to reduced airway diameter. It would be generally recommended not to use a connector with an internal diameter any smaller than that of the patient's endotracheal tube.

Sidestream analyzers can be connected to the patient airway with the addition of minimal to no apparatus dead space (Figure 30.2d). The cost of making this choice is reduced accuracy. A general figure for total minute ventilation is 200 ml/kg/minute. Gas aspiration rates of the monitor must be considerably lower than the patient's total minute ventilation to prevent dilution of expired gas with fresh gas during sampling. If the sampling rate is too near minute ventilation, the capnograph waveform will be deformed and end-expired CO_2 underestimated. However, slow aspiration rates mean a long delay before gas is analyzed and distortion of the capnographic waveform. The sampling line should thus be as short as possible to minimize this delay time. Microstream aspiration technology with miniaturized sample chambers should be used if expired CO_2 is to be measured on spontaneously breathing small patients (< 3–4 kg) for any length of time for improved accuracy and reduced dead space [7, 8].

The closer to the alveolus that respiratory gas is sampled, the more faithfully the capnogram represents alveolar gas. However, a major problem with sampling from within the airways as is required with the sidestream technique is machine aspiration of secretions and water vapor. In intubated patients, sampling catheters placed within the lumen of the endotracheal tube reduce mucus aspiration (Figure 30.2b).

Technology of Carbon Dioxide Measurement

Several techniques are available for measuring CO_2 including infrared absorption, Raman scattering, and mass spectrometry.

Infrared Absorption

Infrared absorption is the most popular technique for CO_2 measurement. Monitors using infrared technology are typically the most compact and least expensive. Infrared absorption is the only technique used for CO_2 measurement in mainstream analyzers. Polyatomic gases like CO_2 have specific and unique absorption spectra of infrared light. The amount of light absorbed in a specific spectrum is proportional to the concentration of the absorbing molecule. The concentration of gas can then be determined by comparing the measured light absorbance with that of a known standard. Infrared absorption can be used to measure any polyatomic gas (e.g. nitrous oxide and the halogenated anesthetic agents), which may be advantageous if purchasing a single monitor for use in anesthetized patients.

Infrared monitors have a short warmup period and a fast response time for CO_2 measurement, allowing them to measure inspired and expired concentrations. Water vapor absorbs infrared light and thus can spuriously increase measured CO_2. Water must therefore be removed from the expired gas by use of water traps or Nafion tubing (Nafion, E. I. du Pont de Nemours and Company). There is some overlap between the absorption characteristics of nitrous oxide and CO_2. Most newer monitors that measure nitrous oxide in addition to CO_2 can correct for the effect of nitrous oxide on CO_2 readings.

Microstream capnographs are based on a modified approach to infrared absorption. Molecular correlation spectroscopy is used to generate a narrow band of infrared light that precisely matches the absorption spectrum of CO_2 and eliminates interference with other gases. The high CO_2 specificity and sensitivity allows for a very short light path, and measurements can be made on a very small gas sample. In turn, this allows the use of low sample rates (50 ml/minute compared with typical rate of 150 ml/minute for conventional infrared analyzers) without compromising accuracy or response time. This reduces entry of moisture into the sample line and reduces the competition for tidal volume that may compromise measurement accuracy in small patients or those with high respiratory rates.

Raman Scatter

Raman scatter is a technique able to measure CO_2, oxygen, nitrogen, nitrous oxide, and halogenated anesthetic agents. Gas is sampled into an analyzing chamber where it is illuminated by a high-intensity monochromatic argon laser beam. When the laser beam hits molecules with interatomic bonds, a fraction of the energy is absorbed and re-emitted at various wavelengths characteristic of the particular molecule that absorbed it. These monitors have a short warmup period, fast response time, require little maintenance, and are very accurate.

Mass Spectrometry

Mass spectrometry is not commonly used for CO_2 measurement in clinical practice because these machines tend to be expensive and bulky. A unique feature of mass spectrometers is that a single unit can be used to measure gas concentration from up to 30 different locations. As such these are most commonly found in large hospitals. Gases are aspirated into a vacuum chamber where an electron beam ionizes and fragments the components of the sample. Ions are then accelerated through a magnetic field that separates them based on their mass-to-charge ratio. Individual detector plates allow for determination of the concentration of each component of the gas mixture. These analyzers typically measure only gases for which they have been preprogrammed to find. Adding the capability to measure new gases may require new hardware and/or software and may be costly. Because these units measure gases in concentrations (as opposed to

infrared analyzers and Raman spectrometers that measure gases as partial pressures), they assume that the sum of the gases they can detect is 100%. If an unmeasured gas is present in significant concentrations, this may result in erroneously high measured CO_2 concentrations.

Indications for Capnography/Capnometry

Indications for performing capnography or capnometry are listed in Box 30.1.

Confirming Correct Endotracheal Tube Placement

Capnography or capnometry may be useful into situations in which it is challenging to determine visually the correct endotracheal tube placement. Repeated upstrokes in the capnogram (repetitive increases in the PCO_2) suggest the presence of respiratory gas (rather than gastrointestinal or atmospheric gas). It is theoretically possible to sample CO_2-containing gas from the stomach, but the values for end-expired partial pressure of CO_2 are likely to be much lower, reduce with time, and not fluctuate in a pattern consistent with respiration. Positioning of the endotracheal tube tip just inside the glottis may produce acceptable end-expired PCO_2 levels and a normal capnogram. Such tube placement risks easy dislodgement and inadequate airway protection. In low perfusion states (e.g. cardiac arrest, shock) verification of correct endotracheal tube placement by capnography is complicated by the presence of abnormally low end-expired PCO_2 and a dampened waveform because little CO_2 is being delivered to the lung for expiration.

Detection of Apnea

If a patient becomes apneic, the capnograph or capnometer typically sounds an alarm when CO_2 stays at zero for a given period of time. Such monitoring is easily achieved if the patient is intubated. The sampling line of a sidestream analyzer can be attached to nasal prongs for the detection of apnea in non-intubated patients.

Box 30.1 Clinical Indications for Capnography

- To ensure correct placement of an endotracheal tube
- To detect apnea
- To monitor adequacy of ventilation
- To monitor pulmonary perfusion during cardiopulmonary resuscitation
- To ensure correct placement of a nasogastric tube
- To detect equipment problems

Monitoring Ventilation

The gas exhaled at the end of expiration should be primarily alveolar gas, and thus end-expired PCO_2 is representative of alveolar PCO_2. Due to the high diffusivity of CO_2, alveolar and arterial PCO_2 equilibrate and end-expired PCO_2 is used to estimate arterial PCO_2. Capnography and capnometry can therefore be used to assess the adequacy of ventilation in spontaneous or mechanically ventilated patients.

Monitoring Pulmonary Perfusion

Large drops in cardiac output (hypovolemic shock or cardiac arrest) result in exponential drops in expired PCO_2. Therefore, very low or precipitously dropping PCO_2 should lead the operator to suspect cardiovascular collapse. Capnography may be useful in monitoring cardiopulmonary resuscitation (CPR) efforts, and a sudden increase in continuously monitored expired PCO_2 during CPR is associated with the return of spontaneous circulation [9]. End-expired PCO_2 has been used to predict the survivability from cardiac arrest. A successful outcome from CPR is more likely if expired PCO_2 levels are greater than 15 mmHg during resuscitation efforts [10].

Correct Nasogastric Tube Placement

Connection of a sidestream analyzer to gastric tubes may provide additional evidence for correct placement. Detection of any significant level of CO_2 should create suspicion of placement in an airway.

Equipment Problems

Capnography may be used to detect malfunctioning or incorrect assembly of breathing circuits, anesthetic machines, and ventilators. Problems such as malfunctioning unidirectional valves, exhausted CO_2 absorbers, and inadequate fresh gas flows may be detected by alterations in the capnogram waveform (see next section).

Interpretation of the Capnogram

The normal capnogram, seen in Figure 30.4, can be divided into four phases (I–IV).

Phase I

Phase I is the normally flat baseline segment of the capnogram. During the first part of this phase, inspiration is occurring. At the very end of this phase, the direction of gas flow reverses as expiration begins. During early expiration, expired gas comes from anatomic dead space. Anatomic dead space has not participated in gas exchange,

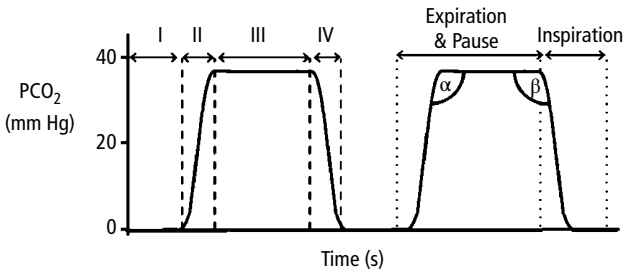


Figure 30.4 The normal capnogram. The capnogram can be divided into four phases, I through IV, and forms two angles, the alpha (α) and beta (β) angles. The phases of the respiratory cycle have been superimposed on the capnogram to the right side of the figure. End-tidal CO_2 is the highest partial pressure of carbon dioxide (PCO_2) value, found at the end of the expiratory plateau, just before the next inspiration begins.

and as such gas from these regions is identical in composition to inspired gas (normally CO_2 free).

Phase II

Phase II is the upstroke of the capnogram waveform. This corresponds to the period of expiration where CO_2-containing alveolar gas begins to be exhaled in a mixture with gas from anatomic dead space (CO_2-free gas). As expiration proceeds the expired gas is composed of rapidly increasing proportions of alveolar gas and the CO_2 levels quickly rise.

Phase III

Phase III is the plateau of the capnogram. During this phase PCO_2 is normally almost constant while alveolar gas, normally of nearly uniform composition, is expired. Expiration actually ends partway through this phase and is usually followed by a pause. During this pause PCO_2 typically remains constant on the capnogram even though no gas is flowing in or out of the patient. This occurs because there is expired alveolar gas remaining stationary within the region of breathing circuit from which the gas is being sampled by the capnograph. This part of the plateau may be cut short by small tidal volumes, high fresh gas flow rates, and/or high gas sampling rates (see abnormal capnograms for more details). The angle between phases II and III of the capnogram is known as the alpha angle and is normally close to 100–110 degrees. End-tidal CO_2 is the highest PCO_2 value, found at the end of the expiratory plateau just before the next inspiration begins.

Phase IV

Phase IV is the rapid downstroke on the capnogram corresponding to inspiration. During this phase fresh, normally CO_2-free gas passes the sampling port as it is inspired into the lungs. The angle between phases III and IV is known as the beta angle and normally close to 90 degrees.

Abnormal Capnograms

Abnormal Phase I

If the capnogram fails to return to baseline during inspiration, then the shape of the waveform should be considered. If the response time of the analyzer is slow, particularly in the face of high respiratory frequencies, then the capnogram may adopt a sine wave formation (Figure 30.5a). This is a relatively common capnographic waveform in cats. Such a waveform has no distinct alveolar plateau, and erroneous values for inspiration and expiration may result (falsely elevated baseline and underestimated peak expired CO_2, respectively).

If the capnogram fails to return to baseline during inspiration and is not the result of a slow response time (i.e. the shape of the waveform is relatively normal), then there must be CO_2 in the inspired gas. Common reasons for this include exhausted CO_2 absorber in a circle system, malfunctioning inspiratory valve in a circle system, or inadequate fresh gas flow in a non-rebreathing system (Figure 30.5b).

Periodic elevations in baseline can occur if external pressure is applied to the patient's chest during the inspiratory period. If this pressure forces gas out of the lungs, a small rise in PCO_2 is registered on the capnogram during what would normally be the baseline period (Figure 30.5c).

Abnormal Phase II

With sidestream capnographs, gas sampling rate affects the shape of the capnogram. Slow sampling rates decrease the slope of phase II, shorten the alveolar plateau, and decrease the slope of phase IV. This delayed equipment response time typically results in increases in both the alpha and beta angle of the capnogram. If the slope of phase II is decreased in the absence of delayed equipment response time, it suggests slow expiration. Such an abnormality is often also associated with a sloped alveolar plateau and increases in alpha angle but normal beta angle (Figure 30.5d). Important causes of slow expiration are patient conditions causing airway narrowing such as bronchoconstriction, or external conditions such as a partially obstructed or kinked endotracheal tube.

Abnormal Phase III

Normally, peak expiratory PCO_2 values are only a few mmHg lower than $PaCO_2$. A normally shaped capnogram

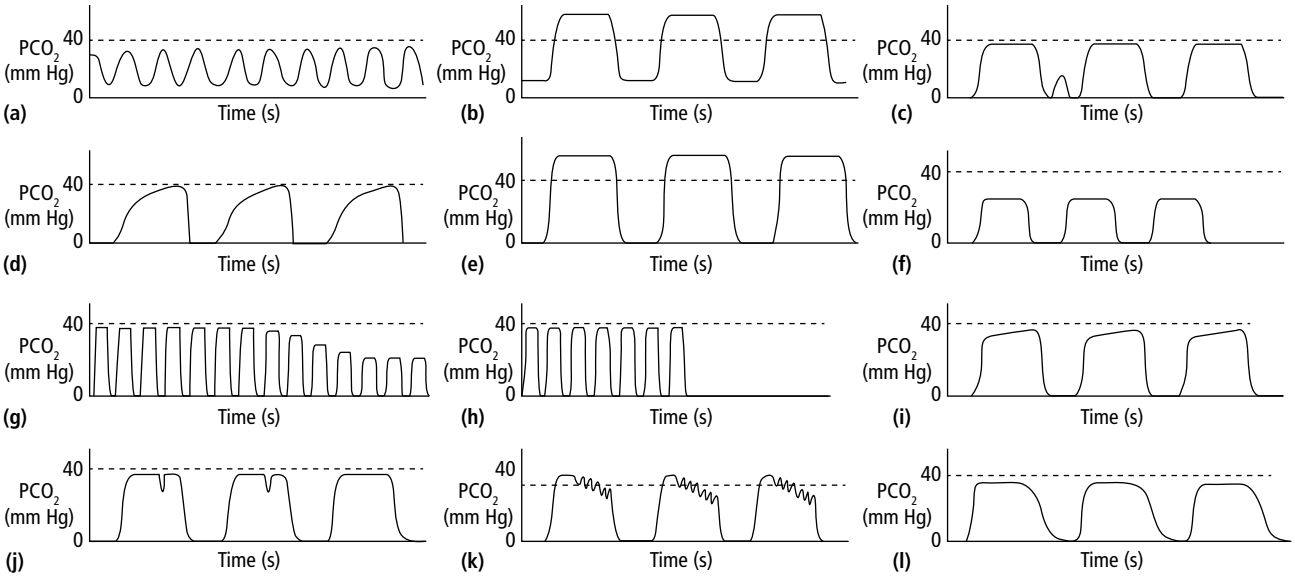


Figure 30.5 Examples of common capnogram waveforms. (a) Sine wave form common with sidestream analysis on small patient with high respiratory rate; (b) rebreathing of CO_2-containing gas; (c) expiratory effort between regular breaths; (d) bronchoconstriction/airway obstruction; (e) hypoventilation; (f) hyperventilation; (g) slow-speed capnogram suggesting reduced pulmonary blood flow; (h) slow-speed capnogram indicating accidental extubation, patient disconnection, or sudden apnea; (i) uneven alveolar emptying; (j) spontaneous inspiratory efforts during mechanical ventilation; (k) cardiogenic oscillations; (l) faulty inspiratory valve. PCO_2, partial pressure of carbon dioxide.

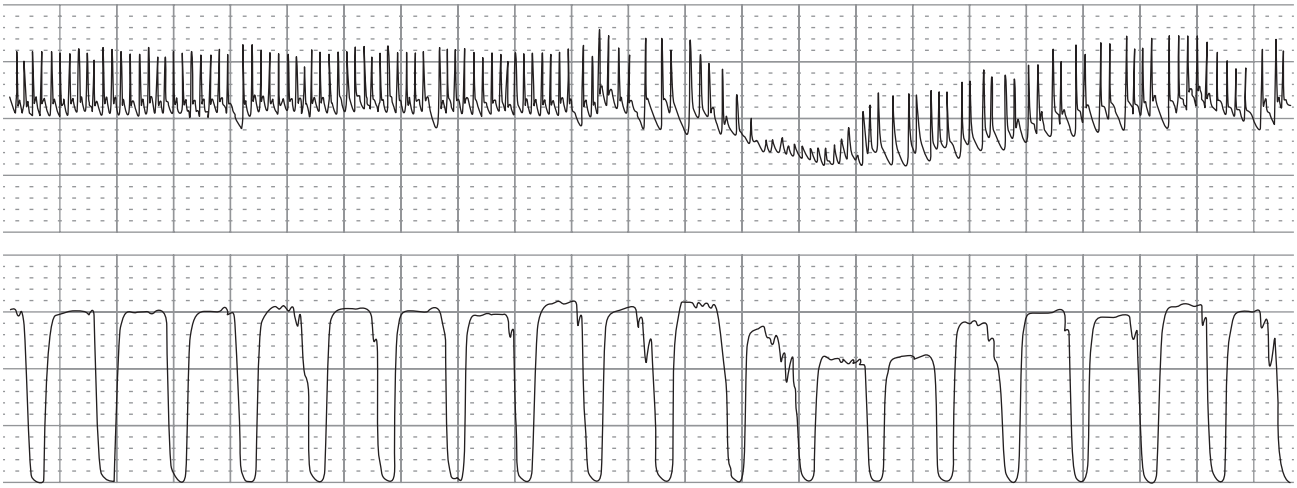


Figure 30.6 The effect of reduced pulmonary blood flow on the capnogram. The upper tracing is arterial blood pressure recorded from the dorsal pedal artery of an anesthetized dog. The lower tracing is a capnogram recorded concurrently from the same patient. Note that the period of hypotension results in a corresponding reduction in the height of the alveolar plateau on the capnogram.

with an elevated alveolar plateau (Figure 30.5e) reflects hypoventilation. This is very common in anesthetized or sedated patients. If the patient is not receiving supplemental oxygen, hypoventilation is a common cause of hypoxemia.

A normally shaped capnogram with lower than normal alveolar plateau (Figure 30.5f) may reflect hyperventilation. If the patient is being mechanically ventilated, ventilator settings should be evaluated. Other causes for lower than normal alveolar plateau include reduced CO_2 production (hypothermia) or reduced delivery of CO_2 to the lungs (low cardiac output). Trends in peak expired CO_2 over time can be useful to demonstrate the effect of reduced pulmonary blood flow on the capnogram. Figure 30.6 displays tracings of a systemic arterial pressure waveform and corresponding capnogram recorded at slow paper speed. A period of hypotension can be seen to correspond to reduced alveolar plateau levels on the capnogram, which returned to previous levels when systemic blood pressure was restored.

The existence of alveolar dead space (ventilated but unperfused lung regions), such as would occur secondary to pulmonary thromboembolism, creates a situation in which peak expired PCO_2 levels are substantially lower than arterial PCO_2 measurements. Unperfused alveoli will not have participated in gas exchange and so contain gas identical in composition to inspired gas, which is normally CO_2 free. During expiration this gas mixes with the gas from perfused alveoli and dilutes the PCO_2 in the expired alveolar gas. When examining a capnogram recorded at slow paper speed, the alveolar plateaus of each wave are typically fairly uniform in a stable patient (see early part of Figure 30.5g). Sudden reductions in pulmonary blood flow, such as occurs with pulmonary thromboembolism, typically result in exponential decreases in the peak alveolar plateau as long as ventilation continues (see progression of Figure 30.5g) as opposed to an abrupt disappearance of the capnogram waveform as would occur with disconnection or accidental extubation of a patient (Figure 30.5h).

An abnormally low alveolar plateau could also be seen if a sidestream analyzer were to have a leak in the gas sampling line and constantly aspirate room air, thus diluting the exhaled gas and creating falsely low PCO_2 measurements.

Conditions that cause filling and emptying of alveoli across the lung to be uneven (regions of ventilation perfusion mismatch) result in a slanted plateau phase of the capnogram (and an increased alpha angle; Figure 30.5i). If exhalation is particularly slow, then peak PCO_2 levels may not be reached before inhalation occurs, and as such end-expired PCO_2 values will be below alveolar and thus arterial PCO_2 values.

The normal alveolar plateau is roughly horizontal (Figure 30.4). Artifactual dips and bumps in the plateau phase may result from pushing on the thorax of an anesthetized patient causing gas to move out and into the lungs. In an animal being mechanically ventilated, spontaneous ventilatory efforts may be interspersed among mechanical breaths and cause dips or clefts in the alveolar plateau (Figure 30.5j). Reasons for these respiratory efforts should be investigated including insufficient anesthetic depth, inadequate mechanical ventilation, hypoxemia, inadequate analgesia, and hyperthermia.

Cardiogenic oscillations are undulations in the capnogram that are synchronous with cardiac contractions (Figure 30.5k). Contraction of the right ventricle and filling of the pulmonary vasculature expels a small volume of gas from the lungs with each beat. In combination with gas aspiration by a sidestream analyzer, oscillations in PCO_2 during the respiratory pause may become evident. This is a common and inconsequential finding in dogs.

Abnormal Phase IV

Normally the capnogram returns briskly to baseline from the alveolar plateau, creating a beta angle of almost 90 degrees as fresh gas is inspired and replaces the CO_2-containing gas at the sampling site. If the slope of this phase is reduced (i.e. the beta angle is increased; Figure 30.5l), then either inspiration is occurring abnormally slowly (not common because it does not take much gas to replace the small volume of exhaled gas at the sampling site) or there is CO_2 in the inspired gas. This could occur with inadequate fresh gas flows on a nonrebreathing circuit or malfunctioning inspiratory valve on a circle system. Box 30.2 describes questions to ask about capnogram interpretation.

Summary

Capnography is a noninvasive method for continuous assessment of ventilation because end-expired CO_2 provides a very good estimate of PCO_2 in most cases. Gas sampling can be either mainstream or sidestream, each of which has advantages and disadvantages. Capnography is most accurate in intubated patients but can also be used in awake, non-intubated patients for continuous noninvasive PCO_2 monitoring. Finally, evaluation of capnographic waveforms can aid in the detection of patient or equipment abnormalities.

Box 30.2 Capnogram Interpretation

1) Are there regular waves of CO_2 providing evidence of ventilation?
2) Does the baseline return to zero (normal) or is there evidence of rebreathing (elevated baseline)?
3) Is the upstroke steep (normal) or is there evidence of slow expiration (slanted upstroke)?
4) Is the alveolar plateau even (normal) or is there evidence of uneven alveolar emptying (slanted plateau) or interruption of the expiratory period by inspiratory efforts (clefts in plateau)?
5) Are end-expired PCO_2 values within an acceptable range, and are they consistent with the patient's respiratory parameters?
6) Is the downstroke steep (normal), or is there evidence of slow inspiration or rebreathing (slanted downstroke)?

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31

Mechanical Ventilation

Kate Hopper and Julie Eveland-Baker

A mechanical ventilator is a machine that performs some or all of the work of breathing. It is used to support respiratory function in patients with respiratory failure. The primary functions of the lung are oxygenation of the arterial blood and removal of carbon dioxide (CO_2) from the venous blood. The ability of the lung to oxygenate the pulmonary capillary blood depends largely on the surface area available for gas exchange and the preservation of the delicate structure of the gas exchange barrier. In contrast, removal of CO_2 primarily depends on the movement of fresh gas into the alveoli, thereby flushing out CO_2-rich gas on exhalation, a process known as ventilation. Patients with respiratory failure can generally be divided into one of two groups: those with oxygenation failure and those with ventilatory failure.

Indications for Mechanical Ventilation

There are three main indications for mechanical ventilation [1]. The first is severe hypoxemia despite oxygen therapy. Severe hypoxemia is indicated by cyanosis, a partial pressure of oxygen in arterial blood (PaO_2) less than 60 mmHg, or an oxygen saturation (SpO_2) less than 90%. Patients with severe hypoxemia have significant lung disease such as pneumonia, acute respiratory distress syndrome, pulmonary edema, or pulmonary contusions.

The second indication for mechanical ventilation is severe hypoventilation despite therapy. Severe hypoventilation is marked by a partial pressure of CO_2 in arterial blood ($PaCO_2$) greater than 60 mmHg, which is discussed in depth in Chapter 26. The $PaCO_2$ is controlled primarily by alveolar minute ventilation. This is the total amount of fresh gas that reaches the alveoli in a minute and is equal to the product of the respiratory rate and alveolar tidal volume. Consequently, causes of severe hypoventilation are diseases that impair the ability of patients to maintain an adequate respiratory rate and/or tidal volume. Such diseases include brain disease, cervical spinal cord disease, peripheral neuropathies, diseases of the neuromuscular junction, myopathies, respiratory muscle fatigue, and airway obstruction. Not all patients that have severe hypoxemia or severe hypoventilation require mechanical ventilation, but these criteria help identify candidates for ventilation. An understanding of the primary disease process and other patient data will help determine the necessity of mechanical ventilation in individual patients.

The third indication for mechanical ventilation is excessive respiratory effort and/or impending fatigue, even if the patient can maintain acceptable blood gas values (i.e. $PaO_2 > 60$ mmHg and $PaCO_2 < 60$ mmHg). Determination of excessive respiratory effort or fatigue is based on clinical judgment, and mechanical ventilation is indicated in these patients to avoid imminent exhaustion and subsequent arrest. These patients most commonly have lung disease.

Ventilator Settings

The ventilator makes gas flow into the lungs by the generation of positive airway pressure in a manner similar to that achieved by squeezing the rebreathing bag on an anesthetic machine when "bagging" a patient. Every ventilator has a variable number of available settings that can be altered to change the nature of the breath delivered. Despite the apparent complexity of many ventilators, there are only a few key settings that are essential to understand to provide effective ventilation. The theory of mechanical ventilation includes many specific terms. Common ventilator terms are defined in Table 31.1. When choosing ventilator settings, the operator must first choose a mode of ventilation and then select machine settings based on general

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Table 31.1 Definitions of common ventilator terms.

Term	Definition	Comments
Mandatory breath	A ventilator breath that is initiated, generated, and ended by the machine	
Assisted breath	A ventilator breath that is initiated (triggered) by the patient but the breath is generated and ended by the machine	Assist control ventilation describes a mode of ventilation with all mandatory breaths with the trigger variable set to allow the patient to increase its own respiratory rate if it chooses
Spontaneous breath	A ventilator breath that is initiated, generated and ended by the patient	Most commonly achieved on a ventilator using continuous positive airway pressure
Supported breath	The breath is initiated and ended by the patient but the machine augments the tidal volume	Most commonly achieved using pressure support ventilation
Compliance	How easily stretched the lung is; defined as the change in volume for a given change in pressure	Healthy lungs have high compliance (large change in volume for small change in pressure); lung disease decreases compliance
Trigger variable or Sensitivity	Determines when the machine will deliver a breath. This variable can be time or a patient-derived variable if the animal is making respiratory efforts	Most commonly set as a change in pressure or a change in flow
Tidal volume	The quantity of gas that moves in or out of the lungs in one breath	Many ventilators measure both inspiratory and expiratory tidal volume
Inspiratory time (I time)	The duration of the inspiratory phase of a ventilator breath	Most commonly set as 0.8–1.0 seconds
Inspiratory to expiratory ratio	The ratio of the duration of inspiration to the duration of expiration	Aim to keep at 1 : 1 or lower
Peak inspired pressure	The highest airway pressure measured during a ventilator breath	

guidelines (described later). These initial settings may be adjusted based on some understanding of the nature of the patient's respiratory disease. Following initiation of mechanical ventilation, the ventilator settings are then titrated as necessary to achieve the respiratory and blood gas goals desired.

Ventilator Breath Types

The three main ventilator breath patterns commonly used in veterinary medicine are assist control ventilation (ACV), synchronized intermittent mandatory ventilation (SIMV), and continuous spontaneous ventilation [1, 2]. Some machines only have one breath pattern option, such as an anesthetic-type ventilator, whereas more modern intensive care unit ventilators usually offer all three options.

The two primary breath patterns used to provide positive pressure ventilation are ACV and SIMV. In ACV, all the breaths delivered are generated entirely by the machine (mandatory breaths). In this mode of ventilation, a minimum respiratory rate is set by the operator (the person adjusting the ventilator settings). If the trigger sensitivity is set appropriately, the patient can increase the respiratory rate, but all breaths delivered will be full ventilator (mandatory) breaths. The size of these ventilator breaths will depend on machine settings entered by the operator. Ventilator breaths can either be pressure- or volume-controlled breaths. In volume-controlled ventilation, the operator presets the desired tidal volume, and the peak inspiratory airway pressure generated depends on the size of the tidal volume chosen and the compliance (how stiff or stretchy the lung is) of the respiratory system. In pressure-controlled ventilation, the operator presets the desired peak airway pressure, and the tidal volume generated depends on the level of airway pressure chosen and the compliance of the respiratory system. ACV provides maximum support of the respiratory system and is used in patients with severe disease or patients with no respiratory drive (those making little or no attempts to breathe on their own).

In SIMV, the operator can set the number of full ventilator (mandatory) breaths delivered, and between these breaths the patient can breathe spontaneously as much or as little as it wishes. The machine tries to synchronize the ventilator breaths with the patient's own respiratory efforts. As this mode combines full ventilator breaths with spontaneous patient breaths, it is generally used for animals that need less than 100% assistance from the ventilator, such as neurologically abnormal animals with a less reliable respiratory drive, or patients with lung disease that are improving and do not need as much support as ACV provides.

The two common options for provision of continuous spontaneous ventilation are continuous positive airway

pressure (CPAP) and pressure support ventilation (PSV). In CPAP, the ventilator delivers no breaths; all breaths are spontaneous breaths, meaning that they are completely patient generated: the respiratory rate, inspiratory time (time spent in the inspiratory phase), and tidal volume are all determined by the patient. CPAP provides just that: a constant level of positive airway pressure (the amount is preset by the operator) throughout the respiratory cycle. It decreases resistance to gas flow and increases respiratory system compliance, enhancing gas exchange and oxygenation. In addition, the machine alarms if the animal does not generate adequate breaths or develops apnea, so it is a useful monitoring mode for weaning patients or for monitoring intubated patients.

As in CPAP, all PSV breaths are spontaneous breaths; the ventilator does not initiate any breaths. In PSV, the tidal volume generated by the patient is augmented by the machine. The amount of support provided during inspiration depends on how much pressure is selected by the operator. This mode reduces the effort required to maintain spontaneous breathing in patients with adequate respiratory drive and inadequate ventilatory strength. PSV can be used alone, in conjunction with CPAP, or to augment the spontaneous breaths during SIMV.

Tidal Volume

The normal tidal volume reported for dogs and cats is in the range of 10–15 ml/kg. Lower tidal volumes (6–8 ml/kg) are generally targeted in animals with significant lung disease. When using volume-controlled ventilation, the operator presets the desired tidal volume. Because overdistension of the lung is extremely dangerous, it is recommended to start with no more than 10 ml/kg as a preset tidal volume; the tidal volume can always be increased if it is determined to be insufficient once the patient is connected to the machine. If pressure-controlled ventilation is used, then the operator presets a desired increase in airway pressure, and once the animal is connected to the machine the tidal volume achieved with the preset pressure is assessed. A tidal volume of around 10–12 ml/kg would be a very acceptable result.

Airway Pressure

Patients with normal lungs such as anesthetic patients or patients with ventilatory failure should only require low peak inspiratory pressures (PIP) in the range of 8–15 cm H_2O, ideally not exceeding 20 cm H_2O. Animals with lung disease have stiff lungs and consequently require higher airway pressures to achieve an adequate tidal volume. Peak inspiratory airway pressures as high as 30–35 cm H_2O may be required in animals with very severe lung disease. When using pressure-controlled ventilation, the desired airway pressure is preset by the operator. Once the animal is connected to the machine, the tidal volume achieved with that airway pressure can be assessed. Initially airway pressures of 8–15 cm H_2O should be targeted; higher airway pressures can be used as necessary.

Trigger Variable

The trigger variable determines when the machine will deliver a breath. If the patient is not making any respiratory efforts the trigger variable is most commonly time. Most modern ventilators allow the patient to trigger machine breaths, allowing ventilation to be better matched to the patient's efforts. The trigger, or sensitivity setting, on the ventilator determines what the machine will recognize as a patient's inspiratory effort. Appropriate trigger sensitivity is essential to ensure the ventilator recognizes genuine respiratory efforts made by the patient. This increases patient comfort and allows the patient to increase its own respiratory rate, if desired. The trigger variable can be too sensitive so that nonrespiratory efforts such as patient handling may initiate breaths; this should be avoided. An airway pressure drop of −2 cm H_2O or a gas flow change of 2 l/minute are reasonable trigger sensitivity settings in medium or larger dogs. In smaller animals, a lower sensitivity is usually more appropriate. Once the animal is connected to the ventilator the trigger setting should be evaluated to ensure that breaths can be initiated by the animal.

Positive End Expiratory Pressure

Positive end expiratory pressure (PEEP) maintains pressure in the breathing circuit during exhalation so that the patient cannot exhale completely. This pressure holds the lung open, improves oxygenating efficiency of the lung, and helps recruit collapsed alveoli. Additionally, it may reduce ventilator-induced lung injury. A small amount of PEEP (2–3 cm H_2O) is commonly set in all ventilator modes to reduce atelectasis. In patients with lung disease, much higher levels of PEEP may be required to improve oxygenating ability. In continuous spontaneous ventilation, CPAP provides PEEP during the exhalation period.

Inspiration to Expiration Ratio/Respiratory Rate

An operator-set respiratory rate is available on most if not all ventilators. A normal respiratory rate of 15–20 breaths/minute with an inspiratory time of around one second is usually selected when the patient is initially established on the machine. This can then be changed as appropriate for the patient. The ratio of the duration of inspiration to

exhalation (I : E ratio) may be preset by the operator, may be a default setting within the machine, or is the byproduct of the respiratory rate and inspiratory time selected by the operator. Ideally an I : E ratio of 1 : 2 is used to ensure the patient fully exhales before the onset of the next breath. As respiratory rates are increased, the expiratory time is sacrificed to "squeeze" in the necessary number of breaths in one minute. High respiratory rates can lead to a situation known as "breath stacking" or intrinsic PEEP because the animal is not able to fully exhale before the start of the next inspiration. To avoid this problem, it is recommended to use an I : E ratio of 1 : 1 or lower. If a higher respiratory rate is required, a shorter inspiratory time will allow maintenance of an acceptable I : E ratio.

Alarms

Before connecting the patient to the ventilator, a review of the ventilator alarm settings is essential. The exact alarms available will vary with machines but changes of particular importance include high- and low-pressure alarms and high- and low-minute ventilation. Alarms should be set with some allowance of patient variability to avoid alarm fatigue but not so extreme that patient harm could occur before the alarm is activated.

Guidelines for Initial Ventilator Settings

As previously stated, the ideal ventilator settings for a given patient cannot be predicted. It is likely that animals with lung disease will need more aggressive settings than those with ventilatory failure. It is necessary to choose some initial settings prior to connecting the patient to the machine. First the type of ventilation must be selected (see ventilator breath types, above). The initial machine settings can be based on guidelines such as those shown in Table 31.2. These settings can then be altered as necessary once the patient is connected to the ventilator. It is best to have the ventilator turned on and confirm it is working appropriately before connecting the patient. It is imperative to have a method by which to perform manual ventilation close at hand at all times during mechanical ventilation in case of equipment malfunction, power failure, or operator error.

Initial Stabilization on the Ventilator

When the patient is first connected to the machine, a fraction of inspired oxygen (FiO_2) of 100% is advised as a safety measure. The FiO_2 can be reduced once the stability of the patient has been verified. After connection to the ventilator, the patient's chest is observed for appropriate movement. If there is insufficient or excessive chest inflation, the ventilator settings should be adjusted appropriately. The chest is then auscultated bilaterally to be sure there is ventilation of both lungs present and all monitoring is evaluated (blood pressure, electrocardiogram (ECG), pulse oximeter, end-tidal CO_2, $ETCO_2$, etc.). Any concerning changes should be addressed immediately. Once the patient appears to be stable, arterial blood gas analysis is ideal for accurate titration of ventilator settings. If an arterial blood gas in not available, venous blood gas and $ETCO_2$ can be used to assess PCO_2, and oxygenation is evaluated by pulse oximetry. Venous PO_2 provides minimal guidance in this scenario because it is not a direct reflection of oxygenation (Chapter 19).

Table 31.2 Suggested initial ventilator settings for patients with normal lungs.

Ventilator parameter	Initial settings: normal lungs
Fraction of inspired oxygen	100%
Tidal volume (volume control)	8–15 ml/kg
Inspiratory pressure (pressure control)	8–15 cm H_2O
Respiratory rate	10–30 breaths/minute
Positive end expiratory pressure	0–5 cm H_2O
Inspiratory time	0.8–1 second
Inspiratory-to-expiratory ratio	1 : 2
Inspiratory trigger	−1 to −2 cm H_2O or 1 to 2 l/minute

Lung Disease

When setting the ventilator up for a patient with lung disease, it is likely that the pressure settings will need to be higher than those needed for animals with normal lungs. This is because pulmonary parenchymal disease reduces the compliance of the lung (i.e. makes the lung stiffer). This means that higher pressures will be needed to achieve the same tidal volume. It is now recognized that excessive distension of the lung is a major cause of ventilator-induced lung injury [2]. For this reason it is recommended to minimize the tidal volume when ventilating patients with significant lung disease. In very severe lung disease, such as the acute respiratory distress syndrome, it may be necessary to target a tidal volume as low as 6–8 ml/kg, whereas in more moderate lung disease tidal volumes no greater

Table 31.3 Suggested initial ventilator settings for patients with lung disease.

Ventilator parameter	Initial settings: lung disease
Fraction of inspired oxygen	100%
Tidal volume (volume control)	6–10 ml/kg
Inspiratory pressure (pressure control)	10–20 cm H_2O
Respiratory rate	10–30 breaths/minute
Positive end expiratory pressure	4–8 cm H_2O
Inspiratory time	0.8–1 second
Inspiratory-to-expiratory ratio	1:1 to 1:2
Inspiratory trigger	−1 to −2 cm H_2O or 1 to 2 l/minute

than 10 ml/kg may be best [3]. Table 31.3 provides some suggested initial ventilator settings for patients with lung disease.

When ventilating patients with lung disease, it can be beneficial to keep them in sternal recumbency for the initial stabilization period. These animals almost always oxygenate better in sternal compared to lateral recumbency.

Goals of Mechanical Ventilation

The goal of ventilator therapy is to maintain acceptable blood gas values with minimally aggressive ventilator settings. The ideal ventilator settings for each individual animal cannot be predicted and are determined through a process of trial and error. The patient should be fully evaluated after every change in ventilator settings, including blood gas analysis if possible. It is advisable to make only one change in ventilator settings at a time to evaluate accurately the effect of each individual change on the patient.

Common blood gas goals of mechanical ventilation are as follows:

- PaO_2 of 80–120 mmHg ($SpO_2 > 95\%$)
- $PaCO_2$ of 35–55 mmHg (35–40 mmHg in patients with brain disease)

PaO_2

If the PaO_2 is higher than the desired range (> 120 mmHg), the first priority is to decrease the FiO_2 until it is less than or equal to 60%. Once FiO_2 is reduced, more emphasis is placed on reducing PEEP and peak airway pressure. If the PaO_2 is lower than desired (< 80 mmHg), the simplest option is to increase the FiO_2. If there is an acute and severe drop in PaO_2, placing the animal on 100% oxygen is appropriate until the issue can be evaluated and more definitive therapy can be provided. Ultimately, it is hoped that manipulation of ventilator settings will increase the oxygenating efficiency of the lung and allow lowering of the FiO_2. Increases in PEEP and peak airway pressure are the main ventilator setting adjustments that may improve pulmonary oxygenating efficiency. An acute hypoxemic episode in a patient that was previously not hypoxemic is a potentially life-threatening complication that requires immediate intervention.

Partial Pressure of Carbon Dioxide in Arterial Blood

The $PaCO_2$ depends primarily on effective alveolar minute ventilation, which in turn is the product of the effective tidal volume and the respiratory rate. If the $PaCO_2$ is higher than the desired range, the respiratory rate or tidal volume or both should be increased and the patient reevaluated. If the $PaCO_2$ is too low, the respiratory rate and/or tidal volume should be decreased. An abrupt increase in $PaCO_2$ in the ventilated patient may indicate a life-threatening complication.

Weaning from Ventilation

Weaning from the ventilator is generally a continuous process of gradually reducing the ventilator settings [1, 2]. Often, as the patient improves, the mode of ventilation may be changed to one that requires the animal to perform a greater proportion of the work of breathing. Such modes include SIMV, pressure support, and CPAP. If animals have been anesthetized for prolonged periods of time, it may take some time for them to wake up (hours to days). It is important to consider reducing the anesthetic dose or changing to shorter acting anesthetic drugs when the animal begins improving and weaning is becoming a possibility.

Prior to disconnection from the machine, the patient must have obtained certain physiologic goals. These include:

- The original disease process is stable or improving
- A normal respiratory drive and initiating its own breaths in a reliable manner
- The patient no longer requires significant ventilator support to achieve acceptable minute ventilation and blood gas values
- Adequate oxygenation (PaO to FiO_2 ratio of at least 150 to 200 is recommended)

Box 31.1 Suggested Standard Contingencies for Clinician Notification for Mechanically Ventilated Patients

- PaO_2 < 80 mmHg or SpO_2 < 95%
- $PaCO_2$ or $ETCO_2$ or $PvCO_2$ > 60 mmHg
- Temperature increase > 1°F
- Persistent tachypnea, panting, fighting ventilator
- Mean arterial pressure < 70 mmHg or systolic arterial pressure < 100 mmHg
- Tachycardia
- Urine output <1 ml/kg/hour

Reasons for an animal not being weaned include cardiovascular instability, requirement of high FiO_2 (> 50%), high PIP (> 25 cm H_2O), and/or high PEEP levels (> 5 cm H_2O).

When a patient has improved sufficiently to consider removing it from the ventilator, it is safest to first test whether the animal can maintain spontaneous breathing while still intubated. During such a spontaneous breathing trial, the patient requires intensive monitoring. The use of CPAP is ideal during a spontaneous breathing trial because the tidal volume, respiratory rate, and $ETCO_2$ can be closely monitored. The development of any of the following may indicate that the trial has failed and that positive pressure ventilation should be reinstituted: hypoxemia, hypercapnia, hyperthermia, tachycardia, hypotension, or tachypnea (Box 31.1). In human medicine, it is now recommended to perform spontaneous breathing trials daily once the patient qualifies according to the physiologic goal criteria previously listed. When the patient can maintain adequate blood gases without anxiety or fatigue, it can be woken up from anesthesia and extubated. As veterinary patients are recovering from anesthesia at the same time as ventilator weaning, signs such as tachycardia, tachypnea and hyperthermia may be acceptable. Clinical judgement is needed to decide if changes seen during the weaning period are anesthesia related or reflect physiologic stress from excessive respiratory demands.

Record Keeping

An essential aspect of management of the ventilated patient is detailed record keeping. This allows evaluation of patient progress and a review of patient responses to changes in ventilator settings. Hourly recording of patient vital signs, blood pressure, pulse oximeter, and $ETCO_2$ readings is ideal. Current ventilator settings should be recorded at the time of every blood gas measurement and every time a change in ventilator settings is made. All ventilator settings and patient values are worth recording, but the most important ones are listed in Box 31.2.

Box 31.2 Minimum Recommended Ventilator Settings and Patient Values to Record

- FiO_2
- Mode of ventilation
- Peak inspiratory pressure
- Tidal volume
- Respiratory rate:
  - Machine set rate
  - Total rate
- Minute ventilation
- I : E ratio
- Positive end expiratory pressure

Ventilator Patient Monitoring

Monitoring of the ventilated patient is very similar to monitoring any anesthetized patient, with an emphasis on real time, continuous monitors where possible in an effort to recognize changes in patient status quickly. Meticulous record keeping of all monitored parameters is essential and an understanding of common complications in the ventilator patient will aid in responding to changes appropriately. While recommended monitoring for the anesthetized patient is reviewed in detail in Chapter 50, a general overview with specific relevance to the mechanically ventilated patient is provided here.

Standard parameters that are monitored in the ventilator patient include continuous ECG, continuous pulse oximetry, continuous $ETCO_2$, and continuous temperature measurement. Of all these parameters, the $ETCO_2$ is generally the best evaluation of life or death in the ventilator patient. A sudden drop of $ETCO_2$ to a very low value is consistent with cardiovascular collapse. The loss of an $ETCO_2$ waveform can also occur if the animal is inadvertently extubated. Like all monitors, the capnograph can malfunction but a sudden change in $ETCO_2$ readings should always prompt immediate patient evaluation.

Most modern ventilators provide a lot of patient data related to the respiratory performance such as tidal volume, respiratory rate, airway pressure, and end expiratory pressure. Calculated parameters of respiratory physiology such as airway resistance and respiratory system compliance also provide valuable information. Continuous ventilator waveforms, such as pressure–time scalars and flow–time scalars, should be displayed at all times if

possible, as these can help identify problems such as a circuit leak, inadequate anesthetic depth, or change in pulmonary function. All ventilator settings and related patient variables provided by the ventilator should be recorded frequently to allow evaluation of trends. It is particularly important to capture these data at the time of arterial blood gas analysis.

Blood pressure measurement is essential as in all anesthetized patients and is further emphasized in the mechanically ventilated patient since positive pressure ventilation can have cardiovascular consequences. In systemically ill ventilator patients, continuous direct arterial blood pressure monitoring is recommended, while in more stable ventilator patients on mild to moderate ventilator settings, intermittent indirect blood pressure monitoring is usually adequate.

Frequent evaluation of anesthetic depth in the anesthetized ventilator patient is important and may need to be performed every five minutes during the initial stabilization period; monitoring frequency often can be reduced to every hour once a stable plane of anesthesia has been reached. Careful documentation of all "ins" and "outs" including blood collected for laboratory analysis, urine output, and any drain production is important since abnormal fluid balance is often a challenge in the ventilator patient. Serial body weights can also be helpful in assessing fluid balance but is not always feasible in these patients.

Blood gas analysis, evaluation of packed cell volume, and plasma total protein, electrolyte, blood glucose and lactate concentrations are all ideally performed frequently in the ventilator patient. The frequency of these tests is determined by individual patient scenario but in animals with rapidly changing disease states it may be necessary to perform blood work every four hours or more frequently. Arterial blood gas analysis to review arterial oxygenation and carbon dioxide is recommended in ventilator patients when possible. PaO_2 is the most accurate measure of oxygenation available and is important for evaluation of lung function and accurate titration of FIO_2. $PaCO_2$ is the ideal measure of ventilatory status (hypoventilation vs. hyperventilation) but in the cardiovascularly stable patient, the venous PCO_2 and $ETCO_2$ provide relevant information. In animals in which arterial catheterization is not possible, intermittent arterial puncture can be a valuable adjunct to pulse oximetry and venous blood gas analysis (Chapter 8). Other blood work such as a complete blood count and biochemistry profile should be performed as indicated by the individual's disease state.

A full physical examination should be performed at least twice daily in the ventilator patient, more frequently in the unstable patient. Particular attention to the oral examination, looking for ulceration or tissue swelling, ocular examination, evaluation of hydration, and surveillance for any evidence of pressure related tissue damage related to recumbency is recommended.

Artificial Airway Care

Appropriate patient care can determine whether mechanical ventilation is successful or unsuccessful. To provide appropriate care, staff training is essential in addition to developing clearly defined patient care protocols and detailed record keeping. Chapter 51 reviews the key points for care of the ventilator patient other than care of the artificial airway, which are reviewed here.

Humidification

The artificial airway should be humidified appropriately to maintain the mucociliary function, keep respiratory secretions from drying out, and reduce the likelihood of airway occlusion from thick secretions [1, 4]. There are two basic options for airway humidification: active heated water devices and passive devices that retain heat and moisture from the exhaled gas and return it to the inhaled gas. There are several types of both humidification options; the most common types are described here. A heat–moisture exchanger (HME or "artificial nose") is an inexpensive passive humidification device that fits between the endotracheal tube the Y-piece of the ventilator circuit. The HME adds dead space to the patient and can increase airway resistance; thus, HMEs are generally avoided in patients under approximately 3 kg. Given their structure, HMEs can easily occlude and for this reason should be avoided in patients with significant airway secretions. HMEs should be changed if they malfunction or become soiled; otherwise they should not be changed more often than every 48 hours [5].

Heated water humidifiers are part of the inspiratory limb of the ventilator circuit and provide excellent gas humidification, but as the gas cools before reaching the artificial airway, condensation or "rain out" can occur, which leads to fluid accumulation in the circuit. This fluid can be a source of ventilator associated pneumonia and in large quantities it can cause occlusion of the circuit. Heated wire circuits provide heating along the length of the inspiratory limb (dual heated wire circuits also provide heating of the expiratory limb) of the circuit to prevent condensation. The optimal form of airway humidification is yet to be determined, as studies to date have not shown a difference in prevention of airway occlusion or pneumonia between HMEs or heated water humidifiers [6].

Artificial Airway

Care of the artificial airway is one of the most important management aspects of the ventilator patient. This care includes securing the airway, maintaining airway patency, and monitoring cuff pressure. Animals can be ventilated via an endotracheal tube (ETT) or a temporary tracheostomy tube. ETTs are usually secured using some form of tie. For long term intubation, it is important to ensure that the ETT tie does not damage tissue or cause tissue edema; repositioning the tube tie every four hours can be of benefit. Gauze or material ETT ties cannot be cleaned effectively, and thus plastic tube ties are ideal. At each evaluation, it is important to ensure the ETT is not causing mucosal injury, or pressure sores in the oral cavity, or commissures of the lips. Temporary tracheostomy tubes are tied or sutured in place. The insertion site should be inspected and cleaned every four hours using aseptic technique and any exposed tissue around the insertion site is covered with sterile gauze or non-adherent dressing material.

Artificial airway suctioning is important for the removal of tracheobronchial and upper airway secretions. Intubated patients cannot cough to clear their airway and accumulated secretions increase airway resistance and can cause airway obstruction. Airway suctioning should be performed every four to six hours, or on an as needed basis. To minimize the risk of infection the inner lumen of any artificial airway and breathing circuit should be handled using aseptic technique.

Whether for a tracheostomy or an ETT, suction technique is essentially the same. For tracheostomy tubes that have an inner cannula, the permanent inner cannula is replaced with a temporary inner cannula during the suctioning procedure. Meanwhile, the permanent inner cannula is placed in a 1:1 dilution of sterile water and 2% hydrogen peroxide for cleaning. Following the suctioning period, the permanent inner cannula is rinsed in sterile water or saline and replaced. The temporary inner cannula is then similarly cleaned and stored aseptically for its next use. For tracheostomy tubes without an inner cannula, the suctioning technique is the same but routine replacement of the tracheostomy tube maybe necessary to allow effective cleaning of accumulated airway secretions.

There are two types of suction catheters available for ventilator patients: open and closed. Open circuit suction catheters require the patient to be disconnected from the ventilator circuit and the catheter is passed down the tracheal tube. The advantage of this approach is that a new sterile catheter can be used each time. A closed-circuit suction catheter is an integrated part of the ventilator circuit and can be used without patient disconnection. This allows maintenance of airway pressure and does not expose the circuit to the outside environment. However, since the catheter remains in the circuit following use, microorganisms may proliferate on the device prior to reintroduction to the airways. Most studies have found no difference in the risk of ventilator associated pneumonia with open versus closed-circuit suction systems. One study found a small benefit associated with closed-circuit suction [7]. The external diameter of the suction catheter size should be less than 50% of the internal diameter of the tracheal tube for most animals. In small patients (< 5 kg), suction of catheter size should be less than 70% of the tracheal tube diameter [8]. As a rough guide, to determine the appropriately sized French catheter, divide the internal tracheal tube size by two and multiply this number by three.

For airway suctioning, the following supplies are required: (i) suction catheter, (ii) suction machine, and (iii) container with sterile water. When an open circuit suction technique is used, two people are required and the person handling the catheter must wear sterile gloves. One person can operate the closed system suction catheter with non-sterile gloves. Prior to suctioning, the patient is placed on 100% oxygen for one to two minutes since airway suctioning can lower the concentration of oxygen in the lungs and can cause small airway collapse. The catheter is passed down the tracheostomy tube or ETT without suction. Airway suctioning can be deep (beyond the tip of the tracheal tube) or shallow. There is limited evidence to show that deep airway suctioning is more effective than shallow suctioning, and it may be associated with more adverse effects. At this time shallow suctioning techniques are recommended [8]. Once the catheter is advanced into the airway, suction is applied for no longer than 10 seconds while the catheter is backed out using a twirling motion. Continuous pulse oximetry during the procedure is recommended and the patient is monitored continuously for desaturation. For open suction techniques, the animal is placed back on 100% oxygen immediately following suctioning. Any secretions removed are evaluated and characterized. Suctioning can be repeated if significant secretions are present and the patient tolerated the procedure.

The practice of instilling sterile saline down the trachea during suctioning to help loosen airway secretions is controversial. Although many feel it makes suctioning more effective there is concern that the saline can wash bacteria from the upper airway or colonized endotracheal tube down into the lung. Saline instillation is not currently recommended.

Summary

Mechanical ventilation can be a lifesaving intervention for patients with severe lung disease or ventilatory failure. Critical care ventilators offer multiple modes and options for ventilation that allow the operator to tailor therapy to the individual. Although complications certainly exist, proper precautions can help minimize risks.

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32

Ventilator Waveform Analysis

Deborah Silverstein and Justina Gerard

Ventilator waveforms can aid the veterinarian and veterinary technician in adjusting ventilator settings, assessing lung function, troubleshooting problems, understanding the interactions between patient and ventilator, reducing the incidence of complications, fine-tuning the ventilator to decrease the patient's work of breathing (WOB), and monitoring patient progress. The three elemental parts to respiratory function monitoring are scalars (also known as waves), loops, and indirect measurements (values that are calculated, such as compliance and resistance). A basic understanding of pulmonary physiology and mechanical ventilation is necessary to interpret ventilatory waveforms. Recent studies in human medicine have also found that the waveform analysis of automated systems is more effective in detecting ineffective triggering and dyssynchrony in mechanically ventilated intensive care unit patients compared with clinician assessment based on visual waveform analysis. It is important that clinicians recognize and properly treat ventilator dyssynchrony to maximize patient comfort, oxygenation, ventilation, and WOB (Video 32.1).

A mechanical ventilator is programmed to deliver a preset pressure, volume, or flow rate; whichever of these is the programmed cause of inspiration is called the *control variable*. The four parameters most useful to examine a mechanical breath include *pressure*, *volume*, *flow*, and *time*. The location of the sensors for detecting these variables depends on the ventilator manufacturer and monitoring system used. For example, most ventilators measure pressure, volume, and flow inside the ventilator, but monitoring devices attached to the end of the endotracheal tube can also be used. In general, the closer the sensor is to the patient, the more accurate is the measurement because the compliance and resistance of the breathing circuit as well as the compressibility of the gas may significantly alter the pressure, volume, and flow values. Using the measured variables, three scalars can be created: flow–time, volume–time, and pressure–time. Time is typically plotted on the horizontal (x) axis and the other parameter is plotted on the vertical (y) axis. Additional graphs, such as flow–volume (F–V) or pressure–volume (P–V) loops plot pressure, volume, and flow against each other without respect to time. The loops provide information regarding changes in lung function.

Five basic flow waveforms are most commonly generated by a mechanical ventilator: a rectangular (or "square") wave, an ascending ramp, a descending ramp, a sinusoidal (or "sine") wave, or an exponential decaying waveform (Figure 32.1). The control variable and ventilator model used determine the possible options because some modes of ventilation only offer certain waveform characteristics. For example, volume control ventilation typically offers several choices of flow patterns, whereas pressure control ventilation commonly uses either a descending ramp or decaying flow pattern only.

Scalars

A mechanical breath can be divided into six stages (Figure 32.2):

1) Beginning of inspiration
2) Inspiration
3) End of inspiration
4) Beginning of expiration
5) Expiration
6) End of expiration.

Which factor initiates inspiration depends on the triggering mechanism (trigger variable) of the ventilator. When using a control mode or in instances where a backup breath is provided by the ventilator, the breath is initiated based on a predetermined amount of lapsed time. When using the assist mode or a synchronized intermittent mandatory ventilation (SIMV) mode, the mechanical breath is

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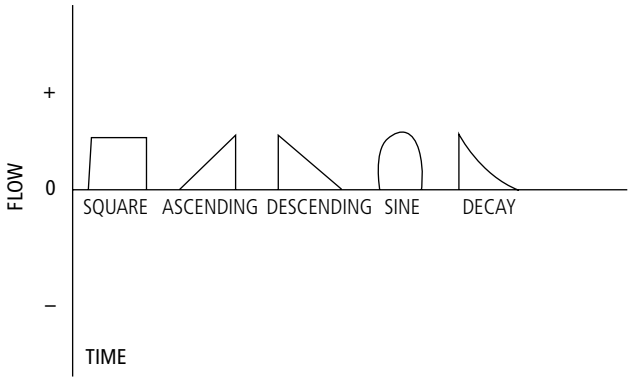


Figure 32.1 The five basic flow waveforms that are most commonly generated by a mechanical ventilator (from left to right): rectangular (or "square") wave, an ascending ramp, a descending ramp, a sinusoidal (or "sine") wave, and an exponential decaying waveform.

initiated by the patient's effort and is referred to as a patient-triggered or patient-initiated breath. During inspiration, the mechanical breath is delivered, and the flow, volume, and pressure of the breath depend on various factors such as airway resistance, lung compliance, type and magnitude of flow, and the delivered volume of each breath. Spontaneous breaths can also be pressure supported to enhance tidal volume.

The attending veterinarian determines the cycling mechanism (cycle variable), or the parameter that is responsible for termination of inspiration. Possible choices include volume cycling, pressure cycling, time cycling, and flow cycling.

Typically, when inspiration ends, the expiratory phase begins. There are specific instances, however, when the expiration valve does not open even though inspiratory gas flow has stopped (e.g. when inspiratory pause or inflation hold controls are activated). The delivered volume is retained within the lungs to obtain static or plateau airway pressures; delayed opening of the expiratory valve then allows expiration to occur.

Expiration is a passive phenomenon and the properties of expiration depend on the resistance of the animal's airways and the artificial airway, as well as pulmonary compliance. The end of expiration is heralded by the beginning of the next breath.

Scalars in Different Modes of Ventilation

The scalar waveforms vary in their appearance depending on the mode of ventilation used. Sample scalars for the five ventilation modes most commonly used (volume assist control, pressure assist control, SIMV, SIMV with pressure support, and continuous positive airway pressure), are described below.

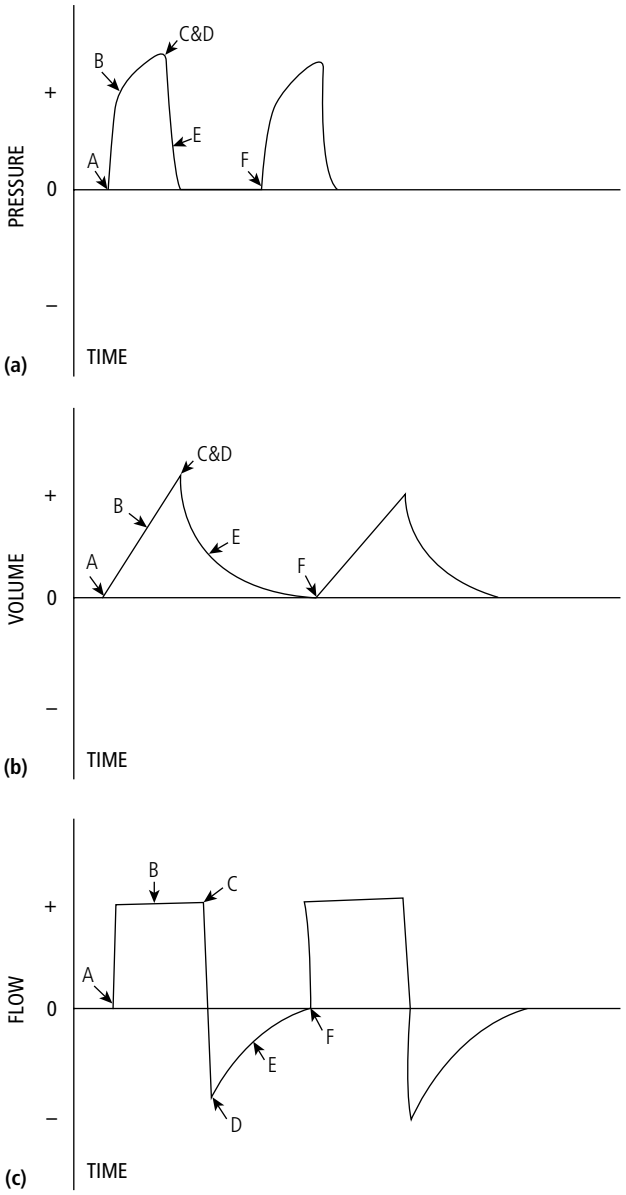


Figure 32.2 (a) The six stages of a mechanical breath as seen in this pressure–time scalar include: A. Beginning of inspiration; B. Inspiration; C. End of inspiration; D. Beginning of expiration; E. Expiration; F. End of expiration. (b) The six stages of a mechanical breath as seen in this volume–time scalar include A. Beginning of inspiration; B. Inspiration; C. End of inspiration; D. Beginning of expiration; E. Expiration; F. End of expiration. (c) The six stages of a mechanical breath as seen in this flow–time scalar include A. Beginning of inspiration; B. Inspiration; C. End of inspiration; D. Beginning of expiration; E. Expiration; F. End of expiration.

Volume Assist Control Mode

Some important curve characteristics to note in Figure 32.3 (volume assist control ventilation) include:

- The inspiratory time and expiratory time on these graphs correspond to termination of inspiration and expiration, respectively.

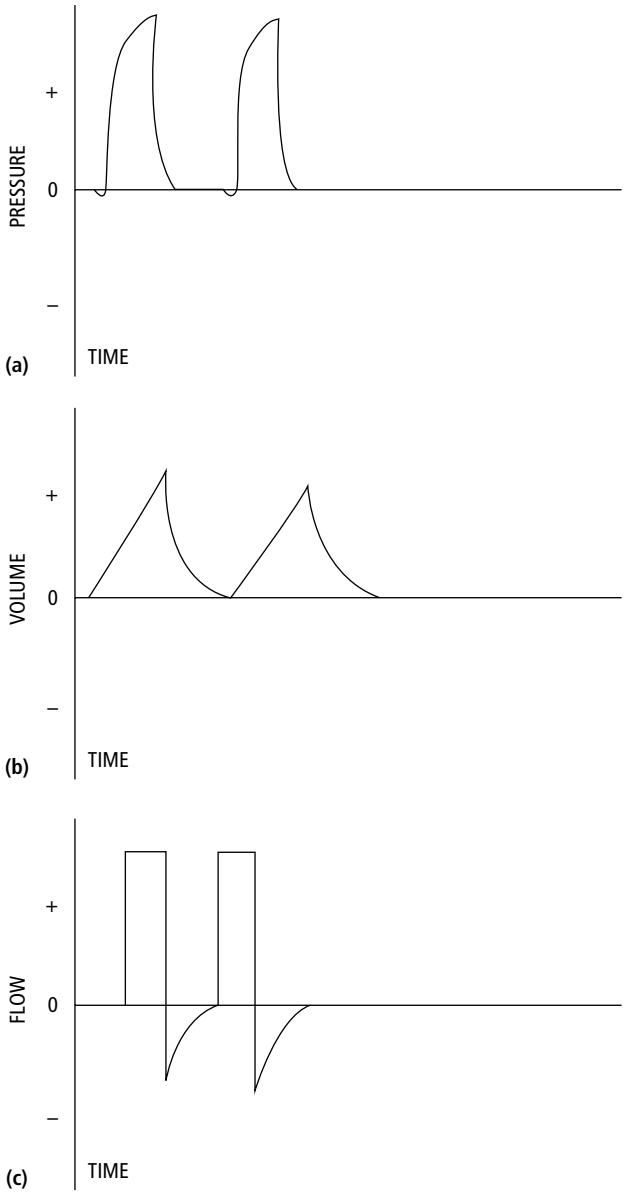


Figure 32.3 (a) A pressure–time scalar from a normal patient receiving volume assist-control mechanical ventilation. (b) A volume–time scalar from a normal patient receiving volume assist control mechanical ventilation. (c) A flow–time scalar from a normal patient receiving volume assist-control mechanical ventilation.

- A negative tracing below baseline is observed during expiration for the flow–time curve. This is because the flow transducer measures inspiratory flow as a positive deflection and expiratory flow as a negative deflection.
- A square flow tracing represents a constant flow pattern.
- Because flow is constant, the delivery of volume is rectilinear.
- When the patient triggers an assisted breath, there is a small negative deflection on the pressure–time graph. This bulge occurs as the patient starts to inhale, thus generating negative pressure and flow that is sensed by the ventilator. The initial rise in pressure corresponds to the pressure necessary to overcome airway resistance. After this point, any increase in pressure depends on pulmonary compliance and volume delivered. As the end of the tidal volume is delivered, the pressure wave has flattened because the lungs are almost full.
- Flow delivery ceases at the end of inspiration because all the tidal volume has been delivered and the peak inspiratory pressure (PIP) has been achieved.
- The dynamic pressure–time scalar has been used to determine stress index, which is the slope of the pressure scalar and may indicate under-recruitment (convex curve) or overdistention (concave curve).

Synchronized Intermittent Mandatory Ventilation Mode

It is important to note the following in the SIMV mode of ventilation (Figure 32.4):

- The inspiratory flow tracing during spontaneous breaths is on the positive side of the graph and is located in the space between mechanical breaths. During the expiratory phase, the flow is depicted below the baseline.
- The inspiratory pressure during spontaneous breaths is traced on the negative side of the baseline (unlike flow and volume) and expiration is shown on the positive side of the tracing.
- The stages of each breath occur in the same order on all three scalars.

Synchronized Intermittent Mandatory Ventilation with Pressure Support

Some important concepts to note in SIMV with pressure support (Figure 32.5) include:

- On the flow–time scalar, the pressure-supported breath delivers a decreasing flow and terminates inspiration when the flow decreases to a certain level (also known as flow cycling).
- A set pressure is maintained throughout the inspiratory phase when a pressure-supported breath is delivered. There is a decrease in pressure down to baseline during the expiratory phase. All breaths are patient triggered in this example, as demonstrated by the small negative deflection at the beginning of inspiration on the pressure–time scalar.

Synchronized Intermittent Mandatory Ventilation with Pressure-Controlled Ventilation and Continuous Positive End Expiratory Pressure Mode

Important facts to note in Figure 32.6 (only a pressure–time scalar is shown because the flow-time and volume-time scalars would be identical to those in Figure 32.5) are:

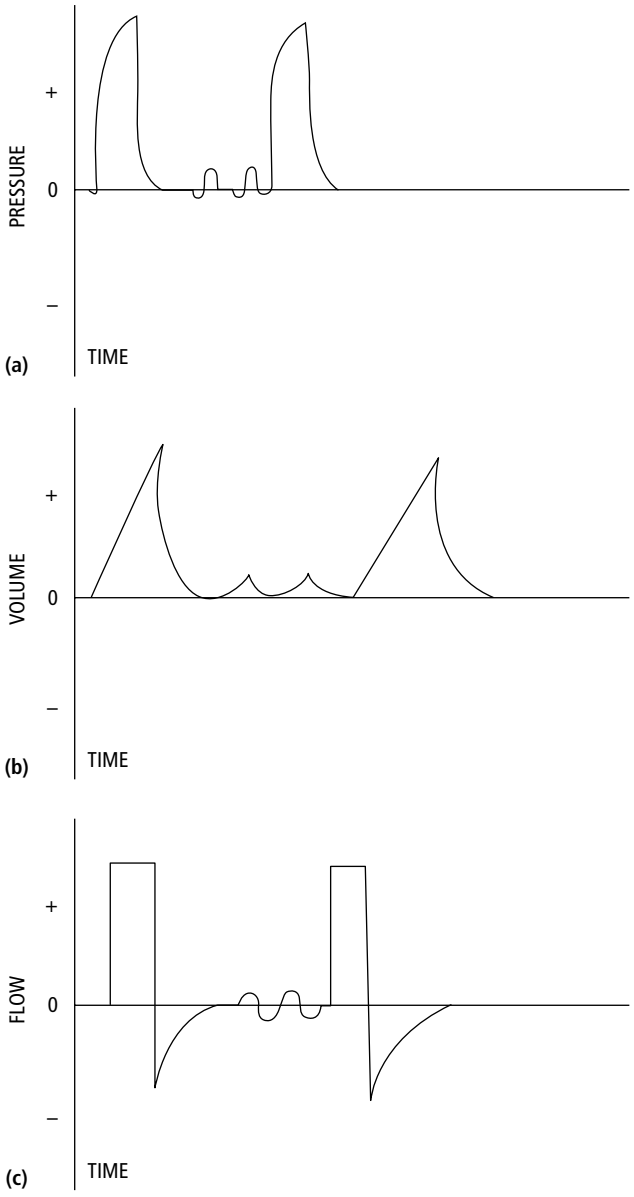


Figure 32.4 (a) A pressure–time scalar from a normal patient receiving synchronized intermittent mandatory mechanical ventilation. The initial mandatory breath is followed by two spontaneous unsupported breaths and another mandatory breath. (b) A volume–time scalar from a normal patient receiving synchronized intermittent mandatory mechanical ventilation. The initial mandatory breath is followed by two spontaneous unsupported breaths and another mandatory breath. (c) A flow–time scalar from a normal patient receiving synchronized intermittent mandatory mechanical ventilation. The initial mandatory breath is followed by two spontaneous unsupported breaths and another mandatory breath.

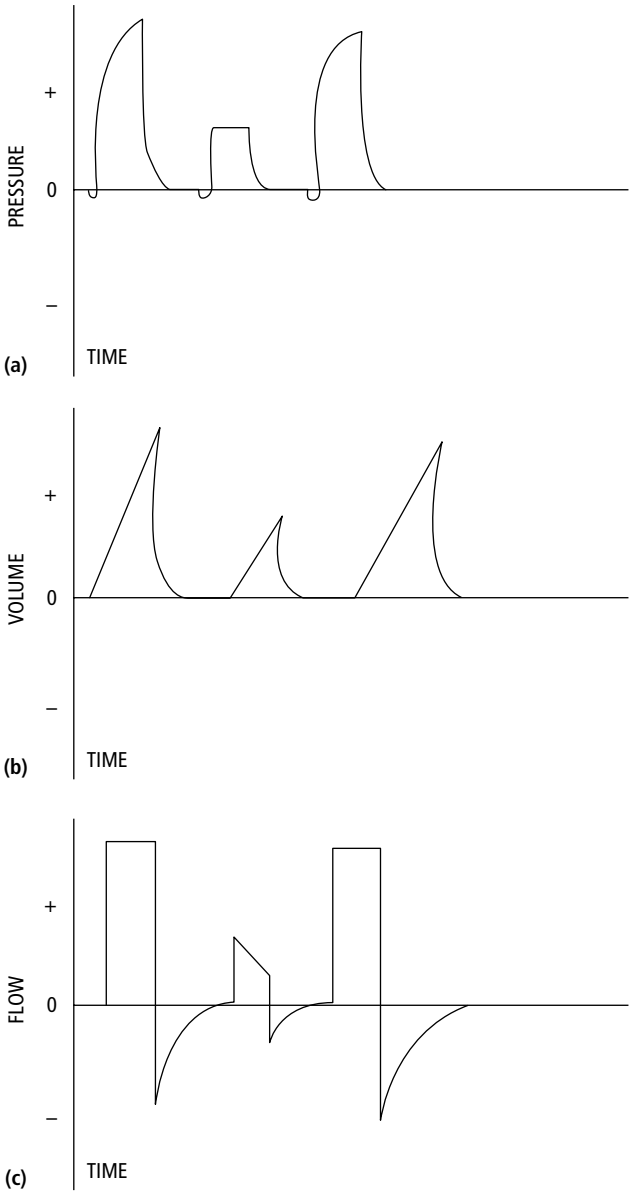


Figure 32.5 (a) A pressure–time scalar from a normal patient receiving synchronized intermittent mandatory mechanical ventilation with pressure support. The figure depicts a mandatory breath followed by a spontaneous breath with pressure support and another mandatory breath. (b) A volume–time scalar from a normal patient receiving synchronized intermittent mandatory mechanical ventilation with pressure support. The figure depicts a mandatory breath followed by a spontaneous breath with pressure support and another mandatory breath. (c) A flow–time scalar from a normal patient receiving synchronized intermittent mandatory mechanical ventilation with pressure support. The figure depicts a mandatory breath followed by a spontaneous breath with pressure support and another mandatory breath.

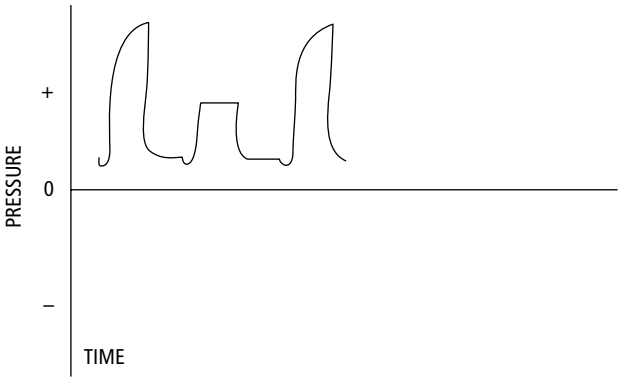


Figure 32.6 A pressure–time scalar from a normal patient receiving synchronized intermittent mandatory mechanical ventilation with pressure support and positive end expiratory pressure (PEEP). The figure depicts a mandatory breath followed by a spontaneous breath with pressure support and another mandatory breath. PEEP is evident by the elevated baseline pressure.

- When positive end expiratory pressure (PEEP) is used, the baseline on the pressure–time graph will be equal to the PEEP value. Upon initiating or increasing PEEP, PIP will also increase.
- The airway pressure decreases to the new baseline PEEP at the end of expiration.
- Although not shown, the baseline does not generally change on the flow–time or volume–time scalars when PEEP is begun.

Pressure-Controlled Ventilation

Some points to note in pressure-controlled ventilation (Figure 32.7) are:

- The ventilator terminates inspiration when a preset time has elapsed. The pressure remains at the set pressure throughout the inspiratory time.
- The flow decreases to zero on the flow–time scalar before the end of inspiration (before expiration begins).

Diagnosing and Interpreting Auto-Positive End Expiratory Pressure from Scalar Graphs

Auto-PEEP (also known as intrinsic PEEP or air trapping) often occurs in patients requiring high respiratory rates, high minute volumes, or PEEP settings greater than 10 cm H_2O. In these situations, inspiration begins before a complete expiration has occurred, leading to air trapping within the small airways and increased patient effort required for a patient-initiated breath. Additionally, auto-PEEP leads to flattening of the diaphragm, which decreases the efficiency of diaphragmatic contractions during inspiration. During expiration on the volume-time scalar, the waveform approaches baseline but then starts upward again before reaching baseline (Figure 32.8a). When examining the flow–time graph, there is an abrupt movement up to baseline at the end of expiration, with an immediate increase in inspiratory flow for the next breath before expiration is completed and reaches baseline (Figure 32.8b).

The presence of auto-PEEP may indicate an airway obstruction. Possible treatment strategies include bronchodilator therapy, placement of a larger endotracheal tube, increasing the inspiratory flow rate (to minimize the ratio of inspiratory to expiratory time), decreasing the tidal

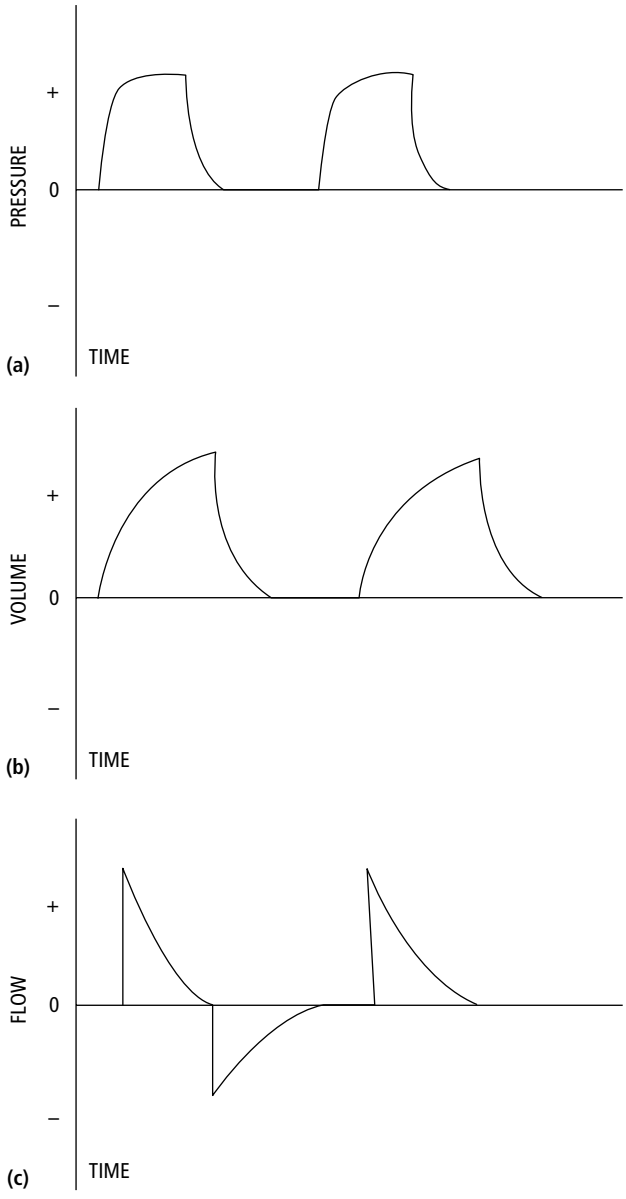


Figure 32.7 (a) A pressure–time scalar from a normal patient receiving pressure-controlled mechanical ventilation. (b) A volume–time scalar from a normal patient receiving pressure-controlled mechanical ventilation. (c) A flow–time scalar from a normal patient receiving pressure-controlled mechanical ventilation. The expiratory phase of the second breath is not shown here.

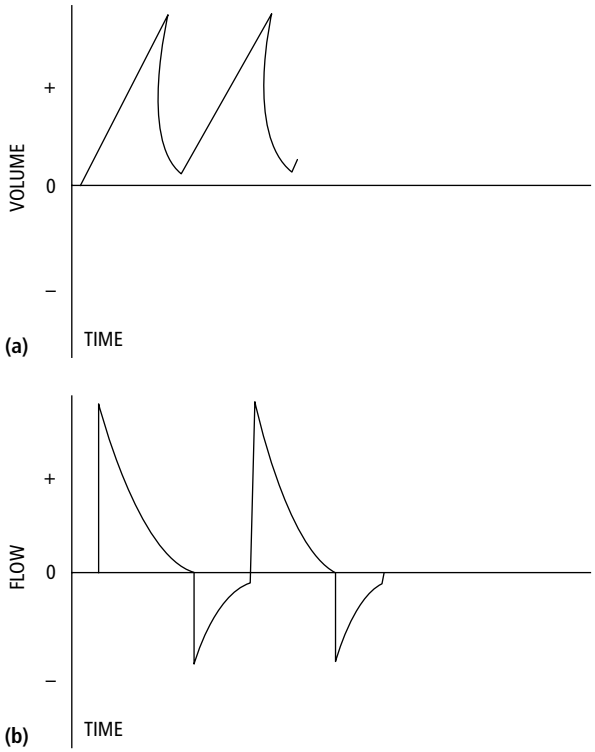


Figure 32.8 (a) A volume–time scalar showing auto-positive end expiratory pressure (auto-PEEP) in an animal with lung disease receiving mechanical ventilation. Note that the waveform approaches baseline but immediately starts upward again before returning to zero. (b) A flow–time scalar showing auto-PEEP in an animal with lung disease receiving mechanical ventilation. Note that there is an abrupt movement up to baseline at the end of expiration, with an immediate increase in inspiratory flow for the next breath before expiration is completed and reaches baseline.

volume, or applying extrinsic PEEP. Alternatively, the respiratory rate may be too high (in which case the clinician can decrease the rate while increasing the tidal volume per breath). Sometimes a different mode of ventilation is beneficial in these animals if the troubleshooting methods previously mentioned are not effective.

Occasionally, auto-PEEP is caused by airway collapse, as in patients with chronic obstructive airway disease. In these cases, increasing PEEP may help to prop the airways open and decrease air trapping. Protocol 32.1 is a rapid reference for initial pressure–time and flow–time scalar evaluation.

Pressure–Volume and Flow–Volume Loops

P–V and F–V loops are best studied after becoming familiar with the pressure, flow, and volume scalars previously described. Similar to the scalar graphs, both the numeric values and waveform graphs reveal information about the patient. A loop is really just inspiratory and expiratory curves that are connected. These loops allow the clinician to analyze the inspiratory and expiratory phases of each breath using either F–V or P–V tracings. These loops can prove challenging initially because there is no unit of time involved; an entire breath is followed throughout the loop without any reference to the passage of time. It is helpful for clinicians to make changes in ventilator and nonventilator treatment

Protocol 32.1 Rapid Reference for Initial Pressure–Time and Flow–Time Scalar Evaluation

Procedure

First look at the pressure–time scalar:

1) Determine the continuous positive airway pressure or PEEP level so baseline pressure is known.
2) Determine whether the patient is triggering any breaths and identify this on the tracings.
3) Determine the intended shape of the pressure wave (e.g. a flat top is pressure controlled; a "shark fin"– shaped top is volume controlled).

Now look at the flow-time scalar:

4) What is the flow pattern? For example, a square-shaped tracing indicates a volume-controlled breath, but a decelerating shape can be present with any mode.
5) Is the patient air trapping? If expiratory flow does not return to baseline before the next breath begins, air trapping (auto-PEEP) is present.
6) Is the patient triggering breaths? Is the breath controlled, pressure supported, or a compulsory breath during SIMV? For example, the pressure-supported breath has a decelerating flow pattern and has a flat-topped airway pressure wave, and the synchronized breath has a triangular-shaped pressure wave.
7) Is the patient triggering a controlled or supported breath? The main difference between these is the duration of the breath because the patient determines the inspiratory time with supported breaths. In other words, the inspiratory time of a supported breath is variable in duration, but the inspiratory time of a controlled breath is uniform and unchanging since it is based on the ventilator settings.
8) Is the patient synchronizing with the ventilator? If the number of triggering episodes is greater than the number of breaths delivered, patient–ventilator dyssynchrony is present. If the peak flow rate of the ventilator is inadequate, the inspiratory flow will be "scooped" inward and the patient may appear to be fighting the ventilator.

variables incrementally so the ventilator waveforms can be used to guide fine-tuning of the ventilator settings and to assess patient changes.

When examining a loop on the monitor screen, the scale of the axes must be set so that the loop images are displayed as large as possible for easy viewing. For example, the slope of the P–V loop, which represents dynamic compliance, is normally around 45 degrees. The clinician can readily observe the slope at a glance to determine whether the dynamic compliance is abnormal.

Unfortunately, there is no convention for how the inspiratory and expiratory limbs of the F–V loops are oriented with respect to the horizontal axis. Traditionally, the F–V loop is displayed with the inspiratory curve below the horizontal axis and the expiratory curve above the axis; however, this is commonly reversed. This chapter depicts the inspiratory curve above the horizontal axis. There is also no consensus as to how the axes should be oriented, but flow is depicted on the vertical scale in this chapter to maintain consistency with the scalars previously discussed.

The change in volume in relation to the change in intrapleural pressure is commonly referred to as *compliance*. Figure 32.9 shows how varying degrees of compliance can affect the inspiratory limb of a P–V curve. The largest volume change for a given pressure is obtained at the steepest portion of the curve, typically in the middle (Figure 32.10). Spontaneous tidal breathing commonly occurs in this region of the curve, which allows for spontaneous ventilation at the most efficient portion of the curve (where the least pressure change is needed for the greatest volume gain). Pulmonary disease may significantly change the baseline volume or pressure prior to each breath (e.g. due to atelectasis or air trapping) and subsequently decrease ventilatory efficiency by forcing the lung into a less efficient portion of the P–V curve. This leads to a decrease in dynamic and static respiratory compliance and distortion (especially of the P–V loop). Table 32.1 lists some common causes of decreased compliance.

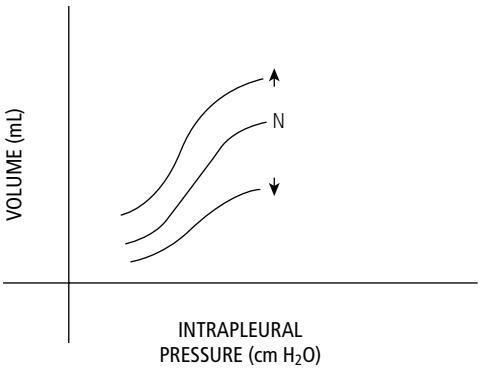


Figure 32.9 A pressure–volume graph from a normal animal receiving mechanical ventilation showing how varying degrees of compliance can affect the inspiratory limb of a pressure-volume curve. N, normal; up arrow, increased compliance; down arrow, decreased compliance.

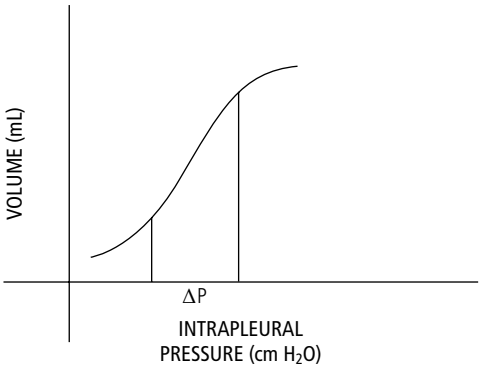


Figure 32.10 A pressure–volume graph from a normal animal receiving mechanical ventilation showing how the largest volume change for a given change in pressure is obtained at the steepest portion of the curve, typically in the middle.

Table 32.1 Common causes of increased pulmonary resistance and decreased pulmonary compliance in mechanically ventilated animals.

Increased resistance (Video 32.3)	Decreased compliance (Video 32.2)
Bronchospasm	Pleural space disease
Airway secretions	Pulmonary parenchymal disease
Small-diameter endotracheal tube	Single-lung intubation
Mucosal edema of airways	Abdominal distention, disease or deformity
	Chest wall disease or deformity

Pressure–Volume Loops

The pressure (horizontal axis) and volume (vertical axis) are typically plotted against each other, and a loop as in Figure 32.11 is generated (A represents the inspiratory limb and B represents the expiratory limb of the breath). Conceptual renderings of the loop are often elliptical or "football shaped," but the loops are not typically so symmetrical. When a ventilator delivers a "control breath," it is initiated in the lower-left corner of the graph and follows the counterclockwise path as indicated by the arrows, eventually returning to the starting point. The upper-right corner represents the end of inspiration and the beginning of expiration. This is the point of maximal pressure and volume, and it represents the dynamic compliance of the respiratory system for that breath. Remember that the loop will not begin at zero pressure if PEEP is applied to the patient.

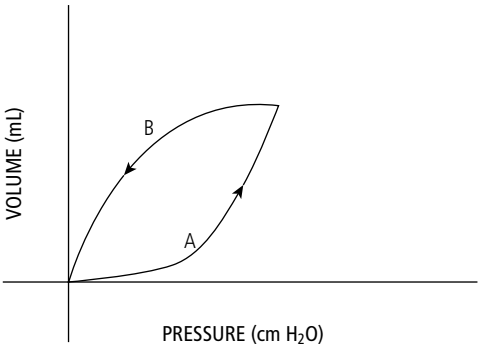


Figure 32.11 A pressure–volume loop from a normal animal receiving mechanical ventilation in which the pressure (horizontal axis) and volume (vertical axis) are plotted against each other, and a loop therefore generated. A represents the inspiratory limb and B represents the expiratory limb of the breath.

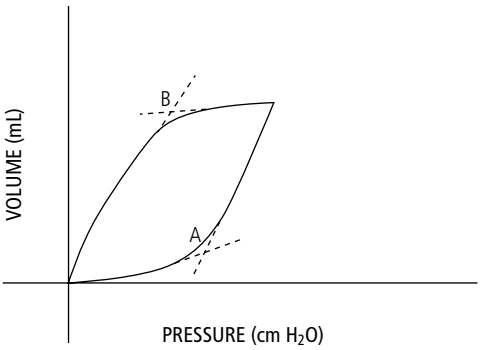


Figure 32.12 A pressure–volume loop from a normal animal receiving mechanical ventilation demonstrating the two inflection points. One point is found during inspiration (a) and the other during expiration (b). Point A is commonly called the lower inflection point and point B, the upper inflection point. If the inflection points are difficult to discern, a straight line can be drawn along the straight portions of the inspiratory and expiratory limbs of the loop, and the point of intersection for the two drawn lines will estimate the inflection point (see dashed lines).

The P–V loop is commonly used to evaluate changes in respiratory compliance. The point of change in the slope of a line is called the *inflection point*. As seen in Figure 32.12, the loop has two inflection points, one during inspiration and one during expiration. Point "A" is commonly called the lower inflection point and point "B" the upper inflection point. If the inflection points are difficult to discern, a straight line can be drawn along the straight portions of the inspiratory and expiratory limbs of the loop, and the point of intersection for the two drawn lines will estimate the inflection point (see dashed lines). When examining a static P–V loop, the inflection points are believed to represent a sudden change in alveolar recruitment during inspiration and de-recruitment during expiration. The dynamic P–V loop includes the effect of resistance to flow; the volume increase lags behind the pressure increase and causes a gap between the inspiratory and expiratory curves of the P–V loop. Subsequently, the inflection points obtained from a dynamic P–V loop are difficult to use when setting PEEP or the upper pressure limit.

When an animal triggers a breath spontaneously (in contrast to a controlled breath, which is triggered solely by the timing mechanism of the ventilator), there will be a small bulge on the negative side of the pressure axis (Figure 32.13) This bulge occurs as the patient starts to inhale, thus generating negative pressure and flow. Spontaneous breaths trace a loop in a clockwise direction (this is discussed further later). If the ventilator senses the patient's effort and begins a machine breath, the line shifts rightward into the positive side of the pressure axis and loops counterclockwise.

Conventionally, compliance is assessed by tracing a line from the beginning to the end of inspiration, with a 45-degree angle created to the horizontal axis in a normal animal. An increase in compliance results in a shift to the left of the 45-degree line (Figure 32.14) because less pressure is required to produce a given change in lung volume, whereas a decrease in compliance causes a rightward shift

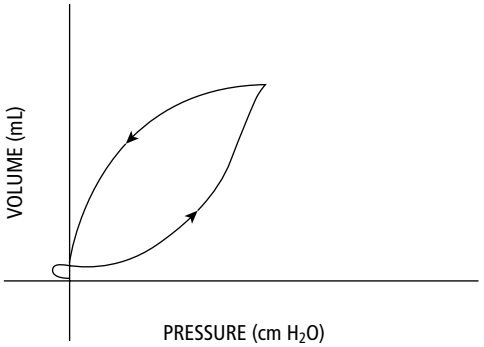


Figure 32.13 A pressure–volume loop from a normal animal that triggers the delivery of an assisted positive pressure ventilation breath (in contrast to a controlled breath that is triggered solely by the timing mechanism of the ventilator). Note the small bulge on the negative side of the pressure axis due to the patient's active inspiratory effort.

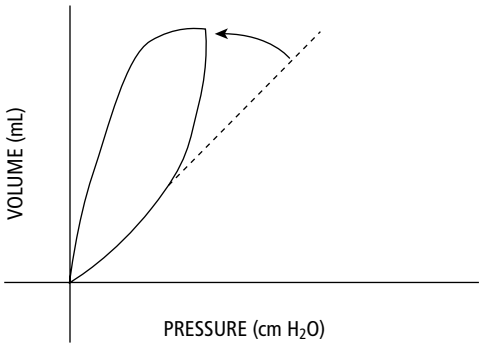


Figure 32.14 A pressure–volume loop from an animal receiving mechanical ventilation with increased compliance. Note how the loop is left-shifted from the 45-degree dotted line.

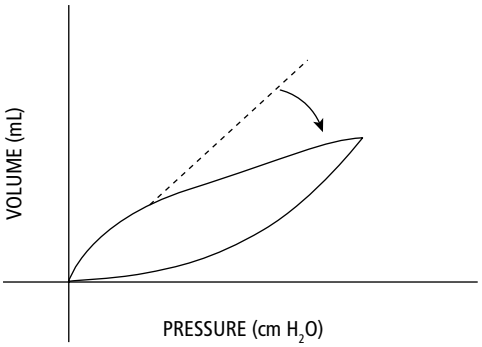


Figure 32.15 A pressure–volume loop from a mechanically ventilated animal with decreased compliance. Note how the loop is right-shifted from the 45-degree dotted line.

in the loop (Figure 32.15) because more pressure is required to produce a given change in lung volume. Refer to Table 32.1 for some common causes of decreased respiratory system compliance and Video 32.2 for an example of how to use waveforms to improve compliance in a dog with aspiration pneumonia.

An increase in the width of the loop indicates an increased resistance in the respiratory system. Subsequently, the area of the P–V loop and its horizontal distance increases (Figure 32.16). These changes are the result of *hysteresis*, a lag in the change in volume compared with the rate of change in pressure that results from resistance to deformation (elasticity) and resistance of the airways. The rightward shift of the loop indicates that the resistance is creating a decreased compliance effect. It is often easier for the clinician to use the F–V loop to evaluate changes in airway resistance because the changes to the P–V loop can be subtle. However, superimposition of two loops on each other may prove helpful in determining changes in the P–V loop. Using both the F-V and P–V loops is the best way to gauge overall resistance and its changes. Table 32.1 lists some common causes of increased airway resistance and Video 32.3 shows an example of how waveforms can be used to diagnose increased resistance and monitor for treatment response in a cat with asthma.

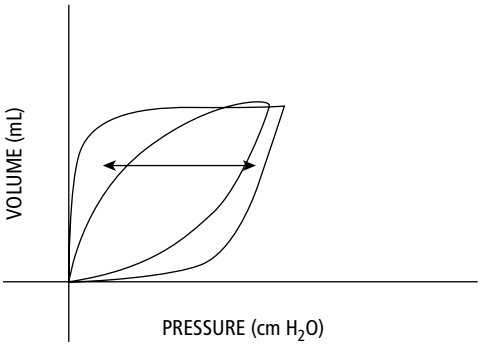


Figure 32.16 Superimposed pressure–volume loops from a mechanically ventilated normal animal (center) and a mechanically ventilated animal with increased airway resistance (outer loop). Note how the increase in airway resistance causes the loop to widen, leading to an increase in the area of the pressure–volume loop and an increase in the horizontal distance (arrows).

The WOB describes the amount of pressure required to move a specific volume of gas. A decrease in compliance or a decrease in functional residual capacity increases the WOB. There are several ways to measure the WOB, but this information focuses on the ventilatory graphics, termed *mechanical* WOB. The patient, ventilator, or both are able to do the WOB. The components of the WOB are shown in Figure 32.17. The WOB to overcome airway resistance is labeled (A) and that required to overcome the elastic nature of the lung is labeled (B). The combination of A and B represent the total mechanical work done during the breath. The WOB equals the area under the changing pressure curve as the volume increases from zero to its peak at the end of inspiration. Most ventilator graphics only display the mechanical work as measured at the endotracheal tube connector, and therefore they do not reveal ventilatory efforts made by the patient. The WOB performed by the patient during the mechanical breaths can be indirectly measured by plotting esophageal pressures because esophageal pressures are a surrogate for measuring intrapleural pressure and intrapleural pressure changes as a result of patient work.

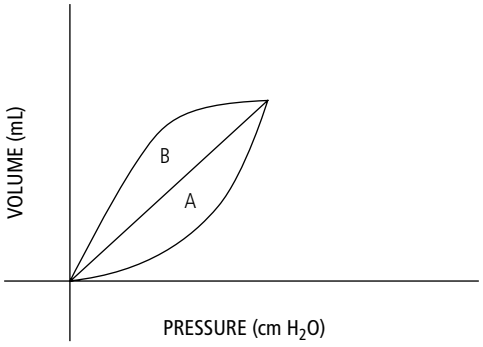


Figure 32.17 A pressure–volume loop from a mechanically ventilated normal animal with the components that determine the mechanical work of breathing (WOB) separated. The WOB to overcome airway resistance is labeled (A), and that required to overcome the elastic nature of the lung is labeled (B). The combination of A and B represent the total mechanical work done during the breath. The WOB equals the area under the changing pressure curve as the volume increases from zero to its peak at the end of inspiration. Most ventilator graphics only display the mechanical work as measured at the endotracheal tube connector, and therefore they do not reveal ventilatory efforts made by the patient. The WOB performed by the patient during the mechanical breaths can be indirectly measured by plotting esophageal pressures.

Flow–Volume Loops

The vertical axis of an F–V loop represents flow rate (liters per minute or per second), and the horizontal axis represents volume (milliliters or liters); however, this is not standardized, so it is important to read the axis labels. The inspiratory portion of the F–V loop is above the horizontal axis, and the expiratory portion is below this line in this chapter but recall that this may be reversed in some graphic displays or references. In Figure 32.18, point A represents the start of inspiration; point B represents the start of expiration. The shape of the F–V curve can be altered by patient changes, ventilator settings, circuit conditions, and the way the ventilator generates and delivers a breath. The transition from inspiration to expiration and back to inspiration is seen where the loop crosses the horizontal axis and the flow rate is transiently zero. The inspiratory curve shape reflects the flow pattern of the ventilator (see note later). The lowest point below the *x* axis depicts the peak expiratory flow rate (PEFR) during passive expiration (point C in Figure 32.18). Anything that leads to obstruction of the airways or endotracheal tube influences the shape of this curve, as discussed later.

Flow–Volume Loops when Flow Delivery Is Constant

When the ventilator uses constant flow to deliver a breath, a square waveform pattern is displayed (Figure 32.19a). This results in a constant volume delivery, since flow is volume per unit of time. Although a descending flow pattern may be used more commonly, the square waveform can be more helpful for detecting abnormalities in the P–V loop because the flow and volume delivered are constant (Figure 32.19b).

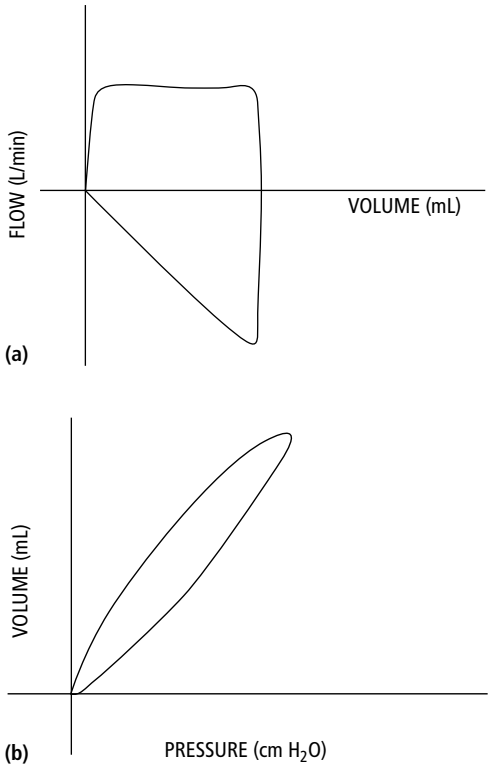


Figure 32.19 (a) A flow–volume loop from a normal animal while receiving positive pressure mechanical ventilation on a machine that delivers constant flow to deliver a breath. Note the square waveform pattern that is displayed. (b) A pressure–volume loop from a normal animal while receiving positive pressure mechanical ventilation on a machine that delivers a constant flow to deliver a breath. This is often the preferred waveform for detecting abnormalities in the pressure–volume loop.

Spontaneous Breath Loops

Spontaneous breaths create loop waveforms that differ from those produced during positive pressure ventilation. The F–V loop is similar, but the inspiratory curve of a spontaneous breath (above the horizontal axis) is rounded (Figure 32.20a). The main difference is a lower peak flow rate during spontaneous breathing. The expiratory waveform (below the horizontal axis) is passive, creating a descending ramp-like shape for both spontaneous and ventilator-created breaths.

The P–V loop during spontaneous respiration is very different than the P–V loop created due to positive pressure. During a spontaneous breath, the negative pressure generated during inspiration causes a leftward bulge of the P–V loop that extends into the negative side of the pressure axis (Figure 32.20b). The loop is then traced in a clockwise fashion, and expiration occurs on the positive side of the pressure axis.

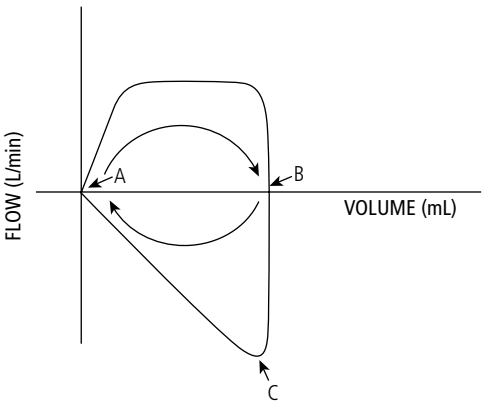


Figure 32.18 A flow–volume loop from a mechanically ventilated normal animal. Point A represents the start of inspiration and point B represents the start of expiration. Point C is showing the peak expiratory flow rate during passive expiration.

Loop Interpretation

The interpretation of abnormal loop patterns requires practice and patience. Some of the abnormalities that may be gleaned from analysis of the loops include airway obstruction, the presence of an air leak, or air trapping.

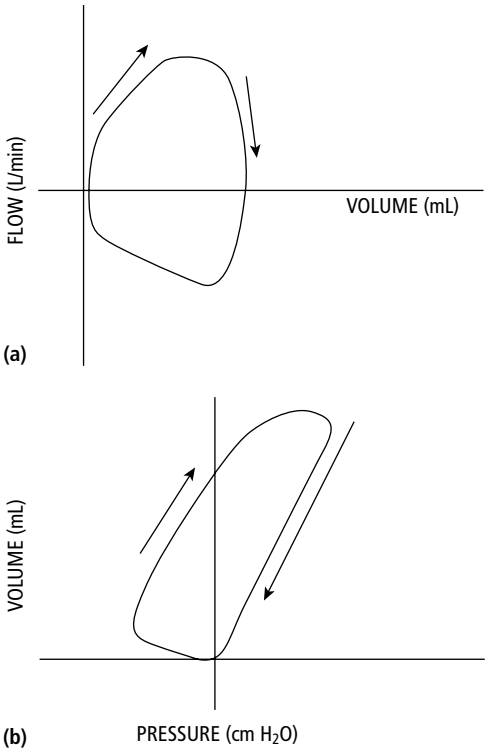


Figure 32.20 (a) A flow–volume loop from a normal animal during a spontaneous breath. The inspiratory curve of the spontaneous breath is above the horizontal axis and appears rounded. (b) A pressure–volume loop from a normal animal during a spontaneous breath. Note that the negative pressure generated during inspiration causes a leftward bulge that extends into the negative side of the pressure axis. The loop continues in a clockwise fashion, and expiration occurs on the positive side of the pressure axis.

Airway Obstruction

The location and severity of airway obstruction determine the changes that result on the F–V loop. Most significant obstructions decrease the PEFR, as seen in Figure 32.21. When the medium and small airways obstruct, the descending segment of the expiratory curve often turns into a more curvilinear shape, referred to as "scooping" (also seen in Figure 32.21). Comparison of F–V loops over time can help the veterinarian assess the effectiveness of bronchodilator therapy in patients with asthma or bronchospasm. The scooped-out appearance often changes to a more linear shape from the peak expiratory flows down to the end of expiration, reflecting the beneficial effect of therapy in relieving the airway obstruction.

Air Leak

The presence of an air leak during a breath is often evident in both the scalar and loop graphs. Leaks may originate from the endotracheal tube cuff, air leaks through chest tubes, or a bronchopleural fistula. The waveform's expiratory volume appears smaller than the inspiratory volume if a leak is occurring during inspiration. If a leak occurs downstream (on the patient side) from the flow transducer used to generate loop graphics, this appears as part of the inspiratory volume in the loop, but the lost volume is not returned through the flow transducer and therefore the loop does not close (see Figure 32.22). If the delivered inspiratory volume is less than the set volume but has an equivalent expiratory volume, a leak in the ventilator circuit between the flow transducer and ventilator should be suspected.

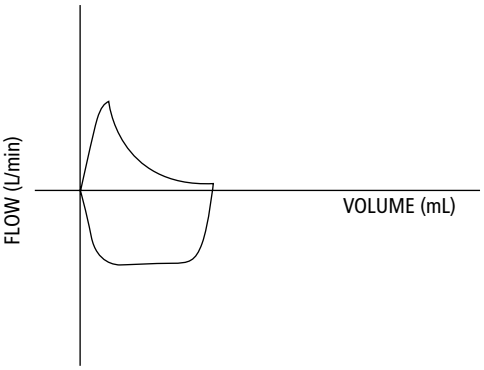


Figure 32.21 A flow–volume loop from a mechanically ventilated animal with airway obstruction. Note the decrease in peak expiratory flow rate. The descending segment of the expiratory curve appears more curvilinear, also known as "scooping," which is commonly seen in animals suffering from obstruction of the medium and small airways.

Air Trapping

If the expiratory time is not sufficient or the smaller airways collapse prematurely, air trapping may occur, causing the expiratory portion of the loop never to return to baseline (to never reach zero flow rate) prior to beginning the next breath (Figure 32.23). It is important to note that the F–V loop for a pressure support ventilation (PSV) breath can appear similar to the loop seen with air trapping. When air trapping is present, the expiratory flow never returns to zero. As with a normal F–V loop in PSV, an abrupt change in the slope of the loop at the end of inspiration is seen. This is due to the ventilator cycling into expiration based upon the preselected flow target. When looking at a P–V loop, it is important to consider the trigger setting for the patient-initiated breaths. This is typically seen around 2 cm H_2O, making it appear that volume is trapped, but this is a normal P–V loop (Figure 32.24).

Basic Pulmonary Mechanics Measured During Mechanical Ventilation

To measure compliance of the lungs, a pressure–time scalar is commonly used. Volume control ventilation is used, and once PIP is reached (Figure 32.25, point 1), an end-inspiratory pause is applied for 0.5–2.0 seconds for

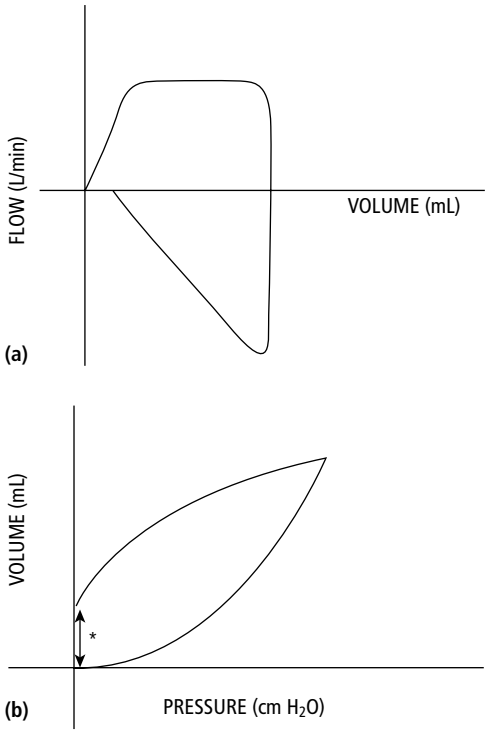


Figure 32.22 (a) A flow–volume loop from an animal receiving mechanical ventilation. There is evidence of an air leak downstream (on the patient side) of the flow transducer used to generate the loop. The lost volume is therefore not returned through the flow transducer upon expiration and the loop does not close. (b) A pressure–volume loop from an animal receiving mechanical ventilation. There is evidence of an air leak downstream (on the patient side) of the flow transducer used to generate the loop. The lost volume is therefore not returned through the flow transducer upon expiration and the loop does not close. The asterisk represents volume loss.

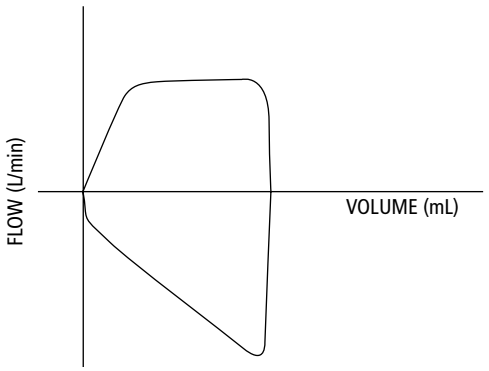


Figure 32.23 A flow–volume loop from an animal receiving mechanical ventilation. The expiratory portion of the loop never reaches a zero flow rate prior to the next breath, indicating the presence of air trapping.

only one breath. During the pause, there is no flow between the patient and the ventilator, which allows for equilibration between proximal airway pressure and alveolar pressure (Palv). The pressure at the end of the pause is the *peak*

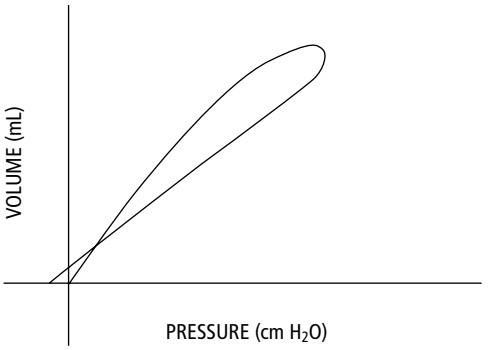


Figure 32.24 A pressure–volume loop from a normal animal receiving a pressure-supported breath. Note how the inspiratory and expiratory lines cross each other at around 2 cm H_2O as the patient attempts to inspire.

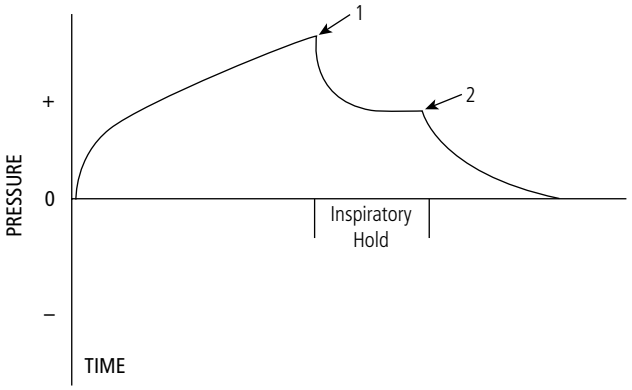


Figure 32.25 A pressure–time scalar during an inspiratory breath hold in a normal animal receiving volume-controlled mechanical ventilation. The peak inspiratory pressure (point 1) is seen prior to the inspiratory breath hold. The plateau pressure, also known as the peak alveolar pressure, is the pressure observed at point 2.

alveolar pressure, commonly referred to as the plateau pressure or static compliance (as seen in Figure 32.25, point 2). The difference between PIP and the peak Palv is due to the resistive properties of the system (either patient airways or artificial airway). The difference between the PIP and end expiratory pressure (EEP; or PEEP if applicable) is referred to as dynamic compliance; dynamic compliance is a less accurate measurement of compliance than static compliance, but its measurement does not require an inspiratory breath hold. Dynamic compliance is more a measure of impedance because it consists of both resistance and compliance components. The difference between the peak Palv and total PEEP is due to the elastic properties of the system (lung and chest wall compliance combined).

During pressure control ventilation, PIP and peak Palv may be equal due to the flow waveform that occurs (Figure 32.7a). Since the pressure often remains at the set point during a majority of the inspiratory time when using pressure control ventilation, static compliance cannot be

determined while using this mode (it must be determined while the patient is receiving volume control ventilation). Flow decreases during inspiration and is typically followed by a period of no flow at the end of inspiration (Figure 32.7c). During the no-flow time, proximal pressure may be equal to the peak Palv. To determine effective respiratory system compliance, the tidal volume is divided by the difference between PIP and EEP. Dynamic compliance is calculated as PIP minus EEP and includes a component of airway resistance.

The P–V and F–V loops can also be used to assess respiratory system compliance. In Figure 32.26a, a constant flow mode is used and the three F–V loops represent varying compliance levels. The "up" arrow shows increased compliance, the "down" arrow shows decreased compliance, and "N" is normal compliance. Note how the tidal volume increases with increases in compliance. Inspiratory peak flows are fairly constant, but expiratory peak flow rates decrease with increases in compliance. Figure 32.26b demonstrates a P–V loop with the same compliance changes as in Figure 32.26a. Airway resistance does not change between the three loops but notice the "right shift" of the P–V loop that occurs as compliance decreases. Although not present in this figure, increased hysteresis sometimes accompanies a decrease in compliance.

An increase in inspiratory airway resistance may be subtle on the F–V loop if the driving force of the ventilator is sufficient to overcome the increased resistance, as seen in the loop labeled with the arrow in Figure 32.27a. The loop does reveal a slight decrease in the PEFR and volume compared with the normal (N) curve. The same scenario is seen in the P–V loop of Figure 32.27b, where the expiratory curves are similar, but the volume of the normal curve is again greater than the abnormal curve and the pressure during inspiration is markedly higher in the abnormal loop labeled with the arrow. Potential causes of increased inspiratory resistance include patient–ventilator dyssynchrony, secretions or exudate within the endotracheal tube or large

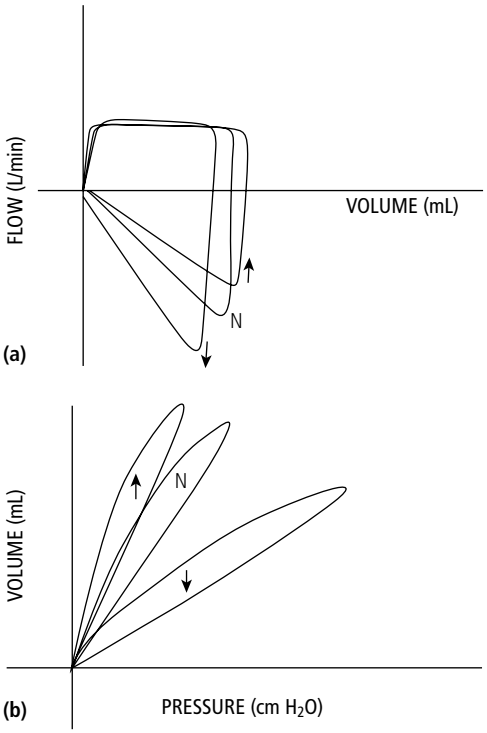


Figure 32.26 (a) Flow–volume loops depicting varying degrees of compliance in three animals receiving mechanical ventilation with constant flow delivery during inspiration. Increased compliance is evident in the loop marked with an up arrow, decreased compliance in the loop with a down arrow, and normal compliance in the loop marked "N." Note how the tidal volume increases with increases in compliance during flow control ventilation. (b) Pressure–volume loops depicting varying degrees of compliance in three animals receiving mechanical ventilation, as in Figure 32.26a. Increased compliance is evident in the loop marked with an up arrow, decreased compliance in the loop with a down arrow, and normal compliance in the loop marked N. Note the "right shift" of the pressure-volume loop that occurs as compliance decreases.

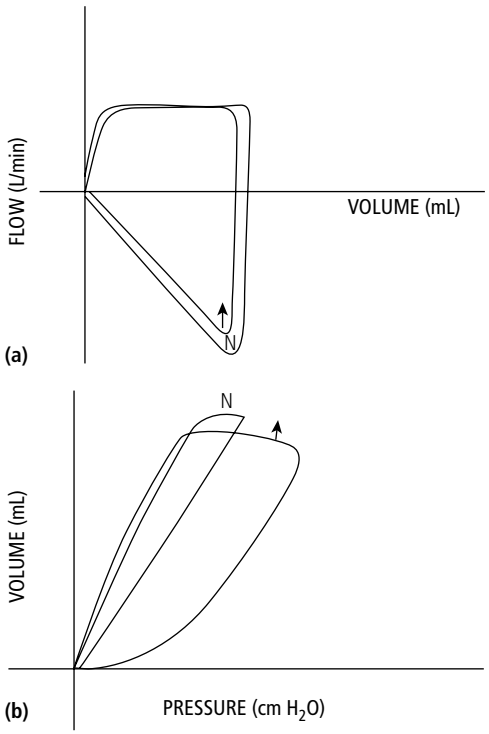


Figure 32.27 (a) Two flow–volume loops from animals receiving mechanical ventilation. The loop labeled with an up arrow depicts the decrease in peak expiratory flow rate and volume in an animal with increased inspiratory airway resistance compared with a normal (N) animal. The changes are subtle because the force of the ventilator is sufficient to overcome the increased resistance. (b) Two pressure–volume loops from animals receiving mechanical ventilation. The loop labeled with an up arrow depicts the decrease in volume and increase in peak pressure in an animal with increased inspiratory airway resistance compared with a normal (N) animal.

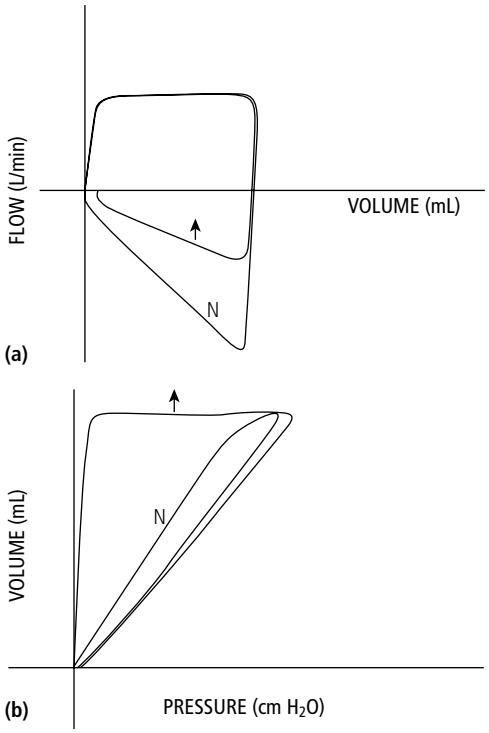


Figure 32.28 (a) Two flow–volume loops from animals receiving mechanical ventilation. The loop labeled with the up arrow shows an increase in expiratory resistance that causes a decrease in the peak expiratory flow rate. The absence of scooping is consistent with a large airway obstruction. A small leak is also present as indicated by the shortened return of the abnormal loop. (b) Two pressure–volume loops from animals receiving mechanical ventilation. Small airway obstruction is evident in the expiratory portion of the loop labeled with an up arrow compared with the normal (N) loop.

airway, or collapse or a mass of the trachea beyond the length of the endotracheal tube.

Resistance during expiration leads only to a decrease in PEFR, as seen in the loop labeled with the "up" arrow in Figure 32.31a; this pattern is consistent with large airway obstruction. Note there is no scooping on the F–V loop. A small leak is also present as indicated by the shortened return of the abnormal loop. Small airway obstruction leading to increased expiratory resistance, as seen in animals with bronchial collapse or narrowing or emphysema, markedly affects the P–V loop as seen in the loop with the "up" arrow in Figure 32.28b.

Practice Problems

A set of eight practice problems follows in this section.

Problem 1

A 20-kg mixed breed dog is on its second day on the ventilator for hypoventilation following ventral slot surgery for a cervical disk herniation. You notice that the inspiratory peak pressures have been getting higher and higher during volume-controlled ventilation, so you decide to perform a one-second inspiratory pause to assess static and dynamic compliance and compare it with yesterday's scalar (A). What is your interpretation of this pressure scalar (B)?

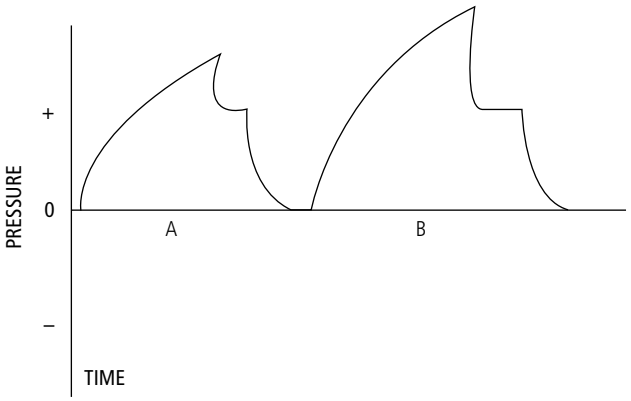


Figure 32.29

Answer

An increase in PIP with an unchanged pause pressure reveals increased respiratory resistance. Upon changing the endotracheal tube, you find a large blood clot inside.

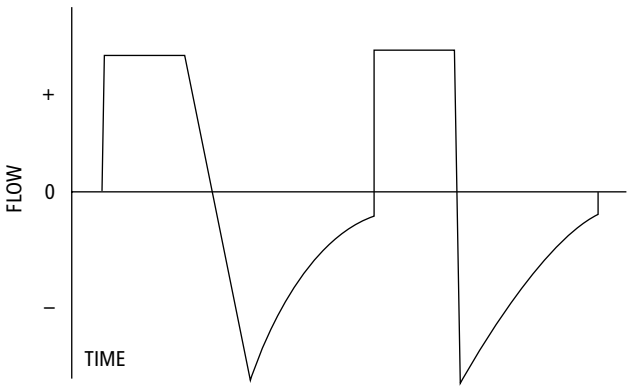


Figure 32.30

Problem 2

A 5-kg domestic shorthair cat is on volume-controlled ventilation following a severe asthmatic attack. You look at the flow scalar shown here. What is wrong?

Answer

Remember that the flow pattern does not change with changes in resistance or compliance. The expiratory flow has not reached zero before the next inspiration begins, indicating air trapping and auto-PEEP. A longer

expiratory time with or without bronchodilator therapy, especially if peak expiratory flow has declined, may prove helpful.

Problem 3

You try switching the cat in Problem 2 to pressure-controlled ventilation and get the flow scalar labeled B here and compare it with the normal flow scalar labeled A. Did this change help the cat?

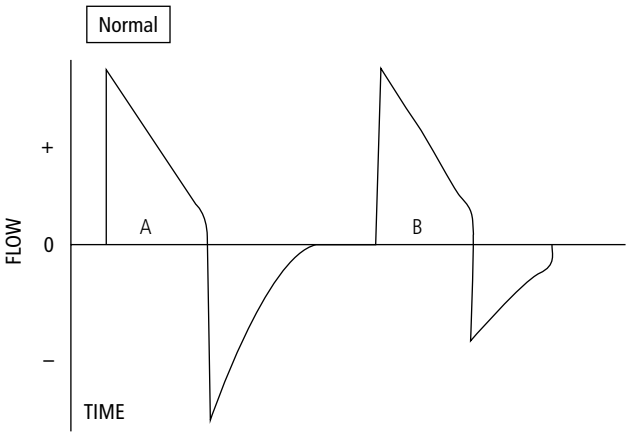


Figure 32.31

Answer

No. The expiratory flow does not reach zero before the next breath begins, and the decrease in expiratory flow indicates a continued expiratory flow limitation.

Problem 4

A nine-year-old Labrador Retriever is placed on positive pressure ventilation following a witnessed vomiting and aspiration episode. Following initial setup, you examine the P–V loop. What do you conclude?

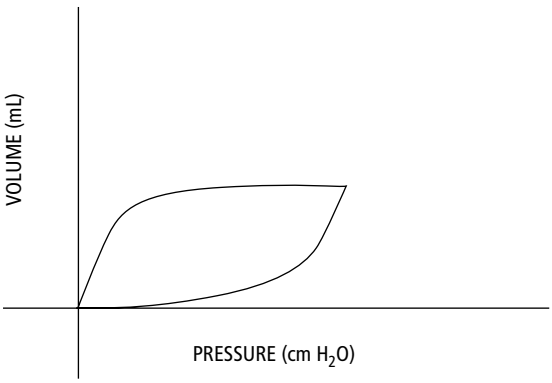


Figure 32.32

Answer

The slope of the loop is well below 45 degrees and the compliance with and without resistance characteristics of the respiratory system is poor. Perhaps an increase in PEEP is a good place to start to maximize pulmonary function by opening airways, allowing for improved volume change for change in pressure and prevent ventilator-induced lung injury.

Problem 5

A five-year-old Chihuahua is on the ventilator for pulmonary contusions after being hit by a car. Volume-controlled ventilation is being used and you see the F–V loop shown here. What do you suspect?

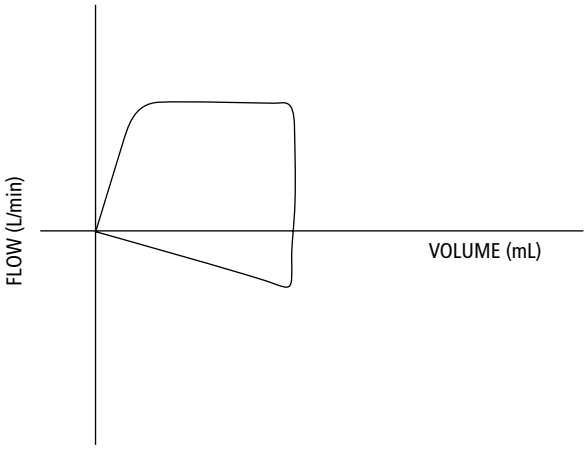


Figure 32.33

Answer

A marked decrease in the expiratory flow rate indicates expiratory flow limitation. Upon examination of the patient, you find no obvious airway obstruction. However, your veterinary technician notices that the water trap in the expiratory limb of the circuit is quite full and overflowing into the breathing circuit, thus increasing resistance to air flow. Upon emptying the water, the F–V loop returns to normal.

Problem 6

This two-year-old German Shepherd required mechanical ventilation for lower motor neuron disease. The dog is currently on SIMV with pressure support, but the clinician reports that the dog is not yet showing any evidence of triggering an assisted or a supported breath. You look at the pressure scalar below and then change a ventilator setting at point A. What did you do?

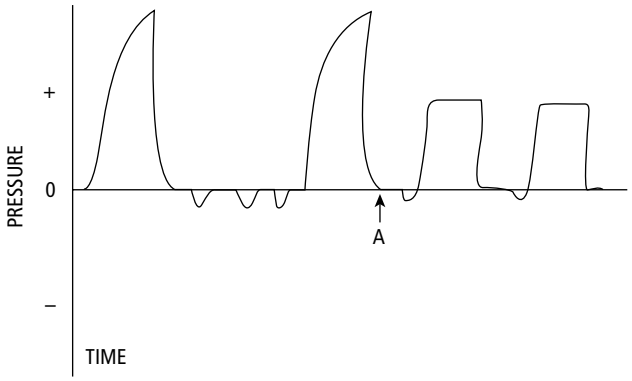


Figure 32.34

Answer

There are negative pressure deflections that are not followed by a delivered pressure support breath, indicating that the trigger sensitivity is set too high. You reduced the trigger sensitivity from 5 to 2 l/minute at point A and then observed two pressure-supported breaths.

Problem 7

A 10-year-old Siamese cat was placed on pressure assist control ventilation for severe pulmonary parenchymal disease of unknown origin. You look at the cat and the flow scalar (Figure 32.35a), F-V loop (Figure 32.35b), and P–V loop (Figure 32.35c). What do you conclude?

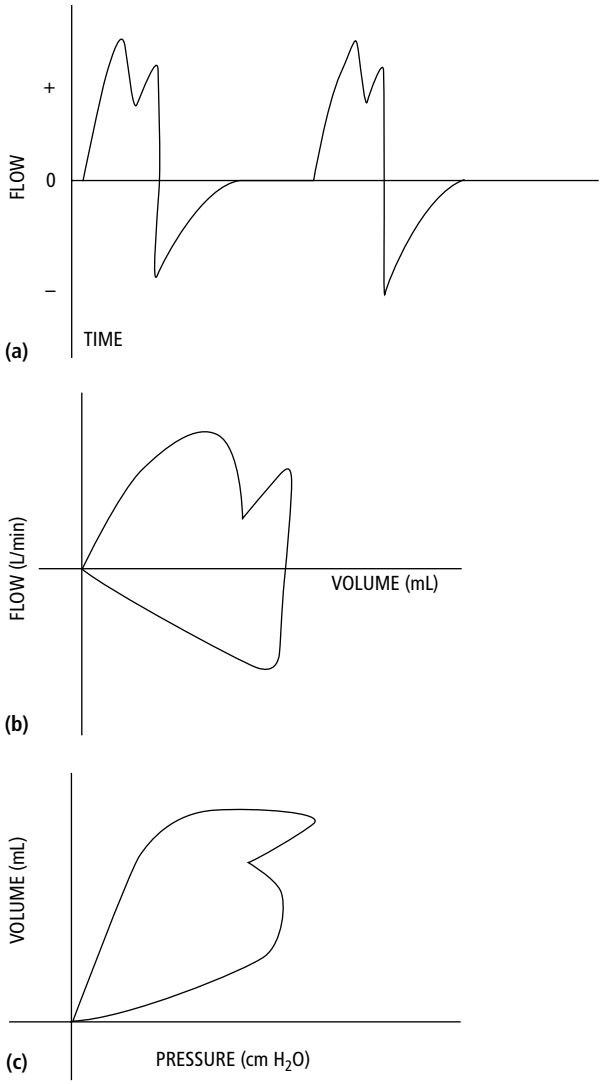


Figure 32.35

Answer

The cat is making an expiratory effort toward the end of inspiratory phase of the breath, thus creating a negative deflection in flow and pressure during the normally positive inspiratory time. This is patient–ventilator dyssynchrony and may lead to inadequate inspiratory volumes and poor oxygenation and ventilation. The clinician should try to change the settings to improve patient comfort based on the specific patient, respiratory parameters, and blood gas values. For example, an increase in inspiratory pressure, respiratory rate, or flow rate may prove beneficial if the animal has inadequate minute ventilation. Unless the cat appears too lightly anesthetized, it is best to give anesthetics or paralytics only after all other strategies have failed. Dyssynchrony is often the patient's way of telling the clinician that the ventilator settings are inappropriate.

Problem 8

A three-month-old Labradoodle has suspected noncardiogenic pulmonary edema after chewing on an electric cord. The puppy is on pressure-controlled assist control ventilation with PEEP, and the following pressure scalar (Figure 32.36a) and P–V loop (Figure 32.36b) are observed. What might you do after seeing this pressure scalar?

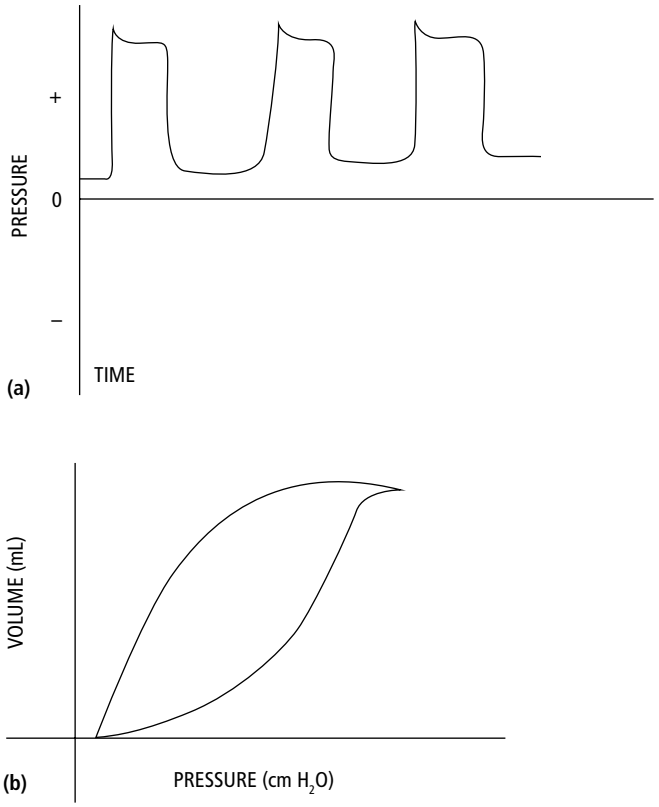
Answer

The spike in the pressure scalar at the beginning of inspiration indicates an excessive flow delivery during a fast rise time. The excessive flow and pressure do not necessarily translate into increased tidal volume delivery and are therefore undesirable. Possible changes that may prove helpful would include increasing the rise time (especially if the endotracheal tube is very narrow) or decreasing the flow rate.

Summary

Respiratory waveforms generated during mechanical ventilation can be helpful in bedside patient monitoring. With practice, interpretation of scalars and loops can be a rapid way to evaluate the patient from a distance using graphic information.

Figure 32.36



Video 32.1 The patient is a 25 kg dog that is on the ventilator for postoperative hypoventilation following cervical disc decompression. The dog displayed ventilatory dyssynchrony due to inadequate ventilator support and inappropriate inspiratory time. This was corrected by increasing the patient's peak inspiratory pressure. The dog also has a shorter inspiratory time than what the ventilator is delivering. The inspiratory time of the ventilator must be decreased so the patient is exhaling at the same time as the ventilator. The patient and the ventilator become in sync after the settings are adjusted.

Video 32.2 The patient is a 25 kg dog that is on the ventilator for severe aspiration pneumonia. The dog has high peak airway pressures along with decreased tidal volumes. As the PEEP is increased on the ventilator, the patient receives larger tidal volumes with the same peak inspiratory pressure. More of the lung is recruited and able to be ventilated.

Video 32.3 The patient is a 7 kg cat who presented with severe asthma. The patient is on a ventilator and given a bronchodilator. After the bronchodilator is administered, the patient's tidal volumes increase, and the peak inspiratory pressure decreases. The patient also has a decreased respiratory rate after medication administration. The patient's ventilator settings can be adjusted. The inspiratory time can be increased to ensure patient–ventilator synchrony.

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33

Alternative Methods of Augmented Ventilation

Jessica Schavone and Elizabeth Rozanski

Intermittent positive pressure ventilation (referred to in this chapter as conventional mechanical ventilation or CMV) can be lifesaving in patients with respiratory or ventilatory failure. However, CMV with high inspiratory pressures exacerbates lung damage and perpetuates lung injury, and so modalities that allow less aggressive settings, or that replace CMV altogether, have gained traction in human and veterinary medicine. Three other methods of alternative ventilation may be considered to support patients with respiratory failure. This chapter briefly describes the following basic alternatives or augmentations to CMV: high-frequency ventilation (HFV), including high-frequency jet ventilation (HFJV) and high-frequency oscillatory ventilation (HFOV); high-flow oxygen therapy (HFOT); and the use of a pediatric helmet.

High-Frequency Ventilation

Compared with CMV, HFV is a method by which to provide ventilation and oxygen supplementation with less potential for biotrauma, which is lung inflammation incited by the over-distention and repetitive opening and closing of lung units. HFV has not been widely adopted in companion animals, although it may be helpful to be familiar with its background, definitions, applications, indications, and contraindications, as well as the equipment required.

Background

The ability of the panting dog to remain normoxemic initially triggered early interest in the concept behind HFV as a therapeutic entity. Panting in normal dogs is used for temperature regulation as heat is lost across the respiratory system. The respiratory rate of panting dogs is commonly approximately 300 breaths/minute (5 Hz), which is very close to the resonant frequency of the respiratory system (RFRS) [1].

The RFRS is the natural frequency of the system at which vibrations will occur with the least amount of energy required. Matching the RFRS is important because it minimizes the metabolic cost of panting. If dogs pant at a rate quite different from the RFRS, it results in heat gain and can negate the cooling benefits. This cost is appreciated in dogs that are tachypneic due to disease, as their energy requirements for ventilation can increase dramatically. The actual influence on caloric needs associated with increased respiratory rate and effort is poorly studied in dogs, although in people there can be marked increase in caloric needs associated with respiratory effort associated with acute and chronic respiratory diseases.

Normal panting dogs also ventilate adequately; dogs can have adequate gas exchange with these small tidal volumes and rapid respiratory rates [2]. In our sea-level lung function laboratory, arterial blood gas samples collected from healthy panting dogs document high values for partial pressure of oxygen in arterial blood, often greater than 100 mmHg, and lower partial pressure of carbon dioxide in arterial blood, often in the 28–32 mmHg range.

Definitions

HFV is any technique that ventilates a patient at a respiratory rate higher than normal for that species, usually greater than 10 times the typical rate. For a cat or a dog, this would be approximately 200 breaths/minute or greater.

HFJV provides ventilation at rates of 60–400 breaths/minute in people. Inspiration is active; expiration is passive. HFOV provides ventilation at rates greater than 400 breaths/minute. In this form, both inspiration and expiration are active, and the gas flow is sinusoidal rather than the

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Companion website: www.wiley.com/go/burkitt/monitoring

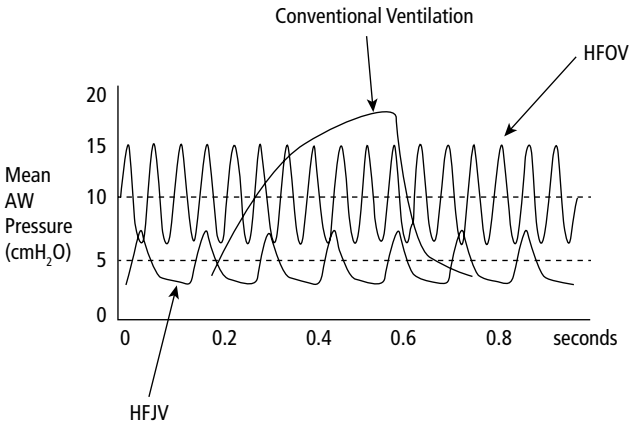


Figure 33.1 Comparison of ventilatory rates and pressure patterns with different ventilatory approaches. AW, airway; HFJV, high-frequency jet ventilation; HFOV, high-frequency oscillatory ventilation. *Source:* Courtesy of Bunnell Inc., Salt Lake City, Utah.



Figure 33.2 High-frequency jet ventilator. The larger green hose coiled on the left of the image is the oxygen input hose. The smaller light green hose coiled on the right is the gas output hose through which HFJV would be delivered to the patient. Note that the supply to the patient has a male tubing adapter at the end to attach to a standard vascular catheter or infant feeding tube, which would be inserted into the animal's trachea.

triangular flow seen with HFJV. Figure 33.1 shows a graphic depiction of the differences in the rates and pressure tracings for these different forms of ventilation.

Any of these HFV techniques can be combined with positive end expiratory pressure (PEEP) if needed to help recruit lung units and improve oxygenation. The application of PEEP is often required in patients with significant pulmonary disease but is generally not required or possible when HFV is used for interventional procedures such as routine bronchoscopy.

How High-Frequency Ventilation Works

Normal CMV relies primarily on bulk convection and diffusion to eliminate carbon dioxide (CO_2) and to provide oxygenation. In all forms of HFV, convection and molecular diffusion are still the primary mechanisms of gas exchange, but additional mechanisms of CO_2 elimination include pendelluft ("sloshing" back and forth of gas), Taylor-type dispersion (gas mixing enhanced by turbulence), and asymmetrical gas velocity profiles. These additional effects help HFV to provide adequate ventilation at lower airway pressures than CMV. Fresh gas is introduced into the system both to displace CO_2-rich gas from the respiratory system and to provide oxygen. This fresh gas is the source of the tidal volume and is introduced at an angle into the high-frequency system; thus, it is often referred to as *bias flow* because it is introduced "on the bias."

HFJV is often used in tandem with a conventional ventilator, with the conventional ventilator providing the bias oxygen flow, PEEP, and occasional sighs (larger breaths). An endotracheal tube adapter is used so the patient does not require reintubation. Ventilation is controlled by altering the rate and peak inspiratory pressure on the jet ventilator. The inspiratory time may be changed but is usually left at 20 milliseconds. As is true of all ventilation (spontaneous as well as mechanical), the volume of the breath in milliliters is determined by the relationship between the pressure difference between the airway opening and the intrapleural cavity as well as the compliance (distensibility) of the lung. Expiration is passive. Sighs are often provided by the ventilator at 2–10 breaths/minute. The ventilatory pressure waveform in HFJV is peaked (Figure 33.1). Figure 33.2 depicts a jet ventilator.

HFOV is provided with a piston and diaphragm, which makes both inspiration and expiration active. There is no need for a conventional ventilator. The inspiratory-to-expiratory ratio is usually fixed at 1 : 2, although it may be adjusted. The mean airway pressure (mPaw) and oxygen concentration can be adjusted. The pressure waveform in HFOV is sinusoidal (Figure 33.1).

Major Applications and Indications

In general, HFV is considered beneficial because it uses smaller tidal volumes and it improves ventilation : perfusion matching in the lungs. The alveoli remain in a relatively constant region on the pressure–volume curve, which may minimize biotrauma.

Practically, HFV has found clinical application in two major areas: in neonatal medicine in people, and for supportive ventilation during interventional procedures in human and veterinary medicine. Occasionally, HFJV is used in human medicine for patients with severe lung disease, although a multitude of clinical trials have failed to

identify a survival benefit in adults with acute respiratory distress syndrome (ARDS). HFJV has been used to some extent during the recent COVID-19 pandemic.

Currently, HFV is used most commonly in neonatal medicine. Techniques for the support of critically ill and premature neonates have improved tremendously in the past 30 years with the advent of surfactant therapy and other advances. Current recommendations for ventilation of neonates include consideration of transition to HFJV or HFOV when peak inspiratory pressures exceed 25 cm H_2O, if tidal volume greater than 6 ml/kg is required, if rapid respiratory rates are required, if air leak syndrome is present, if extracorporeal membrane oxygenation is being considered, or in extreme prematurity [3]. The use of HFV is advised when high respiratory rates are required because conventional ventilators are not responsive at such rates, and air trapping (auto-PEEP) is a potential concern due to breath stacking (see Chapters 31 and 32 for more information). HFOV is emerging as the more popular method of HFV in neonates.

Interventional procedures commonly use HFJV to provide adequate oxygenation during a procedure when a routine endotracheal tube is impractical. Some veterinary internists and criticalists use this technique during bronchoscopy or other airway manipulations. HFV is used during bronchoscopy through a catheter placed with its tip at the level of the mid-trachea or carina, depending on the area being evaluated. Some veterinary pulmonologists simply provide supplemental oxygen during the procedure rather than jet ventilation.

The goals for both HFJV and HFOV are maintenance of optimal lung volume and adequate blood gases. For HFJV and HFOV, lung volume is primarily maintained by PEEP, which limits the need for high peak inspiratory pressures. As with all ventilators, the training and skill set of those using the ventilator is more important than the specific type of ventilator chosen.

Patient assessment is similar to that performed during other forms of ventilation. Animals that require HFV generally are critically ill animals, and minute-to-minute assessments may be required. As in CMV, oxygenation goals may be reduced, such that oxygen saturation of 88% or greater could be acceptable. Depending on the technique used, capnography may or may not be possible (Chapter 30); standard capnographic waveforms would not be expected due to the very rapid respiratory rate.

Contraindications

There are few absolute contraindications to HFV. The major one is lack of familiarity with the equipment and monitoring techniques. Asthma is occasionally listed as a contraindication, though recent studies suggest this is incorrect. The initial fears were based on the potential for air trapping or hyperinflation because lungs with a delayed time constant (high resistance/low compliance) do not have as a favorable response to HFV.

Veterinary Studies of High-Frequency Ventilation

Although HFV and airway pressure release ventilation (see later) have been used extensively in research settings [4], there are no clinical descriptions of their use in dogs or cats with naturally occurring acute lung injury. HFJV is used during bronchoscopy procedures by some clinicians [5], and there exists a report of a premature foal supported by HFJV [6]. Owing to the propensity of dogs to pant, it is conceivable that HFV would be a favorable ventilation scheme in dogs with ARDS. Most recently, HFJV was described as a method of limiting motion in dogs receiving radiation therapy for heart-base tumors [7].

Other Novel Ventilation Strategies

While CMV can be lifesaving, it is invasive, carries its own risks, and requires considerable client emotional and financial investment. Two other modalities of respiratory support that are gaining popularity for treatment of small animals are HFOT and continuous positive airway pressure support using a pediatric helmet.

High Flow Oxygen Therapy

HFOT delivers up to 40 l/minute of warmed, humidified gas to a patient. Studies have shown that HFOT is well-tolerated in dogs, and that it may decrease the need for CMV. HFOT is delivered using specialized equipment but does not require one-to-one nursing care as does CMV. HFOT requires nasal cannulas, and importantly consumes a large amount of oxygen, which can be challenging if only small oxygen tanks are available. Cats tolerate HFOT less well than dogs. Brachycephalic dogs in particular may benefit from HFOT due to the added benefit of the gas pressure stenting open some of the upper airway. See Chapter 24 for more details about the use of HFOT (or high-flow nasal cannula) in dogs and cats.

Pediatric Helmet

Oxygen therapy may also be delivered by pediatric helmet, which effectively delivers continuous positive airway pressure without the need for tracheal intubation. This technique has been used in cats and dogs and may offer another less invasive option compared to intermittent positive pressure ventilation in some cases.

Summary

HFV, particularly HFOV, is an exciting and possibly underused option for ventilatory support in dogs with acute lung injury. Future studies would be helpful to determine whether HFV is more effective than CMV in companion animal medicine, particularly in ARDS. Noninvasive ventilatory methods, including HFOT and helmet-associated ventilation, may provide novel opportunities for veterinary medicine.

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34

Pleural Space Drainage

Amanda Arrowood and Lori S. Waddell

The pleural space is a potential space that can be occupied by tumor, fluid, gas, or abdominal organs. Drainage of the pleural space is indicated for patients with pleural fluid or gas accumulation. The choice of technique, whether thoracocentesis or thoracostomy tube placement, depends on several factors, including the patient's stability, the rate of reaccumulation of gas or fluid, and the underlying disease process. Pleural effusion can be secondary to a wide variety of disease processes and may be characterized as exudative, transudative, chylous, or hemorrhagic. These may be caused by a variety of disease processes. Septic exudative effusions are seen with pyothorax, which may arise secondary to penetrating chest wounds, hematogenous spread, extension from adjacent structures or fascial planes, aspirated foreign bodies, ruptured pulmonary abscesses, ruptured esophagus, and iatrogenic causes. Non-septic exudates can occur from infection with feline infectious peritonitis virus. Transudates (pure and modified) are most often caused by congestive heart failure but can also develop or be exacerbated by factors such as hypoproteinemia or vasculitis. They can be seen in patients with sepsis or pancreatitis and in patients with pulmonary thromboembolism. Neoplasia and lung lobe torsions may also cause a modified transudate. Chylous effusions can be caused by trauma, neoplasia, cardiac disease, and idiopathic causes. Hemothorax is most often caused by trauma, coagulopathy, or neoplasia, but can also be caused by infection with *Spirocerca lupi* or *Dirofilaria*; or by jugular venipuncture or catheter placement, thoracocentesis, or fine needle aspiration or biopsy of intrathoracic structures.

Blunt thoracic trauma can result in rupture of the lung and cause gas leakage from the alveoli or airways into the pleural space, resulting in lung lobe collapse and pneumothorax. Pneumothorax is the most common complication of blunt thoracic trauma in dogs [1]. Pneumothorax can also occur spontaneously, most commonly secondary to gas leakage from ruptured pulmonary bullae or blebs. Spontaneous pneumothorax has also been associated with leakage of gas from sites of pulmonary abscessation, primary and metastatic pulmonary neoplasia, foreign body migration, pneumonia, and feline asthma. In addition, parasitic disease such as *Dirofilaria*, *Paragonimus,* and *Filaroides osleri* have been associated with acute pneumothorax in dogs [2]. Physical examination findings include dull lung sounds and often dull heart sounds. If the lung sounds are dull ventrally, a pleural effusion should be suspected, whereas dullness dorsally is usually associated with a pneumothorax. The respiratory rate is often increased, and an asynchronous or inverse breathing pattern is associated with pleural space disease is both dogs and cats. The findings of dull lung sounds, together with this respiratory pattern, have a high sensitivity but low specificity for pleural space disease [3].

Thoracocentesis

Thoracocentesis is a quick and relatively easy method of removing gas or fluid from the pleural space. If a patient is in respiratory distress and pleural effusion or pneumothorax is suspected, thoracocentesis should be performed before radiographs to optimize patient stability. Thoracocentesis can be both diagnostic and therapeutic. When either gas or fluid is found, the chest cavity should be evacuated, which often eliminates the patient's respiratory distress.

Indications

Any patient that presents in severe respiratory distress with decreased lung sounds is a candidate for immediate thoracocentesis. In particular, patients that have sustained thoracic trauma such as being hit by a car, sustaining bite wounds, or falling from a height may develop pneumothorax or, less commonly, hemothorax. Thoracic radiographs

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can be used to confirm the diagnosis before thoracocentesis but are not always tolerated by unstable patients. Thoracic ultrasound can be used to confirm the presence of gas or fluid and is usually much less stressful to the patient (Chapter 27). Patients that present with pleural effusion benefit from thoracocentesis to remove the fluid, both to stabilize the patient's respiratory status and to obtain samples for diagnostic testing. Thoracocentesis can be a valuable diagnostic tool, even in cases with small volume pleural effusion that is not causing clinical signs. Thoracic ultrasound is helpful in determining the location of any fluid pockets and can help guide the site for thoracocentesis. When fluid is obtained, it should be submitted for cytology, fluid analysis, and culture and sensitivity if indicated.

Patients receiving positive pressure ventilation have an increased risk of pneumothorax and warrant thoracocentesis if they develop signs of respiratory distress, hypoxemia, decreased tidal volumes, or increased airway pressures with diminished lung sounds.

The major contraindication for thoracocentesis is severe coagulopathy, due to the risk of creating a potentially life-threatening hemothorax. Efforts should be made to correct the coagulopathy prior to thoracocentesis, if possible.

Procedure for Thoracocentesis

Thoracocentesis (Protocol 34.1) is an essential skill because it can be both therapeutic and diagnostic for patients with pleural space disease. The required equipment is listed in

Protocol 34.1 Procedure for Thoracocentesis

Items Required

- Clippers
- Surgical scrub
- Large syringe (10–60 ml), ideally with Luer lock
- Sterile gloves
- Three-way stopcock, ideally with Luer lock
- Extension tubing, two sets if expecting fluid
- Needle or a butterfly catheter
  - Large dogs: 1.5-inch needle or longer catheter
  - Medium dogs and large cats: 1-inch needle or catheter
  - Cats and small dogs: ¾- to ⅞-inch butterfly needle
- Bowl and tubes for samples including one with EDTA and one without anticoagulant (if expecting fluid)

Procedure

1) Gather supplies.
2) Perform hand hygiene and don clean examination gloves.
3) Position patient, preferably in sternal recumbency or standing. Lateral recumbency is also acceptable for pneumothorax. Patient comfort should determine position.
4) Have assistant available to restrain patient or give sedation as needed. In many cats, a minimal restraint technique is preferred and generally better tolerated.
5) Clip and aseptically prepare the appropriate rib space:
   a) If expecting fluid, the seventh or eighth intercostal space, at approximately the level of the costochondral junction, or as directed by ultrasound.
   b) If expecting gas, the eighth or ninth intercostal space approximately one-third of the way down the chest (about halfway between the spine and costochondral junction).
6) Perform hand hygiene and don sterile gloves.
7) Insert the appropriate size needle or butterfly catheter, bevel directed dorsally, slowly perpendicular to the chest wall, just cranial to the rib to avoid intercostal blood vessels.
8) Once through the chest wall, the needle can be directed either dorsally (if gas is expected) or ventrally (if fluid is expected) so that the needle is almost parallel to the chest wall.
9) Observe hub of needle for fluid.
   a) If small amount of frank blood is aspirated suddenly or unexpectedly or if lungs can be felt rubbing against needle, needle should be removed and replaced at a slightly different location.
   b) If a larger volume of blood is obtained, place 1–2 ml in a blood collection tube that does not contain anticoagulant, to evaluate for clotting.
   c) Blood from hemothorax should not clot; blood from the heart or a blood vessel should clot normally, if patient does not have a significant coagulopathy.
   d) For any other fluid, aspiration should continue until no more can be removed.
10) Directing the needle ventrally, rolling the patient slightly to the side that thoracocentesis is being performed, and re-aspirating from a more ventral location can facilitate removal of as much fluid as possible.
11) Fluid is saved for analysis, cytology, and culture if indicated.
12) Aspiration of gas will turn the tubing a slightly foggy white color as the warm, humid gas from the thoracic cavity encounters the room temperature tubing.
13) Aspirate until negative pressure is reached. If negative pressure is never achieved, a tension pneumothorax may be present, and chest tube(s) with continuous suction are needed (see section on thoracostomy tube placement). Continued aspiration of gas may be required while thoracostomy tube supplies are collected to optimize patient stability.

Box 34.1 Equipment for Thoracocentesis

- Clippers
- Surgical scrub
- Large syringe (10–60 ml), ideally with Luer lock
- Three-way stopcock, ideally with Luer lock
- Extension tubing and a needle or a butterfly catheter
- Bowl and tubes for samples including one with EDTA and one without anticoagulant (if tapping for fluid)

Box 34.1. Extension tubing is used when tapping with a needle to prevent movement of the needle in the chest as the three-way stopcock is operated. A short over-the-needle catheter can be used to reduce the risk of laceration of the lung or blood vessels, but these often kink once the stylet is removed. Cardiovascularly-sparing sedation may be needed depending on the patient's stability and temperament. Oxygen supplementation should be provided if the patient is in respiratory distress. With an assistant restraining the animal (preferably in sternal recumbency or standing), the appropriate rib space should be clipped and aseptically prepared. Patient comfort often determines which position is best, as well as whether gas or fluid is expected to be aspirated. If gas is expected, sternal or lateral recumbency may be acceptable because the gas will rise to the top of the chest in either position. If fluid is to be aspirated, sternal recumbency or standing is best because the fluid will accumulate in the ventral portion of the pleural space. When pleural fluid is expected, the seventh or eighth intercostal space is recommended, at approximately the costochondral junction. If available, bedside ultrasound can be used to determine where the largest accumulation of fluid is and therefore the best site for thoracocentesis. If gas is expected, a more dorsal approach, about one-third of the way down the chest, in the eighth or ninth intercostal space is used (about halfway between the spine and costochondral junction; Figure 34.1). Sterile gloves should be worn for the insertion of the appropriate size needle or butterfly catheter. Large dogs may require a 1.5-inch needle to penetrate the chest wall, whereas a three-quarters or seven-eighth-inch butterfly needle is sufficient for most cats and small dogs. In very large dogs, a 16-gauge, 3.25 inch or 14-gauge, 5.25-inch catheter can be used, which are longer and much less prone to collapsing than most smaller catheters due to their larger gauge. The needle tip should be placed just cranial to the rib to avoid intercostal blood vessels and nerves, which run along the caudal border of each rib. The needle is then gently inserted into the thorax perpendicular to the chest wall with the bevel of the needle directed dorsally while carefully observing the hub of the needle and extension tubing for any signs of fluid. Once through the chest wall, the needle can be directed either dorsally (if gas is expected) or ventrally (if fluid is expected) so that the needle is almost parallel to the chest wall (Figure 34.2).

If a small amount of frank blood is suddenly and unexpectedly aspirated from the thorax, or if the lungs can be felt rubbing against the tip of the needle, the procedure should be stopped, and the needle removed and replaced at a slightly different location. If a large amount of blood is withdrawn from the thorax, 1–2 ml should be placed in a blood tube containing no anticoagulant, to make sure the blood does not clot. Blood from a hemothorax should not clot within the tube, whereas blood from the heart or a

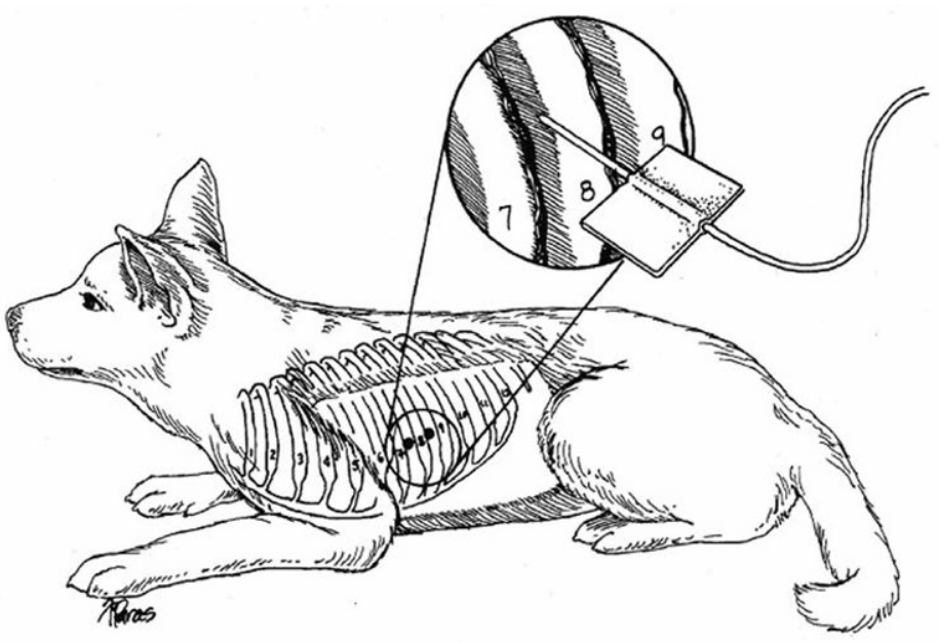


Figure 34.1 Schematic showing thoracocentesis in a dog in sternal recumbency using a butterfly catheter. The needle is inserted in the eighth intercostal space, just cranial to the rib.

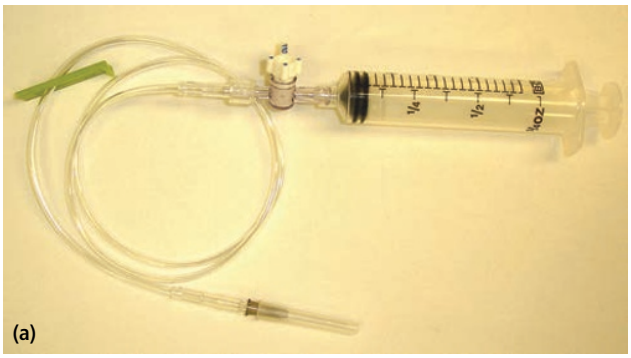






Figure 34.2 (a) Equipment for thoracocentesis using a 1-inch, 22-gauge needle; an extension set; a three-way stopcock; and a syringe. (b) Thoracocentesis in a bearded collie that presented with a spontaneous pneumothorax. Note that the ninth intercostal space is being tapped, one-third of the way down the chest (about halfway between the spine and costochondral junction) because gas is expected. (c) Close-up of thoracocentesis in the bearded collie. The needle is inserted with the bevel of the needle oriented dorsally.

blood vessel is expected to clot normally, providing there is no significant concurrent coagulopathy. If any other fluid type is seen in the hub of the needle, aspiration of fluid should continue until no more fluid can be removed. If the patient tolerates it, the needle can be directed ventrally while the patient is rolled slightly toward the side on which thoracocentesis is being performed, and reaspirating from a more ventral location can facilitate removal of as much fluid as possible. A fluid sample should be saved for fluid analysis, cytology, and possibly culture in appropriate tubes (Chapter 59). If gas is aspirated from the thorax, it usually turns the butterfly tubing a slightly foggy white color as the warm, humid gas from the thoracic cavity encounters the room temperature tubing and condensation occurs. If gas is aspirated, continue aspiration until negative pressure is reached. If negative pressure is never achieved, a tension pneumothorax may be present, and placement of a chest tube(s) with application of continuous suction is recommended. Diagnostic thoracocentesis is a relatively quick procedure (< 5 minutes), though therapeutic thoracocentesis can be prolonged if a large volume of fluid or gas needs to be removed or if the effusion has a lot of fibrin or is very pocketed.

Complications

Complications of thoracocentesis include iatrogenic pneumothorax from lung laceration, intrathoracic hemorrhage from laceration of blood vessels, or rarely re-expansion pulmonary edema in situations of chronic pleural effusions. Thoracocentesis without imaging confirmation of pleural space disease risks tapping an empty pleural space. Although this can lead to iatrogenic pneumothorax or hemorrhage, these are relatively uncommon complications and are usually self-limiting unless the patient has severe, chronic pulmonary pathology. Acute death from the stress of restraint is also possible. Appropriate sedation may reduce these risks, and care should be taken in choosing drugs with minimal respiratory suppression. After thoracocentesis, the patient should be monitored for reoccurrence of respiratory distress that may indicate return of either gas or fluid to the

pleural space or the development of an iatrogenic pneumothorax or hemothorax (less common).

Thoracostomy Tube Placement

Indications

Indications for placement of a thoracostomy tube (chest tube) include recurrent pneumothorax requiring repeated thoracocentesis, tension pneumothorax, pyothorax, rapidly forming pleural effusion, and postoperative management of patients having undergone thoracotomy. A tension pneumothorax occurs as gas progressively accumulates in the pleural space through a one-way valve leak of the lungs or airways into the pleural space. This leads to pressure atelectasis of the lungs and decreased venous return to the heart. A tension pneumothorax can cause fatal respiratory arrest in seconds to minutes. If a tension pneumothorax is present, thoracocentesis and chest tube placement should be performed immediately. The other indications for chest tube placement usually allow a less urgent approach to placement.

Thoracostomy tube placement allows for frequent intermittent evacuation of the pleural space or continuous evacuation if attached to continuous suction. As with thoracocentesis, a major contraindication for thoracostomy tube placement is a severe coagulopathy, which should be corrected prior to placement of a thoracostomy tube if possible.

Thoracostomy tubes should be placed under cardiovascularly-sparing sedation or intubation and general anesthesia. In unstable patients, general anesthesia with intubation is recommended so that ventilation can be assisted if needed. The patient's oxygenation should be monitored with pulse oximetry while sedated or anesthetized.

Positive pressure ventilation worsens a closed pneumothorax, so if the patient requires ventilation, the chest cavity should be evacuated by thoracocentesis while preparing for thoracostomy tube placement. This can be accomplished by either continuous thoracocentesis or via an open pneumothorax. An open pneumothorax may be created by inserting a catheter into the thoracic cavity or by performing a mini-thoracotomy. If a tension pneumothorax is present, continuous evacuation of the pleural space via thoracocentesis will be needed until the chest tube is in place.

Thoracostomy Tube Placement

Box 34.2 lists the equipment needed for chest tube placement. To secure the connections of the chest tube, catheter adapter, and three-way stopcock, 20-gauge orthopedic wire or zip ties may be used. Bandaging material should also be available.

Box 34.2 Equipment for Thoracostomy Tube Placement

- Clippers
- Surgical scrub
- Surgical blade
- Local anesthetic
- Small surgical pack
- Suture material
- An assistant (if possible)
- Thoracostomy tube or guidewire-assisted thoracostomy tube kit
- Catheter adapter (or Christmas tree adapter)
- Chest tube clamp
- Three-way stopcock with Luer lock
- Injection caps with Luer lock
- 20-gauge orthopedic wire or zip ties to secure connections

There is a variety of commercially available thoracostomy tubes made of silicone or polyvinyl that are packaged with stylets. These tubes typically have a radiopaque line that allows for easier visualization on radiographs. Small-bore chest tubes that are placed via a guidewire are available and can usually be placed quite rapidly but may not be ideal for thick or loculated effusions [5]. Pigtail catheters are used as thoracostomy tubes in human medicine but currently have not been widely adopted in veterinary medicine. Red rubber tubes can also be used but are more difficult to place (must use surgical technique) and are more likely to collapse; red rubber tubing is also irritating to tissues and can thus perpetuate fluid accumulation.

The size of the tube should be based on the size of the patient and whether gas or fluid is expected. Smaller tubes can be used for aspiration of gas. If fluid is expected, larger sized tubes are used, but care should be taken not to place a tube as large or larger than the patient's intercostal space to avoid discomfort. If the fluid is thick or loculated, extra drainage holes can be made in the tube prior to placement with a scalpel blade, making sure the holes are less than 50% the diameter of tube. If the fenestrations are more than 50% the tube's diameter, or are made too closely together, there is risk of the distal portion of the tube breaking off in the thorax, requiring surgical removal.

Three common techniques for placement of a thoracostomy tube are described: noninvasive surgical (Protocol 34.2), guidewire-based (Protocol 34.3) and trocar (Protocol 34.4) methods. All methods require the same approach.

Protocol 34.2 Noninvasive Surgical Method of Chest Tube Placement

- For items required see Box 34.2.

Procedure

1) Gather supplies.
2) General anesthesia or sedation is usually required. General anesthesia allows for more control of patient's respiratory function and is preferred.
3) Place patient in lateral recumbency.
4) Clip the lateral thorax from just caudal to the scapula to the last rib and from dorsal spine to ventral midline.
5) Aseptically prepare the hemithorax.
6) Perform hand hygiene and don sterile gloves.
7) Drape the area.
8) Loosen stylet from chest tube. Extra holes can be carefully made in the chest tube to aid drainage if fluid is present in pleural space. The holes are made with a scalpel blade and should not exceed 50% the diameter of the tube to prevent tube breakage within the thoracic cavity.
9) Make a small stab incision in the skin over the widest point of the thorax when the patient is in lateral recumbency at intercostal space 9–10 (about halfway between the spine and costochondral junction).
10) Pull the skin cranially (assistant) to allow the chest tube to be placed in intercostal space 7–8.
11) For dogs, 0.25–1.0 ml of 0.25 or 0.5% bupivacaine (maximum total dose of 3 mg/kg) can be injected into the subcutis and intercostal muscles at the planned tube insertion site. Alternatively, an intercostal nerve block can be performed by injecting 0.25–1.0 ml of 0.25 or 0.5% bupivacaine per site just ventral and caudal to the transverse processes of the thoracic vertebrae/head of ribs one space cranial and caudal, and at the site of insertion. Before injecting, the syringe should be aspirated to ensure that the needle is not in an artery or vein. Small dogs should receive 0.25 ml/site, medium dogs 0.5 ml/site, and large dogs 1.0 ml/site [4]. The total feline dose should not exceed approximately 1 mg/kg bupivacaine.
12) Bluntly dissect into the pleural space using hemostats, and then spread them wide enough to allow the tube to be passed between the hemostat's jaws and through the hole that was created. Insert the tube and stylet into the pleural space and advance cranio-ventrally 1–2 inches, orienting the tube parallel to the thoracic wall. The tube should then be fed off the stylet cranio-ventrally. Tubes without trocars can also be placed using this technique.
13) Stop positive pressure ventilation while inserting the tube, to allow lungs to deflate and decrease risk of trauma to lungs.
14) Assess placement of the tube by using the stylet (or a same-size tube, if using a tube without a stylet) to measure the distance the tube has been advanced within the thorax. Ideal placement results in the tube tip lying just caudal to the ipsilateral elbow. Once placement is determined to be adequate, the assistant can release the skin and allow skin to create a subcutaneous tunnel for a portion of the tube that remains outside the thorax; this helps create an airtight seal to prevent room air entering the thorax through the insertion site.
15) Connect the tube to a Luer-locking three-way stopcock and screw-on injection caps or a pleural drainage system.
16) As soon as the tube is in place, aspirate the gas or fluid present.
17) Once finished aspirating, place chest tube clamp on tube.
18) Secure the tube to the skin with a purse-string suture around the tube at the entry site and a finger trap suture pattern.
19) Place sterile dressing and light bandage over the insertion site.
20) Secure tube connection with orthopedic wire or zip ties.
21) Perform thoracic radiographs (two orthogonal views) to check tube(s) placement.

Noninvasive Surgical Method

The patient should be placed in lateral recumbency, and the lateral thorax should be clipped from just caudal to the scapula to the last rib and from dorsal spine to ventral midline. The area should be aseptically prepared (Figure 34.3a) and draped. The chest tube should be loosened from the stylet if a styletted tube is being used. For dogs, 0.25–1.0 ml of 0.25% or 0.5% bupivacaine can be injected into the subcutis and intercostal muscles at the planned tube insertion site, or alternatively, an intercostal nerve block can be performed by injecting 0.25–1.0 ml of 0.25% or 0.5% bupivacaine per site just ventral and caudal to the transverse processes of the thoracic vertebrae/head of ribs one space cranial and caudal, and at the site of insertion. Before injecting, the syringe should be aspirated to ensure that the needle is not in an artery or vein. The total dose of bupivacaine should not exceed 3 mg/kg in dogs. Small dogs should receive 0.25 ml/site, medium-size dogs 0.5 ml/site, and large dogs 1.0 ml/site [4]. For cats, 1 mg/kg bupivacaine is an appropriate total (maximum) dose for all sites combined.

Protocol 34.3 Guidewire-Assisted Thoracostomy Tube Placement

- For items required see Box 34.2.

Procedure

1) Gather supplies.
2) Provide local anesthesia and sedation as needed.
3) Initial preparation as previously described (Protocol 34.2).
4) A stab incision is made in the skin of the seventh or eighth intercostal space.
5) An 18- or 14-gauge introducer catheter is placed cranial to the rib and advanced cranially into the pleural space.
6) A 60-cm guidewire is inserted through the catheter and directed cranially, leaving approximately 20 cm or more outside the thorax.
7) The catheter introducer is removed, leaving the guide wire in place.
8) If using the kit's semi-firm dilating catheter, at this stage it is threaded over the guidewire, twisted firmly but gently through the soft tissues to create the tunnel, and removed.
9) The 14-gauge catheter is fed over the guidewire and inserted into the pleural space, ensuring that the wire can be grasped at the proximal end of the catheter before the catheter is fully advanced into the chest.
10) The guidewire is removed, and the catheter sutured in place via the eyelets and the neck of the catheter. Adapters can be used to adjust the length of the catheter that remains in the pleural space, with additional sutures through the eyelets of the adapters to secure the tube.
11) Stop positive pressure ventilation any time a stylet or other sharp object enters the thorax, to allow lungs to deflate and decrease risk of trauma to lungs.
12) Connect the tube to a three-way Luer-locking stopcock and screw-on injection caps or a pleural drainage system.
13) As soon as the tube is in place, aspirate the gas or fluid present.
14) Once finished aspirating, clamp the tube.
15) Place sterile dressing and light bandage.
16) Perform thoracic radiographs (two orthogonal views) to check tube(s) placement.

Protocol 34.4 Trocar Method of Chest Tube Placement

- For items required see Box 34.2.

Procedure

1) Gather supplies
2) Provide sedation or anesthesia if possible.
3) Initial preparation as previously described (Protocol 34.2).
4) Incise just the skin, tunnel the tube subcutaneously two to three rib spaces, and then position it perpendicular to the chest wall and grasp in a tight fist to allow only 1–2 inches of the tube to penetrate the chest (to prevent iatrogenic trauma from the tube penetrating too deeply).
5) Bluntly strike the top of the tube with the palm of other hand, popping the tube through into the pleural space.
6) Lower the top portion of the tube toward the table, decreasing the angle of insertion as the tube is advanced slightly. Once an additional 1–2 inches of the tube and stylet are in the chest cavity, the tube is advanced off the stylet, directing it cranially and ventrally.
7) Assess placement of the tube by using the stylet outside the animal to measure the distance the tube has been advanced within the thorax.
8) Connect the tube via an adapter piece to a Luer-locking three-way stopcock and screw-on injection caps or a pleural drainage system.
9) As soon as the tube is in place, aspirate the gas or fluid present.
10) Once finished aspirating, place chest tube clamp on tube.
11) Secure the tube to the skin with a purse-string suture around the tube at the entry site and a finger trap suture pattern.
12) Place sterile dressing and light bandage.
13) Secure tube connection with orthopedic wire or zip ties.
14) Perform thoracic radiographs (two orthogonal views) to check tube(s) placement.

This is a very rapid placement technique but is not recommended unless in an emergency situation in dogs with no other options, due to increased risk of iatrogenic trauma. It is never recommended in cats.



Figure 34.3 (a) A bearded collie is anesthetized, placed in lateral recumbency, and has had the lateral chest clipped and aseptically prepared in preparation for thoracostomy tube placement. (b) An assistant pulls the skin cranially to create the subcutaneous tunnel for the thoracostomy tube. (c) The chest is draped, and an incision approximately 1 cm in length is made between the 9th and 10th ribs, approximately one-third of the way down the chest. (d) Carmalt clamps are used to bluntly dissect through the subcutis and muscles of the chest wall. Carmalt clamps were chosen because of the size of this patient. (e) The thoracostomy tube is inserted into the chest between the jaws of the Carmalt. (f) The thoracostomy tube is introduced into the pleural space while still on the trocar, angled to become more parallel with the chest wall, advanced several centimeters, and then the tube is fed off the stylet. (g, h) The thoracostomy tube is secured with a purse string suture around the tube and then a finger trap pattern to prevent the tube from slipping.

For the surgical technique, a small stab incision should be made in the skin over the highest point of the thorax at intercostal space 9–10 (about halfway between the spine and costochondral junction). The skin should be stretched forward by an assistant (Figure 34.3b,c) to allow the chest tube to be placed in intercostal space 7–8, causing a tunnel under the skin of two to three intercostal spaces. Blunt dissection with hemostats through the intercostal muscles and parietal pleura is used to enter the thorax (Figure 34.3d). Then, without removing the hemostats,

the tip of the chest tube is placed into the thorax at a right angle to the chest wall (Figure 34.3e). During this part of the procedure, the anesthetist should cease positive pressure ventilation to allow lung deflation, therefore decreasing the chance of iatrogenic trauma to the lungs. Then the external end of the chest tube is lowered to decrease the angle of insertion and allow the tube to be placed along the inside of the thoracic wall, and the tube and stylet are advanced in a cranio-ventral direction as the tube is fed off the stylet (Figure 34.3f). The tube is generally advanced toward the thoracic inlet. Placement of the tube can be assessed using the stylet to measure the distance that the tube has been advanced within the thorax, or if using a red rubber catheter, by using another catheter of the same size. The tube is sutured in place with a purse-string around the base of the tube and a finger trap (Figure 34.3g,h). This suture pattern helps prevent the tube from slipping out of the thoracic cavity by tightening if the tube is pulled. This is accomplished by first suturing with a single knot to the patient's skin just cranial to the purse-string suture, leaving both ends of the suture long, and then crossing the ends of the suture beneath the chest tube, making a single throw on top of the tube, then repeating this pattern five to six times, finally tying off the suture. This allows the suture to climb the tube in a crisscross pattern. The tube is then connected via an adapter piece to a Luer-locking three-way stopcock with screw-on injection caps and clamped with a chest tube clamp or connected to a pleural drainage system for continuous suction. The insertion site is covered with a sterile dressing and light bandage. Both lateral and ventrodorsal or dorsoventral thoracic radiographs should be taken to confirm the cranial and ventral position of the tube(s) (Figure 34.4a,b).

Guidewire-Assisted Thoracostomy Tube Placement

The guidewire-based technique allows for placement of small-bore chest tubes very quickly with just local anesthesia and sedation. (Figure 34.5a)

A modified Seldinger technique is used, in which an introducer catheter is placed first, then a guidewire, followed by the catheter or thoracostomy tube. This method requires the same initial approach as the surgical method. Local anesthesia should be provided as described above in the surgical method and sedation given as needed. A small stab incision should be made in the skin where the catheter will be placed, typically in the seventh or eighth intercostal space. An 18- or 14-gauge introducer catheter is placed into the pleural space along the cranial aspect of the rib (Figure 34.5b) and directed cranially (Figure 34.5c). A 60-cm guidewire is inserted through the catheter, directing the wire cranially, leaving at least 20 cm of wire outside the thorax. (Figure 34.5d) The introducer catheter is removed, leaving the guidewire in place. The kits usually contain a

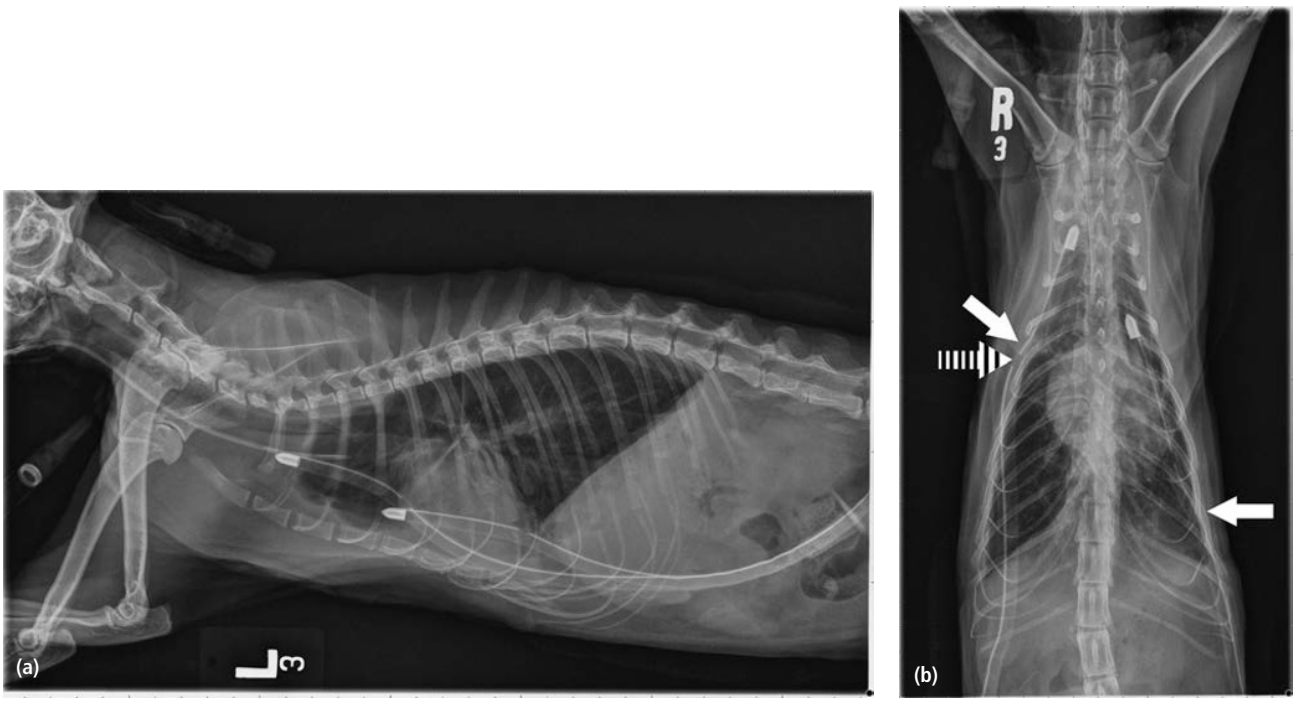


Figure 34.4 (a) Lateral radiograph of a cat with pyothorax to check placement of bilateral thoracostomy tubes. (b) Ventrodorsal radiograph of the same cat. Both views are needed to confirm correct placement. Note the right thoracostomy tube enters the chest too cranially (solid white arrows show where tubes enter thoracic cavity), at the fourth intercostal space, and it has one fenestration outside the pleural space (striped arrow). This tube should be repositioned.

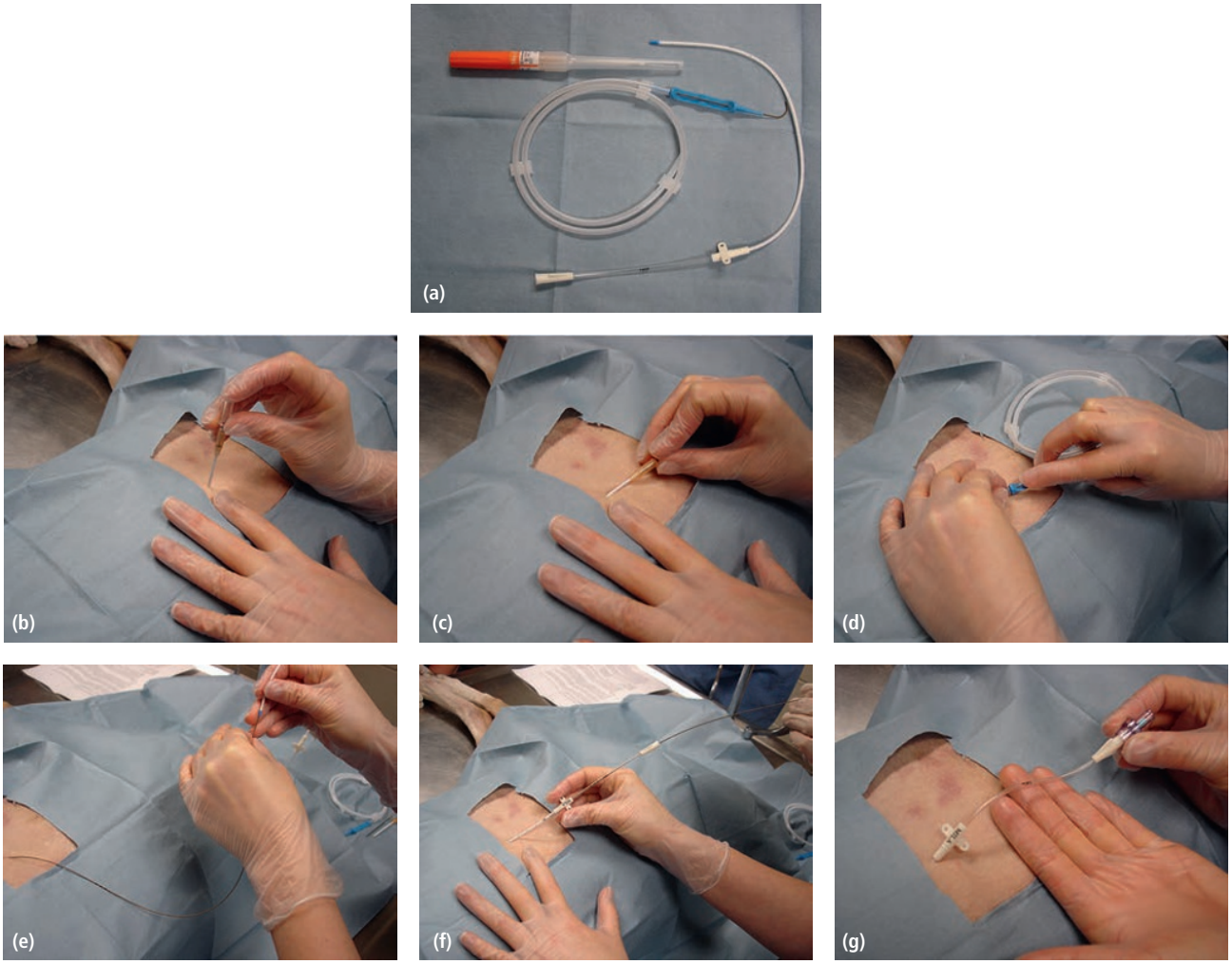


Figure 34.5 Placement of a guide wire based thoracostomy tube in a cadaver dog. (a) A guidewire based thoracostomy tube kit. (b) The introducer catheter is passed through a skin nick perpendicular to the chest wall just cranial to the rib. (c) The catheter is angled cranially as it is advanced into the pleural space. (d) The catheter's stylet is removed and the guidewire is fed through the catheter. (e) The introducer catheter is removed and the thoracostomy tube catheter is passed over the wire. (f) Once the wire is grasped at the proximal end of the catheter, the catheter is fed fully into the pleural space. (g) The wire is removed and the catheter is sutured in place.

semi-firm dilating catheter, which can be used to widen the diameter of the soft tissue tunnel through which the chest drain will be placed, if desired. If it is used, the dilator is threaded over the guidewire, twisted firmly but gently through the soft tissues to create the tunnel, and removed before placing the indwelling catheter. The 14-gauge thoracostomy catheter is then fed over the guidewire and inserted into the pleural space (Figure 34.5e), ensuring that the wire can be grasped at the proximal end of the catheter before the catheter is fully advanced into the pleural space, to prevent loss of the wire into the thorax (Figure 34.5f). The guidewire is then removed, and the catheter is sutured in place via the eyelets and the neck of the catheter (Figure 34.5g). Adapters can be used to adjust the length of catheter that remains in the pleural space, with additional sutures through the eyelets of the adapters to secure the tube. The tube is then connected and secured as described earlier for surgical placement, with radiographic confirmation of correct placement.

The small diameter and flexible material of the guidewire-placed catheter makes it well tolerated, especially in cats and small dogs. These thoracostomy tubes should be used with caution in some cases; they can become obstructed if the effusion is very thick or loculated, and can kink if placed in animals with a thick chest wall such as large or obese dogs.

Trocar Method

The trocar technique is an alternative technique that is more rapid but also has more risk of complications. This method requires the same initial approach as the surgical method in that it begins with a stab incision through only

the skin over the widest point of the thorax when the patient is in lateral recumbency (about halfway between the spine and costochondral junction) at intercostal space 9–10. The tube is then tunneled subcutaneously two rib spaces cranial to the skin incision. The tube is then positioned perpendicular to the chest wall and the end closest to the animal's chest grasped tightly in the surgeon's fist, so that only an additional 1–2 inch of the tube will be available to penetrate into the thoracic cavity. The top of the tube is struck bluntly with the palm of other hand, popping the tube through intercostal musculature and the pleura, into the chest cavity. The angle of the tube is then lowered, decreasing the angle of insertion as the tube is advanced slightly cranially into the thorax. Once 1–2 inch of the tube and stylet are in the chest, the tube is advanced off the stylet, directing it cranially and ventrally. The tube is then connected and secured as described earlier for surgical placement, with radiographic confirmation of correct placement.

Complications that can occur with the trocar technique include impaling the heart or lungs with the trocar (especially if the hand at the distal end of the tube slips), pulmonary contusions, and placement of the tube into the abdominal cavity. This technique is generally not used unless it is an emergency situation with no other options, and it is never recommended in cats because it is ineffective and even more dangerous, due to cats' more compliant chest walls. With any method of placement, pain management is important for patients with chest tubes. See the section on pain management later in this chapter.

Thoracostomy Tube Maintenance

A thoracostomy tube requires 24-hour monitoring because of the risk of detachment, obstruction, or leakage. The tube should be capped with a Luer-locking three-way stopcock fitted with screw-on injection caps. The stopcock must be positioned "off" to the patient when not in use. The chest tube itself should also be clamped using a chest tube clamp in the event the three-way stopcock detaches from the tube (Figure 34.6). A cerclage wire or zip tie may help secure the stopcock to the tube. Thoracostomy tubes should be checked daily for migration and removed as soon as possible to reduce discomfort and the risk of nosocomial infection.

Thoracostomy tube handling and insertion site evaluation require strict aseptic technique. Prophylactic use of antimicrobials is not recommended [6]. The thoracostomy tube insertion site should be evaluated at least once daily for signs of inflammation or infection including redness, pain, heat, swelling, subcutaneous emphysema, and/or purulent discharge. The integrity of the purse-string, finger trap, and any sutures used to fix the tube to the body wall should also be noted. Presence of subcutaneous emphysema or seroma formation around the insertion site should be documented



Figure 34.6 Use of orthopedic wire to secure thoracostomy tube, clamp, three-way stopcock, and injection caps to prevent accidental uncapping and iatrogenic pneumothorax.

in the patient's record. An increasing amount of subcutaneous emphysema or an enlarging seroma may indicate the chest tube has migrated out of the pleural space. To prevent accidental removal of the tube, it may be secured to the patient several ways, such as wrapping the tube with bandage material, placing a stockinette, or making butterfly tags from 1-inch tape that can be sutured to the patient's skin. Additionally, an Elizabethan collar may be necessary to prevent the patient from removing the chest tube.

The condition of the chest tube bandage should be monitored several times daily for strikethrough. Soiled or damp bandages should be changed immediately to prevent bacteria from the patient's surroundings migrating through the bandage toward the thoracostomy tube insertion site. The pleural space may be evacuated either by continuous suction drainage or manual evacuation. Generally, if continuous suction is not being used, the thoracostomy tube should be manually evacuated every four to six hours. Each time the tube is aspirated, the pleural space should be evacuated until negative pressure is obtained, unless re-expansion pulmonary edema is a concern. This is most commonly a concern in patients with prolonged pleural space disease such as a postoperative chronic diaphragmatic hernia. In these cases, the pleural space should not be fully evacuated initially, allowing the lungs to reinflate gradually thus reducing the risk of pulmonary edema [7]. Depending on the patient's disease, the tube may require evacuation more or less often. Frequent assessments are required for patients with thoracostomy tubes. Bilateral chest auscultation, pulse oximetry, and respiratory rate and effort should be monitored regularly for any changes [8]. Increased respiratory rate and effort, dyspnea, diminished lung sounds, and/or patient's posture (i.e. orthopnea), as

well as a rise in gas or fluid production, may indicate the need for more frequent evacuation or further diagnostics.

During chest tube evacuation, the tubing should be evaluated to ensure there are no leaks in the system, kinks, accumulation of fibrin material or other proteinaceous material. An unnoticed kink or obstruction in the chest tube yields negative pressure on aspiration. This "false" negative pressure is misleading and leads to the incorrect assumption that the pleural space has been evacuated. If the tube is clogged it can be flushed, paying strict attention to aseptic technique, with a small amount of sterile 0.9% NaCl instilled to dislodge the blockage [9].

A loose connection within the evacuation system can lead to the incorrect assumption that the patient has a continuous pneumothorax or has developed a pneumothorax if this was not the patient's initial problem. To check the tube for leaks, first all connections of the chest tube and three-way stopcock should be tightened. A chest tube clamp should be placed on the tube proximally. If all connections are tight and the chest tube is clamped, aspiration should yield negative pressure. If gas is aspirated, there is a leak in the system. All connections should be rechecked, and the chest tube should be evaluated for any small holes or cracks. If repeated attempts to evacuate the thoracotomy tube fail to yield negative pressure and a leak is not identified during inspection of the system, this suggests a rapidly accumulating pneumothorax and continuous suction drainage may be indicated.

After manually evacuating a thoracostomy tube and achieving negative pressure, the patient's clinical status should be reassessed. Any increase in respiratory rate and effort, orthopneic posture, or diminished lung sounds on auscultation may warrant further diagnostics such as pulse oximetry, arterial blood gas analysis, and/or thoracic imaging. Radiographs may reveal tube malposition/migration leading to unaspirated fluid or gas in the pleural space or the presence of pulmonary parenchymal disease. Other causes of increased respiratory rate and effort, such as pain, should be considered.

The volume of gas and/or fluid obtained from the chest tube should be monitored daily. The tube should be removed once there is negative pressure in the chest on several consecutive aspirations or the fluid obtained on aspiration decreases significantly. Normally, small volumes of fluid (up to 1–2 ml/kg/day) are generated because of the body's natural inflammatory response to a foreign object (thoracostomy tube) in the pleural space [10]. In general, if tube aspiration yields little or no gas and no greater than 2–4 ml/kg of pleural fluid over a 24-hour period, the tube can be removed. If it is yielding negative pressure and disease within the pleural space is still suspected, diagnostic imaging such as thoracic ultrasound or thoracic radiographs are indicated to determine whether a new chest tube should be placed, and the existing chest tube removed.

Thoracostomy Tube Removal

When removing the thoracostomy tube a smooth, quick motion should be used. Prior to removal, analgesia should be considered to reduce patient discomfort. The thoracostomy tube insertion site should be left to heal by second intention; no sutures are generally needed. The site should be covered with a light bandage including a sterile, nonadherent pad [9]. The bandage should be monitored for any strikethrough of residual fluid and changed as indicated.

Chest Tube Drainage Systems

Chest tube drainage systems are indicated when large quantities of gas or fluid are accumulating in the pleural space rapidly. These systems prevent the patient from developing respiratory distress between aspirations of the tube and help to keep the lungs fully inflated in their normal expanded position. Otherwise, each time the chest tube is aspirated, the lung reinflates from a relatively collapsed position, which may cause a seal that is forming to break apart. For patients with pyothorax that are producing a large volume of effusion, continuous drainage of the pleural space is indicated to reduce the volume of purulent material in the chest cavity. For other causes of pleural effusion including chylothorax, continuous drainage may be indicated if the effusion is accumulating rapidly.

Continuous Suction Drainage Systems

Active drainage of the pleural space can be accomplished by connecting the patient to a continuous suction source and a drainage system. The various drainage systems are based on the three-bottle system (Figure 34.7). In this model, the first bottle acts as a fluid trap, the second bottle provides the underwater seal, and the third bottle regulates the amount of suction that is applied to the pleural space. The three-bottle system is cumbersome and can be difficult to use because the bottles are hard to transport and maintain in the upright position [10].

For ease of use, the commercially available continuous chest drainage systems combine the three-bottle system into one compact plastic unit (Protocol 34.5). The left-most section of the unit is the collection chamber. The patient's chest tube connects to the collection chamber of the drainage unit via the included tubing and tube adapter in most cases. If the chest tube is small, the adapter may be too large to connect to it and a Christmas tree adapter and a short piece of sterile nonconductive connecting tubing (6-mm diameter is commonly used, available from Medline Industries, Mundelein, IL) may be needed to attach to the

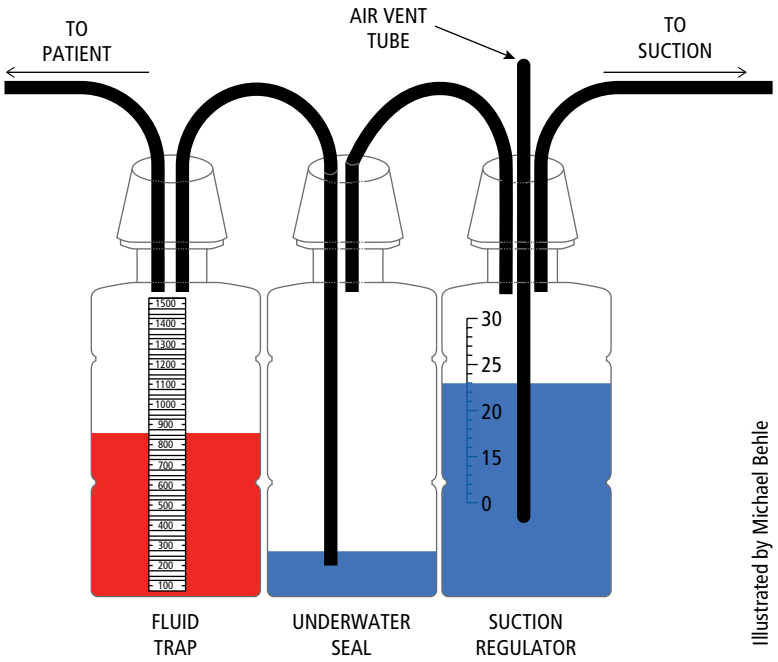


Figure 34.7 Schematic of an airtight three-bottle system for continuous drainage of a chest tube. The chamber on the left accumulates and measures the volume of fluid from the pleural space. The middle chamber is the water seal to prevent gas from flowing backward into the patient's pleural space, and the chamber on the right is the suction control chamber. It is the depth of the vent tube below the water level that determines the amount of suction.

tubing adapter. Fluid from the chest tube collects in this chamber, which allows for easy measurement and recording of the volume of drainage. The middle chamber of the chest drainage system provides the water seal. The purpose of the water seal is to prevent gas from flowing backward through the tubing and into the pleural cavity. It is recommended that the water seal chamber be filled with sterile water up to the 2-cm line, so a 2-cm water seal is established. To maintain an effective seal, it is important to maintain the chest drainage unit upright at all times and to monitor the water level in the water seal because it may evaporate. Bubbling in the water seal chamber is caused by gas flowing from the tubing into the chamber. This indicates gas is being removed from the patient's pleural space or there is a leak in the system. The amount of gas bubbling in this chamber cannot be quantitated but does allow a subjective evaluation of the amount of gas coming off the pleural space. The right-most chamber of the unit is the suction control chamber. Traditional chest drainage units regulate the amount of suction by the height of the column of water in the suction control chamber. It is important to remember that it is the height of the water, not the setting of the suction source, that limits the amount of suction transmitted to the pleural cavity. The recommended height of water is 15–20 cm [9, 10]. The unit is then attached via a 6-mm diameter sterile nonconductive connecting tubing to a suction source such as wall suction or a portable suction machine like the Schuco Vac (Allied Healthcare Products, Inc., St. Louis, MO).

A few brand-name continuous suction drainage units are commonly used in critical care. Some examples include the Argyle™ Thora-Seal™ III chest drainage unit (CardinalHealth™, Dublin, OH) and the Pleur-evac® chest drainage system (Teleflex Medical OEM, Gurnee, IL).

The chest drainage system must be kept on a flat, level surface. If the unit is not level, it could disrupt the water seal. The gentle bubbling causes evaporation of the water in the chambers over time. It is necessary to check the water levels every few hours to ensure the proper water seal and suction. Careful inspection of all chest tube connections to the chest drainage system is warranted to avoid potential leaks. The system can be checked for leaks by briefly clamping off the chest tube and watching for bubbling in the water seal chamber. If bubbles are still occurring in the water seal chamber while the chest tube is clamped, there is a leak in the system distal to the clamp. All connections should be checked, and then the process should be repeated.

The chest drainage tubing should be checked for kinks. Any fluid that accumulates in the tubing should periodically be encouraged to flow into the collection chamber by elevating the tubing (but not above the level of the chest tube) so gravity will aid in its drainage. The fluid collection chamber has a capacity of approximately 2000 ml, depending on the type of drainage system. The fluid collection chamber should be monitored closely and emptied or replaced if it becomes filled. Aseptic technique should be used if the collection chamber is emptied. Many units do not allow for emptying, so a new unit is required if the collection chamber fills. If the chest drainage tubing becomes clogged with thick secretions, the chest drainage unit needs to be replaced.

Non-suction Drainage

A passive continuous drainage method is also available. The Heimlich valve consists of a rubber one-way valve inside a plastic tube that connects to a standard chest tube. These units only allow gas and fluid to move from the chest into the environment or collection bag. They are not recommended for animals under 15 kg because smaller patients do not generate sufficient increases in intrapleural pressure during exhalation to allow the valve to operate properly. Heimlich valves (Figure 34.8) are best suited for removal of gas from the pleural space because fluid may cause the valve to stick and no longer function [11]. Even when pneumothorax is the primary problem, small amounts of fluid, blood, or fibrin may cause the Heimlich valve to stick, so they should only be used with constant supervision [12].



Figure 34.8 A Heimlich valve, which allows for passive continuous drainage of a thoracostomy tube.

Manual Drainage of the Thoracostomy Tube

Strict attention to aseptic technique must be followed whenever the chest tube is handled or aspirated. The chest tube may be manually evacuated by attaching a chest tube adapter to a Luer-locking three-way stopcock. An empty syringe should be attached to the stopcock, and the stopcock should be positioned open to both the patient and the syringe. To avoid pleural damage, a maximum of 3–5 ml of negative pressure should be applied during chest tube drainage [13]. The chest tube should be evacuated via gentle aspiration of the syringe plunger, until negative pressure is obtained. Once negative pressure is achieved, the patient should be repositioned and the chest tube re-aspirated to determine whether changing the patient's position yields any additional gas or fluid from the tube. Occasionally, there may be a pocket of fluid that cannot be drained by the chest tube unless the patient is repositioned.

Protocol 34.5 Setting up a Continuous Chest Drainage System

Items Required

- Continuous chest drainage system (Thora-Seal III™ or Pleur-evac™)
- Christmas tree adapter
- Short piece of sterile nonconductive connecting tubing (6-mm diameter)
- Sterile water
- Y connector (optional)
- Suction source

Procedure

1) Place the suction unit on a flat surface, and secure if needed to prevent it from falling over.
2) Pour water into the water seal chamber to the level indicated on the unit, then pour water into the suction control chamber to the required level (usually 15–20 cm of water) [9, 10].
3) The level of water in the suction control chamber determines the degree of suction, not the vacuum meter.
4) Connect drainage tubing to the chest tube, and connect the suction tubing to the vacuum source, then unclamp the chest tube(s) to allow evacuation of the pleural space.
5) Two chest tubes can be connected into one chest drainage system by using a Y connector (Figure 34.9) and a piece of 6-mm diameter sterile nonconductive connecting tubing.



Figure 34.9 A sterile plastic Y piece and tubing are used to connect bilateral chest tubes to a single drainage system.

The total volume of gas and fluid aspirated from the chest tube should be measured and documented in the patient's record. Changes in the volume of gas or fluid aspirated from the thoracostomy tube from one intermittent evacuation to the next should be closely monitored. If increased gas is aspirated, the system should be evaluated for any leaks. If negative pressure is suddenly obtained and the patient is still showing signs of respiratory distress, an obstruction should be considered. If the chest tube is proven functional, it may indicate improvement of the patient's pleural space disease. In addition to changes in the volume of aspirated fluid or gas, any gross changes in the appearance of fluid (e.g. changes in consistency and color of pleural fluid) should be noted. A sudden increase in the volume or change in the type of fluid being aspirated could indicate a secondary problem such as infection, hemorrhage, severe hypoalbuminemia, or other causes. Fluid analysis may be indicated to further investigate.

Pain Management

Indwelling chest tubes can be very painful; therefore, it is important to provide analgesia to these patients. Bupivacaine 0.5% (Hospira Inc., Lake Forest, IL) can provide local analgesia when administered via the thoracostomy tube at a total dose of 1.5 mg/kg every six to eight hours for dogs [14, 15]. A lower dose of 1 mg/kg every six to eight hours is recommended for cats [16, 17]. Cats are more sensitive to local anesthetics than dogs and should be carefully monitored when these drugs are used. After injecting into the chest tubes, 2–3 ml of sterile 0.9% NaCl should be used to flush the bupivacaine out of the tube and into the patient's pleural space. If a single chest tube is present, placing the patient with the chest tube side down after instillation of bupivacaine may allow for better local analgesia. The bupivacaine dose can be split and given via two tubes in patients that have bilateral thoracostomy tubes. Bupivacaine should not be used in patients that are attached to a chest drainage unit with continuous suction because it will be suctioned back out of the thoracostomy tube immediately after administration. Similarly, the thoracostomy tube should not be aspirated immediately after administration of a local anesthetic. Bupivacaine can sting on initial injection due to the acidity of the solution. Sodium bicarbonate can be used to buffer the solution at a dose of one part sodium bicarbonate to nine parts bupivacaine [16]. If the total volume of bupivacaine or bupivacaine plus sodium bicarbonate is very small, it can be diluted with sterile 0.9% NaCl to a total volume of 10–20 ml, depending on the size of the patient.

The use of bupivacaine in animals that have had a pericardiectomy is controversial due to the possibility of cardiotoxicity [18]. Injectable opioids offer excellent analgesia but may be associated with decreased respiratory drive at higher doses (Chapter 48). The combination of opioids and intrapleural bupivacaine can provide excellent pain relief for most patients. A tranquilizer such as acepromazine (Butler Animal Health Supply, Dublin, OH) or a benzodiazepine can be administered with an opioid to treat anxiety. If the patient is eating, oral trazadone at 3–10 mg/kg orally every eight hours (Teva Pharmaceutical Industries) may also help reduce anxiety [19]. It is important to keep patients calm to prevent them from disconnecting the chest drainage system or pulling out the chest tube. Nonsteroidal anti-inflammatory drugs may also be used for analgesia if the patient has no contraindications (Chapter 48) and may provide the best pain relief.

Handling Samples for Fluid Analysis

Analysis of the pleural fluid may indicate whether the effusion is a transudative, modified transudative, chylous, exudative, neoplastic, or hemorrhagic effusion [20, 21]. A sample of the effusion should be submitted for fluid analysis to aid in identification of the disease process causing the effusion. Fluid analysis should include cell counts, protein concentration, and cytology [14] (Table 34.1). Fluid should be submitted for bacterial culture (aerobic and anaerobic) if bacteria or suppurative inflammation are seen on cytology or an infectious process is suspected. If a chylothorax is suspected, triglyceride concentrations of both the effusion and serum should be measured and compared [21]. A diagnosis of chylothorax can be made if triglyceride concentrations are greater in the effusion than in the serum and fluid cytology shows a large number of mature lymphocytes. A sterile red top tube (without anticoagulant) should be used for cell counts, cytology, and triglyceride levels. Hemorrhagic fluid samples should be submitted in a purple-top tube (with EDTA anticoagulant). An iatrogenic hemothorax may result if the needle used during thoracocentesis contacted the patient's heart or a blood vessel. Active hemorrhage can be ruled out by placing a small volume of the effusion into a red top tube or a tube with diatomaceous earth and monitoring for clot formation. If samples are to be submitted for culture, a sterile red top tube or anaerobic and aerobic culturettes should be used.

Table 34.1 Pleural effusion types and characteristics [21].

Fluid type	Appearance	Cell counts (cells/µl)	Protein concentration (g/dl)	Triglyceride present	Cytology
Transudate	Clear, pale yellow	< 1500	< 2.5	No	Predominately acellular, occasional RBC
Modified transudate	Yellow or pink; clear to slightly cloudy	1500–5000	~ 3.0	No	Moderately cellular, some RBC, macrophages, and mesothelial cells
Chylous	Milky/white; turbid	500–20 000	≥ 3.0	Yes (triglyceride more than serum triglyceride)	Mature lymphocytes, neutrophils, and macrophages
Exudate	Yellow to orange-brown	> 5000	> 3.0	No	Primarily nondegenerate to degenerate neutrophils; bacteria may or may not be present
Hemorrhagic	Red	Resembles peripheral blood	> 3.0	Yes; triglyceride level equal to serum triglyceride level	Resembles peripheral blood

RBC, red blood cells

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Section Four

Urinary and Gastrointestinal Procedures

35

Urethral Catheterization

Jamie M. Burkitt Creedon

Urethral catheterization is a reasonably simple procedure that is performed most commonly in emergency and critical care practice to relieve obstruction of the lower urinary tract or to monitor urine output. Catheters are generally placed retrograde from the urethral orifice into the urinary bladder, opposite the natural flow of urine. As with all devices, insertion and maintenance of a urethral catheter carries risks, and the risks should be weighed against the potential benefits whenever the procedure is considered.

Indications for Urethral Catheterization

Indications are to alleviate urethral obstruction, empty the bladder, monitor urine output, acquire samples for analysis, and aid in diagnostic procedures and urologic surgery [1]. Indwelling urethral catheters should not be used in place of good nursing care to keep patients clean and dry. Urine samples for analysis and culture are generally better procured by cystocentesis or free catch.

Urethral Catheters

Urethral catheters are made of materials such as silicone, latex, or a latex base with various coatings. The ideal material would be atraumatic and would not elicit inflammation; it would resist kinking and would inhibit bacterial adherence. Latex and latex-based catheters may cause more inflammation than some other flexible materials of which indwelling urethral catheters are made. For instance, latex-based catheters may be more likely to cause urethritis in people compared with silicone catheters [2]. In an experimental dog model, latex catheters tended to cause more inflammatory changes in urethral cells than did silicone, Teflon-coated latex, or red rubber (polyvinylchloride) catheters [3]. General-use catheters such as polyvinyl, polypropylene, or feeding tubes are also used. While its stiffness makes polypropylene the easiest insert, it may also be the least desirable because it can directly traumatize the urethra and, if left indwelling, can traumatize the bladder wall. An experimental study in cats showed that both polypropylene and polyvinyl catheters caused inflammatory lesions in the urethra and bladder, with polypropylene causing the most lesions [4]. That being said, one more recent retrospective study in male cats found no difference in recurrence of urethral obstruction when the indwelling urethral catheter was made of polypropylene as opposed to polyvinyl chloride, although overall recurrence rate was so low that finding a difference would have been unlikely [5]. Silicone catheters appear to resist kinking better than latex-based catheters [6].

Bacteria colonize indwelling urinary catheters and grow as biofilms embed in a gel-like polysaccharide matrix. Bacteria growing in the biofilm are resistant to antimicrobials, and unfortunately all types of catheters, including antimicrobial-coated ones, are vulnerable to biofilm formation [7]. One small study in dogs showed a decrease in biofilm formation with use of a sustained-release chlorhexidine varnish coating on indwelling urethral catheters [8]. In an extensive review of scientific studies in humans comparing types of standard catheters (silicone, latex, hydrogel-coated latex, siliconized latex), none of the standard catheter types was found better than another at reducing bacteriuria [2]. Marked bacterial adherence to polyvinyl (red rubber) catheters compared with other materials has been shown [9]. Antimicrobial or antiseptic-impregnated and hydrophilic catheters may reduce infection in people, but they can be less comfortable and are more expensive; more research is needed [2]. One small veterinary in vitro study and one

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small clinical study in dogs suggest that silver-coated urinary catheters may decrease biofilm formation and bacteriuria [10, 11]; further investigations are needed to determine whether silver coating leads to decreased bacterial cystitis in small animals.

Design

Foley-type urinary catheters have an inflation balloon at the distal end, which, when inflated with sterile water, retains the catheter in the bladder. We prefer these for indwelling use. There are lengths suitable for both males and females (Figure 35.1) and sizes as small as 4 Fr. Some catheters come fitted with an integrated stylet.

For male cats, tomcat-style urethral catheters of 3.5 Fr or 5 Fr do not have an inflation balloon and must be sutured in place. Some have a removable stylet. These catheters are either open ended (having a single opening at the tip) or closed ended (having openings along the side). The open-ended catheter allows retrograde flushing of an obstructed urethra. The rounded tip of the closed-ended catheter might reduce urethral trauma. The Minnesota olive-tip urethral catheter is a 22-gauge metal catheter with an olive-shaped open-ended tip. It is inserted into the distal urethra of an obstructed male cat to assist in retropulsion of debris from the urethra into the bladder.

Stylets

A stylet can make it easier to advance a catheter, especially one of small diameter. However, a stylet can cause mucosal trauma or even rupture of the urethra because it allows excessively vigorous efforts to advance the catheter. A stylet can also be dislodged during placement attempts such that it exits through a side hole in the catheter, thus preventing passage of the catheter into the urethra and traumatizing the mucosa with the exposed stylet tip (Figure 35.2). Use a stylet with proper caution. Make sure that it is properly positioned before attempting insertion. With practice and a good technique, most catheters can be placed without a stylet.

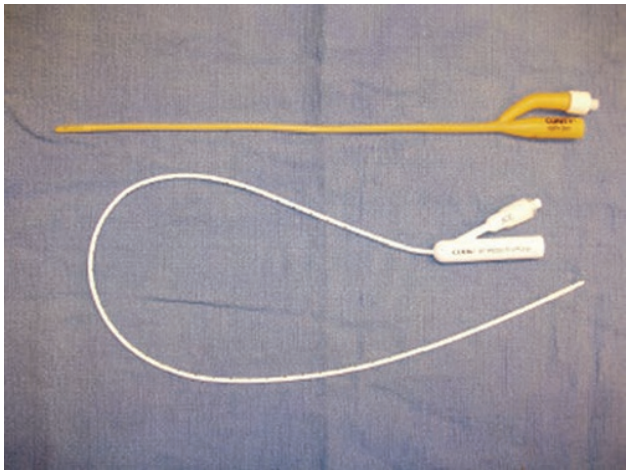


Figure 35.1 Foley catheter in lengths for females (above) and males (below).

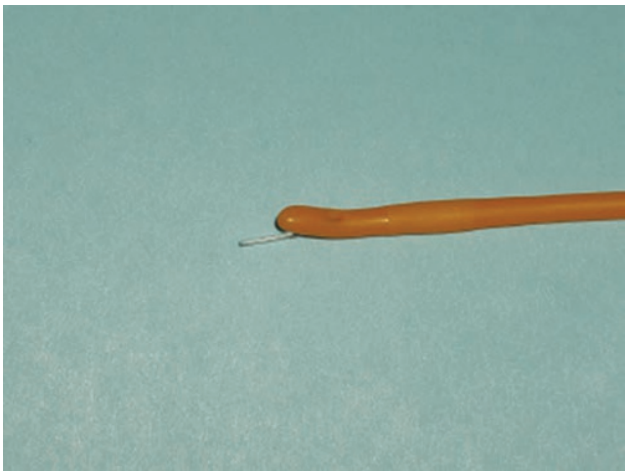


Figure 35.2 Stylet protruding from side hole of a closed-ended catheter.

Diameter

We recommend using the smallest diameter catheter that will achieve good urine flow and will not kink. Approximate requirements are 3.5 Fr or 5 Fr for cats or small dogs, 8 Fr for medium dogs, and 8 Fr to 12 Fr for large dogs.

Length

Catheters should be premeasured so that the operator knows when the tip should reside within the lumen of the trigone – not in the urethra and not at the bladder apex. This is particularly true for catheters without a Foley balloon. In general, the trigone of the bladder lies at or just caudal to the cranial aspect of the ilial wing; this is the point to which we premeasure when placing non-Foley urethral catheters. When correctly placed, the catheter tip rests in the bladder near the trigone. As the patient moves or the bladder size changes, the catheter should remain in the bladder (not retract into the urethra) but should not be so far advanced that it contacts the cranial bladder wall. Catheter tip (or Foley balloon) placement can be confirmed with point-of-care abdominal ultrasound if desired (Chapter 39).

Closed Collection Systems

In human medicine, it is accepted practice and strongly recommended to connect all indwelling urinary catheters to a sterile closed urine collection system [1]. Closed urine

drainage systems facilitate aseptic emptying of the urine from the bag without disconnection from the catheter. They have a check valve to prevent retrograde flow of urine from the bag to the bladder, and often include a sampling port for acquiring urine samples. In veterinary practice, urine collection systems are often created from available materials using a macrodrip fluid line set and a sterile, empty fluid bag. This is considered an open system because, when the fluid bag is filled with urine, it must be disconnected from the tubing and replaced with a new bag. The disconnection carries the risk of introducing bacteria into the system. These systems were reported not to be associated with nosocomial bacteriuria in dogs with short-term urinary catheterization, but the authors cautioned that asepsis must be maintained when changing the collection bag [12, 13]. These open urine collection systems must not be confused with leaving a catheter unattached to any urine collection system, referred to as an open catheter. Forty years ago, it was common practice to treat urethral obstruction in male cats by placing an indwelling urinary catheter and not attaching a urine collection system but instead leaving the catheter open to the environment. Although an experimental study in cats with these open indwelling catheters showed that 20 of 36 developed bacteriuria [14], this practice may still be fairly common [15]. We recommend that all indwelling catheters be attached to a sterile urine collection system. If an open urine collection system using a sterile empty fluid bag as the reservoir is used, it is essential to maintain sterility in setting up and maintaining the system. The purpose-specific closed urine collection systems have advantages as previously discussed.

Catheter Placement and Maintenance

Asepsis and Infection Control

Infection is a complication of urinary catheterization. Bacteria might be introduced into the bladder during catheterization or while maintaining an indwelling system. Organisms can ascend into the bladder from the catheter insertion site or through the catheter lumen from the collection system. In people, urinary tract infection is the most common hospital-acquired infection, and indwelling urinary catheters are the major associated cause [2]. One-time urethral catheterization in healthy dogs resulted in catheter-associated urinary tract infection in 20% of female dogs and 0% of the males [16]. Hospitalized dogs and cats with indwelling urinary catheters develop catheter-associated urinary tract infection at a rate from 10% to 52% [12, 17–22]. Increasing duration of indwelling urinary catheterization increases the risk of catheter-associated urinary tract infection [19, 21, 22]. In the studies with the lowest catheter-associated urinary tract infection, the authors speculated that their use of a strict protocol to maintain asepsis during insertion and maintenance of indwelling catheters contributed to the lower rate [12, 19]. Those protocols are the ones recommended in this chapter.

Antimicrobial treatment of patients with indwelling urinary catheters is variously reported to increase [21], decrease [19], or not affect [22] the development of a urinary tract infection. In human medicine, the Centers for Disease Control and Prevention (CDC) strongly recommend against the routine use of systemic antimicrobials to prevent catheter-associated urinary tract infections either with short-term or long-term catheterization [1]. The available evidence does not support administering prophylactic antimicrobials for an indwelling urinary catheter but does not preclude administering them for other purposes. Moreover, the International Society for Companion Animal Infectious Diseases guidelines for the diagnosis and management of bacterial urinary tract infections in dogs and cats recommends against the routine use of antimicrobials as prophylaxis for urinary tract infection during indwelling urethral catheterization in small animals [23].

The risk of infection is reduced by proper patient selection; aseptic practices by trained caregivers in insertion and maintenance of indwelling catheters and collection systems and by removing indwelling catheters as soon as possible [24]. The known risk of catheter-associated urinary tract infections should be considered in patient management after catheter removal.

Basic Guidelines

Based on our experience and informed by the CDC 2009 guidelines [1] for prevention of catheter-associated urinary tract infections in people, we use and recommend the following guidelines.

1) Ensure that only properly trained caregivers who know the correct aseptic techniques perform catheter insertion and maintenance.
2) Perform hand hygiene before and after insertion or manipulation of catheter or collection system.
3) Use sterile gloves, drape, and sterile lubrication for catheter insertion.
4) Follow a protocol (preferably written) for catheter insertion and indwelling catheter care to ensure consistency in maintaining good technique between caregivers.
5) Use the smallest bore, softest catheter possible consistent with good drainage to minimize urethral and bladder trauma.

6) Secure indwelling catheters to prevent movement and urethral traction.
7) Attach all indwelling catheters to a closed, sterile collection system. If breaks in aseptic technique occur, replace the catheter and collection system.
8) For indwelling catheters attached to a sterile collection system, maintain unobstructed flow, keep the catheter and collecting tubes from kinking, and keep the collecting bag lower than the bladder to prevent retrograde urine flow. For closed systems, empty the collecting bag regularly using a clean container, avoid splashing, and prevent contact of the collecting bag drainage tube with the collecting container. For open systems, follow aseptic technique when breaking the line to replace the urine collection bag. Use sterile gloves and gowns as needed for aseptic technique and for protection of caregivers if public health considerations are present.
9) Do not change indwelling catheters and drainage bags at fixed intervals but rather based on clinical indications such as infection, obstruction, or compromise of the closed collection system.
10) Do not routinely instill antiseptics or antimicrobials into the bladder or collection system.
11) Use good nursing care to minimize contamination of the catheter and periurethral area from contact with soiled hospital surfaces, wound discharge, or feces.

Catheter Placement

Catheter placement must be according to good aseptic practice taking into consideration the patient, environment, operator, and equipment. Assemble the material for placement before beginning the procedure (Box 35.1). The procedure varies according to species and sex. A summary of the steps is provided for female dog or cat (Protocol 35.1), male dog (Protocol 35.2), and male cat (Protocol 35.3).

Catheter Selection

Measure from the vulva or prepucial tip to the bladder to determine the length of catheter needed. The bladder can be located by palpation or estimated to be just cranial to the pubis. Alternatively, estimate the bladder position as the cranial aspect of the proximal femur with the patient in lateral recumbency and the limb in neutral position. Select the softest, smallest diameter catheter that will serve the purpose.

Sedation

Sedation is needed for conscious cats and is often needed for dogs. Patient compliance is important for success, so sedation or anesthesia should be provided as indicated.

Box 35.1 Preparation of Materials for Urethral Catheter Placement

Items Required

- Urinary catheter in sterile wrap.
- If using a Foley catheter: sterile syringe containing appropriate volume of sterile water for Foley balloon inflation (volume indicated on catheter hub)
- If catheter will be indwelling: sterile urinary collection system, cable ties
- Sterile barrier drape
- Sterile gloves
- Examination gloves
- Clean gauze pads soaked in chlorhexidine (or other surgical) scrub
- Clean gauze pads soaked in water to rinse off the scrub
- 0.05% chlorhexidine solution
- Sterile syringe to use to flush the vestibule or prepuce with chlorhexidine solution
- Sterile lubricant
- For females: sterile 2% lidocaine jelly and sterile 1- or 3-ml syringe for injecting jelly into vestibule
- Clippers for fur
- If catheter will be indwelling: suture material, instruments, medical tape, and cable ties for securing catheter and collection system
- Sterile syringe for collection of urine sample, if desired

Patient Preparation

Clip fur from the perivulvar or peripreputial area to establish a 5-cm fur-free zone that can be cleaned and to allow for insertion of sutures if needed. Take care to avoid damage to the skin during clipping. Local irritation causes discomfort, increases risk of skin infection, and decreases patient tolerance of the indwelling catheter. Clean the skin with antiseptic scrub such as chlorhexidine scrub and rinse well with tap water. Do not contact mucosal surfaces with the scrub.

Next, use 5–10 ml of 0.05% chlorhexidine solution (add 6.25 ml of 2% chlorhexidine to 250 ml sterile water) as an antiseptic solution to flush the vulva and vestibule or prepuce five times. Sterile water or saline, or another antiseptic solution at concentrations suitable for mucosal contact, could be used as the flush. The remaining portion of the sterile solution is maintained aseptically and stored for catheter maintenance if the catheter is to be indwelling.

In males, after positioning the patient and extruding the penis, gently clean the area of the external urethral orifice with the solution. Thereafter, do not allow the penis to retract into the prepuce until the catheter has been placed into the bladder and will not be further advanced.

Protocol 35.1 Patient Preparation and Urethral Catheter Placement Using Digital Technique in a Female Dog or Cat

Items Required

- See Box 35.1 for appropriate supplies.

Procedure

1) Ensure that only properly trained caregivers perform catheter insertion, and that sterility is maintained throughout.
2) Sedate patient if indicated.
3) Position the patient.
4) Determine the length of catheter needed to reach the bladder trigone by measurement on the patient. This is generally the length from the urethral opening to a point at or just caudal to the cranial aspect of the ilial wing.
5) Clip fur to maintain an adequate fur-free zone adjacent to the vulva, ideally ≥ 5 cm in each direction from the vulva.
6) Perform hand hygiene and don clean examination gloves.
7) Wash off visible dirt from the patient's perineal area.
8) Perform surgical scrub of the skin surrounding the vulva; rinse off the scrub with water.
9) Flush the vulva and vestibule five times with 0.05% chlorhexidine solution.
10) Instill sterile 2% lidocaine jelly into the vestibule using a 1- or 3-ml sterile syringe.
11) Don sterile gloves (operator).
12) Position barrier drape to provide adequate sterile field: there should be an opening for catheter insertion and a tabletop field caudal to the animal's perineal area where the catheter can rest and remain sterile.
13) Remove catheter from sterile wrap.
14) If the catheter has a stylet, verify that it is in the correct location.
15) If using a Foley catheter, test the balloon with the volume of sterile water indicated on the catheter's hub.
16) While maintaining sterility, mark the catheter or otherwise indicate the length needed to reach the bladder.
17) Lubricate the end of the catheter.
18) Lubricate the operator's gloved palpating finger (generally the index finger of the dominant hand).
19) Insert the gloved palpating finger between the labia of the vulva.
20) If patient size permits, advance the finger to palpate the urethral papilla on ventral midline.
21) If patient size does not permit advancing, leave the finger between the labia of the vulva.
22) Insert the catheter ventral to the finger and advance into the urethra and bladder.
23) If urine is not obtained, verify the proper placement of the catheter in the bladder or reposition if needed.
24) Withdraw the palpating finger without disturbing the placement of the catheter.
25) Inflate the Foley balloon with the indicated volume of sterile water if using a Foley catheter.
26) If the catheter is to be indwelling, attach a sterile collection system; secure the catheter and the collection system to prevent urethral traction and catheter movement.

Catheter Preparation

While maintaining sterility, mark the catheter or otherwise indicate the spot where it will exit the body when the tip is in the bladder. During insertion it is easy to lose track of how much catheter has been inserted. If a flexible catheter curls up in the bladder, it can form a knot that requires surgical removal, so it is important to avoid overinsertion.

If using a Foley catheter with a balloon, test the balloon before insertion (Figure 35.3). The inflation port is imprinted with the volume of sterile water required. Follow the manufacturer's directions regarding the fluid type and volume for inflation. Under or overinflation can cause an asymmetrical balloon that can deflect the catheter tip and cause occlusion or irritation of the bladder wall. Water is recommended because saline can cause crystal formation in the balloon and prevent deflation of the balloon at the time of removal. Inflation with air causes the balloon tip to float in the urine [25]. To test the balloon, fill a syringe with the recommended volume of sterile water and attach it to the inflation port. Inflate and verify that the balloon maintains inflation with the syringe disconnected from the valve; then deflate the balloon in the opposite order. The proper method to deflate the balloon is to attach the empty syringe to the inflation port and let the fluid drain without aspirating. If the balloon does not deflate, reseat the syringe firmly and try again.

Lubrication and Local Anesthesia

In males, apply lubricant to the tip of the catheter and the tip of the extruded penis. As the catheter is passed, continue to apply lubricant on the catheter body at the tip of the penis.

In females, we have found that both local anesthesia and lubrication are important to improve patient compliance. After aseptic preparation we use 2% lidocaine jelly in addition to sterile water-based lubricant. While maintaining sterility, fill a syringe barrel with a suitable volume of sterile lidocaine jelly, replace the plunger, and gently insert the

Protocol 35.2 Patient Preparation and Urinary Catheter Placement in a Male Dog

Items Required

- See Box 35.1 for appropriate supplies.

Procedure

1) Ensure that only properly trained caregivers perform catheter insertion, and that sterility is maintained throughout.
2) Position the patient.
3) Determine the length of catheter needed to reach the bladder by measurement on the patient. This is generally the length from the urethral opening, following the path of the urethra, to a point at or just caudal to the cranial aspect of the ilial wing.
4) Clip fur to maintain an adequate fur-free zone adjacent to the opening of the prepuce; ideally ≥ 5 cm in each direction from the preputial opening, including on the ventral abdomen.
5) Perform hand hygiene and don clean examination gloves.
6) Wash off visible dirt from the prepuce and peripreputial area.
7) Perform surgical scrub of the skin surrounding the prepuce; rinse off the scrub with water.
8) Flush the prepuce five times with 0.05% chlorhexidine solution.
9) Assistant extrudes the penis with gloved hands and maintains it in that position until catheter is placed.
10) Perform surgical preparation of the extruded penis with 0.05% chlorhexidine solution.
11) Don sterile gloves (operator).
12) Position barrier drape to provide adequate sterile field: there should be an opening for catheter insertion and a tabletop field ventral to the dog's abdomen where the catheter can rest and remain sterile.
13) Remove catheter from sterile wrap.
14) If using a Foley catheter, test the balloon with the volume of sterile water indicated on the catheter's hub.
15) While maintaining sterility, mark the catheter or otherwise indicate the length needed to reach the bladder.
16) Lubricate the end of the catheter.
17) Insert the catheter into the penis and gently advance it into the bladder.
18) Verify correct position of the catheter in the bladder radiographically or with point-of-care ultrasound and reposition if needed.
19) Inflate the Foley balloon with the indicated volume of sterile water if using a Foley catheter.
20) If the catheter is to be indwelling, attach a sterile collection system; secure the catheter and the collection system to prevent urethral traction and catheter movement.

catheter tip between the labia and into the vestibule. Then inject the jelly. Lidocaine is absorbed through the mucosa, so limit the total volume to no more than 0.2 ml of 2% lidocaine jelly per kilogram of body weight. Wait 10 minutes for the lidocaine to take effect. Use plain sterile water-based lubricant on a gloved finger and the catheter during insertion, as the lidocaine jelly is not as effective a lubricant as the purpose-made lubricant products. If you plan on using a speculum or otoscope cone technique for placing the catheter, lubricate the instrument and catheter tip, but do not fill the vestibule with jelly because it will obscure visualization of the urethral papilla.

Species and Sex-Specific Instructions

This section outlines instructions for catheter placement in female dogs (digital, guidewire, speculum, and otoscope cone techniques); female cats or small female dogs (guidewire, two-catheter, speculum); male cats; and male dogs. If the catheter is to be indwelling, see the section "Indwelling Catheters" for instructions on securing and maintaining the catheter and collection system.

Female Dog

Female dog urethral catheterization is a skill that takes concentration, correct technique, and some practice. The required skills are similar to those needed to place a venous catheter dependably. In both cases, the target is identified by knowing the relevant anatomy and by palpation. In both cases, the operator must line up the catheter carefully along the long axis of the structure one is attempting to enter (vein or urethra), and the catheter tip must be directed downward. Small, controlled motions are better than large ones. As with any procedure, it is important to know when to stop and seek assistance. The patient is the primary concern.

We prefer the digital technique with the patient comfortably restrained in lateral recumbency. This has been successfully used at the University of California Davis's Veterinary Medical Teaching Hospital and taught in wet labs for many years. Other techniques using a guidewire, speculum, or otoscope cone require specialized equipment, and some may be less comfortable for the animal. However, it is valuable to know more than one way of performing a procedure, and so we also describe these techniques.

Protocol 35.3 Patient Preparation and Urinary Catheter Placement in a Male Cat

Items Required

- See Box 35.1 for appropriate supplies.
- For difficult placements, consider different catheter options, including polypropylene (open- or closed-tip); 22-gauge vascular catheter with the stylet removed; olive-tipped catheters.
- For difficult placements, consider a hydrophilic guidewire (Weasel Wire, Infiniti Medical, Palo Alto, CA) lubricated with sterile isotonic crystalloid.

Procedure

1) Ensure that only properly trained caregivers perform catheter insertion, and that sterility is maintained throughout.
2) Sedate the cat unless it is severely obtunded or already anesthetized.
3) Position the patient.
4) Determine the length of catheter needed to reach the bladder by measurement on the patient. This is generally the length from the urethral opening to a point at or just caudal to the cranial aspect of the ilial wing.
5) Clip fur to maintain an adequate hair-free zone adjacent to the opening of the prepuce, ideally ≥ 5 cm in each direction from the preputial opening.
6) Perform hand hygiene and don clean examination gloves.
7) Wash off visible dirt from the patient's perineal area.
8) Perform surgical scrub of the skin surrounding the prepuce; rinse off the scrub with water.
9) Flush the prepuce five times with 0.05% chlorhexidine solution.
10) Assistant or operator uses gloved hands to extrude the penis and maintain it in that position until catheter is placed. (This step performed after donning sterile gloves if operator will extrude penis.)
11) Perform aseptic preparation of the extruded penis with 0.05% chlorhexidine solution.
12) Don sterile gloves (operator).
13) Position barrier drape to provide adequate sterile field: there should be an opening for catheter insertion and a tabletop field caudal to the cat's perineal area where the catheter can rest and remain sterile. Try to exclude the anus from the opening.
14) Remove catheter from sterile wrap.
15) While maintaining sterility, mark the catheter or otherwise note the length needed to reach the bladder.
16) Lubricate the end of the catheter.
17) Insert the catheter into the penis and advance it toward the bladder while applying caudodorsal traction to the penis to straighten the sigmoid flexure. For difficult placements, consider:
    a) Different catheter types as above
    b) Decompressive cystocentesis prior to catheter placement
    c) Hydrophilic guidewire placement first, followed by urethral catheter placement over the guidewire
18) Verify correct position of catheter in the bladder using radiography or point-of-care ultrasound as desired; reposition if needed.
19) If the catheter is to be indwelling, attach a sterile collection system; secure the catheter and the collection system to prevent urethral traction and catheter movement. As these are usually non-Foley catheters, they are generally secured by suturing the catheter flange to the prepuce and taping the collection system to the ventral aspect of the tail at a length that allows for normal tail movement.



Figure 35.3 Test the balloon of the Foley before placing the catheter.

Anatomy

The relevant anatomy from caudal to cranial is the vulva, vestibule, and vestibulovaginal junction (Figure 35.4). The vagina is cranial to this junction and not entered during this procedure. When attempting to enter the vestibule, avoid the clitoris, which is in a blind-ending pouch located just inside the labia of the vulva (Figure 35.5). The urethral opening is in the vestibule on the ventral midline, at or just caudal to the vaginovestibular junction. Various strips and bands of tissue can form strictures in the vestibule or, uncommonly, in the vulva. They may prevent digital palpation. Nonetheless, a catheter can often still be placed by directing it without palpation as described in the section for small dogs.



Figure 35.4 Contrast vaginourethrogram showing the relevant anatomy in the female dog.



Figure 35.5 The clitoris is in a blind ending pouch, which must be avoided when inserting the finger into the vestibule.

Digital Technique

Positioning The dog should be sedated and provided analgesia, as indicated. Position the patient in lateral recumbency. For a right-handed operator, position the patient in right lateral recumbency and use the right index finger for palpation; for left-handed operators, the opposite is recommended. Let the pelvic limbs rest in a relaxed, normal position. Move the tail out of the way, but do not elevate it because that position can narrow the vulvar opening and make entering through it more difficult.

Palpation First, familiarize yourself with the anatomy in this patient by gentle digital palpation. In this procedure, you are not palpating in the vagina but rather in the vestibule. Insert the palpating finger under the dorsal fold of the vulva in a vertical (not horizontal) orientation and direct it dorsally toward the spine so the fingertip passes under the dorsal fold and avoids the clitoris. Once your finger passes over the pelvic brim, change the angle to one parallel with the spine, and advance the finger cranially into the vestibule until the vestibulovaginal junction is palpated as a circumferential thickening that usually does not allow passage of the palpating finger into the vagina. This is a normal structure, not a stricture. With the fingertip in contact with the vestibulovaginal junction, move the fingertip ventrally and caudally to palpate on ventral midline for the urethral papilla as a soft mound of tissue surrounding the urethral opening, about 0.5 cm caudal to the vestibulovaginal junction in a medium-size dog. It may be obvious or subtle. It is not absolutely necessary to palpate the papilla because the correctly directed catheter will enter the urethra if the catheter is properly aligned and moved forward exactly along the ventral midline.

Placing the Catheter with Digital Technique Once you have familiarized yourself with the anatomy, withdraw the palpating finger and use that hand to grasp the catheter near the tip. Use the non-dominant hand to keep the length of the catheter aligned along the midline. With the palpating finger, re-enter the vestibule as before, taking the catheter tip along (Figure 35.6). Use the palpating finger to guide and the non-dominant hand to guide and advance the catheter. Make sure not to twist or turn the hand as you maneuver the catheter in. Position the catheter tip in the vestibule caudal to the perceived location of the urethral papilla and exactly on the midline. Place the palpating finger back into position over the papilla (but not compressing it) and check the position of the finger and catheter to ensure that all is aligned along the midline. Use the non-dominant hand to keep that portion of the catheter protruding from the vulva lined up along the midline. It is better if the catheter enters between the dorsal rather than the ventral aspect of the vulvar labia because this

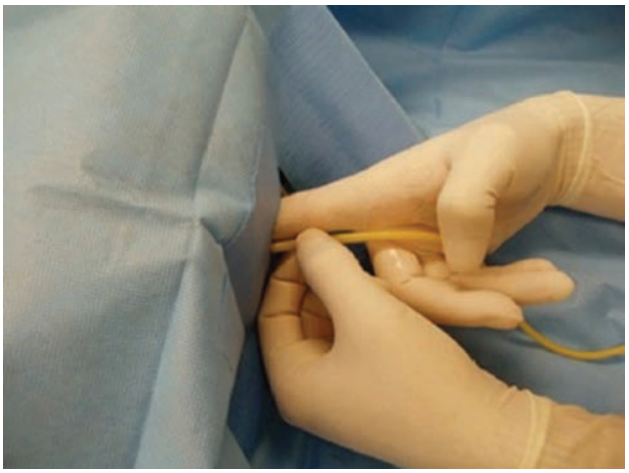


Figure 35.6 Digital technique for a right-handed operator inserting a urethral catheter in a female dog. The dog's head is to the left of the image and tail toward the right; the dog is positioned in right lateral recumbency. Note the angle of the right wrist and index finger to help direct the catheter ventrally – the wrist should be straight or nearly so. The non-dominant (here, left) hand holds the catheter very close to the vulva and makes advancing motions to push the catheter forward. No stylet in needed with this technique.

will help direct the catheter ventrally. Let the tip of the palpating finger hover over the urethral papilla, and gently advance the catheter using the other hand (Figure 35.7). When you think the catheter tip is near to engaging the slit of tissue that is the urethral papilla, flex the palpating finger, press very gently down on the catheter just behind the tip, and extend the finger to push the catheter tip cranially under the slit of tissue (Figure 35.8). Attempt to move the catheter only a few millimeters at a time. Think of sliding a piece of cooked spaghetti along the top of a wet surface. The idea is to get the catheter tip to engage the slit of tissue that is the papilla instead of moving over it (Figure 35.9). Be sure the palpating finger is not pressing down on the urethral papilla



Figure 35.7 Cadaver preparation of sagittal section of female dog in right lateral recumbency with the head toward the left of the image. The cut surface of the midline of the pubic bone is visible. The urethral papilla is seen just cranial to the tip of the catheter. Note that the guiding finger is not pressing down on the urethral papilla. To do so is likely to close off the papilla and prevent the catheter from entering.

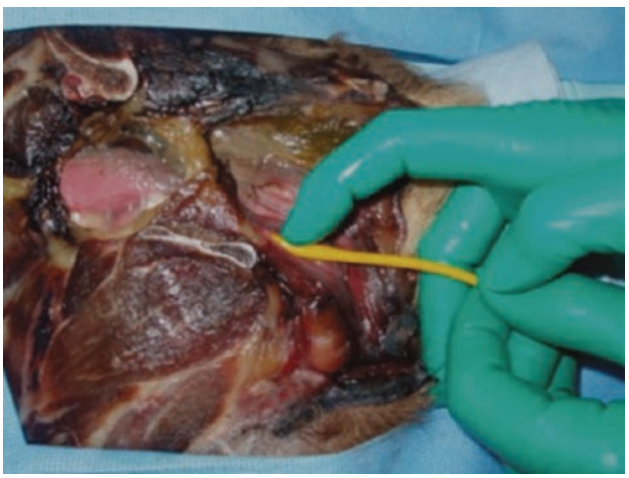


Figure 35.8 Cadaver preparation of sagittal section of female dog in right lateral recumbency with the head toward the left of the image. The cut surface of the midline of the pubic bone is visible. The catheter has just contacted the urethral papilla and the finger is being flexed to help advance the catheter. (See text for description of this technique.)

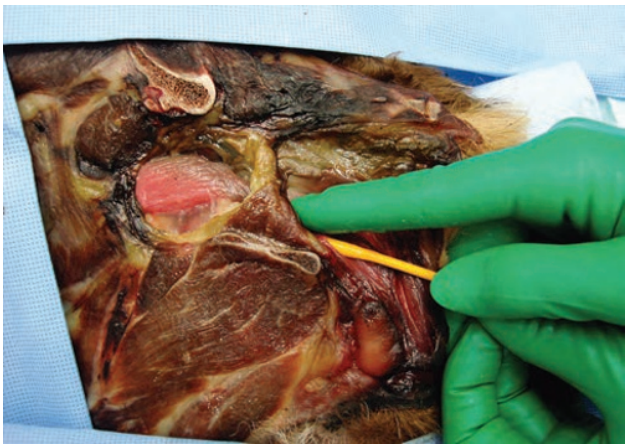


Figure 35.9 Cadaver preparation of sagittal section of female dog in right lateral recumbency with the head toward the left of the image. The cut surface of the midline of the pubic bone is visible. The catheter has now entered the urethral papilla and can be advanced into the bladder.

because that pressure will close the opening. Other operators insert the palpating finger firmly into the vestibulovaginal junction to obstruct it and prevent the catheter from entering it. They then use the non-dominant hand to advance the catheter and let the catheter tip engage the papilla on its own.

During the blind catheterization procedure, the catheter tip will either slip into the urethral papilla and enter the urethra or will slide over the top of it and move cranially toward the vestibulovaginal junction. With experience, you will be able to discern the friction as the catheter passes through the urethra compared with the relatively free passage if it slips over the papilla and moves forward in the vestibule. If it seems that the catheter has entered the papilla, continue to advance it in small increments until the tip of the catheter has reached the premeasured distance to the bladder or until there is good urine flow.

Verify Catheter Placement If urine is not obtained (and the bladder is known to contain urine), palpate along the length of the catheter in the vestibule from caudal to cranial to determine its location. If the catheter has entered the urethra, you will feel the catheter disappear into a hole on the ventral surface of the vestibule, and no catheter will be palpable cranial to the hole. If the catheter has passed over the papilla, you will be able to palpate the catheter going through the vestibulovaginal junction or curling up in the vestibule. If the catheter needs to be repositioned, pay particular attention to meticulous technique in aligning the entire catheter along the vestibule's ventral midline and keeping it on the midline as you advance it. Be sure to withdraw it sufficiently caudally to ensure that the urethral papilla has not already been passed before the catheter is advanced again. If you are not sure of the location of the papilla, advance the catheter on ventral midline in small increments. If the catheter is correctly oriented, it is likely to engage the urethral papilla on its own.

If you think the catheter tip is in the bladder, but no urine is flowing, attach a sterile syringe to the catheter and gently aspirate for urine or infuse sterile saline and aspirate. If injected fluid comes back around the vulva, the catheter tip is likely in the vestibule and should be repositioned. One can also place an ultrasound probe over the bladder and either see the tip of the catheter in the bladder or see the flow of fluid as it is flushed through the catheter.

Speculum or Otoscope Cone Technique

Preparation is as described for the digital technique except the vestibule is not filled with sterile anesthetic lubricant because that would obscure visualization. Lubricate the device and the catheter before insertion.

Patient Positioning Sedation is generally required for these techniques. The patient may be positioned in lateral or sternal recumbency. Some operators place the patient sternally and drape the pelvic limbs off the end of a table.

Speculum A separate light source is needed, preferably a headlamp. The correct orientation for a speculum is with the handles pointing dorsally (toward the spine), not down. This allows an assistant to grasp the handles once the speculum has been inserted into the vestibule with the assistant's hand out of the way of the operator.

Otoscope Cone Attach the cone to the otoscope handle in the usual way and use the handle to manipulate the cone into the vestibule. Visualize the urethral papilla, pass the catheter through the cone, and advance it into the urethral papilla. Once the catheter is placed into the bladder and the cone removed from the handle, the cone is left on the portion of the catheter outside the body because the external end of the catheter will not fit through the tip of the cone. Although this may look a little odd, it does not cause problems (other than the potential to act as a fomite), and the cone is retrieved when the catheter is removed.

Placing the Catheter Insert the speculum (in closed position) or otoscope cone between the labia of the vulva, directing it first vertically to avoid the clitoris and then horizontally to enter the vestibule. If using a speculum, open the blades and have an assistant hold it in position. Look through the device and locate the urethral papilla as a slit of tissue on the ventral floor of the vestibule slightly caudal to the vestibulovaginal junction. Because you will not be able to touch the catheter tip to guide it into the papilla, a stylet will be required unless you are using a catheter made of stiff material, such as polypropylene.

Insert the catheter between the blades of the speculum or through the otoscope cone and advance it into the urethral papilla and into the bladder. If you cannot see the papilla, you may still be able to place the catheter by gently advancing the catheter tip into the mucosal tissue in the area where the papilla should be located, in other words, by using the tip of the catheter as a probe to identify the papilla. When the catheter tip is positioned in the bladder, inflate the balloon if there is one, and gently withdraw the catheter to seat the balloon at the neck of the bladder. Withdraw the device. If using an otoscope, disconnect the cone from the handle and leave the cone on the catheter outside the body.

Guidewire Technique

Recently a guidewire-assisted technique has been described for placement of urethral catheters in female dogs [26]. Patient preparation and positioning are identical to that described above for the digital technique; sedation is likely to be required because the guidewire's introducer is semi-rigid and may be more uncomfortable to the patient than a more flexible catheter. A specialized catheter kit is available (MILA International, Inc., Florence, KY) that contains a semi-rigid, flexible-tip introducer catheter with an open end through which a flexible, stainless-steel guidewire is passed into the urethra to facilitate finding and entering the urethral opening. After being lubricated with isotonic fluid, the introducer catheter is inserted using the digital or speculum technique; the guidewire is then advanced through the introducer and the introducer removed. A specialized fluid-lubricated, open-ended, balloon-tipped (Foley) catheter is then fed over the guidewire and into the urinary bladder; the guidewire is removed and the remainder of the procedure is the same as for other Foley catheter placements.

Female Cat or Small Female Dog

If the patient is too small to allow digital palpation of the urethral papilla, the catheter can nonetheless often be placed successfully.

Anatomy

The relevant anatomy for female cats is as described for female dogs, except the urethra opens to the floor of the vestibule in a groove that facilitates catheterization.

Positioning

Sedation is generally required in female cats. Position the patient in lateral recumbency. For cats, some operators prefer dorsal recumbency with the pelvic limbs drawn cranially to expose the vulva. However, this position tends to decrease the opening of the vulva and can make it more difficult to insert the catheter into the vestibule.

Placing the Catheter

Follow the preceding instructions for preparation for insertion. Then gently separate the labia of the vulva and pass the catheter between the most dorsal aspects of the labia,



Figure 35.10 Urinary catheter placed by blind (without internal palpation) technique. Note that the catheter is entering the dorsal aspect of the vulva and directed ventrally to engage the urethral papilla which helps avoid the more ventrally located clitoral fossa.

directing it to the ventral midline aiming toward the bladder (Figure 35.10). Even in small patients, the operator can usually insert at least the tip of the finger between the labia to help direct the catheter ventrally. The urethral papilla is very close to the vulva, and most missed insertions occur because the catheter tip has already passed the urethral opening before it is properly positioned on the ventral midline. The operator can usually feel the slight resistance as the catheter tip enters the urethra. Advance the catheter into the bladder.

Recently, a two-catheter technique has been described for urethral catheter placement in anesthetized small female dogs and female cats [27]. In this technique, a larger-bore red rubber (polyvinyl chloride) catheter is first advanced into the vestibule until abrupt resistance is noted. In the study, a 10 Fr red rubber catheter was used for cats and 18 Fr for small dogs. Once this large catheter can be inserted no farther, it is grasped in the operator's nondominant hand and flexed dorsally. The operator then introduces the intended indwelling catheter into the vestibule along midline, ventral to the larger catheter. In this study, authors reported more successful catheterizations using this two-catheter technique compared with the traditional blind insertion method, regardless of prior familiarity with the technique.

Verify Catheter Placement

If you think that the catheter tip is in the bladder but no urine is flowing, attach a sterile syringe to the catheter and gently aspirate for urine, or infuse sterile saline and aspirate. If injected fluid comes back around the vulva, the catheter tip is likely in the vestibule and should be repositioned. You can also place an ultrasound probe over the bladder and either see the tip of the catheter in the bladder or see the flow of fluid within the bladder as it is flushed through the catheter.

Male Cat

Many male cats in need of a urinary catheter have urethral obstruction. Sometimes the obstructing material is at the distal tip of the penis and can be removed by gently massaging the most distal part of the penis to dislodge obstructing substances. If successful, this will result in immediate urine flow because of increased pressure from the overdistended bladder.

Anatomy

The feline prepuce is a short, fur-covered sheath facing caudally and covering the nonerect penis. The penis is directed caudoventrally and covered by the prepuce. When the penis is extruded sufficiently by reflection of the prepuce off the surface of the penis, about 1–1.5 cm of penile tissue can be seen. Penile barbs are visible near the tip of the penis in the non-castrated male cat.

In its course from the bladder to the tip of the penis, the urethra has a marked sigmoid flexure. To pass a catheter up the urethra, the penis must be drawn caudally and dorsally to straighten out the urethra.

Positioning

Heavy sedation to general anesthesia is generally required unless the cat is severely obtunded. Position the patient in lateral or dorsal recumbency. In dorsal recumbency, the pelvic limbs can be drawn cranially to expose the prepuce.

The goal is to extrude the penis caudally and dorsally out of the prepuce and keep it in this position as the catheter is passed. To extrude the penis, gently grasp the prepuce with thumb and finger and press gently onto the ischial arch to provide a firm base for the procedure and to align the penis parallel to the spine. If the penis is extruded facing in a ventral direction, it will be difficult to pass the catheter. Then, gently move the prepuce cranially to expose the penis. The penis should now be extruded far enough to see the reflection of the prepuce off the penile surface, about 1–1.5 cm from the tip. The operator can digitally grasp the penis near the reflection of the prepuce to stabilize it or can use an instrument to grasp the preputial tissue (not the penile tissue) at the site of its reflection off the penis. Hold all structures in a horizontal straight line with the tip of the penis facing caudally, not ventrally, and apply gentle but firm traction caudodorsally to straighten the sigmoid flexure. Insert the lubricated catheter into the tip of the penis, and gently advance into the bladder without losing control of the extruded penis. As the catheter is moved cranially, continue to put traction on the penis in a caudodorsal direction; otherwise, the catheter might not advance into the bladder. Once the catheter is positioned

properly in the bladder, allow the penis to withdraw into its normal position within the prepuce.

Male Dog

Anatomy

The prepuce is the tubular sheath of integument covering the nonerect penis. The penis in nonerection is entirely withdrawn into the prepuce. Within the penis, the os penis surrounds the dorsal surface of the urethra. The distal end of the os penis has a fibrocartilaginous projection that is curved slightly ventrally.

Positioning

With the male dog in left lateral recumbency (for a right-handed operator) the assistant stands at the patient's spine with the patient's head on the assistant's left, reaches over the abdomen, presses the left hand against the body wall at the place where the prepuce reflects from the ventral body wall, and exerts pressure caudally. With the right hand, the assistant grasps the os penis through the prepuce at its most proximal aspect (meaning away from the tip) and pushes the penis out of the prepuce while moving the left hand more caudally to stabilize it and keep the penis as parallel to the spine as possible. The penis must now be held in this position and not allowed to withdraw into the prepuce while the catheter is being placed. Gently wipe the tip of the penis with 0.05% chlorhexidine solution (Protocol 35.2).

Placing the Catheter

Gently insert the catheter tip into the fibrocartilaginous projection of the os penis and then direct it ventrally to advance within the urethra. Orienting the catheter in a direction parallel to the body wall and slightly ventrally once the fibrocartilaginous tip has been negotiated facilitates passage. The penis must remain exposed during catheter passage to assure that the catheter tip is entering the penis and to prevent contamination of the catheter. Resistance is usually felt as the catheter tip enters the os penis because it is narrow at this site and again as the catheter changes direction at the ischial arch and passes through the prostatic section of the urethra. Use the softest catheter possible to ease passage and minimize discomfort. Advance the catheter into the bladder.

Care and Maintenance of Indwelling Catheter Systems

Securing the Catheter and Collection System

Once the catheter is properly placed, it must be secured if it is to remain indwelling. For Foley catheters with a balloon, the inflation port is imprinted with the volume of sterile water required. Fill a syringe with this amount and firmly attach it to the inflation port. Slowly inflate the balloon and thereafter, gently withdraw the catheter to seat the balloon at the neck of the bladder. If the patient exhibits discomfort during inflation, the balloon may be in the urethra. Deflate the balloon, reposition the catheter, and try again. Point-of-care ultrasound guidance can be used to visualize a Foley balloon in the urinary bladder.

Catheters without an inflation balloon must be secured at the site where the catheter exits the body to keep the catheter tip in the bladder. To do this, first dry the catheter and apply an adhesive tape butterfly on the catheter where it exits the vulva or prepuce. Catheters can easily slip through a tape butterfly unless the tape is kept closely adhered to the catheter surface, so we recommend placing an encircling suture around the catheter: insert the needle through the tape as close as possible to the catheter, draw some suture material through, and insert the needle up through the tape on the opposite side of the catheter. Tie the suture securely but do not occlude the catheter. To secure the adhesive butterfly to the patient, we recommend placing stay sutures in the patient and then suturing the adhesive butterfly to them. Some veterinary-specific catheters come fitted with a plastic disk with prepared holes for suturing to the prepuce.

All indwelling catheters must be attached to a sterile collection system. Cable ties can be useful to provide extra security, especially between the catheter and the collection system, if an integrated Luer-locking mechanism is not present. Secure a portion of the collection system tubing to the patient's leg, tail, or ventral abdomen (using tape or sutures) to prevent dislodging or discomfort caused by pulling on the catheter as the patient moves.

Management of Indwelling Urinary Catheter and Collection System

Make sure that personnel handling catheters and collection systems are properly trained and perform hand hygiene before and after handling the system. Inspect the system several times a day. Make sure all connections are secure. Use good nursing care to minimize contamination of the catheter and periurethral area from contact with soiled hospital surfaces, wound discharge, or feces. Maintain unobstructed flow, keep the catheter and collecting tubes from kinking, and keep the collecting bag lower than the bladder to prevent retrograde urine flow.

Catheter Care

Perform catheter care every eight hours or whenever the system is visibly soiled. Use surgical scrub followed by water rinse to clean any visible soiling on the exposed

portion of the catheter, the collection system, or the patient's skin in the perivulvar or peripreputial area. Do not let scrub contact mucosal surfaces. Wipe the exposed portion of the catheter and the skin of the perivulvar or peripreputial area with 0.05% chlorhexidine solution. This solution can be that saved from the preparation for catheter placement as described earlier. Flush the prepuce or vulva and vestibule five times with the 0.05% chlorhexidine solution using a sterile syringe.

Emptying or Changing the Urine Collection Bag

Wash hands and put on examination gloves. Use gowns and barriers as needed for aseptic technique or if public health considerations are present. For closed systems, drain the urine into a clean container. Do not let the drainage tube contact the container and avoid splashing. Close the drainage tube.

For open systems, assemble the needed materials to replace the bag while maintaining sterility. Disconnect the full bag from the macrodrip set and aseptically attach an empty sterile fluid bag.

Do not change indwelling catheters and drainage bags at fixed intervals but rather based on clinical indications such as infection, obstruction, or compromise of the closed collection system (Protocol 35.4).

Basic Techniques for Difficult Catheterizations

Decompressive Cystocentesis for Urethral Obstruction

For difficult urethral catheterizations in cats and dogs with urethral obstruction, decompressive cystocentesis may be considered. Decompressive cystocentesis may decrease the anterograde pressure applied to an obstructive object (stone, plug, grit) in the urethra, allowing easier retrograde urethral catheter passage. Also, if repeated urethral catheterization attempts are unsuccessful and, for example, a surgical procedure is planned to resolve or circumvent the obstructive process, decompressive cystocentesis can be used as a bridge therapy to definitive care.

Decompressive cystocentesis appears to be relatively safe in cats [28, 29], though little information is available about the procedure in dogs. It is unclear whether the technique improves one's ability to pass a retrograde urethral catheter, but it should be considered in challenging cases in which other options pose challenges.

We prefer to perform decompressive cystocentesis using point-of-care ultrasound guidance to assess for free abdominal fluid prior to and following the procedure.

Protocol 35.4 Indwelling Catheter Maintenance

To be performed by trained veterinary healthcare workers every eight hours or whenever the system is visibly soiled.

Items Required

- Water
- Gauze pads
- Surgical scrub
- Sterile syringe
- Examination gloves
- 0.05% chlorhexidine solution
- Empty, sterile fluid bag if the current bag is full and the patient does not have a purpose-made, closed urinary collection system attached

Procedure

1) Gather supplies.
2) Perform hand hygiene and don clean examination gloves.
3) Minimize contamination of the catheter, collection system, and periurethral area from contact with soiled hospital surfaces, wound discharge, or feces during handling and procedure.
4) Make sure that the catheter and the collection system are properly secured. Keep the collection bag lower than the patient to prevent retrograde flow of urine.
5) Use surgical scrub followed by water rinse to clean soiling on catheter, collection system, or the perivulvar or peripreputial area as needed.
6) Wipe the exposed portion of the catheter and the skin of the perivulvar or peripreputial area with gauze sponges soaked in 0.05% chlorhexidine solution.
7) Clean the perivulvar or peripreputial area with 0.05% chlorhexidine-soaked gauze sponges and flush the vulva and vestibule or prepuce with 0.05% chlorhexidine solution using the syringe.
8) Empty or change the urine collection bag as needed.
   d) For the closed system, open the urine drainage spout and drain the urine into a container. Avoid touching the container with the drainage tubing and avoid splashing. Close the urine drainage tube.
   e) For the open system, disconnect the full bag from the macrodrip and sterilely attach an empty sterile fluid bag.
9) Perform hand hygiene after discarding urine and examination gloves.

With ultrasound guidance, a 22-gauge vascular catheter of appropriate length can be inserted percutaneously into the urinary bladder, the stylet removed, and the flexible vascular catheter attached to an extension set and syringe. The bladder can then be emptied partially or completely, and the percutaneous catheter removed.

Retropulsion for Urethral Obstruction

Retropulsion is the process of expanding the urethra with fluid to flush an obstructing substance retrograde and deposit it back into the bladder. It is not desirable to use a catheter to force an obstructing substance retrograde because this may damage or even rupture the urethra. Retropulsion is most commonly needed in males and required uncommonly in females because their urethra is shorter and of larger diameter. The goal of retropulsion is to expand the diameter of the urethra with fluid to suspend the obstructing substance in a fluid column that will carry it back into the bladder. Be aware that the urethra can be ruptured by overly aggressive flushing technique or by overly aggressive attempts to advance the catheter against an obstruction (Protocol 35.5).

For male dogs, a supplement to retropulsion is to also occlude the urethra proximal to the obstruction (Figure 35.11). To occlude the urethra proximal to the obstruction, an assistant inserts a gloved finger into the rectum and occludes the urethra by compressing it ventrally onto the pelvic floor. In a large dog, two fingers may be inserted to better trap the urethra against the pelvis. Briskly inject 5–20 ml of flush into the urethra, depending on the patient's size. The assistant occluding the urethra should be able to feel the urethra dilate. The assistant then abruptly releases the urethral occlusion as injection of flush continues and the operator attempts to advance the catheter.

If unsuccessful, and the patient is not already anesthetized, consider providing full general anesthesia before retrying the procedure (Protocol 35.6).



Figure 35.11 Retropulsion with occlusion of the urethra per rectum in a male dog.

Protocol 35.5 Retropulsion for Urethral Obstruction

Items Required

- See Box 35.1 for appropriate supplies

Procedure

1) The procedure can be painful. Provide analgesia and sedation as indicated. The relaxation of the urethra that occurs during general anesthesia might be needed for difficult obstructions.
2) Evaluate bladder size to determine whether it would be safe to add more fluid. If not, perform cystocentesis before retropulsion. If performed correctly (small-gauge needle inserted as atraumatically as possible), cystocentesis even of a distended bladder is probably lower risk than adding fluid to an already pathologically distended bladder.
3) Fill a syringe (5- to 20-ml size) with sterile saline for flush.
4) Follow all the protocols as previously described to maintain sterility. Pass the catheter as far as possible up the urethra. If the catheter cannot be advanced far enough to seat it inside the urethra, try a smaller or stiffer catheter. Remember that in male cats, the penis must be pulled caudally to straighten the sigmoid flexure in the urethra before the catheter can be passed. For male cats, an olive-tip catheter or a 22-gauge venous catheter (with the stylet removed) may be easier to place in the most distal urethra. Once the obstruction is relieved, a regular catheter can be placed indwelling.
5) Attach the syringe to the end of the catheter.
6) Occlude the tip of the penis around the catheter with digital pressure to prevent the flush from flowing back out.
7) An assistant should briskly inject flush while the operator gently advances the catheter. If the catheter can be advanced, continue to inject fluid until the catheter tip is in the bladder. Keep track of the volume of flush injected, do not overdistend the bladder, and decompress the bladder by cystocentesis if needed.

Protocol 35.6 Retropulsion with Proximal Urethral Compression for Challenging Urethral Obstruction in Male Dogs

Items Required

- See Box 35.1 for appropriate supplies
- Sterile flush solution
- Several sterile syringes (for flushes)
- Two assistants
- Examination gloves
- Lubricant

Procedure

1) Gather necessary supplies.
2) Ensure that only properly trained caregivers perform the procedure, and that sterility is maintained throughout.
3) Consider heavy sedation or anesthesia.
4) Evaluate bladder size. Perform decompressive cystocentesis if needed.
5) Assure that sufficient personnel are available to perform the various aspects of the procedure: catheter insertion by operator; flushing the catheter by assistant 1; digital occlusion of the proximal urethra per rectum by assistant 2; and patient restraint or anesthetic monitoring, as appropriate by assistant 3.
6) Follow procedure for catheter insertion and advance the catheter gently until the obstruction is reached.
7) Operator occludes tip of penis around the catheter to prevent backflow of flush.
8) Assistant 1 injects sterile flush briskly while operator gently attempts to advance the catheter.
9) Occlusion of the urethra per rectum, if step 7 was unsuccessful:
   a) Assistant 2 inserts a gloved finger into the rectum and prepares to apply firm ventral digital pressure on the proximal urethra to occlude it.
   b) Repeat step 6.
   c) Assistant 2 firmly occludes the urethra per rectum.
   d) Assistant 1 injects flush briskly and continues injecting.
   e) Assistant 2 feels the urethra dilate and abruptly releases the pressure while assistant 1 continues the flush and the operator attempts to advance the catheter.
10) If unsuccessful and the patient is not yet anesthetized, consider anesthesia before repeating the procedure.

Deflating a Foley Balloon

The correct method for deflating a Foley balloon is as follows. Attach a Luer slip syringe to the catheter valve. Allow the pressure in the balloon to force the water into the syringe to deflate the balloon completely. Do not apply aspirating pressure at this time. If the balloon does not deflate, reseat the syringe gently and try again. If unsuccessful, reposition the patient; ensure there is no traction on the catheter, and then try again. If the balloon still does not deflate, apply gentle, slow aspiration, remembering that rapid or forceful aspiration can collapse the inflation tube and prevent balloon deflation. If the balloon cannot be deflated, cut the channel through which the balloon was inflated to allow fluid to egress, which will deflate the balloon. In the rare situation in which cutting the channel does not facilitate balloon deflation, the Foley balloon can be aspirated percutaneously with ultrasound guidance in the anesthetized animal to allow catheter removal.

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36

Peritoneal Dialysis

Michael D. Santasieri, Carolyn Tai, and Mary Anna Labato

Despite its long-term use in human medicine for the treatment of both acute kidney injury (AKI) and chronic kidney disease, the use of renal replacement therapies in veterinary medicine is still uncommon. The most likely explanation for this is that owners are deterred by the relatively high cost and clinicians by the relatively high amount of specialized knowledge required for these therapies. However, peritoneal dialysis is an accessible therapy for most specialty hospitals and may be less financially taxing than extracorporeal hemodialysis modalities.

Indications

In veterinary medicine, the primary indication for peritoneal dialysis is AKI. AKI is defined as the inability of the kidneys to regulate water and solute balance, which is typically recognized by an accumulation of nitrogenous wastes such as urea and creatinine [1]. Dialytic therapies may be the only option to treat severe uremia that is unresponsive to fluid therapy and post-renal uremia resulting from ureteral obstruction [2]. Severe electrolyte derangements, refractory metabolic acidosis, and the metabolites and neurotransmitters responsible for the clinical signs of hepatic encephalopathy are all responsive to peritoneal dialysis. Volume overload, which can occur iatrogenically in oliguric and anuric patients with AKI, or secondary to congestive heart failure, can also be treated with peritoneal dialysis through the use of hyperosmolar dialysate to cause ultrafiltration.

Peritoneal dialysis can also be used for the treatment of some toxicities if the offending toxin is diffusible across the peritoneal membrane. Such toxins include ethylene glycol and its toxic metabolites, ethanol, and barbiturates [2, 3]. Hemodialysis is about 75% more efficient than peritoneal dialysis, so it should be considered as a first-line treatment if available [3]. However, if transfer to a hemodialysis center is not possible due to local availability or cost, peritoneal dialysis is a reasonable alternative. It may also be preferable in cases where vascular access is difficult to obtain, where refractory hypotension or small patient size (Figure 36.1) make the risks of performing hemodialysis greater than the potential benefits.

Even though peritoneal dialysis is less efficient and arguably as labor intensive as hemodialysis, there are still some definite therapeutic advantages. Peritoneal dialysis is technologically simple, relatively inexpensive, and more efficacious in removing uremic middle molecules that are in the 500–15 000 Da range, including parathyroid hormone, leptin, β-2 microglobulin, and tumor necrosis factors [4, 5]. Hemodialysis is more efficacious overall at altering water and solute balance but requires a high level of expertise as well as expensive equipment and supplies.

Performing Peritoneal Dialysis

Dialysis is defined as the transfer of water and solutes from one compartment to another across a semipermeable membrane. Peritoneal dialysis uses the lining of the abdominal cavity, the peritoneum, as this membrane. Dialysate solution is instilled into the abdominal cavity, and allowed to dwell so that solutes can pass through the peritoneum, and then the solution is drained and discarded. This is repeated as frequently as needed for resolution of clinical signs. This process is governed by diffusion and osmosis, convection, and ultrafiltration.

Diffusion is the movement of molecules from an area of high concentration to an area of low concentration in an attempt to reach equilibrium. *Osmosis* refers to diffusion of water across a semipermeable membrane from an area of low solute concentration to an area of high solute

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Figure 36.1 Peritoneal dialysis is an effective dialysis modality for patients that are too small for extracorporeal therapies, such as small dogs and cats, or exotic mammals like rabbits and ferrets.

concentration. For diffusible molecules, the processes of solute diffusion and water osmosis ultimately results in equal concentrations on both sides of the membrane.

Ultrafiltration is the movement of water across a semipermeable membrane caused by differences in hydrostatic pressure or osmolality in the two compartments, which results in excess fluid removal from the patient. This is accomplished during peritoneal dialysis by instilling dialysate into the peritoneal cavity that is of a higher osmolality than plasma, usually accomplished by adding dextrose or glucose to the dialysate.

Convection is the movement of solutes when carried along with the flow of water (called "solute drag") during ultrafiltration. Convection does not play a significant role in peritoneal dialysis but can explain the loss of some higher weight molecules, such as albumin, during aggressive ultrafiltration.

The necessary equipment needed for peritoneal dialysis consists of items that are readily available in most specialty practices (Box 36.1). Catheters designed specifically for peritoneal dialysis are recommended, but alternatives are listed.

Box 36.1 Items Required for Performing Peritoneal Dialysis

- Peritoneal dialysis catheter
- Dialysate solution
  - Note that some commercially available dialysate solutions require brand-specific adaptors to "spike" the bag
- Volumetric infusion pump
- Fluid warmer and/or inline fluid line warmer
- 2 sterile intravenous fluid administration sets
- Sterile collection bags
- Three-way stopcock
- Sterile gauze sponges
- 2- to 4-inch conforming gauze bandage
- 2- to 4-inch cast padding
- 2- to 4-inch cohesive bandage (e.g. Vetrap)
- (optional) 2- to 4-inch fabric elastic tape (e.g. Elastikon)
- Waterproof bandage tape
- Chlorhexidine solution 1–2%
- Sterile gloves
- Exam gloves (non-sterile)

Catheter Types

There are many different types and brands of peritoneal dialysis catheters (Table 36.1); most are variations of a fenestrated silicone elastomer or polyurethane tube [6]. According to the International Society for Peritoneal Dialysis, the ideal peritoneal dialysis catheter "provides reliable, rapid dialysate flow rates without leaks or infections" [7]. For acute short-term dialysis, a percutaneously placed catheter can be used. These catheters are typically functional for 12–36 hours before they become occluded with omentum, which prevents dialysate from draining out of the body. Peritoneal dialysis catheters for long-term use should be placed surgically. Catheters explicitly made for this purpose often have polyethylene terephthalate cuffs, usually referred to by the brand name Dacron™ (Invista, Wichita, KS), to promote fibrous attachments at the peritoneal and cutaneous exit sites. The use of these cuffs has been shown to reduce the incidence of dialysate leakage

Table 36.1 Some peritoneal dialysis catheter options for veterinary patients.

Catheter	Manufacturer
Argyle™ Tenckhoff peritoneal dialysis catheter Argyle™ Swan Neck Missouri peritoneal dialysis catheter Argyle ™ Swan Neck Curl Cath peritoneal dialysis catheter	Covidien, LLC www.covidien.com
Universa® Malecot drainage catheter	Cook Medical, Inc. www.cookmedical.com
Blake® Silicone drain	Ethicon, Inc. www.ethicon.com
Percutaneous peritoneal drain kit (peritoneal dialysis) Jackson Pratt drain with trocar Silicone chest tube Centesis catheter (fenestrated)	MILA International, Inc. www.milaint.com

and peritonitis in both human and veterinary patients [1, 3, 4, 8–13]. Owing to the high prevalence of omental occlusion of peritoneal dialysis catheters, it is highly recommended to perform an omentectomy at the time of placement.

Historically, the authors have found the T-fluted catheter (Ash Advantage, Ash Access Technology, Lafayette, IN) to be most successful for long-term peritoneal dialysis; however, at the time of writing, these catheters are no longer manufactured. This catheter featured long channels (called "flutes") rather than fenestrations to prevent omental adhesion and to decrease resistance during the influx and efflux of fluid [3, 14].

A readily available alternative is the Blake® surgical drain (Ethicon Inc., Somerville, NJ). Although not specifically manufactured for peritoneal dialysis, this catheter would be expected to perform similarly owing to its employment of the same type of "flutes" as the T-fluted catheter. In a retrospective study conducted in 2008, the use of a Blake surgical drain combined with an intermittent closed suction system was an effective peritoneal dialysis system for 100% of the cats included in the study [15]. Although the study was underpowered (only six cases were included), it demonstrates that the Blake surgical drain may be a reasonable alternative to the T-fluted catheter. The main disadvantage is that there are no Dacron cuffs to help prevent leakage from the peritoneal space.

The Tenckhoff catheter is a straight catheter with either a straight or curled fenestrated end, and one or two Dacron cuffs. It is the most frequently used catheter for humans undergoing peritoneal dialysis, and versions are made by several manufacturers (Figure 36.2) [5–7, 16]. We have

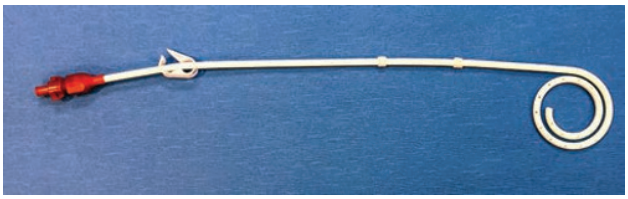


Figure 36.2 A Tenckhoff-style catheter with a curled tip (Medcomp, Harleysville, PA).

found the Swan Neck Missouri Catheter (Covidien, Mansfield, MA) to be successful for many long-term applications. It consists of a fenestrated tube that is curled at the end, with two Dacron cuffs and a hard silicone bead to help prevent dialysate leakage. However, because of these cuffs and bead, this catheter requires surgical removal. The Swan Neck Curl Cath (Covidien, Mansfield, MA) is a similar catheter that does not include the silicone bead but still has two Dacron cuffs (Figure 36.3).

Based on our experience, although the Tenckhoff, Swan Neck Missouri, Swan Neck Curl Cath, and T-fluted catheters are all designed to be placed by either laparoscopy or blind trocarization in humans, we recommend placing them surgically in veterinary patients for better results.

For emergent, short-term peritoneal dialysis, a percutaneously placed catheter can be considered. The authors have the most experience with the Malecot Drainage Catheter (Cook Medicial, Bloomington, IN), which is easy to place with minimal experience and without anesthesia (Figure 36.4). However, we find that it only tends to work for 12–36 hours before becoming occluded with omentum. Other alternatives include the previously mentioned Blake surgical drain, the Jackson Pratt surgical drain, fenestrated chest tube, or centesis catheter (Figure 36.5). At the time of

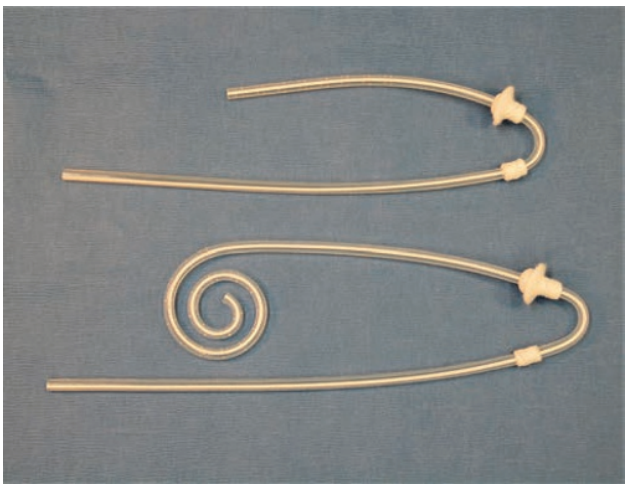


Figure 36.3 Argyle™ Swan Neck Missouri Catheter and Argyle Swan Neck Curl Cath (Covidien, Mansfield, MA).

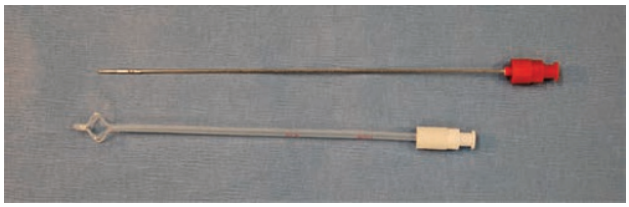


Figure 36.4 Universa® Malecot Drainage Catheter (Cook Medical, Bloomington, IN).

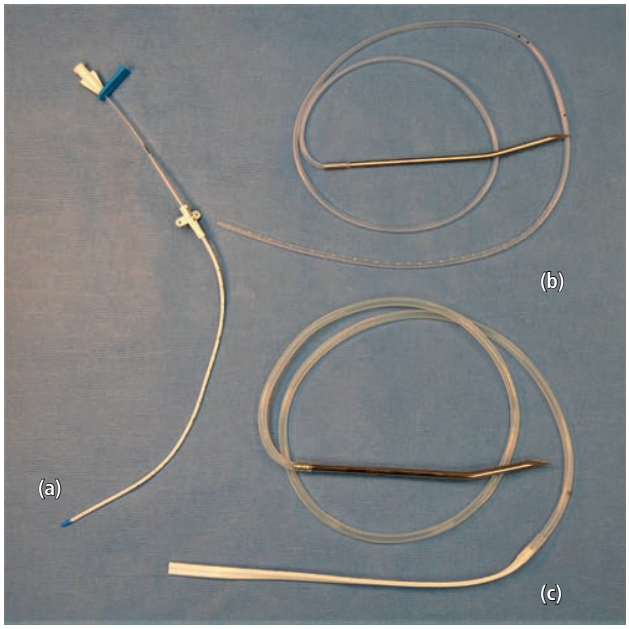


Figure 36.5 Improvised peritoneal dialysis catheters: (a) chest tube (MILA International, Florence, KY); (b) Jackson Pratt drain (Jorgensen Labs, Loveland, CO); (c) Blake® silicone drain (Ethicon, Somerville, NJ).

this writing, MILA International Inc. has just released a new "percutaneous peritoneal drain kit" that looks potentially helpful for short-term use, but the authors have no personal experience with this product [17].

Site Selection, Preparation, and Catheter Placement

For emergent percutaneous placement of a peritoneal dialysis catheter, the patient should be administered mild sedation and a local anesthetic block. An indwelling urinary catheter should be placed to ensure that the urinary bladder is empty so that the trocar does not inadvertently pierce it. With the patient restrained in dorsal recumbency, the abdomen should be shaved from the xiphoid to the pubis and aseptically cleaned with a suitable antiseptic, such as chlorhexidine or povidone-iodine. A stab incision should be made 3–5 cm lateral to the umbilicus on either side. The trocar should be inserted subcutaneously and tunneled for several centimeters, pointing toward the pelvis and inguinal canal, before piercing through the abdominal muscles. The catheter should then be advanced over the trocar until it is seated fully in the abdomen. A purse-string and finger-trap suture pattern should be used to secure the catheter in place.

Surgical placement is highly preferable when treatment is expected to last longer than 24 hours. The patient should be prepared in the same way as for emergent catheter placement: in dorsal recumbency, shaved from xiphoid to pubis, and cleaned with a suitable antiseptic. Current human guidelines recommend the use of a single perioperative dose of a prophylactic antibiotic that provides antistaphylococcal coverage, such as cefazolin, and the authors consider that this is also beneficial to veterinary patients [6, 13, 18]. Surgical omentectomy is necessary to prevent omental entrapment and should be performed at the same time as catheter placement. After omentectomy, an incision should be made off midline by 3–5 cm on either side, and a subcutaneous tunnel made as previously described for percutaneous placement. The catheter should terminate in the inguinal area and be oriented in a roughly cranial to caudal plane. Care should be taken to ensure that the catheter does not kink at either the internal or external exit sites.

If a cuffed catheter is used (current human guidelines recommend silicone catheters with at least two Dacron cuffs), the cuffs should be soaked in sterile saline before placement to remove air and facilitate fibroblast invasion [13]. The deeper cuff should be placed in the rectus muscle, and the more superficial cuff should be placed in the subcutaneous tunnel. The silicone bead of a Missouri catheter, if used, should be positioned just inside the peritoneum to prevent dialysate leakage.

Dialysate Solutions

Dialysate solutions are buffered, slightly hyperosmolar crystalloid solutions designed to osmotically pull fluid, creatinine, urea, electrolytes, phosphorous, and other solutes from the plasma into the dialysate. These solutions also provide diffusible buffer and other needed compounds such as magnesium and calcium [19]. Most commercially available dialysate solutions do not contain potassium, as many dialysis patients are hyperkalemic. For normokalemic and hypokalemic patients, potassium chloride can be added to the dialysate up to a maximum concentration of 4 mmol/l (4 mEq/l), or potassium can be supplemented orally or parenterally [20, 21].

Hyperosmolar dialysate solutions are effective in minimizing edema in overhydrated patients and enhancing

Table 36.2 Recommended hyperosmotic sugar concentrations.

Concentration of dextrose/ glucose monohydrate	Patient fluid status
1.5%/1.36 mg/dl	Euvolemic or dehydrated
2.5%/2.27 mg/dl	Mildly overhydrated
4.25%/3.86 mg/dl	Severely overhydrated

ultrafiltration. The most frequently used hypertonic solution is sugar, either as dextrose or as glucose monohydrate, though other solutions have seen use in human medicine in recent years [16]. These sugars appear to favor capillary vasodilation and promote solute drag, so small concentrations are frequently used in even euvolemic or dehydrated patients to maximize the permeability of the peritoneal membrane (refer to Table 36.2 for recommended hyperosmotic sugar concentrations). Intermittent use of 4.25% dextrose or 3.86 mg/dl glucose monohydrate dialysate may increase the efficiency of dialysis in all patients, regardless of fluid status [2].

Heparin (250–1000 iu/l) should be added to dialysate for the first few days after catheter placement to help prevent occlusion of the catheter by fibrin deposits (Figure 36.6) [2, 22]. There is minimal systemic absorption of this heparin, making it unlikely to prolong clotting times [2, 22–25].

Many different formulations of dialysate solutions are commercially available (Table 36.3). Dialysate solutions should be chosen based on patient serum electrolyte and

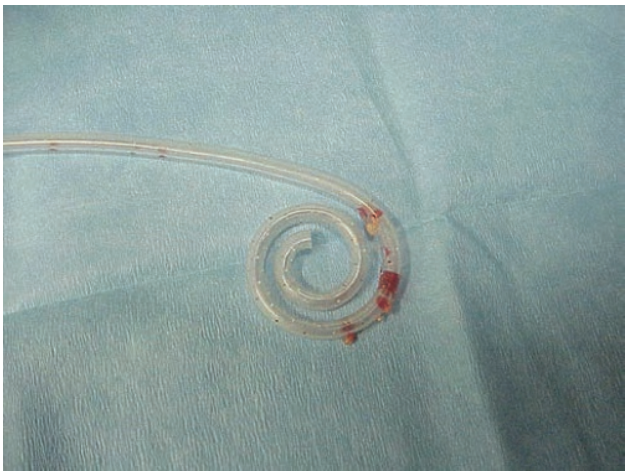


Figure 36.6 Fibrin deposits, which may occlude the catheter (as pictured here), can be avoided by adding heparin to the dialysate for the first few days after catheter placement.

fluid volume status. It is also possible to make a suitable dialysate solution for immediate short-term use with supplies readily available in all specialty practices until commercial dialysate solutions can be acquired (Box 36.2, Figure 36.7). Once treatment has been initiated, commercial solutions should be used as soon as possible. It is worth noting that although 0.9% NaCl (physiological saline) may be considered in severely hyperkalemic patients as it contains no potassium, it has also been shown to predispose human chronic peritoneal dialysis patients to the formation of peritoneal adhesions and fibrosis [6]. Because of this, it is advisable to change to

Table 36.3 Common commercially available and homemade peritoneal dialysis dialysate solutions [6, 16, 20, 26].

Brand	Name[a]	Osmotic agent	pH	Na (mmol/l)	Ca (mmol/l)[b]	Mg (mmol/l)[c]	Lactate (mmol/l)	Bicarb (mmol/l)
Baxter	Dianeal™ PD-1	Glucose	5.5	132	1.75	0.75	35	0
	Dianeal™ PD-4	Glucose	5.5	132	1.25	0.25	40	0
	Extraneal™	Icodextrin	5.5	132	1.75	0.25	40	0
	Nutrineal™	Amino acids	6.5	132	1.25	0.25	40	0
	Physioneal™ 35	Glucose	7.4	132	1.75	0.25	10	25
	Physioneal 40	Glucose	7.4	132	1.25	0.25	15	25
Fresenius Medical Care	Balance®	Glucose	7.4	134	1.25/1.75	0.5	35	2.5
	Bicavera®	Glucose	7.4	134	1.25/1.75	0.5	0	34
	Stay Safe® 2/4/3	Glucose	5.5	134	1.75	0.5	35	0
	Stay Safe 17/19/18	Glucose	5.5	134	1.25	0.5	35	0
Homemade	Lactated Ringer's solution	Dextrose	6.5	130	1.4	0	28	0
	0.9% NaCl and sodium bicarbonate	Dextrose	5.5	154	0	0	0	35

[a] Solutions may differ slightly in name and formulation depending on region.
[b] To convert to mg/dl: Ca × 4
[c] To convert to mg/dl: Mg × 2.43.

Box 36.2 Recipe for Homemade Dialysate Solutions

- 1 liter crystalloid solution:
  - lactated Ringer's solution
    or
  - 0.9% NaCl (physiological saline); if using, add 35 ml 8.4% sodium bicarbonate (35 mEq)
- 50% dextrose solution:
  - 30 ml = 1.5%
    or
  - 50 ml = 2.5%
    or
  - 85 ml = 4.25%

Before adding dextrose or sodium bicarbonate, aseptically remove the same volume of crystalloid from the bag as the volume of dextrose and/or sodium bicarbonate, and discard. See references [2, 12, 22].

either a buffered electrolyte solution or commercially available dialysate as soon as possible. Sodium bicarbonate can be added to 0.9% NaCl to buffer the solution and potentially negate some of these effects [6]. If renal function is still not adequate after acute peritoneal dialysis and the decision is made to pursue chronic peritoneal dialysis, the use of amino acid- or icodextrin-based dialysate will likely be advantageous [16, 27–29].

Catheter Management

To avoid catheter-related infections, asepsis should be practiced and maintained at all times. After placement of the peritoneal dialysis catheter, the exit site should be bandaged and connected to the inflow and outflow lines. All manipulations of lines, daily bandage changes, and daily line changes should be performed wearing sterile gloves. All contact with the patient, including physical exams and treatments, should be performed wearing clean examination gloves. Ports should be swabbed with 70% isopropyl alcohol prior to any injections.

Central to managing the peritoneal dialysis catheter is proper bandaging of the exit site and stabilization of the catheter and exiting lines. The items needed are shown in Figure 36.8.

An antimicrobial ointment should be applied over the exit site after placement of the catheter, followed by a sterile dressing. Current human guidelines recommend either mupirocin or gentamycin ointments, although the authors have also used bacitracin/neomycin/polymyxin (triple antibiotic ointment) to good effect [13, 18]. For dressings, the authors prefer the Tegaderm™ +Pad (3M), which is available in several sizes. This dressing has an adhesive backing which allows it to stick to the catheter and the skin around it. In the middle of the dressing there is a non-adherent, absorbent pad that will wick away any effusion from the catheter exit site.



Figure 36.7 Commercially available and homemade dialysate solutions.

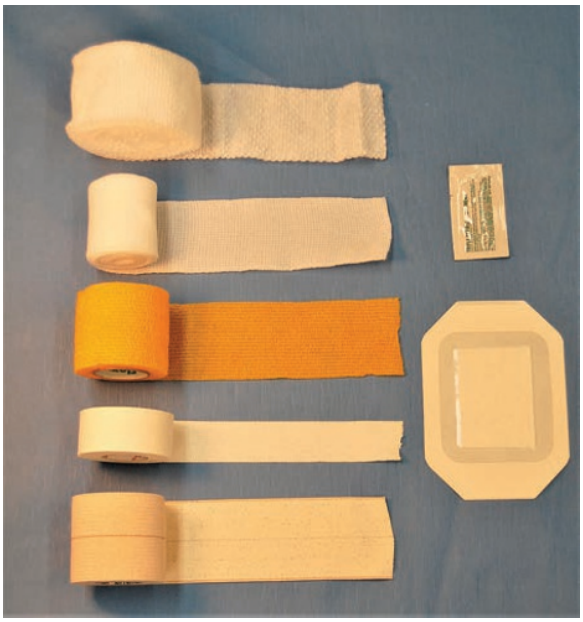


Figure 36.8 Supplies needed for bandaging an indwelling peritoneal dialysis catheter.

The abdomen should then be wrapped with cast padding, taking care that the catheter is stabilized without kinking or occluding it. This should be followed by a layer of conforming gauze and finished with a layer of cohesive bandage (e.g. Vetrap™, 3M). If needed, a layer of four-inch fabric elastic tape (e.g. Elastikon®, Johnson & Johnson) can be applied around the cranial aspect of the bandage to keep it from sliding.

Once the bandage is applied and the catheter is stabilized, it should be attached to lines in preparation for exchange. In veterinary peritoneal dialysis, there are two main types of catheter connecting systems. In the standard or straight connecting system, the catheter is connected to the dialysate solution bag using a straight piece of tubing and intravenous (IV) fluid administration set. The container of dialysate is drained into the patient and the empty bag is rolled up and remains attached until the next exchange is initiated. At the beginning of the next exchange, the effluent is drained into the empty dialysate bag, removed, measured, and discarded. A new connection must be made for each exchange.

Studies in humans have shown the Y-connection system, as compared with the standard connection, is associated with a lower incidence of peritonitis [30]. With the Y-connection system, one line is connected to the patient, the second line is connected to an empty "effluent" bag, while the third line is connected to the dialysate. The items needed are shown in Figure 36.9.

An extension set should be connected to the peritoneal dialysis catheter and then attached to a three-way stopcock. The dialysate should be spiked with an IV fluid administration set. The authors recommend using a volumetric fluid pump to control and monitor the volume of infusion for each exchange. The fluid administration set should be primed with dialysate and attached to the T-side of the three-way stopcock. A second IV fluid administration set should be connected to a sterile empty collection bag and attached to the remaining port of the stopcock. This line should be



Figure 36.9 Supplies needed for Y-connection. (a) Volumetric intravenous (IV) fluid administration set. (b) Extension set. (c) Three-way stopcock. (d) IV fluid administration set. (e) Empty fluid bag (sterile).

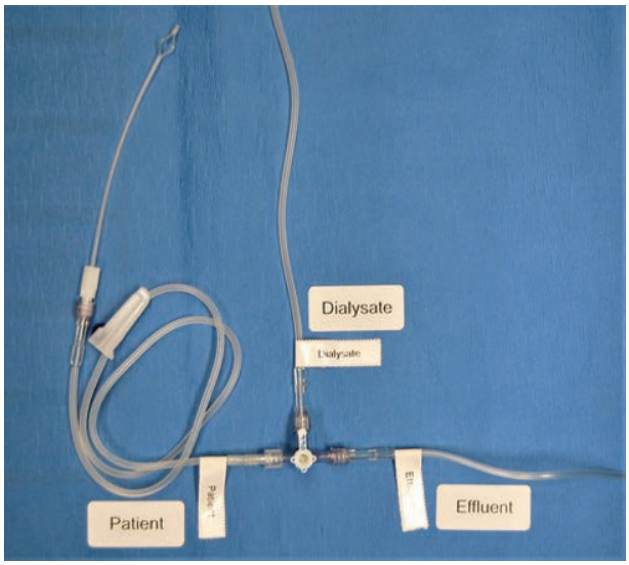


Figure 36.10 Peritoneal dialysis Y-connection system set up with appropriate labeling.

positioned so that it is in a direct, straight route from the patient's dialysis catheter through the stopcock and into the empty collection bag. To maintain sterility, all connections should be wrapped in gauze soaked with chlorhexidine 1–2% and secured with waterproof tape. All the lines and connections should be labeled in a manner that all personnel will understand: "dialysate in" or "inflow;" "patient;" "drain" or "outflow" or "effluent" (Figure 36.10). Figure 36.11 shows all equipment set up as it would be for a patient.

The abdominal bandage should be removed every 24 hours, and the catheter exit site should be inspected. Any redness, swelling, discharge, catheter slippage, or other abnormalities should be reported to the doctor immediately. Before rebandaging, the catheter exit site should be cleaned with chlorhexidine 1–2%, dried thoroughly, and new antimicrobial ointment applied. All lines, including the effluent bag and any unused dialysate, should also be changed every 24 hours. If the dialysate bag is consumed in less than 24 hours, only the bag needs to be changed at that time. Any direct contact with the catheter, including changing the lines, should be performed in an aseptic manner while wearing sterile gloves. The exposed portion of the catheter and all lines should be wiped down with chlorhexidine 1–2% every four to six hours.

Performing Peritoneal Dialysis Exchanges

The type of dialysate, the amount infused, and the dwell and dry times are collectively referred to as the *dialysis prescription*. A *dialysis cycle* refers to how often exchanges occur. Initially, in the acute setting, these may be hourly. Patients undergoing chronic peritoneal dialysis may have dwell times of two to four hours, though they can last up to 12 hours. These are interspersed with 4 to 12 hour "dry" times, during which the peritoneal cavity is devoid of dialysate.

Once everything is attached, secured, and labeled, exchanges may begin. The dialysate dose is usually infused over 10–20 minutes, depending on the volume and speed at which it can be given. The use of a fluid pump is

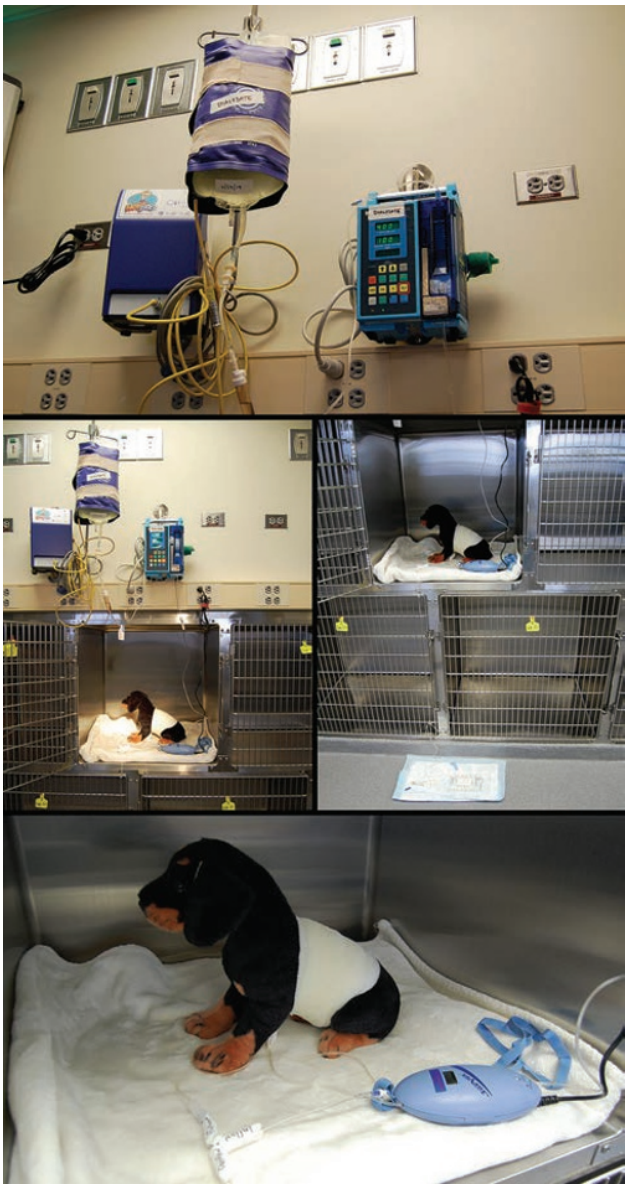


Figure 36.11 Mock peritoneal dialysis setup featuring the authors' dialysis training model "Lepto Louie." For this setup, both a heating pad to warm the dialysate and an inline warmer have been used to keep the dialysate at an optimal temperature, as well as a volumetric fluid pump for accurate infusion dosing.

advantageous in assuring accuracy in the delivery of prescribed infusions over a set amount of time.

The dialysate should be warmed to physiologic temperatures, 100.4–102.2°F (38–39°C), prior to instillation. This will improve the permeability of the peritoneum and will be more comfortable for the patient [23]. Instilling room-temperature dialysate may dramatically and detrimentally lower the patient's body temperature. An inline fluid warmer may be used on the end of the inflow line between the stopcock and dialysate bag. However, it is important to ensure that it will sufficiently warm the fluid at the rate at which the dialysate is being infused. A drawback to this methodology is the extra weight pulling on the catheter, especially if the patient is ambulatory.

Another strategy is to wrap the dialysate fluid bag in a warming blanket. A limitation of this method is that the fluid in the line cools during the dwell and drain period. With small infusion volumes, the cool dialysate fluid in the fluid line could amount to a large portion of the total infusion. A combination of the inline and around-the-bag fluid warming techniques is ideal.

At the beginning of each exchange, a minimum of 2 ml of dialysate should first be flushed through the stopcock. This serves to rinse bacteria that are potentially trapped in the stopcock into the outflow line, and subsequently, the effluent bag. This is referred to as the "drain first protocol" [18]. An added benefit to this technique is that the volume that has cooled in the fluid line can be added to the rinse volume so that only warmed fluid will be infused into the patient. The "rinse" volume must be excluded from the measured volumes during monitoring.

To initiate an exchange, the pump should be set for the rinse volume. The stopcock should be turned "off" to the patient, or "on" to the effluent bag, depending on the design of the stopcock. After the rinse volume is infused into the effluent bag, turn the stopcock "off" to the effluent bag or "on" to the patient. The fluid pump should be programmed to deliver the prescribed amount of dialysate over the prescribed amount of time. When the infusion is complete, turn the stopcock "off" to the patient, or "on" to the effluent bag. The patient is now "dwelling" with dialysate remaining within the peritoneum. Any time the patient is not receiving an infusion or actively draining, the stopcock should be turned "off" to the patient. At the end of the prescribed dwell time, the stopcock should be turned "off" to the dialysate or dialysis inflow line. Dialysate fluid, now referred to as effluent, should flow freely from the patient into the outflow line and effluent collection bag. After the drain period, the effluent should be measured and discarded, and another exchange can be initiated.

Monitoring

Accurate and complete record keeping is a critical aspect of peritoneal dialysis. The following values should be recorded with each exchange: IV fluids infused, including all IV medications, dialysate infused, effluent drained, and urine output. A running total comparing the volumes infused to the outputs should also be recorded. A digital spreadsheet is effective in recording and tracking these values (Figure 36.12). The advantage of a digital spreadsheet is that it can be programmed to calculate totals and deficits automatically.

Body weight, which can be a useful tool in assessing fluid volume status, should be measured every six to eight hours. This should be done in a "dry" state before an infusion of dialysate. Rapid decreases in body weight may be an indicator that the patient is becoming volume-depleted, or alternatively could signify successful fluid removal from an overhydrated patient (ultrafiltration). Acute increases in weight may indicate that an oliguric patient is in jeopardy of developing volume overload, or that a significant volume of dialysate is being retained during draining.

Initially, temperature should be monitored every six to eight hours. Temperature should be evaluated at least once after the first infusion of dialysate to determine if the exchanges are affecting body temperature. As with any patient that has compromised kidney function, blood pressure should be assessed every six to eight hours to ensure that the patient does not become either hypo- or hypertensive.

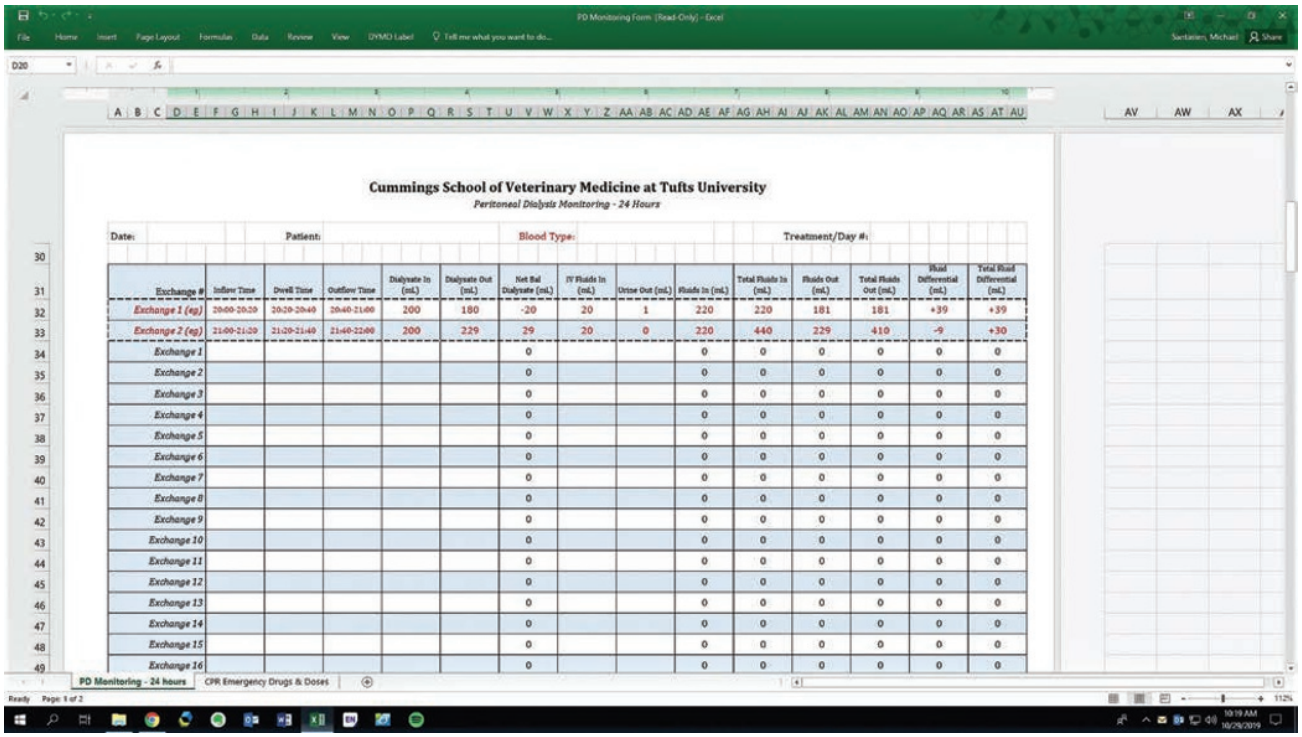
Heart rate, respiratory rate, and respiratory effort should be evaluated every one to two hours. If there is any change in respiratory rate or effort, it should be noted how these changes correlate with dialysate infusion. Distension of the peritoneal cavity with dialysate will increase the pressure on the diaphragm, potentially making respiration difficult. Increases in heart rate, respiratory rate, and respiratory effort could indicate hypovolemia, volume overload, or pain, and should be addressed appropriately.

Changes in kidney and electrolyte values can occur rapidly in the first few days of peritoneal dialysis. These values should be checked two to three times a day during this critical period, and then daily when it is determined that the patient is tolerating exchanges well.

Complications

Complications of peritoneal dialysis include dialysate retention, dialysate leakage, hypothermia, electrolyte disturbances, hypoalbuminemia, and bacterial peritonitis.

Poor fluid efflux generally occurs if the catheter was placed percutaneously, and it has become occluded by



Cummings School of Veterinary Medicine at Tufts University

Peritoneal Dialysis Monitoring - 24 Hours

Date: Patient: Blood Type: Treatment/Day #:

Exchange #	Inflow Time	Dwell Time	Outflow Time	Dialysate In (mL)	Dialysate Out (mL)	Net Bal Dialysate (mL)	IV Fluids In (mL)	Urine Out (mL)	Fluids In (mL)	Total Fluids In (mL)	Fluids Out (mL)	Total Fluids Out (mL)	Fluid Differential (mL)	Total Fluid Differential (mL)
Exchange 1 (eg)	20:00-20:20	20:20-20:40	20:40-21:00	200	180	-20	20	1	220	220	181	181	+39	+39
Exchange 2 (eg)	21:00-21:20	21:20-21:40	21:40-22:00	200	229	29	20	0	220	440	229	410	-9	+30
Exchange 1						0			0	0	0	0	0	0
Exchange 2						0			0	0	0	0	0	0
Exchange 3						0			0	0	0	0	0	0
Exchange 4						0			0	0	0	0	0	0
Exchange 5						0			0	0	0	0	0	0
Exchange 6						0			0	0	0	0	0	0
Exchange 7						0			0	0	0	0	0	0
Exchange 8						0			0	0	0	0	0	0
Exchange 9						0			0	0	0	0	0	0
Exchange 10						0			0	0	0	0	0	0
Exchange 11						0			0	0	0	0	0	0
Exchange 12						0			0	0	0	0	0	0
Exchange 13						0			0	0	0	0	0	0
Exchange 14						0			0	0	0	0	0	0
Exchange 15						0			0	0	0	0	0	0
Exchange 16						0			0	0	0	0	0	0

Figure 36.12 A peritoneal dialysis monitoring sheet that has been programmed to perform calculations automatically in Microsoft Excel®.

omentum, thus resulting in dialysate retention. The ideal way to avoid this complication is to perform an omentectomy at the time of surgical placement of the peritoneal dialysis catheter, discussed earlier in this chapter. However, even with a surgically placed peritoneal dialysis catheter in an omentectomized patient, there are times that fluid will not drain freely.

Manipulating the patient's position may facilitate drainage. Changing recumbency or standing the patient on its rear limbs with the head elevated or elevating the pelvis with the thoracic limbs on the ground may move dialysate within the abdomen and encourage drainage through the catheter. Instilling or flushing a small amount of dialysate through the catheter may clear an outflow problem due to omentum covering the drainage holes. These methodologies are sometimes successful, but often, once the catheter is clogged with omentum, it is difficult to correct.

Another potential cause of slow fluid outflow may be a kink in the catheter or drainage tubing. Manual inspection of all lines leading from the patient should reveal any kinks or occlusions. If there are no obvious occlusions in the distal lines, but fluid efflux remains poor, the bandage should be removed so that the catheter and catheter exit site can be inspected for possible kinks or occlusions. Unfortunately, kinks within the patient are difficult to distinguish from omental clogging. However, if dialysate flows freely into the patient but efflux is slow, an omental clog is more likely than a kink in the catheter, which should lead to difficulty with both fluid influx and efflux.

At our institution, Cummings School of Veterinary Medicine, pericatheter leakage into the subcutaneous tissue, usually through the abdominal incision site, is the most frequent complication (Figure 36.13). A total of 62% of cats with percutaneously placed catheters and 50% of cats with surgically placed catheters experienced subcutaneous leakage [31]. Ensuring a closely apposed abdominal incision closure, using a simple interrupted suture pattern



Figure 36.13 Dialysate leakage into the subcutaneous space.

only, can minimize dialysate leakage. Generally, patients receiving peritoneal dialysis require immediate therapy; however, if it is possible to delay the first exchange for 12–24 hours, it may help the site to "seal" and therefore minimize leakage. If treatment cannot be delayed, initial exchange volumes should be started at a quarter of the calculated infusion amount. If leakage does occur, intermittently wrapping the limbs may assist in promoting mobilization of the edema. The dialysate solution should be changed to the lowest possible osmolality formulation available; otherwise, the hyperosmolar glucose solution in the dialysate will continue to bring body water with it into the subcutaneous tissue.

Hypokalemia and hypoalbuminemia may both develop in patients undergoing peritoneal dialysis, so both serum potassium and albumin concentrations should be monitored daily. Hypokalemia develops due to the nature of diffusion of potassium across the peritoneal membrane from the patient into the dialysate and subsequently into the effluent. Hypoalbuminemia may be the result of low dietary protein intake, gastrointestinal or renal protein loss, loss into the dialysate due to inflammatory changes in the peritoneum, uremic catabolism, or concurrent diseases. Protein losses can be clinically important in patients undergoing peritoneal dialysis [32]. In a review of peritoneal dialysis cases in dogs and cats, hypoalbuminemia was the most common complication, with 41% of animals affected [33]. In another study, 16% of cats developed hypoalbuminemia during peritoneal dialysis [33]. Adequate enteral nutrition may be difficult to achieve in uremic patients. Nutritional support should be initiated early in the course of therapy for the uremic patient. This includes feeding tubes, partial or total parenteral supplementation, and the technique of using 1.1% amino acid solutions during peritoneal dialysis exchanges [22, 28, 29, 34, 35]. Gastrostomy and jejunostomy tubes are contraindicated during peritoneal dialysis due to increased risk of infection and pericatheter dialysate leakage.

Regular temperature monitoring is crucial to maintaining an appropriate body temperature for the patient. Thermoregulation is challenging due to the constant changes in the patient's treatment, influx versus dwell time versus efflux, which all affect the body temperature. As previously discussed, hypothermia can be prevented by adequate warming of the dialysate solution before instillation. Additionally, providing the patient with external warming support, such as a warming blanket or heating pad, can help prevent hypothermia. An increase in body temperature may be a sign of infection or peritonitis and should be thoroughly investigated.

The prevalence of peritonitis in veterinary patients receiving peritoneal dialysis has been reported as higher (22%) than that reported in human patients (15%) [33, 36]. Additionally, exit site infection is a reported complication in humans [37]. In studies performed at our institution, peritonitis was not identified in any of the peritoneal dialysis cases in dogs reviewed during a four-year period and was reported in only 1 of 22 cats over a five-year period [31, 38]. Peritonitis is diagnosed when two of the following criteria are recognized: cloudy dialysate effluent, greater than 100 inflammatory cells per microliter of effluent or positive culture results, and/or clinical signs of peritonitis. The most common source of peritonitis is contamination of the bag spike or tubing by the handler, although intestinal, hematogenous, and exit site sources of infection do occur [37]. It is important to recognize pericatheter leaks to minimize exit site sources of infection [22]. At our institution, the incidence of peritonitis has dramatically decreased with the use of the closed Y-system and drain first protocol.

Contraindications

There are only a few situations in which peritoneal dialysis is absolutely contraindicated, including recent gastrointestinal surgery (due to the risk of dehiscence from increased intrabdominal pressure), pleuroperitoneal leaking, diaphragmatic herniation, or the presence of peritoneal fibrosis or adhesions [1–3, 12]. Adhesions are frequently reported in humans but much more rarely so in dogs and cats [3, 12]. Relative contraindications include recent abdominal or thoracic surgery, the presence of inguinal or abdominal hernias or masses, marked obesity, or patients with severe catabolic disease states that may be worsened by protein loss during peritoneal dialysis exchanges [1–3, 12].

The Future of Peritoneal Dialysis

Although currently only used for the treatment of acute reversible kidney injury in veterinary medicine, it is possible that continuous ambulatory peritoneal dialysis could be a viable maintenance modality for patients with chronic kidney disease in the future; especially for patients for whom hemodialysis or transplantation is not an option. This would require a dedicated owner that is not intimidated by the complex nature of the home medical care required.

Peritoneal dialysis may continue to be more accessible to clinicians and owners as automatic peritoneal dialysis cycler machines, such as those used for human patients, become more available and less expensive. A few veterinary specialty centers have already begun using human peritoneal dialysis cyclers to good effect, but patient size is limited to medium and large breed dogs as these machines are designed to deliver higher volumes of dialysate than

can be administered to smaller veterinary patients. Although there are no veterinary-specific peritoneal dialysis cyclers currently on the market in the United States, the authors are currently testing a prototype that may become available soon. This would significantly reduce the burden on technical staff when treating peritoneal dialysis patients, and could, in theory, be useful to owners performing peritoneal dialysis at home.

Recently, a new dialytic therapy has been developed in human medicine called "continuous flow peritoneal dialysis." This technique uses two peritoneal dialysis catheters and a hemodialysis machine capable of performing continuous renal replacement therapy [21]. Rather than dialyzing blood, dialysate is run continuously in and out of the abdomen and filtered through a dialyzer ("artificial kidney") in a continuous loop [21]. This allows for solute clearance rates close to that of traditional hemodialysis modalities, but for patients who cannot tolerate hemodialysis due to hemodynamic instability or poor vascular access [21]. Further research is warranted to see if this procedure can be adapted for veterinary use, but the authors postulate that it could be a safer and more effective therapy for small patients than our current practices of priming the hemodialysis circuits with donor blood or performing traditional peritoneal dialysis .

Finally, new advances in dialysate and catheter products may lead to safer and easier administration of peritoneal dialysis. The addition of amino acid-based dialysates to the market for nutritional support of peritoneal dialysis patients is relatively recent and shows the potential for further advancement in dialysate technology. Likewise, new catheter development is expected to be ongoing and may shape future peritoneal dialysis treatment strategies in human and veterinary patients alike.

Summary

Peritoneal dialysis is a viable and technically simple option for the treatment of patients with AKI that is accessible to almost every veterinary practitioner. The objectives of peritoneal dialysis are to resolve the clinical signs of uremia, reduce azotemia, and to correct electrolyte, fluid, and acid–base abnormalities. Peritoneal dialysis is also a realistic treatment for dialyzable toxin exposure if referral to a hemodialysis center is not possible.

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37

Technical Management of Hemodialysis

Karen Poeppel and Cathy Langston

Hemodialysis is a therapy by which blood is removed from the patient, run through an artificial kidney called a dialyzer where uremic toxins are removed, and then returned to the patient (Figure 37.1). Removal of these toxins is achieved by diffusion across a semipermeable membrane in the dialyzer. Blood is circulated on one side of the membrane and a balanced electrolyte solution called dialysate is circulated on the other side. Molecules small enough to pass through the pores in the membrane move from the side with higher concentration to the side with lower concentration. Used dialysate, which is on the opposite side of the semipermeable membrane from the patient's blood and contains the patient's uremic waste products, is then washed down the drain. Blood continuously circulates in this loop for the duration of the dialysis treatment, usually four to five hours for intermittent hemodialysis (IHD). This way the entire blood volume of the patient is treated many times over while minimizing the actual volume of blood in the extracorporeal circuit at any given time. Vascular access for this circuit is obtained by placing a large double-lumen catheter into the jugular vein. In addition to removing uremic waste products, hemodialysis can restore appropriate patient hydration via ultrafiltration, as well as electrolyte and acid–base balance via diffusion or convection. Ultrafiltration is the removal of excess patient fluid from the vascular compartment and is achieved when a pump on the side of the outgoing dialysate creates a negative pressure across the semipermeable membrane in the dialyzer.

IHD is a renal replacement therapy performed for a set period of time per day, generally three days per week during the maintenance phase of treatment. Continuous renal replacement therapies (CRRT) exist that rely on the same concepts of diffusion, ultrafiltration, and convection across an extracorporeal semipermeable membrane. As the name implies, patients are treated continuously rather than intermittently. There are increasingly more veterinary facilities that provide forms of extracorporeal therapies, including IHD, CRRT, hybrids of the two, and some non-renal therapies [1–3].

Patient Selection

Acute Kidney Disease

There are a number of indications for IHD (Box 37.1), but in veterinary medicine IHD is used most commonly to treat patients with acute kidney injury (AKI) or failure [4, 5]. Standard medical therapy should always be attempted before initiating hemodialysis, but a certain number of patients do not respond adequately. Anuria or oliguria is often present in patients with more severe kidney injury. Life-threatening volume overload can develop in these patients as a result of aggressive intravenous (IV) fluid diuresis, excessive volumes of medications, or total or partial parenteral nutrition. In the absence of urine production, the body has very limited methods of removing extra fluid, so hemodialysis is indicated to remove the accumulated fluid [4, 5].

Anuric or oliguric patients, patients with severe renal impairment, and patients receiving overly aggressive potassium supplementation are at risk of hyperkalemia. Emergency treatment (i.e. insulin, dextrose, bicarbonate) only shifts the potassium to the intracellular space, but if urine production cannot be established, there is no way for the patient to excrete the excess potassium. Hemodialysis is indicated in these patients to remove the potassium [4]. Presence of uremic signs, progressive azotemia, or azotemia that does not improve over a

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Figure 37.1 A cat being dialyzed. Patients are not sedated for dialysis treatments.

Box 37.1 Indications for Hemodialysis

- Severe electrolyte or acid–base disturbances
- Life-threatening volume overload
- Azotemia refractory to conventional medical management
- Chronic kidney disease
- Toxicities and drug overdoses

24-hour period with standard medical therapy is also an indication for dialysis.

In all cases of AKI, the ultimate goal of dialytic therapy is to provide a supportive therapy to allow time for the kidneys to recover sufficiently so dialysis can be discontinued. Renal recovery generally takes at least a few weeks, and sometimes months, so without dialytic therapy the patient would die of uremic complications before renal recovery could occur. Of the patients that survive, a certain percentage have full renal recovery, some no longer require dialysis but have renal insufficiency, and the final group remain dialysis dependent for the remainder of their lives.

Chronic Kidney Disease

Hemodialysis can also be used to treat patients with chronic kidney disease. In these patients, there is no hope for renal recovery because the disease is degenerative. The patients are thus dialysis dependent for the remainder of their lives. Chronic dialysis is indicated when medical management fails to control uremic symptoms, which include vomiting, nausea, anorexia, and weakness.

It is not uncommon for patients with end-stage chronic kidney disease to present in an acute uremic crisis. In this setting treatment progresses similarly to treatment for AKI until the crisis is stabilized. The difference is that the owner and the medical staff know that the patient will always be dialysis dependent, so they can make decisions accordingly. If the patient is severely decompensated, prolonged intermittent renal replacement therapy or CRRT may be appropriate, but as the patient stabilizes, it would be transitioned to intermittent therapy that would allow the patient to leave the hospital and return for outpatient dialysis.

Toxin Removal

Hemodialysis can be used to treat certain toxicities and drug overdoses. For effective removal by dialysis, the substance must be small enough to pass through the semipermeable membrane, and it cannot be protein bound or sequestered in extravascular tissues [4, 5]. The dialysis treatment must be initiated before the toxin causes irreversible damage to the patient. Antifreeze (ethylene glycol), alcohol, and digoxin are a few substances that can be effectively removed by dialysis. For a more complete list, see Box 37.2.

Hemodialysis is superior to CRRT for clearing most toxins because diffusion happens much more quickly in the dialyzer during an IHD treatment than it would during a CRRT treatment. A continuous therapy may be beneficial for removal of toxins that have a high post-dialysis rebound, meaning that they are sequestered in the extravascular space and diffuse more slowly into the blood compartment [6].

For toxins that are larger or have more extensive protein binding, addition of carbon hemoperfusion will enhance clearance [7–10]. With carbon hemoperfusion, the blood is passed over a carbon substrate, or other substrate that can bind the offending molecule, and the toxin/offending molecule binds to the carbon and is thus removed from the circulation. The carbon cartridge is generally connected to the blood tubing delivering blood to the dialyzer, although machines or tubing sets are available that do not simultaneously provide hemodialysis.

Box 37.2 Substances Removed by Dialysis or Carbon Hemoperfusion [11]

Removed by hemodialysis
- Acetaminophen
- Asprin
- Aluminum
- Baclofen
- Bromides
- Caffeine
- Diethylene glycol
- Ethanol
- Ethylene glycol
- Isopropyl alcohol
- Lithium
- Mannitol
- Metaldehyde
- Methanol
- Metformin
- Methyl alcohol
- Theophylline

Removed by Carbon Hemoperfusion
- Amanita toxins
- Baclofen
- Cannabinoids
- Cyclosporine
- Ibuprofen
- Metaldehyde
- Methotrexate
- Paraquat
- Pentobarbital
- Phenobarbital

Patient Considerations

Hemodialysis involves prolonged and intimate operator contact with the patient. It would be unsafe for both the patient and technician to treat an aggressive patient. Repositioning of the catheter or patient for continuous blood flow is frequently necessary. Inability to handle the patient can lead to inadequate dialysis treatment, significant blood loss from clotting in the dialyzer, physical harm to the technician, or inadvertent dialysis catheter removal. Sedation for several hours daily is likely counterproductive to renal recovery.

Patients weighing less than 2.5 kg are difficult to treat due to their low blood volume. The smallest priming volume currently available for the extracorporeal circuit is 52 ml. Treatment of small patients requires priming the circuit with blood from the blood bank.

Equipment

Machines

The IHD and CRRT machines used in veterinary medicine are all manufactured for human use. In the United States, most units performing IHD or CRRT use either Gambro or Fresenius machines (Figure 37.2). Machines for CRRT are different in appearance than IHD machines (Figure 37.2c). Regardless of the model or manufacturer, all modern dialysis machines have certain common characteristics. First, they all contain a display screen. This screen displays the current operating mode (such as "set-up," "autotest," "dialysis"), all options available in that mode, treatment parameters, alarm conditions, and any necessary instructions. During the dialysis treatment, the screen also displays treatment status information such as time remaining in treatment, total fluid removed, and liters of blood processed (passed through the dialyzer).

The main difference between IHD and CRRT machines is the source of dialysate. IHD machines house a dialysate proportioning system. This system mixes incoming purified water with the appropriate volume of bicarbonate and electrolyte concentrate solutions to create dialysate. It is essential for patient safety that the dialysate is proportioned consistently and accurately to the operator's specifications. To that end, the machines also have sensors to assure the dialysate meets concentration and temperature requirements. CRRT machines use prepackaged dialysate that is generally supplied in 5-l bags, and thus, machines of this type do not need a water treatment system.

Each machine has a blood pump and clamps for the blood lines. There are housings for the blood cartridge, blood lines, and dialyzer. The vast majority of patients are anticoagulated with heparin to prevent blood clotting in the extracorporeal circuit, so all modern dialysis have a built-in syringe pump for heparin administration.

Finally, the machines contain a number of additional sensors that monitor for pressure changes in the extracorporeal circuit, air in the return line, blood leaks in the dialyzer, and other unsafe conditions. In any alarm situation, the machine automatically takes actions to ensure patient safety. For example, if a problem is detected in the dialysate, dialysate is diverted from the dialyzer so as not to affect the patient, but the blood continues to circulate to minimize the chances of clotting. If a problem is detected in the extracorporeal circuit, the blood pump stops and a clamp occludes the return line to prevent further removal of blood from the patient or unsafe return of blood to the patient. In extreme situations, the machine

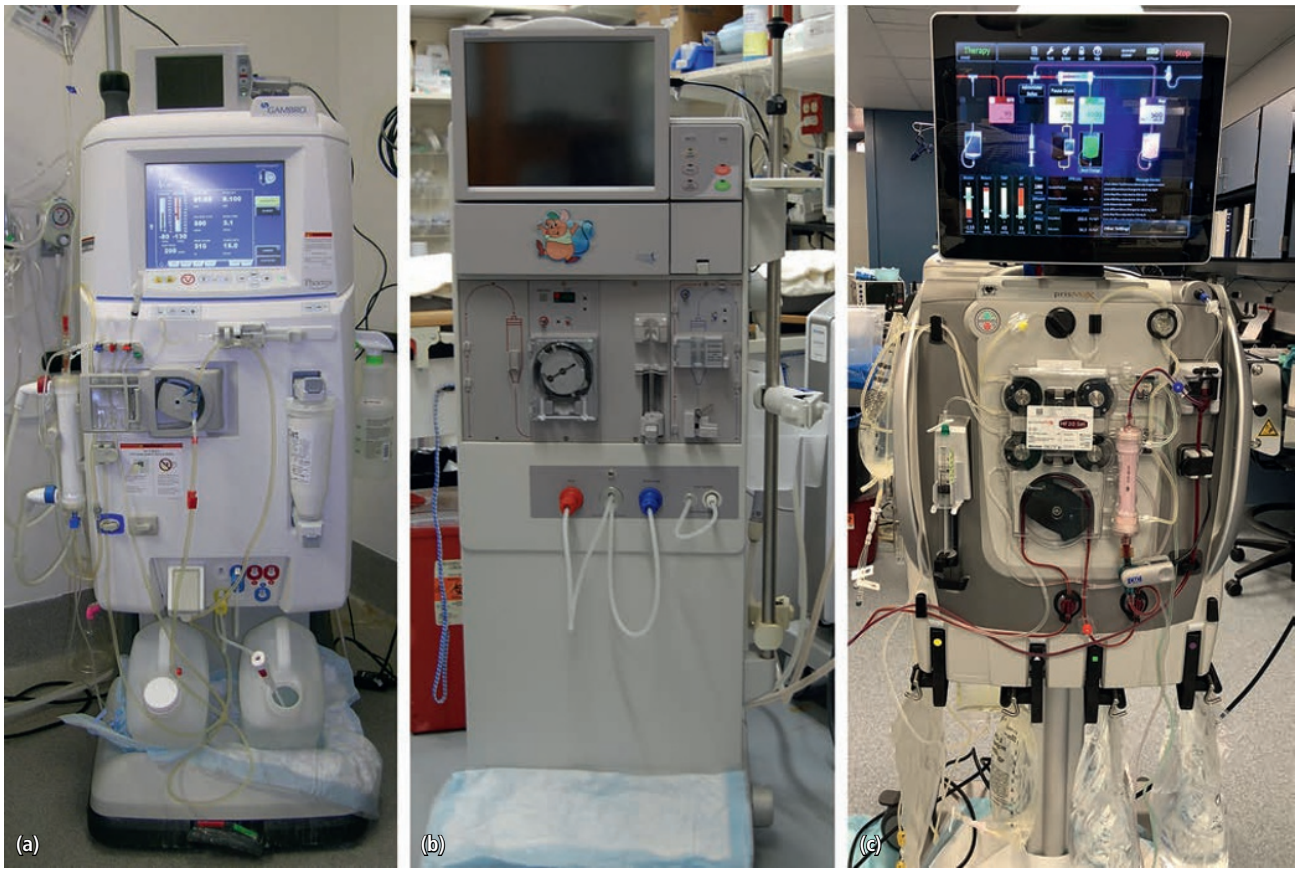


Figure 37.2 (a) Baxter Phoenix hemodialysis machine; (b) Fresenius 2008T hemodialysis machine (c) Baxter PrisMax CRRT machine.

requires the operator to perform an emergency stop treatment procedure.

Water Treatment System

A water treatment system is essential to providing a safe hemodialysis treatment with an IHD machine. The patient is exposed to roughly 20 gallons of water in the form of dialysate in an average IHD treatment, so even trace amounts of impurities can have detrimental effects [12, 13]. Water treatment systems vary in size and water output, from a small unit that fits the back of a dialysis machine (Figure 37.3) to an entire room full of equipment that provides water for up to 30 dialysis machines (Figure 37.4), but they all have certain common features. A typical system contains a mixing valve (to blend hot and cold water to the optimal temperature), sediment filter (to remove debris), potentially an ion exchange tank (to remove calcium and magnesium), carbon tanks (to remove organic components), and reverse osmosis or deionization (to remove any remaining contaminants and ions). Daily monitoring of the product water is required to assure patient safety. The technician performing the hemodialysis treatment generally performs this task.



Figure 37.3 A portable water treatment system. (a) carbon filter; (b) pre-filter; (c) reverse osmosis system.



Figure 37.4 Water treatment system with, from left to right, an ion exchange tank, carbon tanks, back-up deionization tanks, and the reverse osmosis system. Not pictured are the mixing valve and sediment filter.

Extracorporeal Circuit

The extracorporeal circuit used in IHD consists of a blood cartridge, blood tubing, and the dialyzer. Infusion lines are generally built into the cartridge for fluids, medications, and heparin. The entire circuit is discarded at the end of each dialysis treatment. The extracorporeal circuits used in veterinary medicine are manufactured for human use, so generally neonatal and pediatric sizes are used. Adult sizes are used for the largest patients (Table 37.1).

The variation in length and diameter of the tubing allows for maximal blood flow in larger patients and minimal priming volumes in smaller patients.

A number of different dialyzers are used for veterinary patients, but they all have certain common characteristics.

Table 37.1 Recommended extracorporeal volumes [4].

	Body weight (kg)	Dialyzer volume (ml)	Total extracorporeal volume (ml)	Blood volume (%)
Cats, dogs	< 6	< 20	< 60	13–40
Cats	> 6	< 30	< 70	< 23
Dogs	6–12	< 45	< 90	9–19
Dogs	12–20	< 80	100–160	6–17
Dogs	20–30	< 120	150–200	6–13
Dogs	> 30	> 80	150–250	6–10

Source: Adapted from Cowgill et al. 2012.

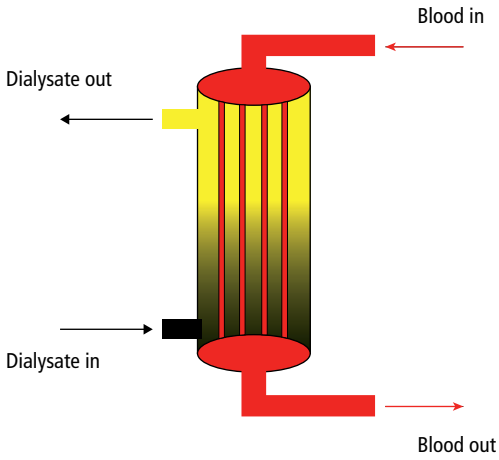


Figure 37.5 Diagram of a hollow fiber dialyzer. Countercurrent flows of blood and dialysate allow for more effective clearance of waste products.

They are a hollow fiber design, which means the semipermeable membranes form thin straw-like tubes through which the blood passes. The dialysate then bathes these blood-filled fibers (Figure 37.5). This design allows for smaller priming volumes than other dialyzer configurations, and it also provides greater membrane surface area for greater efficiency.

Dialyzer membranes are made of either natural or synthetic material. The natural fiber membranes are no longer readily available in the United States. Synthetic membranes usually have larger pore sizes, which allow for better clearance of middle molecular weight uremic toxins in addition to small molecule clearance. They are also reportedly more biocompatible and less thrombogenic than the natural fiber membranes [14, 15].

Dialyzers come in a variety of different sizes. Priming volumes for dialyzers commonly used in veterinary medicine range from 28 ml to more than 150 ml. The larger dialyzers have more membrane surface area, so they are more efficient at clearing waste products, and the smaller dialyzers, although less efficient, allow for treatment of very small patients because of the small priming volumes.

Catheters

Consistent long-term vascular access is key in providing adequate dialytic therapy via intermittent and continuous modes. In veterinary medicine, this is almost always achieved by placing a double-lumen catheter into the jugular vein [4, 14]. The catheter should be large enough to supply a blood flow of 80–125 ml/minute in cats or small dogs and 250–500 ml/minute in medium or large dogs. This generally means placing the largest-bore catheter that will fit into the patient's vein. A number of different catheters manufactured for human dialysis patients are suitable for

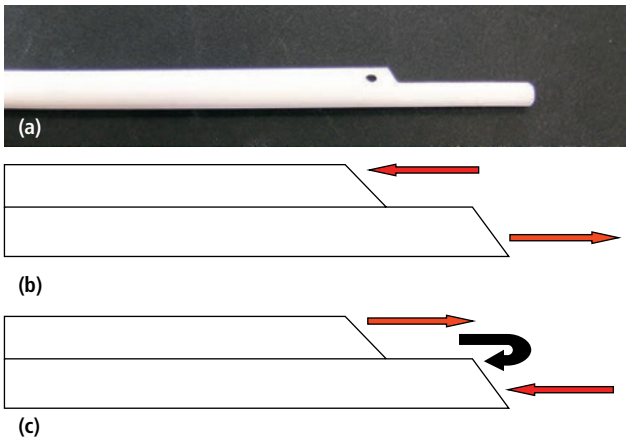


Figure 37.6 (a) Picture of the tip of a hemodialysis catheter demonstrating the staggered lumens. (b) Diagram indicating preferred direction of blood flow into the proximal lumen and out of the distal lumen. (c) Diagram showing how access recirculation occurs when blood flows into the distal lumen and out of the proximal lumen.

veterinary use. Most of these catheters have two lumens, referred to as the access lumen and the return lumen. The access lumen, sometimes called arterial, has blood flow from the patient to the extracorporeal circuit, and the return lumen, sometimes called venous, is for return of blood to the patient from the dialysis machine. Regardless of terminology, all blood flowing through the catheter is venous blood due to its location in the jugular vein or vena cava. The ends of the lumens are staggered such that the tip of the access lumen is proximal to the tip of the return lumen (Figure 37.6). This is meant to reduce the reuptake of "clean" blood by the arterial lumen (called access recirculation), which could reduce the clearance of waste products during the dialysis treatment.

Dialysis catheters are referred to as temporary (nontunneled) or permanent (tunneled). Temporary catheters are noncuffed, tapered at the tip, and usually placed percutaneously. These catheters are meant to stay in place for only a few weeks. In most cases, temporary catheters are the appropriate choice, as long as chronic dialysis therapy is not anticipated, and they are the mainstay in veterinary dialysis. Permanent catheters have an external cuff, frequently are blunt at the end, and they must be placed surgically. The catheter is tunneled in a subcutaneous pocket that extends from the skin exit site to the vessel before being inserted into the vessel. The subcutaneous pocket is a few centimeters in length, and the external cuff of the catheter is positioned in this pocket. Fibroblasts attach to the cuff, which secures the catheter in the pocket and creates a physical barrier inhibiting bacteria around the skin exit site from moving along the catheter and into the vessel. These catheters can stay in place one to two years and are the preferred choice for a patient receiving chronic dialysis [14].

Several double-lumen or multilumen catheters may be adequate for small patients (Mila, Arrow). These catheters are designed to be placed percutaneously. The proximal lumen is used as the access lumen, and the distal lumen is used as the return lumen. Use of a multilumen catheter is not recommended for dialysis because the more lumens in a catheter, the smaller the size of each lumen, and therefore the slower the blood flow. Also, use of a catheter smaller than 7 Fr is not recommended for the same reason, although a smaller catheter can be placed when there is no other option.

Placement

As previously discussed, hemodialysis catheters can be placed percutaneously or surgically, depending on the type of catheter and the personnel involved. Strict attention to aseptic technique during placement is mandatory for both temporary and permanent catheters [15]. These catheters are generally placed in a clean procedure room with restricted traffic. All personnel involved in the procedure should wear caps and masks. A large barrier drape and sterile gloves are necessary. Because of the "springiness" of the guidewire, a surgical gown should be worn to decrease the risk of contaminating it during placement. Permanent catheters are placed with a surgical technique and so should be placed in an operating room.

The person placing the catheter must be skilled and should use the method with which he or she is most comfortable. In addition to dialysis personnel, emergency and critical care personnel are often equally skilled at placing percutaneous catheters. A dialysis nephrologist, criticalist, or surgeon may have the most experience in placing a permanent dialysis catheter. In some cases, catheter placement is facilitated by either ultrasound guidance or a cutdown procedure to isolate the vessel, followed by the percutaneous technique.

Sedation or anesthesia is often needed for catheter placement. Some compliant or severely depressed animals may only require a local anesthetic. A short-acting or reversible drug can be used for percutaneous placement. General anesthesia should be used if catheter placement is expected to be problematic, and it is required if an esophagostomy feeding tube is being placed at the same time or for placement of a permanent, tunneled catheter.

Location

The jugular veins generally are the only vessels large enough for a dialysis catheter in most animals because of the catheter size needed relative to patient size. All attempts

should be made to preserve at least one jugular vein in any patient that may eventually need hemodialysis. There is no documented difference in veterinary medicine between the right and left jugular vein in dialysis treatment aside from individual preference during placement.

The tip of the catheter should be positioned at the junction of the cranial vena cava and the right atrium to provide maximum blood flow. Whether placed percutaneously or surgically, fluoroscopy can be used during the procedure to assure proper placement [16]. If fluoroscopy is not used, a post-procedure radiograph should be taken. In either case, blood flows are evaluated by rapidly aspirating blood into a 10- or 12-cc syringe before the procedure is complete. Blood should flow from both lumens with ease.

Care and Maintenance

The dialysis catheter should be handled aseptically at all times. The catheter should only be handled by personnel trained in dialysis catheter care, and it should not be used for purposes other than dialysis. Each time the catheter is unwrapped for treatment, the catheter exit site should be cleaned and assessed (Figure 37.7). The bandaging material should be changed as needed if it becomes wet, blood soaked, or otherwise compromised [14]. If dialysis is not being performed, the catheter locking solution (see later) should be changed at least every three to four days, and the exit site can be cleaned at that time. Guidelines for accessing the catheter for dialysis treatments or changing the catheter locking solution are outlined in Protocol 37.1.

When the catheter is not in use, it is wrapped securely to protect it from dislodgement or inadvertent opening. The wrap should completely cover the catheter, and the catheter should not be accessed regularly for heparinized saline flushes. Instead, an anticoagulant locking solution is placed in each lumen to prevent clotting between dialysis treatments. The locking solution has historically been sodium heparin (500–5000 iu/ml), but a number of studies have shown that sodium citrate at a 4% or higher concentration is as effective an anticoagulant as heparin, does not stimulate biofilm production, is bacteriostatic, and is less expensive than heparin [17–19]. For that reason, 4%

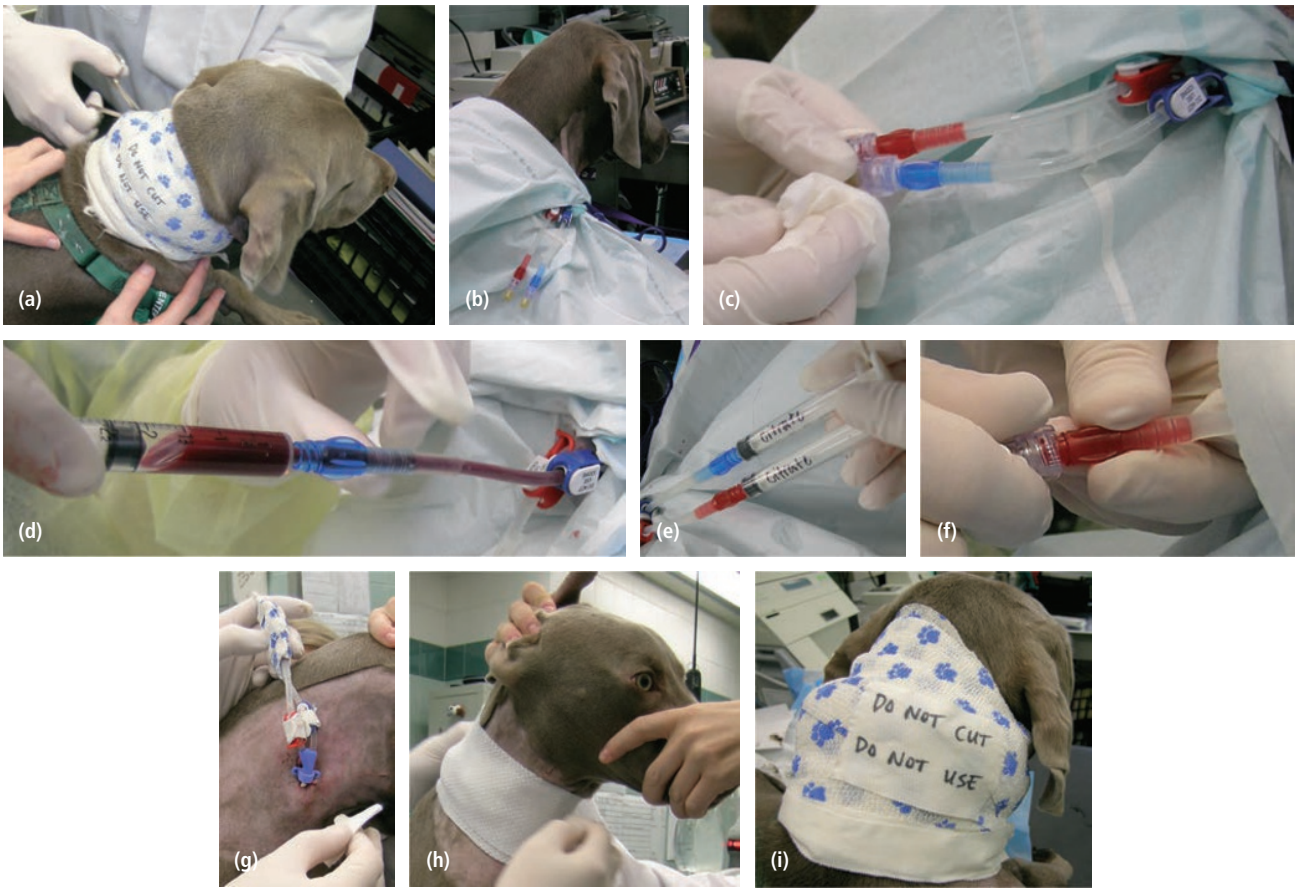


Figure 37.7 Hemodialysis catheter care. (a) Careful removal of catheter bandage. (b) Draped catheter. (c) Scrubbing the catheter ports. (d) Aspirating the anticoagulant lock. (e) Replacing the anticoagulant locks. (f) Placing caps on the catheter. (g) Secured catheter ports. (h) Wrapping the catheter. (i) Final bandage with reminder not to use the catheter.

Protocol 37.1 Hemodialysis Catheter Care

Items Required

- Bandage scissors
- Two sterile barrier drapes
- Clean examination gloves
- Surgical mask
- Surgical scrub and solution (chlorhexidine or povidone-iodine)
- Sterile gauze pads
- Syringes
- Heparinized saline
- Injection caps
- Antiseptic cream
- Porous tape
- Cohesive bandage
- Cast padding
- Conforming bandage

Procedure

1) Gather materials.
2) Perform hand hygiene.
3) Unwrap catheter bandage by cutting bandage on opposite side of neck from where catheter is (Figure 37.6a).
4) Clean area around catheter exit site.
5) Assess catheter exit site for redness, swelling, odor, or discharge, and assess subcutaneous tunnel for signs of infection or excess bruising.
6) Remove cohesive bandage and the tape that is on the clamps.
7) Place a sterile barrier around catheter to prevent ports from touching fur or skin (Figure 37.6b).
8) Don a surgical mask and exam gloves (mask and gloves should be worn from here until you begin wrapping the catheter again).
9) Perform a surgical-type scrub on both ports, extending from the clamps to the tops of the injection ports (Figure 37.6c).
10) Place another sterile barrier around catheter.
11) Have two squares of *sterile* gauze within reach, as well as all syringes that are needed.
12) Open access/proximal port by removing injection cap.
13) Wipe port opening with sterile gauze.
14) Withdraw the exact volume (the exact volume for each side is printed on the catheter) of the lumen and discard. This is the locking solution, so you must *never* flush the catheter first (Figure 37.6d).
15) Flush lumen with 6 cc fresh (prepared within 24 hours) heparinized or 0.9% saline.
16) Repeat steps 13–16 on the return/distal side.
17) Replace the locking solution by injecting the exact volume of each lumen (Figure 37.6e).
18) Place a new injection cap on each lumen (Figure 37.6f).
19) At this point, you can remove your gloves and mask.
20) Tape both clamps shut.
21) Place a piece of cohesive bandage around both ports.
22) Place a gauze square with an antiseptic cream over the catheter exit site (Figure 37.6g).
23) Wrap catheter with cast padding, conforming bandage, and then a flexible cohesive bandage. Wrap tightly enough that the bandage stays in place but not too tightly (Figure 37.6h).
24) Place a strip of porous white tape around both ends of bandage to anchor it to skin and prevent slipping. This is especially important for active animals.
25) Place a final piece of tape with the words "DO NOT CUT/DO NOT USE" on the outside of the wrap (Figure 37.6i).

sodium citrate is used as a locking solution in some units. Higher concentrations of citrate can be used but create a risk of hypocalcemia if inadvertently flushed into the patient [20]. Each dialysis catheter has the exact volume of the two lumens printed on the catheter and/or in the package insert, which informs the handler of the volume of anticoagulant solution to instill.

Performing Hemodialysis

Hemodialysis Machine Preparation

The structure and function of dialysis machines was described earlier; the following is instruction regarding the steps necessary to prepare a machine for a hemodialysis treatment. Each model of dialysis machine has a specific and detailed set-up protocol, but all include these same general steps.

First, for IHD machines, the water treatment unit is turned on and any daily water testing is done. Then the dialysis machine is turned on and the acid and bicarbonate concentrate containers are attached. The machine runs through a series of internal tests as the dialysate is being proportioned. During this time, the extracorporeal circuit is loaded onto the machine. The next step is to prime the extracorporeal circuit, which on some machines can be done immediately, and on others must wait until the internal tests are complete or the dialysate is ready. Priming involves filling with saline all sections of the extracorporeal circuit that will contain blood, thus removing all air. Air

removal is essential because blood that comes into contact with any trapped air will be more likely to clot.

Once the circuit is filled with saline, the machine is put through a recirculation phase. The access and return patient lines are connected to each other, and the saline is then circulated throughout the loop created by the tubing and the dialyzer. The purpose of this phase is to remove any residual substances from the manufacturing and sterilization process of the dialyzer and tubing [21]. During recirculation, the blood pump is running quickly, so saline flow through the circuit is rapid. This aids in propelling any remaining air bubbles from the dialyzer fibers or the sides of the blood tubing into the pressure chambers, which also act as air traps. With most machines, there is no one specific point during set-up at which the treatment parameters (treatment time, fluid to be removed, dialysate concentration, anticoagulant protocol) must be set. Each hemodialysis unit, though, should have an established protocol that defines when these parameters are set to ensure that they are set appropriately for each treatment.

When the recirculation phase is complete, the saline in the tubing and dialyzer is flushed out by the priming solution. The priming solution is the fluid given to the patient to replace the volume of blood removed when the dialysis treatment begins. The blood pump is used to flush the circuit with twice the priming volume, using the desired priming solution (e.g. 0.9% NaCl, hetastarch solution, blood). At this point, some machines are ready to start a treatment. Other machines need to run through another set of internal tests before they are ready.

With CRRT machines, set-up involves loading the circuit, priming it with saline, attaching the bags of dialysate and replacement fluid, followed by automatic testing to ensure correct machine function. After preparation, the prescription parameters are entered.

For most machines, whether IHD or CRRT, machine set-up and preparation will take 15–30 minutes with an experienced operator.

Patient Preparation

Patient preparation includes an assessment of a standard set of pretreatment parameters as well as preparation of the hemodialysis catheter. Parameters to be assessed prior to each dialysis treatment should include a blood pressure, heart rate, packed cell volume (PCV), total protein, body weight, temperature, activated clotting time or other measure of coagulation, and the patient's attitude, mentation, and hydration status. If the systemic arterial blood pressure is less than 80 mmHg systolic, we generally recommend use of pressor agents to increase blood pressure before initiating hemodialysis. If the blood pressure cannot be maintained above 80 mmHg, the patient can experience life-threatening hypotension when the dialysis treatment is started. In our experience, if the PCV is not at least 22%, a blood prime or blood transfusion will likely benefit the patient. The body weight and total protein will help assess the patient's hydration, and the weight pre- and post-treatment will aid in assessing fluid balance during the dialysis treatment because any weight changes in that short a period of time are due to fluid gain or loss. Finally, the activated clotting time will help to determine how much heparin to give the patient initially, and how much to infuse during the dialysis treatment.

Prior to each dialysis treatment, a set of serum biochemical parameters is generally measured that includes, at the least, urea, creatinine, phosphorus, and potassium concentrations. It is not always essential to have these results before starting the dialysis treatment, but it is often useful to see the results within an hour of beginning treatment. Re-measuring these same parameters at the end of the treatment provides a useful method of determining the adequacy of the dialysis treatment, which is discussed in the monitoring section of this chapter.

The hemodialysis catheter needs to be prepared with great care. As discussed previously, only properly trained personnel should handle this catheter. The catheter should be opened using the same protocol as for changing the locking solution (Protocol 37.1). After the locking solution is removed, blood samples for pretreatment blood work are taken through the catheter. The loading dose of heparin can be administered through the catheter at this time if prescribed.

Starting Treatment

When both the machine and the patient are ready, it is time to connect the patient and start dialyzing. The access patient line of the extracorporeal circuit is connected to the proximal lumen of the dialysis catheter, and the return patient line of the circuit is connected to the distal lumen of the catheter. This connection should be covered with gauze soaked in antiseptic to minimize contamination during the treatment. The patient lines are anchored to the patient in some manner (attached to harness or thoracic limb) to prevent excess pressure directly on the dialysis catheter.

As the blood is removed from the patient through the arterial circuit line, the priming solution is infused into the patient through the venous circuit line. The blood is removed slowly to try to prevent a sudden drop in blood pressure. The blood will fill more and more of the extracorporeal circuit and eventually fill the dialyzer. A button is pressed to start the dialysis treatment when blood has filled the dialyzer. When this button is selected, treatment time begins to count down, heparin infusion begins, programmed fluid removal

begins, and any other programs that run during the dialysis treatment will begin.

Ending Treatment

The dialysis machine alerts the operator when the specified treatment time is finished. The operator can also elect to end the treatment early, usually in emergency situations. About 10 minutes before the end of treatment, the catheter is prepared for disconnection by uncovering and scrubbing the connection site. The procedure is the same as for opening the catheter. When treatment time is complete, blood for post-treatment laboratory work is drawn. The exact time of sampling (i.e. immediately, two minutes after treatment ends) and site of sampling (i.e. extracorporeal circuit, dialysis catheter) may vary between different dialysis units but should be consistent within a unit. Then the patient's blood is returned in a procedure called rinseback. Most commonly, the access patient line is attached to saline. The blood pump is started and the saline is drawn in so that it flushes the blood from the circuit back into the patient. In especially monitored situations, the access line can be left open to the air without attaching the saline bag, if there is a concern about volume overload in the patient, but this risks creating a massive air embolus for the patient. When rinseback is complete, the locking solution is infused into both lumens of the dialysis catheter and the catheter is wrapped. Finally, post-treatment patient parameters, which are the same as pretreatment parameters, are assessed and recorded.

Monitoring During the Hemodialysis Treatment

There are a number of patient and machine parameters that should be monitored throughout the hemodialysis treatment (Box 37.3). The following is a summary of the parameters that are routinely measured; additional parameters may need to be assessed in individual patients. These parameters are recorded in the patient chart.

Box 37.3 Parameters to Monitor During Dialysis

Recommended	Optional[a]
Blood pressure	Blood volume
Heart rate	Hematocrit (inline)
Ultrafiltration rate	Temperature
Blood flow/Access pressures	Oxygen saturation
Heparin rate	
Activated clotting time	
Adequacy	

[a]Based on patient status.

Blood Pressure

Blood pressure may decrease for a variety of reasons, including acute decrease in effective circulating blood volume associated with filling the extracorporeal circuit with blood, inflammatory reactions associated with exposure of blood to the dialysis membrane, rapid ultrafiltration, excessive ultrafiltration, and bleeding from excessive anticoagulation or uremic thrombocytopathy. The patient's underlying disease can also lead to hypotension.

The frequency of blood pressure monitoring depends on the circumstances, and certainly patients with unstable or marginal blood pressure measurements or those developing clinical signs of hypotension should be monitored frequently. In our dialysis unit, blood pressure measurements generally are recorded 15 and 30 minutes after starting the dialysis treatment and then every 30 minutes thereafter.

Coagulation

When blood is removed from the patient and circulated through the tubing and dialyzer, it usually clots within an hour in the absence of an anticoagulant. The majority of IHD treatments are performed using heparin as the anticoagulant. The ACT is generally used to monitor heparin therapy. As previously mentioned, the ACT is assessed prior to starting treatment, and this value is used to determine the initial heparin dose. The ACT should be checked 30 minutes after starting dialysis to determine if a dose adjustment is necessary [4]. The normal range for ACT using a Medtronic ACT-Plus machine is 80–100 seconds in dogs and 100 seconds in cats (Poeppel and Bogue, unpublished data). The target ACT during a dialysis treatment is 1.6–2 times normal [5]. If the ACT is in the target range and no dose adjustments are needed, it is monitored hourly thereafter. The ACT is measured 30 minutes after any dose adjustment. Partial thromboplastin time evaluates the arm of the coagulation cascade affected by heparin and could be used instead of ACT.

For patients where systemic anticoagulation is contraindicated, regional citrate anticoagulation can be used. This involves infusing citrate into the blood as it is removed from the patient, to chelate calcium and thus prevent clotting. Simultaneously, calcium is infused into the patient to prevent hypocalcemia. The ionized calcium concentration in the circuit needs to be monitoring to ensure adequate chelation/anticoagulation, and the ionized calcium of the patient needs to be monitored to avoid symptoms of

hypocalcemia. This process is more labor intensive than heparin anticoagulation [22].

Regional citrate anticoagulation may become the preferred method of anticoagulation in continuous therapies because continuous systemic anticoagulation is more problematic than intermittent. Citrate anticoagulation is relatively contraindicated in patients with severe liver failure, due to their inability to metabolize citrate [5].

Access Pressure and Blood Flows

The pressure transducers that attach to the blood cartridge allow the dialysis machines to determine pressure in the access and return tubing segments. The dialysis technician should monitor these pressures throughout the treatment. The access pressure is negative when the blood pump is running. The access pressure will be excessively negative if the arterial lumen of the dialysis catheter is functioning poorly. Causes of poor function include kinking, being lodged against the vessel wall, or partial occlusion by a thrombus. Return pressure is positive when the blood pump is running. This return pressure will be excessively positive if there is an obstruction to the return of blood to the patient. High return pressure could be due to kinking or thrombosis of the venous lumen of the dialysis catheter or a clot obstructing the filter in the venous chamber.

In addition, two specific values are routinely measured for each dialysis treatment. One is the maximum blood flow maintained during the treatment. If this value decreases over time, it may indicate impending catheter malfunction. The other value is the blood flow at a certain access pressure, for example at minus 200 mmHg. If the blood flow at this same pressure decreases over time, it may also indicate an impending problem with the catheter. Blood flow can be affected by other factors, such as the patient's blood pressure and intravascular volume, which must be taken into account when blood flows are being evaluated. These factors have usually stabilized within the first week of dialytic therapy.

Hematocrit

The patient's hematocrit is measured at the beginning and end of every dialysis treatment. An inline hematocrit monitor (Crit-Line®, HemaMetrics, Kaysville, UT) records real-time hematocrit throughout the dialysis treatment. This information is very helpful in some cases. The hematocrit tends to drop at the beginning of the treatment because the patient's blood volume is diluted with the priming solution. Blood transfusions are sometimes administered during a dialysis treatment, so an inline monitor immediately displays the efficacy of the transfusion. Finally, if fluid therapy is required to maintain blood pressure during the dialysis treatment, the inline monitor provides information that helps the clinician avoid severe hemodilution.

Blood Volume

Changes in blood volume during a dialysis treatment can be measured using the inline hematocrit monitor also. Presuming that the red blood cell mass remains constant (i.e. there is no continuing bleeding or blood administration), any changes in the hematocrit reflect changes in plasma volume. An increase in hematocrit concentration indicates plasma volume removal (via ultrafiltration), whereas a decrease in hematocrit concentration would be expected if fluid is being administered at a rate exceeding fluid removal via ultrafiltration. A rapid decrease in intravascular volume may precipitate symptomatic hypotension. It is not advisable to have more than a 10% decrease in blood volume within one hour [4, 5]. Some of the newest dialysis machines include an integrated hematocrit monitor so that an external monitor is not necessary.

Oxygenation

It is not always necessary to measure oxygen saturation in a stable patient undergoing hemodialysis, but it is generally part of the overall treatment plan in the unstable patient or the patient with respiratory or cardiovascular compromise. It can be useful to measure oxygenation in patients receiving aggressive ultrafiltration because a decrease in oxygen saturation is often a precursor to a decrease in blood pressure [4]. An inline hematocrit monitor measures the oxygen saturation of the blood in the extracorporeal circuit, which in veterinary patients is central venous blood. This is an effective method of measuring changes in oxygen saturation that can adversely affect the patient's blood pressure. The inline monitor is convenient because the sensor is attached to the tubing and thus is not dislodged by patient movement.

Adequacy

The adequacy of dialysis treatments, meaning the amount of waste product that has been cleared from the patient, should be measured, ideally every dialysis treatment initially, then on a routine basis (i.e. weekly). By tracking adequacy, the dialysis team is assured that the patient is receiving the prescribed dose of dialysis. A complete discussion of dialysis adequacy is beyond the scope of this chapter, so only the two most commonly used methods are briefly discussed here. More detailed discussions have been published for IHD [4, 23].

The most straightforward method of measuring adequacy is by calculating the urea reduction ratio, which is

calculated by subtracting the post-treatment blood urea nitrogen (BUN) from the pretreatment BUN and then dividing that value by the pretreatment BUN. The ratio typically exceeds 90%, except during the initial few dialysis treatments for each patient.

The most common measure of dialysis dose in human hemodialysis is Kt/V, in which K is a clearance constant of the dialyzer, t is time on dialysis, and V is volume of distribution [24]. The constant K for each dialyzer is calculated in vitro under specific conditions and is published on the package insert. The actual K during the treatment can be calculated using the blood flow and the simultaneous BUN of blood flowing into and out of the dialyzer. Most newer dialysis machines have a program that measures a treatment Kt/V, so this may become the most common measure of adequacy in veterinary medicine as well.

Other Parameters

Any patient parameters that are monitored in the intensive care unit or patient ward should also be monitored during hemodialysis. Examples are continuous electrocardiogram or temperature monitoring, pulse oximetry for arterial oxygen saturation, and urine output.

Record Keeping

Record keeping for a hemodialysis treatment should be thorough and descriptive. This entails documenting the dialysis prescription itself, a summary of what was accomplished during the treatment, patient or machine complications, all values obtained while monitoring the patient, and any medical treatments done on the patient during dialysis. As with any monitoring record, the best way to assure all parameters are recorded is to have a template for recording. This template can be a preprinted form or an electronic veterinary dialysis database. It is essential that the dialysis technician performing the treatment maintain meticulous records to keep track of what parameters work best for a particular patient and to aid in troubleshooting if problems should arise.

Complications and Special Considerations

Patients with kidney disease severe enough to require hemodialysis are complex patients, and case management is rarely straightforward. Patients exhibit the usual manifestations of uremia commonly encountered in uremic patients managed with traditional medical therapies. Hemodialysis patients may also develop complications directly related to the dialytic therapy. Finally, the clinician may encounter long-term complications of uremia that are rarely seen because of limited patient survival time with traditional medical therapy of the end-stage renal disease patient. This section focuses mainly on complications directly related to dialysis therapy.

Technical Complications

Technical complications due to machine errors or malfunctions are rare because of the number of redundant monitors and alarms built into modern hemodialysis machines. When they occur, they may range from mild to devastating problems. Complications related to the water treatment system include chemical or infectious contamination [13]. It is therefore essential to maintain and monitor the function of both the dialysis machine and the water treatment system to achieve peak performance. The operator must also have superb knowledge of the machines so as to be able to troubleshoot quickly during a dialysis treatment to prevent minor problems from escalating. The hemodialysis technician is generally responsible for maintaining the dialysis equipment and monitoring dialysis treatments.

Operator errors can also occur, and the number and severity depend on a variety of factors, including the training and experience of the operator, the work environment, and some patient factors. Again, it is important to have highly trained personnel to minimize these errors and their consequences.

Hypotension

A decrease in blood pressure is common at the start of a hemodialysis treatment [13]. The volume of blood required to fill the extracorporeal circuit in relationship to the patient's total blood volume can be considerable, up to 40% in smaller cats (Table 37.1). Priming the circuit with a colloid (i.e. 50 : 50 mixture of 0.9% NaCl and hetastarch) helps mitigate the drop in blood pressure in cats and small dogs but is not generally necessary for medium to large dogs (in which a saline prime suffices). There are concerns about the use of synthetic colloids such as hetastarch inducing AKI. Most patients are able to autoregulate and return blood pressure to almost baseline values within 30–60 minutes of the start of dialysis, but some cannot. These patients may require intervention if the blood pressure drops too low. The blood pressure generally returns to predialysis values when all the patient's blood is returned at the end of the dialysis treatment.

Exposure of the blood to a bioincompatible dialyzer membrane can activate the complement and coagulation cascades, releasing several mediators that may cause

hypotension [25, 26]. Use of synthetic membranes can minimize this problem. Rapid ultrafiltration may lead to hypotension, if the rate of removal from the vascular compartment exceeds the capacity for refilling from the interstitial compartment. A general guideline of keeping the ultrafiltration rate below 10–20 ml/kg/hour is recommended, although this value may be adjusted up or down depending on patient status. Monitoring blood volume (e.g. with an inline hematocrit monitor) may predict symptomatic hypotension, allowing intervention before hypotension occurs [3, 4]. If hypotension occurs despite decreasing or stopping ultrafiltration (temporarily or for the duration of the treatment), small boluses of crystalloid or colloidal solutions, or use of pressor drugs may be used to correct the blood pressure.

Dialysis Disequilibrium Syndrome

Dialysis disequilibrium syndrome (DDS) is a syndrome induced by rapid, or highly efficient, dialysis in severely azotemic patients. The cause of this syndrome is not well described, but it results secondary to the development of cerebral edema induced by rapid changes in the osmolality of the blood [4, 5, 27]. More widespread adoption of long slow initial treatment prescriptions has made DDS a rare occurrence. It was most likely to occur during the first few dialysis treatments when uremia was more severe. However, DDS can occur at any time, even with chronic dialysis.

Clinical signs of DDS include agitation, disorientation, seizures, vomiting, coma, and death. Dogs usually have premonitory signs such as restlessness in a previously quiet dog. Cats frequently have no noticeable premonitory signs and may rapidly go from a normal appearance to a coma-like state. DDS may occur at any time during dialysis or up to 24 hours after dialysis [4]. Therefore, it is essential that patients be monitored for signs of DDS continually for a full day after each of the first few dialysis treatments. The signs of DDS may reverse entirely within a few hours, particularly if the signs are mild or treatment for DDS was started early. More severe signs may persist for up to 24 hours. Some patients do not regain consciousness or die acutely.

Treatment of DDS involves dissipating the blood–brain osmotic gradient by infusing osmoles into the bloodstream. Mannitol is the most commonly used treatment in veterinary hemodialysis [4]. Hypertonic saline has the same short-term effect as mannitol but creates an undesirable sodium load. Some dialysis machines have the capacity to increase the sodium concentration of the dialysate quickly, to cause rapid diffusion of sodium from dialysate into the bloodstream, having the same effect as an IV bolus of hypertonic saline.

Prevention of DDS is clearly desirable. Decreasing the efficiency of hemodialysis during the first few treatments is the main method of prevention. Methods of accomplishing this goal include slow blood flows, slow dialysate flow or intermittently interrupting dialysate flow, reversing the dialysate lines so dialysate flows concurrent to blood, and reversing the access and return ports on the catheter to increase recirculation. To achieve adequate clearance, treatment times need to be extended, providing a long, slow, gentle treatment. Mannitol may be given prophylactically in high-risk patients such as severely uremic patients (BUN >150 mg/dl), small patients (<5 kg), or those with preexisting central nervous system disease. The dose is administered in the first one-third to one-half of the dialysis treatment for the first one to three treatments, although using the prescription guidelines above make this unnecessary in most situations [4, 14]. Sodium profiling, in which the dialysate sodium is initially higher and gradually decreases during the treatment, is another preventive measure. Sodium profiling is a specific program set on the dialysis machine when prescribed.

Hemorrhage

Anticoagulation is necessary during hemodialysis, so a risk of hemorrhage is present if the anticoagulation dose is too high for the patient's metabolism. Mild forms may involve bleeding from the skin exit site of the dialysis catheter or other insertion sites. Internal bleeding, including bleeding from gastric ulceration, central nervous system hemorrhage, or massive pulmonary hemorrhage, have been encountered [28]. Discontinuation of the anticoagulant (usually heparin), administration of a reversal agent (i.e. protamine sulfate for heparin), and red cell or plasma transfusion may be required. Bleeding problems can be minimized by careful control of anticoagulation and minimizing sources of bleeding. It is best not to do any procedures, place catheters, or even pierce the skin in any manner immediately before or after a dialysis treatment.

To prevent or minimize hemorrhage after the hemodialysis treatment, heparin is usually stopped 30 minutes before the end of treatment. The patient should receive no needle sticks for eight hours after the treatment. Even removal of a catheter can lead to excessive bleeding in a heparinized patient. All procedures such as thoracocentesis or feeding tube placement should be performed at least eight hours after the dialysis treatment. Surgeries or more invasive procedures are best scheduled on nondialysis days. If the patient requires a treatment or procedure that could cause bleeding within the eight-hour period, a reversal agent (such as protamine sulfate) should be used.

Respiratory Complications

Mild to severe hypoxemia is common during dialysis both in human and animal patients. Contact of blood with the dialyzer membrane activates the alternate complement pathway. This causes leukocyte and platelet aggregation in the pulmonary microvasculature that interferes with oxygen diffusion [29]. The maximal effect of this is seen within 30–60 minutes of the start of dialysis and resolves within 120 minutes after discontinuation of dialysis. Therefore, oxygen therapy that began during a dialysis treatment may need to be continued for a few hours after the treatment, but improvement is expected. Some patients come to dialysis with respiratory compromise from pulmonary edema or pleural effusion resulting from volume overload. Pulmonary hemorrhage is common in dogs with leptospirosis [30].

Gastrointestinal Complications

Anorexia, nausea, and vomiting are common complications of kidney disease but also may be seen at the start of hemodialysis secondary to diversion of blood flow from the gastrointestinal tract caused by hypotension, bioincompatibility reactions to the membrane, or contaminants in the dialysate. Dialysis disequilibrium can also cause centrally mediated nausea and vomiting [31]. Using slow blood flow rates at the beginning of dialysis treatments with a gradual increase to the prescribed rate minimizes these signs and patient discomfort. It is not standard to fast a patient the morning of a hemodialysis treatment, but it is advisable to allow at least an hour between feeding and initiation of dialysis in case one of these complications arise. If the patient routinely vomits at the start of dialysis regardless of preventive measures, a morning fast may be considered.

Thrombosis

Catheter

Catheter thrombosis may be intraluminal or extraluminal. It can occur at any time but is uncommon within the first week after catheter placement unless there have been major problems with anticoagulation in that time period [15]. Intraluminal thrombosis can affect catheter flow when severe enough. Aggressive flushing of the affected lumen may restore blood flow but usually does not remove all remnants of thrombi. Mechanical disruption of thrombi by feeding a stylet into the lumen may be slightly more effective. Despite the ever-present risk of inducing significant or fatal thromboembolic complications with these maneuvers, rarely have we encountered clinically detected problems. A thrombolytic agent such as tissue plasminogen activator can be instilled in the catheter for a period of time. This seems to be effective in the short term, but treatment frequently needs to be repeated within a week [32].

Thrombolytic therapy should be initiated as soon as aggressive flushing is not effective so that thrombi do not continue to grow and completely obstruct the catheter. If these measures are unsuccessful, the affected catheter can be replaced. A percutaneous catheter is relatively easy to replace over a guidewire with no or minimal sedation [14, 33]. Replacement of a tunneled catheter requires heavy sedation or general anesthesia and is technically more difficult. Both techniques carry a risk of contamination of the new catheter if the exit site or tunnel are infected, and they are more likely to result in loss of the vessel. If the catheter cannot be replaced due to technical issues or other factors, a new catheter may be placed in the opposite jugular vein. Extraluminal thrombosis can be even more troublesome. A thrombus may be attached to the outside of the catheter, but it can act as a flap or ball valve that occludes catheter flow. Aggressive flushing may remove the thrombus if the attachment is weak. Thrombolytic therapy may not be very effective because it is hard to direct this therapy to an extraluminal thrombus. Systemic thrombolytic therapy can lead to uncontrollable hemorrhage [34]. Replacement of the affected catheter is the last option, which carries with it the risk that the thrombus will be stripped off the catheter during removal and enter the patient's circulation.

Systemic

Patients who have hemodialysis catheters in place for more than two weeks are at risk of developing a right atrial thrombus. Pulmonary thromboembolism from platelet aggregation or thrombus formation induced by the catheter can cause acute onset of mild to severe dyspnea during or between dialysis treatments [14].

Prevention

Patients with indwelling hemodialysis catheters routinely receive anti-platelet therapy (e.g. clopidogrel or low-dose aspirin) to prevent systemic thrombus formation. As previously mentioned, the catheter lumens are filled with an anticoagulant lock between dialysis treatments to prevent intraluminal thrombosis.

Infection

There are multiple potential sites of infection in the hemodialysis patient. Uremia decreases immune

function, and indwelling catheters (vascular or urinary) and feeding tubes are potential portals of entry for bacteria [35, 36]. Other open sites, such as surgical incisions or pressure sores, are possible entry sites for bacteria as well. Finally, the dialysis catheter and the extracorporeal circuit are potentially large sources of bacteria. If bacteria from any site enters the vascular system, they can then reach the dialysis catheter and adhere to it. Once bacteria adhere to the dialysis catheter, they can produce a biofilm that adheres to the walls of the catheter and protects the bacteria from removal or destruction. Then the only way to eliminate all bacteria is to remove the dialysis catheter [15].

For this reason, extra vigilance is required to prevent infections from developing in hemodialysis patients. All drugs and flushes given through any catheter should be freshly prepared (we prefer less than 24 hours old). Aseptic technique must be maintained for catheter placement, feeding tube placement, and other procedures. Urinary catheters are typically removed once hemodialysis has been initiated. Examination gloves are worn when setting up the dialysis machine, and examination gloves and a surgical mask are worn when accessing the dialysis catheter. Finally, as mentioned previously, only trained personnel should handle the dialysis catheter.

Edema

Volume overload may manifest as pulmonary edema, pleural effusion, ascites, and generalized peripheral edema. Ultrafiltration during dialysis and careful attention to fluid balance can help minimize this problem [4]. The problem may persist longer in anuric or oliguric patients than nonoliguric or polyuric patients. Facial, intermandibular, and forelimb edema may occur in dogs over time and can be severe. Edema in these specific locations may be an indication of partial cranial vena caval occlusion by the catheter itself, or thrombosis or stenosis induced by the catheter [37]. In many dogs, hypoalbuminemia is a concurrent problem due to ongoing renal albumin loss, loss in the extracorporeal circuit, gastrointestinal loss, and suppressed synthesis due to systemic inflammation. Hypoalbuminemia can exacerbate edema formation.

Malnutrition

Malnutrition is common in uremic patients due to uremia-induced gastrointestinal complications and high metabolic needs associated with AKI and other critical illness. Early and aggressive nutritional support is prudent. It is unclear how to determine the exact protein requirements of the AKI patient [38, 39]. Certain amino acids are lost during the dialysis treatment, particularly taurine and carnitine, so patients on long-term dialysis therapy should receive taurine and carnitine supplementation [40].

Nausea, vomiting, and the dialysis treatments themselves can interfere with feedings, which adds to the problem of malnutrition. A feeding tube is often placed at the time of dialysis catheter placement to allow early nutritional support. Nausea and/or vomiting can often be alleviated by medications and control of uremia via hemodialysis. Patients do not need to be fasted before dialysis treatments, and feedings scheduled during dialysis should not be skipped unless necessary. If feedings have to be missed during a treatment, they can be supplemented when dialysis is complete.

Aluminum Toxicity

Aluminum is used in many municipal water treatment plants as one stage of the water purification process. If the hemodialysis water treatment process is not sufficient, trace amounts of aluminum can appear in the dialysate. Aluminum-containing phosphate binders provide an additional and greater source of aluminum. Acute aluminum toxicity can occur if water treatment is inadequate, but it is generally encountered only after long-term exposure with chronic dialysis. Aluminum accumulation can lead to a microcytic, hypochromic anemia. Clinical signs associated with aluminum toxicity include neurologic or neuromuscular signs including mild weakness, paresis, dullness, obtundation, or coma. There have been two cases of hemodialysis-related aluminum toxicity reported in veterinary medicine [41].

Anemia

Small amounts of blood are lost with each dialysis treatment, and this combined with any gastrointestinal losses and iron deficiency leads to anemia in almost all dialysis patients [39]. Patients receiving dialysis treatment for more than two to three weeks generally require iron supplementation and some erythropoietin hormone replacement therapy such as darbepoetin.

Medication Dosing

It is often necessary to adjust the dose and timing of medications given to a patient receiving hemodialysis. Many drugs are removed from the blood during dialysis, some to a significant degree [42]. For this reason, it is best to give all once-a-day medications in the evening. It also may be necessary to withhold a medication until after dialysis or to supplement the dose when dialysis is complete. This may be particularly important when administering antibiotics,

pain medications, cardiac medications, or antiseizure medications.

Patient Care

The typical IHD treatment lasts from four to five hours, which means an animal is away from the patient care ward for a significant period. In addition to feedings and medication, therapies such as peripheral catheter care, wound management, turning, bathing, and physical therapy may be indicated during this time. Considerations must be made to determine if these therapies are safe to perform while the patient is undergoing hemodialysis.

During an IHD treatment, patients are essentially tethered to the hemodialysis machine because the blood lines of the extracorporeal circuit are attached to the dialysis catheter. The IHD machine must remain connected to the incoming water source, so it is not freely mobile. This means the patients are not free to walk around to urinate, defecate, and exercise during the dialysis treatment, so it is beneficial to allow ambulatory patients time to walk outside or around a room before and after the treatment.

Patients are generally anticoagulated during an IHD treatment, so any therapies that have the potential to cause bleeding, such as wound debridement or nail trims, should be avoided. Peripheral catheter care should be avoided if there is a high probability that the catheter will be removed. Gentle wound cleaning and application of topical ointments is acceptable.

Patient temperament will dictate whether certain therapies, such as oral medication administration, can be performed. The dialysis catheter is unwrapped during hemodialysis, so any therapy that would cause the patient to struggle and possibly damage or remove the catheter must be done only after the catheter has been safely wrapped.

Finally, dialysis catheter flow can dictate how much the patient should be moved. A well-placed catheter in a medium to large patient often flows very well when the patient is in a variety of different positions. In this case, turning the patient, expressing the bladder, doing passive range-of-motion exercises, and any similar therapy is unlikely to pose problems while the patient is undergoing hemodialysis. Changes in intrathoracic pressure can affect catheter flow, so coupage is not recommended until after the treatment. Catheter flow problems can occur in any patient during any given treatment, so the hemodialysis technician often determines on a day-to-day basis how much to allow or prevent movement of a patient. There will be times when it is best to keep the patient very still throughout the entire hemodialysis treatment.

Outcomes

Overall, 40–60% of veterinary patients with acute uremia treated with hemodialysis survive [4, 43]. In recent studies, the reported survival rates for AKI from infectious causes were 58–100% [44–46]. Hemodynamic and metabolic causes of AKI had a 40–72% survival rate [46, 47]. Only 20–40% of patients with AKI from toxic causes survive [44, 46]. Of the patients receiving hemodialysis that do not survive, about half of those die or are euthanatized due to extrarenal conditions (e.g. pancreatitis, respiratory complications). About one-third of nonsurvivors are euthanized due to failure of recovery of renal function. Continuing uremic signs, dialysis complications, and unknown causes account for the remaining patient deaths. As with patients treated medically, approximately half of hemodialysis patients regain normal renal function (defined by normal serum creatinine concentration) and half have persistent chronic kidney disease [5].

Summary

Box 37.4 provides a summary of the chapter. IHD is a highly advanced therapy used primarily to treat patients with kidney disease. For patients with AKI, IHD is initiated when medical management fails, and it is a means of stabilizing the patient long enough to allow for renal repair. For patients with chronic kidney disease, IHD is a method of extending the animal's life. IHD can also be used to remove excess fluids or electrolytes, restore acid–base balance, or treat toxicities and drug overdoses.

Overall survival rate of animals undergoing IHD for AKI is 40–60%. The patient population in this case generally has a prognosis of 0% survival without some type of renal replacement therapy. Hemodialysis is therefore a viable alternative to euthanasia for veterinary patients.

Box 37.4 Summary

- Hemodialysis is a method of clearing the blood of uremic toxins when the kidneys are not functioning properly.
- Patients with acute kidney injury, chronic kidney disease, toxicities, and fluid or electrolyte imbalances are candidates for hemodialysis.
- Hemodialysis requires equipment that must be maintained and operated by highly trained personnel.
- Patients receiving hemodialysis can experience complications directly related to treatment in addition to complications related to their disease.
- Overall survival of hemodialysis patients is 40–60%.

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38

Peritoneal Evaluation

Laura Osborne and Lindsey Strang

Peritoneal evaluation, particularly via point of care ultrasound (POCUS), has become a routine part of a comprehensive and thorough assessment of small animal emergency and critical care patients. Dogs and cats with an acute condition of the abdomen, often characterized by abdominal pain, represent a common emergency presentation. Complications involving the peritoneal cavity, such as ileus and ascites, are common in hospitalized critically ill patients. Patients with abdominal pain typically have abnormalities within the peritoneal cavity. However, disease of the retroperitoneal cavity, as well as the lumbar and sacral spine, can also result in referred or simulated abdominal pain. Clinical signs that accompany abdominal pain, such as nausea, vomiting, diarrhea, and abdominal distention, can aid in making an accurate diagnosis.

In general, abdominal pain is caused by capsular stretch of solid organs, or distention, traction, or forceful contractions of hollow organs. Additionally, inflammation and ischemia initiate production of proteinases and other vasoactive substances that can stimulate abdominal nerve endings. Intra-abdominal pathology can result in tissue necrosis and loss of organ function; therefore, rapid identification and treatment of the underlying abnormality is essential to minimize the occurrence of serious complications.

Diagnostic procedures used to evaluate the peritoneal cavity include physical examination with thorough abdominal palpation; abdominal imaging such as radiographs, ultrasound, or computed tomography (CT); and evaluation of peritoneal fluid obtained by abdominocentesis, diagnostic peritoneal lavage, or from a previously placed abdominal drain. Additionally, intra-abdominal pressure (IAP) can be monitored in patients at risk of the development of intra-abdominal hypertension (IAH). In some cases, an exploratory laparotomy may be indicated for diagnostic and therapeutic reasons. Clinically, the terms "peritoneal" and "abdominal" synonymously describe the space in which the intra-abdominal contents reside, and the terms are used interchangeably throughout this chapter.

Signalment and History

Signalment can help to raise the index of suspicion for the cause of acute abdominal pain. For example, foreign body or toxin ingestion and infectious disease should be considered in young animals, while prostatic disease and pyometra should be considered in older, intact animals. Furthermore, an accurate and complete history can be informative in patients presenting for acute abdominal pain. In some circumstances, complete history taking will need to be delayed until after stabilization efforts are performed. Potential for exposure to toxins or dietary indiscretion and the possibility of foreign material ingestion should be assessed. The patient's past medical history should be established, and current medications and supplements documented. Vaccination status and preventive routine, as well as tick exposure and travel history should be recognized. The possibility of trauma should be considered. It is important to establish the time course and progression of clinical signs. This will help to establish the urgency of further diagnostic pursuit, with severe, acute, or rapidly progressive clinical signs warranting a more proactive approach.

Physical Examination

Physical examination with an emphasis on careful abdominal palpation is the initial step in evaluation of the peritoneal cavity. The abdomen should be examined systematically, starting with the spinal column and abdominal wall before evaluation of the deeper structures. Direct palpation of the spine and body wall will aid in the differentiation of back

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and/or muscle pain from conditions affecting the peritoneal structures. The integrity of the abdominal wall should be carefully evaluated to rule out penetrating injuries. Abnormalities in the contour of the abdomen suggest distention and/or enlargement of intra-abdominal structures or accumulation of fluid and/or air in the peritoneal cavity.

Gentle, persistent digital pressure should be used to evaluate the size and position of organs and for the presence of abdominal masses. Pain should be noted and localized. Pain localized to the cranial right quadrant suggests a pancreatic, duodenal, or pyloric abnormality. Caudal abdominal pain is associated with abnormalities of the reproductive or urinary tract. Diffuse abdominal pain suggests peritonitis or involvement of a significant portion of the intestinal tract.

Percussion (striking the body wall with short sharp blows and noting the tone of the resultant sound) aids in the detection of fluid or gas accumulation within the peritoneal cavity or within a distended hollow viscus. Abdominal auscultation has been recommended to detect hypermotility or ileus but lacks reliability (see Chapter 40 for more information).

A rectal examination should be completed to evaluate for the presence of abnormal stool, especially melena or hematochezia. In male dogs, the prostate gland should be assessed for size, symmetry, and presence or absence of pain. The urethra should be smooth and compressible, and any masses or irregularities should be noted. The pelvic canal should be palpated for evidence of pain, crepitus, or asymmetry and the lumbosacral lymph nodes evaluated. The anal sacs should be palpated for any masses.

Abdominal Imaging

Veterinary Point of Care Ultrasound (POCUS)

Abdominal imaging should be performed in all patients presenting with acute abdomen. In the emergency setting, POCUS is an invaluable first step and can provide a wealth of information with limited training and minimal compromise to the patient. It is also an essential monitoring tool for serial evaluation of patients hospitalized in critical care for peritoneal complications such as surgical site dehiscence or ileus.

The utility of POCUS in the emergency and critical care setting is vast and continuing to expand (Chapters 6 and 39) [1]. One of the most familiar techniques is the abdominal focused assessment with sonography for trauma scan (AFAST), which aids in rapid detection of free abdominal fluid [2, 3]. Serial AFAST examination may allow for detection of delayed fluid accumulations and can be used to monitor progression or resolution of free fluid accumulations over time. A further application of this technique is the abdominal fluid scoring system, which helps to characterize internal bleeding and the likelihood of blood transfusion requirement [4]. While more challenging, with some additional training, POCUS can also be used to identify pneumoperitoneum by identifying the enhanced peritoneal stripe sign, typically at the paralumbar region when the patient is placed in lateral recumbency [5].

Beyond evaluation for the presence of free fluid and air within the peritoneal cavity, POCUS can be used to identify gastric stasis and intestinal ileus. This can be achieved through identification of a distended, fluid-filled stomach and the absence or decreased frequency of peristaltic contractions [6]. This may help support the suspected diagnosis of gastroenteritis or post-operative ileus, identify patients at risk of regurgitation and enteral nutrition intolerance, and prompt timely medical intervention such as nasogastric tube placement for gastric evacuation and initiation of prokinetic therapies.

POCUS can also be used to select an area and provide guidance for diagnostic or therapeutic abdominocentesis. Causes of abdominal fluid accumulation are listed in Table 38.1. Urinary bladder volume can also be estimated using ultrasound in dogs and cats. Using measurements from the cystocolic ultrasonographic view, a noninvasive estimate of urine production can be made [9]. This avoids the technical challenges, risks, and financial implications associated with urinary catheterization. It can be used in the management of acute kidney injury with anuric or oliguric renal failure, as well as to tailor fluid therapy.

In addition to aiding diagnosis and monitoring of peritoneal disease, abdominal POCUS can be employed to further assess patient stability and guide resuscitation efforts. The technique shows promise in estimating the intravascular volume status of small animal patients, with evaluation of the caudal vena cava (CVC) diameter, CVC to aorta ratio, and the change in CVC diameter between inspiratory and expiratory phases of respiration reflecting volume status [10–12]. The CVC diameter normally changes approximately 50% throughout the respiratory cycle, being larger at the end of expiration. A flat or small-diameter CVC that lacks dynamic change (<10%) during inspiration and expiration indicates hypovolemia. Alternatively, a wide, large-diameter CVC that lacks dynamic change during inspiration and expiration is consistent with volume overload [13]. Information regarding volume status can be combined with evaluation of the gall bladder to provide further insight in some cases. The gall bladder halo sign (double-rimmed gall bladder wall) indicates gall bladder wall edema and supports the diagnosis of anaphylaxis when the CVC is flat. This is in comparison to right-sided volume overload seen with right-sided heart failure, pericardial effusion, or pulmonary hypertension when the CVC is concurrently wide [14]. See Table 38.2 for a summary of diagnostic criteria regarding abdominal imaging.

Table 38.1 Effusions classified by cause and associated disorders [7, 8].

Classification of effusion	Cause/associated disorders
Transudate:	
Protein-rich	Congestive heart failure
	Post-sinusoidal hypertension (portal hypertension)
Protein-poor	Hypoalbuminemia (PLN, PLE, burns)
	Lymphatic obstruction (neoplasia, abscess, thrombosis, lymphangiectasia)
	Presinusoidal and sinusoidal hypertension (cirrhosis, idiopathic portal hypertension)
Exudate:	
Septic	Gastrointestinal/biliary tract leakage
	Penetrating wounds
	Migrating foreign bodies
	Disruption of the urogenital tract
	Bacterial translocation
	Hematogenous spread
Nonseptic	Pancreatitis
	Feline infectious peritonitis
Ruptured vessel or viscus	Hemorrhage
	Chylous effusion
	Uroabdomen
	Bile peritonitis
Cell exfoliation	Neoplasia
	Reactive mesothelial proliferation

PLE, protein-losing enteropathy; PLN, protein-losing nephropathy.

Table 38.2 Imaging peritoneal evaluation diagnostic criteria.

Abnormalities	Diagnostic criteria
Radiographic	
Dog ratio of maximal small intestinal diameter to the narrowest width of L5 on lateral radiograph	< 1.4 unlikely to be obstructed
	> 2.4 likely to be obstructed
Cat ratio of maximal small intestinal diameter to the height of cranial endplate of L2	> 2 consistent with obstruction
Cat maximal small intestinal diameter	> 12 mm consistent with obstruction
Ultrasonographic	
CVC characterization	Flat or fat with < 10% dynamic change is consistent with hypovolemia
Gall bladder halo sign	Double-rimmed gall bladder wall
	With concurrent flat or wide CVC consistent with anaphylaxis
Tree-trunk sign	Distended hepatic veins draining into a distended CVC is consistent with right-sided volume overload
Urinary bladder volume estimation formula (ml)	Length (cm) × width (cm) × height (cm) × 0.2 × Pi (0.625)
Peristaltic contractions of the stomach and proximal duodenum	< 4–5 contractions/minute consistent with hypomotility

CVC, caudal vena cava.

Abdominal Radiographs

A complete set of three-view survey abdominal radiographs should be obtained in any patient presenting with abdominal pain. The abdominal and extra-abdominal structures should be evaluated in a systematic fashion. Deviation from normal density, shape, size, and/or location of abdominal organs may provide clues to abnormalities within the peritoneal cavity. Radiographs are most valuable in detecting organ size and location, and distention of a hollow viscus; they are less useful in detecting solid organ injury or dysfunction.

The serosal detail of both the peritoneal and retroperitoneal space should be evaluated. Decreased visualization of the kidneys, distention, or a streaky or mottled appearance of the retroperitoneal space is seen with abnormal fluid accumulation (urine, blood) or a space-occupying mass in the retroperitoneum. Differentials for decreased detail in the peritoneal space include free abdominal fluid, lack of abdominal fat, and carcinomatosis. Loss of serosal detail should trigger further evaluation for free abdominal fluid via ultrasound or abdominocentesis.

Radiographs should be examined for the presence of free gas in the peritoneal cavity. In the standard lateral view, free gas can be most easily detected between the stomach or liver and the diaphragm. The sensitivity for detecting free gas can be increased by positioning the patient in left lateral recumbency and making a horizontal beam radiograph focused at the least dependent area of the abdomen. The presence of free peritoneal gas in a patient with no previous open needle technique abdominocentesis or recent abdominal surgery that could introduce air indicates a penetrating injury of the abdominal wall, rupture of a hollow viscus, or rupture of an abscess or necrotic mass containing gas-producing microorganisms into the peritoneal cavity. These conditions are considered surgical emergencies.

Next, intestinal gas patterns should be evaluated. Ileus, resulting in the accumulation of fluid and gas within the intestine, may be seen with a number of conditions. Segmental ileus supports a diagnosis of intestinal obstruction. The normal diameter of the small intestine in the dog is

approximately two to three times the width of a rib, or less than the width of an intercostal space. In dogs, bowel obstruction is considered likely if the internal diameter of a small bowel segment is four times or more the width of a rib, two times or more the width of a vertebral body, or if the ratio of the diameter of the bowel to the height of the narrowest point of the body of the L5 vertebra is greater than 1.6 [15]. A more recent study found that dogs with a maximum small intestinal diameter-to-L5 vertebral body height ratio less than 1.4 are very unlikely to be mechanically obstructed, while dogs with a ratio greater than 2.4 are very likely obstructed, particularly if segmental dilation is present. Dogs with a ratio between 1.4 and 2.4 should be evaluated further [16]. In cats, a ratio of the maximum diameter of small intestine to the height of the cranial endplate of the L2 vertebra greater than 2.0 or intestinal diameter greater than 12 mm is concerning for intestinal obstruction [17, 18]. Additionally, all small bowel loops should be of a similar diameter. The presence of "two populations" of small bowel, where one segment is 50% larger than the other segment of small bowel, strongly suggests bowel obstruction secondary to a foreign body, neoplasia, or intussusception. Generalized small bowel distention can be associated with a distal bowel obstruction, mesenteric torsion, or with a number of nonobstructive conditions that cause generalized ileus. Gastric dilation and volvulus (GDV) can be identified on a right lateral abdominal radiograph showing compartmentalization of the stomach and displacement of the pylorus dorsally, giving the stomach a "double bubble" or "Popeye arm" appearance. Identification of compartmentalization or displacement allows distinction between GDV and bloat or food bloat, where the stomach is distended with air or food but not displaced.

If gastrointestinal obstruction is suspected but not identified radiographically, additional imaging should be pursued. One option is to fast and rehydrate the patient and repeat plain radiographs 4–24 hours later. If the bowel remains distended in the same area, this suggests a bowel obstruction. When the position of the colon cannot be determined on survey radiographs, pneumocolonography can be performed to allow identification of the colon by creating a pneumocolon. This allows differentiation of colon from dilated small intestine and determination of the location of foreign material to the colon or small intestinal tract. Approximately 10–12 ml/kg of air is instilled into the rectum and colon via a lubricated red rubber catheter to fill the colon to the cecum [19]. Another technique that can be of assistance in detecting the presence of foreign material is compression radiography. With the patient in lateral recumbency, the area in question is mildly compressed with a large wooden or plastic spoon or paddle to displace adjacent abdominal contents and increase radiographic conspicuity, resulting in improved visualization of potential foreign material, plicated bowel, or intestinal masses.

An upper gastrointestinal positive contrast study may be needed to make a definitive diagnosis of gastrointestinal obstruction in a patient with generalized ileus or inconclusive findings on plain radiographs. Barium sulfate provides the highest diagnostic quality contrast study but has a propensity to cause severe intraperitoneal inflammation and granuloma formation if leakage occurs. This complication can be mitigated if abdominal surgery with extensive peritoneal lavage is performed immediately, as would be pursued for gastrointestinal perforation. Iodine-based contrast media can be used to reduce this risk; however, the quality of the study is reduced. When performing a contrast study, it is important to use an adequate volume of the contrast agent. Low volume administration causes inadequate filling and a nondiagnostic study. The recommended dose of barium (60% wt/wt) is 5–10 ml/kg for large dogs and 10–12 ml/kg for small dogs and cats given by mouth or orogastric tube [20].

Diagnostic Abdominal Ultrasound

Abdominal ultrasound by a trained radiologist can be used to further evaluate the peritoneal cavity. Ultrasound provides valuable information about the abdominal viscera, lymph nodes, and vascular structures. While a thorough discussion of the diagnostic utility of abdominal ultrasound in peritoneal evaluation is beyond the scope of this chapter, a few points can be highlighted.

Ultrasound is superior to radiography to evaluate solid organ structure and abnormalities in blood flow to organs, for instance as seen with splenic torsion or portal vein thrombosis. Abdominal ultrasound, when performed by an experienced radiologist, also has greater accuracy than radiographs at identifying gastrointestinal obstruction, with the presence of distension of the jejunal lumen greater than 1.5 cm a useful supportive finding of small intestinal obstruction [21].

Abdominal ultrasound is particularly useful for evaluation for pancreatic and biliary disease. Ultrasonographic findings consistent with pancreatitis include an enlarged pancreas that can be hypo- or hyperechoic or have a mixed pattern of echogenicity, hyperechoic surrounding mesentery, and localized peritoneal effusion. Partial or complete obstruction of the biliary tract characterized by dilation of the common bile duct and gall bladder enlargement can also be seen in patients with pancreatitis. Gall bladder mucoceles are characterized by the presence of nongravity-dependent granular biliary sludge that is organized, typically having a kiwi-like pattern or stellate appearance. Ultrasonographic signs of gall bladder rupture include the presence of echogenic free fluid around the gall bladder, hyperechoic fat near the gall bladder, inability to confirm gall bladder wall continuity, or a mucocele protruding from the gall bladder or free in the abdomen [22].

Abdominal Computed Tomography (CT)

Abdominal CT is the gold standard primary imaging technique for the diagnosis of acute abdominal pain in people [23]. Contrast-enhanced CT has also been shown to differentiate surgical from non-surgical causes of acute abdominal pain in dogs accurately [24]. Despite its superiority, CT is not used extensively in veterinary medicine due to its limited availability, requirement for sedation/anesthesia, and associated cost. It should be pursued in cases where other imaging studies have not achieved a diagnosis.

Abdominal Fluid Sampling

The detection, collection, and analysis of free abdominal fluid is extremely useful in peritoneal evaluation.

Blind and Ultrasound-Guided Abdominocentesis

Blind abdominocentesis, also known as abdominal paracentesis, is a simple way to obtain an abdominal fluid sample for evaluation. The procedure is usually performed with manual restraint, eliminating the need for sedation or anesthesia, with a local block used to facilitate patient compliance during the procedure if needed. The veterinary team should prepare for the procedure and have all necessary equipment in place before attempting abdominocentesis. The procedure is detailed in Protocol 38.1. It is simple and specific, but not sensitive. It has been estimated that 5–6 ml of fluid/kilogram body weight within the abdominal cavity is required to obtain fluid by blind centesis [5]. Ultrasound guidance increases the sensitivity of the procedure by allowing the clinician to view and aspirate small accumulations of fluid (Figure 38.1). In addition, with the use of ultrasound guidance the procedure can be performed with the patient in sternal recumbency with the catheter placed into the fluid pocket through the lateral body wall and directed ventrally; this will reduce dripping and seroma formation afterwards as the insertion site is not gravity dependent.

Open Compared With Closed Needle Technique

Either a closed or open needle technique can be used. In the closed technique, the needle is slowly inserted

Protocol 38.1 Abdominocentesis

Items Required

- Clippers
- Surgical scrub, alcohol, and gauze sponges for skin preparation
- Sterile gloves
- Blood tube with EDTA additive (lavender-top tube)
- Blood tube with no additive (red-top tube)
- Culturette swab
- Needles: 18–22-gauge × 1.5 inches (gauge should be based on the patient's size and body wall thickness; alternatively, an over-the-needle catheter of similar size may be selected. A larger bore, fenestrated catheter is preferred when draining large volume effusions)
- Several ≥ 3-cc syringes for sample collection
- Sedative agent(s) (if required)
- Ultrasound for guidance (preferred)

For therapeutic abdominocentesis, also include:

- Three-way stopcock
- Two fluid extension sets
- Appropriately sized syringes
- Collection bowl or graduated cylinder

Procedure

1) Gather supplies and administer patient sedation (if required).

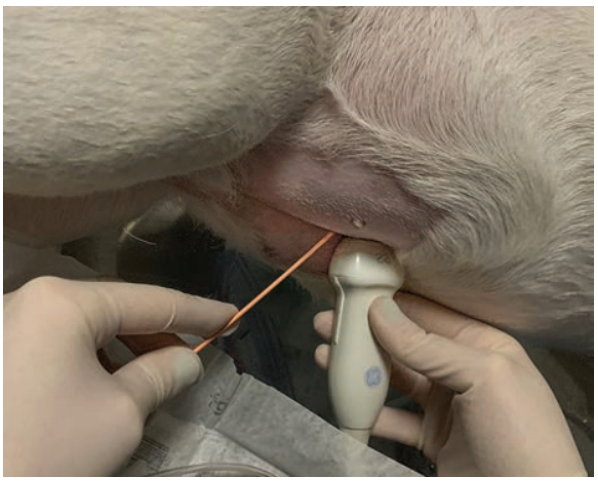


Figure 38.1 Position catheter in line with the ultrasound probe, at a 30–45 degree angle. This will allow for optimal visualization of the needle within the field of view.

2) Place patient in left-lateral recumbency or in a standing position. Positioning in left-lateral recumbency is preferable to avoid inadvertent puncture of the spleen.
3) Clip and scrub the ventral abdomen. If using ultrasound guidance, clip over the proposed site and perform a scrub of the ultrasound probe.

4) Perform hand hygiene and don sterile gloves.
5a) *Ultrasound guided:* Locate the largest fluid pocket with an unobstructed path. Using one hand to stabilize the ultrasound probe (this becomes the non-sterile hand), advance the needle or catheter with the sterile hand. The needle should be advanced in plane with the probe, entering the skin at a 30–45 degree angle to allow adequate visualization of the needle (Figure 38.1)
5b) *Blind:* Insert the needle or catheter caudal to the umbilicus (avoiding the falciform fat), at or within 2 cm of midline.
   a) *Closed technique*: The needle or catheter is attached to a syringe (or extension set) before penetration into the abdominal cavity.
   b) *Open technique*: An unattached needle or catheter is inserted into the abdominal cavity.
6) Once the bevel of the needle has been advanced through the skin and body wall (depth of needle insertion is relative to patient size), pull back on the plunger of the syringe to apply suction if using the closed technique.
   a) If using the open technique, observe for a flash of fluid in the hub of the needle.
   b) If using an over-the-needle catheter, after the catheter is inserted into the abdominal cavity, feed the catheter off the stylet and remove the stylet to collect the sample.
7) If blood is aspirated unexpectedly, the needle should be removed from the abdomen and the sample placed in a red-top tube to observe for clot formation.
   a) Blood from inadvertent laceration of a vessel or organ will clot.
   b) Hemorrhagic free abdominal fluid will not clot.
8) Allow fluid to drip into sterile collection tubes or apply gentle suction with a syringe to collect sample. The fluid should be collected into sterile tubes for further analysis.
9) If no fluid is retrieved:
   a) *Blind*: Gently rotate the needle within the abdomen. If required, advance or withdraw the needle slowly then reapply gentle pressure on the syringe once stationary.
   b) *Ultrasound guided*: Identify the needle or catheter in the field of view and redirect within the fluid pocket.
   c) Consider four-quadrant centesis, or diagnostic peritoneal lavage.

Four-Quadrant Centesis Technique

1) Place patient in left-lateral recumbency.
2) Clip and scrub the ventral abdomen 10 cm cranially and caudally from the umbilicus and laterally to the mammary chain on either side.
3) Perform hand hygiene, and don sterile gloves.
4) Using the umbilicus as a center point, divide the abdomen into four quadrants. Serially sample the right cranial, left cranial, right caudal, and left caudal quadrants (Figure 38.2) as directed in the abdominocentesis protocol. Avoid puncturing the superficial and deep epigastric vessels that lie parallel to, and in the vicinity of the mammary chain.
5) Continue collection of the sample as described in the abdominocentesis protocol.

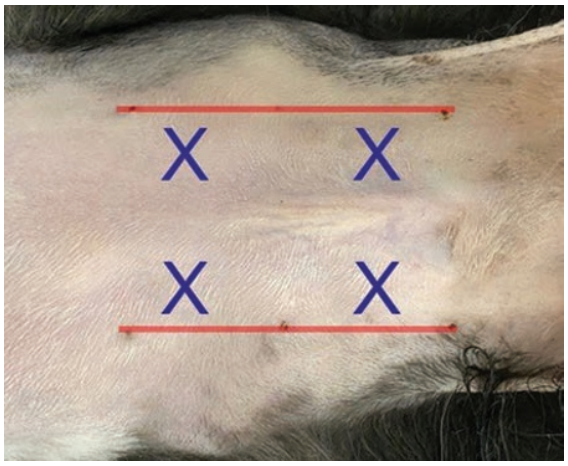


Figure 38.2 Four-quadrant centesis technique: sample the right cranial, left cranial, right caudal, and left caudal quadrants in turn, avoiding the superficial and deep epigastric vessels (marked in red). The dog's head is to the left of the image and the tail to the right.

perpendicular to the abdomen and advanced a few millimeters at a time. Following each advancement, the syringe is aspirated. If fluid is not obtained by the closed technique, conversion to the open technique can be done by removing the syringe from the needle and slowly backing the needle out. The open needle technique reduces the chance of omentum or viscera occluding the needle, but it may introduce free air into the abdominal cavity. Ideally, open abdominocentesis should only be performed after abdominal radiographs have been obtained to prevent misinterpretation of iatrogenically introduced abdominal free air. There are minimal associated risks, but risks may be increased in patients with a coagulopathy, or with marked organomegaly or distention of an abdominal viscus. The patient should be placed in left lateral recumbency to decrease the chance of accidental puncture or laceration of the spleen. Negative pressure should not be applied while the needle and syringe are advanced, as this can cause the omentum

to occlude the end of the needle, resulting in a false negative abdominocentesis. The needle should not be redirected once within the abdominal cavity, as this may increase the chance of lacerating an organ.

Four-Quadrant Technique

To increase diagnostic yield, the four-quadrant technique can be used, as described in Protocol 38.1 and depicted in Figure 38.2. The four areas for abdominocentesis are 1–2 cm cranial and caudal to the umbilicus on the right and left side of the abdomen. This is a modification of the open needle technique, with abdominocentesis performed at all four sites in turn. Gravity dependency or changes in transabdominal pressure between the needles may increase the likelihood of successful fluid retrieval. If a needle becomes occluded by mesentery during active aspiration or passive drainage, rapid reinfusion of a small volume of the retrieved fluid or gentle external abdominal palpation can sometimes restore patency by displacing the mesentery from the needle tip.

A possible complication of large volume evacuation of ascites is paracentesis-induced circulatory dysfunction, which can result in faster reaccumulation, electrolyte derangements, and renal impairment [25].

Diagnostic Peritoneal Lavage

If no fluid can be obtained by abdominocentesis using the above techniques and abdominal pathology is highly suspected, diagnostic peritoneal lavage (DPL) can be performed to obtain abdominal fluid samples for evaluation. This technique is seldom required in patients that have been adequately fluid resuscitated if ultrasound is available to enhance the detection of small fluid pockets. The technique is described in Protocol 38.2. The procedure is performed with local anesthesia, with or without additional sedation. General anesthesia should be avoided as turgid abdominal musculature will facilitate catheter placement. There are several different commercially available, multifenestrated catheters that can be used including peritoneal dialysis catheters, chest tubes, or abdominal drainage catheters (Figure 38.3). Alternatively, readily available over-the-needle catheters, red rubber catheters, or large single-lumen intravenous catheters can be used if

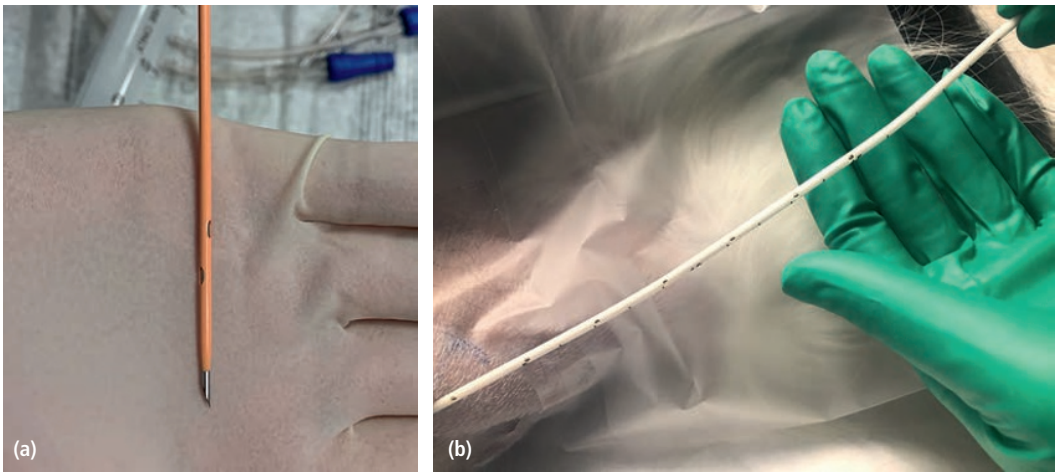


Figure 38.3 Examples of multifenestrated catheters: MILA fenestrated centesis catheter (a) and guidewire-inserted chest tube (b) shown.

Protocol 38.2 Diagnostic Peritoneal Lavage

Items Required

- Clippers
- Surgical scrub, alcohol, and gauze sponges for skin preparation
- Sterile gloves
- Sterile drape
- 2% lidocaine
- No. 11 scalpel blade
- Sterile 10–14-gauge catheter for abdominal drainage
  - Peritoneal dialysis catheter
  - 14–16 gauge × 2–5.5 inch over-the-needle catheter (with manual fenestrations, no further than one-third of the catheter length)
  - Fenestrated chest tube
- Warmed, sterile 0.9% sodium chloride (attached to drip set)
- Sterile collection system (drip set, three-way stopcock, collection bag)
- Blood tube with EDTA additive (lavender-top tube)
- Blood tube with no additive (red-top tube)
- Sedative agent(s)

Procedure

1) Gather supplies and administer patient sedation.
2) Ensure that the urinary bladder is empty (manual expression or catheterization).
3) Place patient in left lateral recumbency.
4) Clip and aseptically prepare a wide margin, centered around the umbilicus.
5) Perform hand hygiene and don sterile gloves.
6) Place drape with the fenestration centered over the umbilicus.
7) Infuse lidocaine into proposed puncture location, from the skin into the peritoneum.
8) Catheter placement will vary depending on the type of drainage catheter used:
   a) Trocar-type catheter (or over-the-needle catheter; Figure 38.4):
      i) Create a small stab incision no larger than the diameter of the catheter either at the umbilicus (avoiding the falciform fat), or within 2–3 cm laterally of midline.
      ii) Introduce the trocar and catheter through the body wall at a 45-degree angle, directed caudodorsally toward the urinary bladder.
      iii) Advance the catheter off the trocar, ensuring that all fenestrations are within the peritoneal cavity.
   b) Catheters may also be introduced via the modified Seldinger and peel-away methods. A slow, rotating motion is recommended while advancing the catheter to facilitate passage through the fascia and linea alba (Figure 38.5).
9) If no fluid is obtained when the catheter is placed, lavage the abdomen by instillation of 22 ml/kg of warmed, 0.9% sodium chloride through the catheter and into the peritoneal cavity.



Figure 38.4 (a) Create a small stab incision over proposed site (no larger than the diameter of the catheter). (b) Introduce the catheter through the body wall at a 45-degree angle, caudodorsally. (c) Advance the catheter off the trocar once approximately 50% of the catheter has been inserted, ensuring that all fenestrations are within the peritoneal cavity. In these images, the animal is in left lateral recumbency with the head to the upper right and the tail to the lower right.



Figure 38.5 Use a slow, rotating motion for large bore drainage catheters to facilitate passage through the fascia and linea alba.

10) While the fluid is being infused, monitor the patient carefully for signs of respiratory distress or discomfort.
11) Clamp the infusion set.



Figure 38.6 Secured catheter for continuous abdominal drainage. Cover with occlusive bandage to prevent contamination.

12) Gently massage the patient's abdomen, or carefully roll the patient to ensure adequate distribution of saline within the peritoneal cavity.
13) Open the infusion set and allow the fluid to drain through the catheter by gravity into a sterile collection bag. Collect samples in sterile empty and EDTA-containing tubes for evaluation.
14) If the catheter is being used for continuous abdominal drainage, suture the catheter in place and cover with a sterile dressing (Figure 38.6).

additional fenestrations are added. Fenestrations in an over-the-needle or red rubber catheter can be made using a scalpel blade or biopsy punch. They should be small and smooth, less than 40% of the circumference of the catheter, and should not be placed directly across from one another as this will weaken the catheter, risking kinking or breakage.

The patient's urinary bladder should be emptied either by voiding or manual expression to avoid accidental puncture. Other complications include omental obstruction of the catheter fenestrations and incomplete fluid retrieval. The stab incision can be made 2–3 cm lateral to the umbilicus to avoid the falciform fat. It is uncommon to aspirate large volumes of fluid following a DPL due to dispersion of the fluid in the abdomen. Contraindications for DPL include pregnancy, marked organomegaly, cardiovascular or respiratory compromise, diaphragmatic hernia, previous celiotomy, or patients suspected to have abdominal adhesions.

Analysis of Peritoneal Effusion

Gross, cytologic, and biochemical evaluation of peritoneal fluid collected either by centesis or peritoneal lavage can provide important diagnostic clues in the patient with abdominal pain, especially if peritonitis is suspected. Evaluated parameters and their clinical associations are summarized in Table 38.3.

The peritoneal fluid sample should be separated into one tube containing EDTA to prevent clotting and another tube containing no additives. The additive-free tube will be used for biochemistries and for cultures because EDTA is bacteriostatic. To prevent contamination by sample handling, any microbial cultures should be performed aseptically, immediately after fluid sampling. Slides should be made soon after fluid collection to prevent degeneration of cells in the fluid (Chapter 59).

Color and clarity should be noted before additional sample handling occurs. Peritoneal fluid color can range from colorless to red tinged, red, white, yellow, brown, green, and anything in between. Fluid color is not specific for, but could indicate, an organ system as the primary cause for the effusion. For example, red-colored fluid suggests intra-abdominal hemorrhage, while green-tinged fluid is seen with bile leakage. Clarity of the fluid is noted as clear to slightly turbid or turbid. The degree of turbidity indicates the presence of cellular material and other particulate matter. Foul-smelling fluid is associated with anaerobic infection.

Table 38.3 Clinicopathologic peritoneal evaluation diagnostic criteria.

Clinicopathologic tests	Diagnostic criteria/interpretation
Peritoneal fluid packed cell volume	> 5% consistent with significant intra-abdominal hemorrhage
Blood to peritoneal fluid glucose difference	> 20 mg/dl consistent with septic peritonitis
Plasma to peritoneal fluid glucose difference	> 38 mg/dl consistent with septic peritonitis in dogs
Blood to peritoneal fluid lactate difference	< −2 mmol/l consistent with septic peritonitis in dogs
Peritoneal fluid to blood potassium radio	Dogs: > 1.4 : 1; cats: > 1.9 : 1 consistent with uroabdomen
Peritoneal fluid to blood creatinine ratio	Dogs and cats: > 2:1 consistent with uroabdomen
Peritoneal fluid to blood bilirubin ratio (also may see bile piment/crystals in abdominal fluid)	> 2:1 consistent with bile peritonitis
Peritoneal fluid lipase activity [7]	> Fourfold upper reference limit for serum lipase activity consistent with pancreatitis; effusion to serum lipase activity ratio > 2 consistent with pancreatitis
Specific cPLI (serum) [26][a]	< 200 µg/l not consistent with pancreatitis
	201–399 µg/l gray zone
	> 400 µg/l consistent with pancreatitis
Specific fPLI (serum) [26][a]	< 3.5 µg/l: not consistent with pancreatitis
	3.6–5.3 µg/l: increased, gray zone
	> 5.4 µg/l: consistent with pancreatitis
Peritoneal fluid triglyceride concentration [7]	Effusion triglyceride concentration > concurrent serum triglyceride concentration or > 100 mg/dl consistent with chylous effusion
Abdominal perfusion pressure	Mean arterial pressure – IAP
	> 12 mmHg consistent with IAH

cPLI, canine pancreatic lipase immunoreactivity; fPLI, feline pancreatic lipase immunoreactivity; IAH, intra-abdominal hypertension; IAP, intra-abdominal pressure.
[a] Insufficient for diagnosis in absence of clinical findings.

Red-colored fluid should be evaluated for clotting by being placed in a non-anticoagulant (red top) tube or observed in the syringe for evidence of clotting. Clotting suggests that the fluid was obtained from inadvertent aspiration of a vessel or organ, or that the hemorrhage is peracute. Typically, hemorrhagic peritoneal effusion does not clot due to the fibrinolytic activity of the mesothelium [27]. If the sample clots, abdominocentesis should be repeated to obtain a true sample of the effusion. Retrieval of non-clotting fluid is most consistent with peritoneal effusion, but a coagulopathy should be ruled out through evaluation of platelet number, platelet function, and clotting times. A packed cell volume (PCV) and total protein concentration should be obtained on any red-colored fluid to differentiate hemorrhage from serosanguineous effusion. A PCV greater than 5% is suspicious for abdominal hemorrhage. Following peritoneal lavage, the volume of blood in the abdomen can be estimated by the following formula [5]:

$$x = L \times V / P - L \quad (38.1)$$

where x = volume of blood in the abdominal cavity; L = PCV of the returned lavage fluid; V = volume of lavage fluid infused into the abdominal cavity; and P = PCV of the peripheral blood before intravenous infusion of fluids.

A direct smear for cytology allows for estimation of cellularity, while cytology of a sedimented sample increases the chances of detecting bacteria or atypical cells. Fluid obtained from a DPL should be evaluated using a sedimented sample due to dilution from the infusate. Total nucleated cell counts can be estimated by running the sample through the in-house complete blood count machine or estimated microscopically by trained personnel (see Chapter 61 for more information). Normal abdominal fluid contains less than 1000 nucleated cells/mm [3]. Cytology includes evaluation for degenerate or toxic neutrophils, intracellular bacteria, neoplastic cells, bile stain or crystals, and any other abnormalities. An elevated white blood cell count (> 5000 nucleated cells/mm) indicates an inflammatory process [3]. Increased numbers of neutrophils and degenerate neutrophils support a diagnosis of peritonitis. Intracellular bacteria can be seen with septic peritonitis. Leukocyte morphology and the presence of bacteria are more important than absolute leukocyte numbers. Identification of many bacteria with no white blood cells is seen with inadvertent aspiration of the gastrointestinal tract. Gram staining of septic effusions can assist with empirical selection of antibiotics.

Once PCV, total protein, cell count, and cytologic morphology has been assessed, the fluid should be classified to narrow differential diagnoses and guide further diagnostics and therapies. Transudation occurs due to altered hydrostatic and oncotic forces within the vessels or lymphatics, while exudation is caused by increased capillary permeability. Traditionally, effusions have been classified by protein concentration and cellularity as transudates,

modified transudates, or exudates. This system is controversial, and classification based by etiology has been proposed to be more clinically useful, with effusions divided into transudates (protein-poor and protein-rich), exudates, effusions resulting from vessel or viscous disruption, and effusions resulting from cell exfoliation (Table 38.1) [28].

Based on the patient's physical examination, history, and suspected disease, specific chemistry analysis on the sample may be indicated. In most cases, the peritoneal chemistries are compared with a simultaneously collected peripheral blood sample. Samples obtained via DPL are diluted by the infused saline, so biochemical analysis is less likely to be diagnostic. Abdominal fluid bilirubin twice as high as the bilirubin in the peripheral blood is diagnostic for bile peritonitis [29]. A uroabdomen can be diagnosed by comparing blood and fluid creatinine and potassium levels. Fluid that has creatinine levels twice as high as blood, and potassium levels 1.4 times as high in dogs and 1.9 times as high in cats is consistent with uroabdomen [30, 31].

The gold standard for differentiating septic peritoneal effusions from inflammatory nonseptic processes such as pancreatitis is the identification of intracellular bacteria in the fluid. Intracellular bacteria are not always seen in patients with septic peritonitis, especially if the patient has been receiving antibiotic therapy or there is a walled-off process such as a hepatic abscess. Comparison of blood glucose in the peripheral blood and abdominal effusion can be helpful in differentiating septic from nonseptic effusions. Peripheral whole-blood glucose $\geq$ 20 mg/dl higher than peritoneal fluid glucose has been shown to be highly specific but insensitive for septic peritonitis in dogs and cats [32]. A plasma glucose to fluid glucose difference of greater than 38 mg/dl using a point of care glucometer is more accurate for detecting septic peritonitis in dogs [33]. Similarly, a peritoneal fluid lactate concentration higher than blood lactate, resulting in a negative blood to fluid lactate difference of less than −2.0 mmol/l is predictive of septic peritonitis in the dog, but not in the cat [32, 34].

Intra-Abdominal Pressure (IAP) Monitoring

Elevated pressure in the abdomen is referred to as intra-abdominal hypertension (IAH), whereas pathologic derangements that occur because of IAH are referred to as abdominal compartment syndrome. IAH is caused by increased pressure within the abdominal cavity, for example due to tissue edema or free fluid accumulation. It is considered primary if the disease process arises within the abdominal cavity (e.g. fractured liver or spleen) or secondary if the inciting disease is extraperitoneal in origin (e.g. high-pressure mechanical ventilation). Elevated pressure in the closed abdominal space can compromise perfusion to the abdominal organs and predisposes patients to developing multiple organ dysfunction and failure. IAP should be measured in patients at risk of IAH.

IAP is most commonly measured in veterinary patients via a catheter placed in the urinary bladder. Excluding gas in the gastrointestinal tract, the abdominal contents are non-compressible. Consequently, bladder pressure measurements reflect the overall intra-abdominal pressure. The technique is relatively easy and is described in Protocol 38.3. Measurements can be taken in either lateral or sternal recumbency, but the position should be consistent across serial measurements, as IAP is affected by body position. The measured value has also shown to be affected by the volume of saline instilled into the urinary bladder, body condition, pregnancy, external abdominal pressure application, and by the presence of abdominal wall or detrusor muscle contractions. End-expiratory readings are standard, and reported normal IAP is 0–5 cm H_2O in dogs, 4–8 cm H_2O in sedated cats, and 6–11 cm H_2O in awake cats [35]. The frequency of IAP monitoring can be adjusted according to patient risk, and should be evaluated every four hours in the at-risk critically ill patient [36]. When an indwelling urinary catheter is contraindicated, measurements can be made from a catheter tip located in the intra-abdominal vena cava.

Exploratory Laparotomy

The decision to proceed with an exploratory laparotomy often needs to be made swiftly in cases of acute abdomen. Clear indications for immediate abdominal surgery include abdominal wall perforation, septic peritonitis, persistent abdominal hemorrhage, complete intestinal obstruction, free abdominal gas (not associated with previous surgery, pneumomediastinum, or invasive procedures), abdominal abscess, ischemic bowel, gastric dilation volvulus, mesenteric volvulus, and bile peritonitis. Uroabdomen is also often listed as an indication for prompt abdominal surgery but can be managed medically initially with an indwelling peritoneal catheter in some cases to improve patient stability prior to general anesthesia for definitive repair. An exploratory laparotomy can also be used as a diagnostic tool in patients with an open cause for acute abdomen.

Protocol 38.3 Intra-abdominal Pressure Measurement

Items Required

- Sterile gloves
- Foley urethral catheter (size appropriate)
- Sterile urine collection system
- Two three-way stopcocks
- Water manometer
- 35–60-ml syringe
- Sterile 0.9% sodium chloride
- Two intravenous (IV) administration extension sets

If using a pressure transducer, also include:

- Pressure transducer kit
- 500 ml bag of sterile 0.9% sodium chloride
- Pressure bag

Procedure (Manometer Method)

1) Gather supplies.
2) Perform hand hygiene, and don sterile gloves.
3) A Foley urinary catheter should be aseptically placed, with the catheter tip located just inside the trigone of the urinary bladder (Chapter 35).
4) Connect the Foley catheter to a sterile urine collection system, with two three-way stopcocks incorporated into the collection system (Figure 38.7a).
5) Attach a water manometer to the first upright stopcock. Attach the syringe to the second stopcock for filling the manometer and infusion of the bladder.
6) Place the patient in lateral or sternal recumbency (patient position should be consistent for repeat measurements).

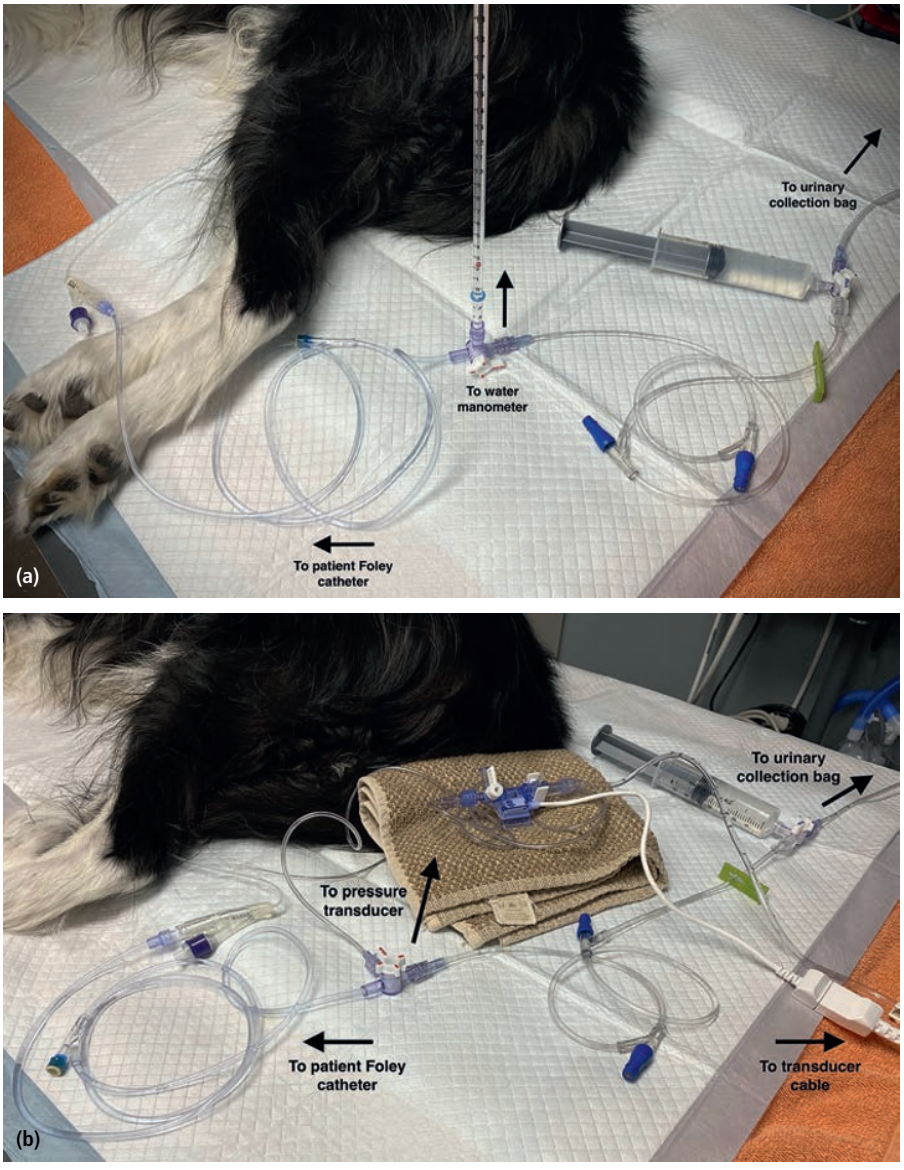


Figure 38.7 Intra-abdominal pressure measurements. (a) Manometer method set-up. Ensure that the manometer is zeroed at the patient's midline (tip of black arrow indicates zero mark in this case). (b) Pressure transducer method set-up. Elevate the transducer to the level of the patient's midline, then zero the transducer.

7) Empty the urinary bladder.
8) Instill 1.0 ml/kg of sterile saline into the urinary bladder (to 25 ml/patient maximum).
9) Zero the manometer to the patient's midline at the symphysis pubis, and fill manometer with sterile saline.
10) Close the stopcock to the fluid source to allow the meniscus in the manometer to equilibrate to the pressure within the urinary bladder.
11) Take the measurement at end expiration. The difference between the equilibration point and the zero reference point is the IAP measurement.

Procedure (Pressure Transducer Method)

1) Complete steps 1–4 as described above.
2) Attach a 500 ml bag of sterile saline to the pressure transducer IV spike set and insert the IV bag into the pressure bag.
3) Inflate the pressure bag to 50 mmHg.
4) Flush the line to remove any air from the system and attach the patient connection to the first upright stopcock. Attach the syringe to the second stopcock for infusion of the bladder (Figure 38.7b).
5) Place the patient in lateral or sternal recumbency.
6) Empty the urinary bladder.
7) Instill 1.0 ml/kg of sterile saline into the urinary bladder (to 25 ml/patient maximum).
8) Position the transducer at the patient's midline at the symphysis pubis (a towel can be used to elevate the transducer as required).
9) Zero the transducer (this may vary depending on the monitor used) to current atmospheric pressure, ensuring the transducer stopcock is turned off to the patient and the cap is removed (see Chapter 12 for further details).
10) Open the transducer stopcock to allow flow between the patient and monitor.
11) Close the stopcock to the fluid source, allowing the monitor to equilibrate to the pressure within the urinary bladder.
12) Take the measurement at end expiration.

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39

Point-of-Care Abdominal Ultrasound

Søren Boysen and Valerie Madden

Most currently published abdominal veterinary point-of-care ultrasound (POCUS) protocols incorporate sonographic windows from the original abdominal focused assessment with sonography for trauma (AFAST) study published in 2004 [1]. Despite abdominal FAST and abdominal POCUS sharing many common applications it is important to highlight the distinction between the two: abdominal FAST was designed to detect the presence of free abdominal fluid while abdominal POCUS incorporates a broader array of clinically relevant questions that can be answered in the point-of-care setting [1–10]. As new research expands the body of evidence, the number of clinical questions and scenarios that can be rapidly answered by non-specialist clinicians using abdominal POCUS will also expand. For example, in addition to searching for free fluid, POCUS includes assessment of duodenal motility at the right paralumbar region, a search for pneumoperitoneum, and assessment of ureteral obstruction at the left and right paralumbar regions, among other advances. It is therefore important to consider the clinical question at hand, as this dictates which abdominal organs, and specific organ anatomy, will be evaluated. For example, the assessment of renal pelvic dilation for suspected ureteral obstruction involves a different approach to imaging the kidney than the detection of free fluid around the kidney [1, 10]. Randomly placing the transducer on the patient without a specific question to interpret should be avoided, as this may lead to false positive results. Failing to assess each site thoroughly for pathology (fanning and/or sweeping through multiple planes in both the longitudinal and transverse orientations) or performing "a quick peek" without asking a specific question is likely to lead to false negative results.

Patient Positioning and Machine Settings

To maximize success, the operator should consider gravitational effects when deciding on patient positioning. Minor protocol adjustments may be necessary when searching for specific pathology: fluid falls while gas rises, and thus the location at which to find fluid or gas changes based on patient positioning.

Given that patient status may preclude positioning patients in particular ways (e.g. in respiratory distress or spinal cord injury), sonographers should become comfortable performing POCUS with patients in all positions (lateral, sternal, standing). The authors prefer to scan patients in the position they are most comfortable, which is often lateral recumbency. Lateral recumbency can be contraindicated when respiratory distress is present, in which case the patient is scanned while standing or sternal. The choice of left or right lateral recumbency is often based on the position of the patient at the time of presentation and operator preference. There is no difference between left or right lateral recumbency for the detection of free abdominal fluid [2], although the time to complete abdominal POCUS is faster in left-lateral recumbency compared with right lateral recumbency [2].

Identification of the right kidney is easier when patients are in left-lateral recumbency than in right-lateral recumbency because the right kidney is located more cranially (under the ribs), compared with the left kidney; this likely explains why it is faster to scan dogs in left-lateral than in right-lateral recumbency [2].

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Patients initially placed in lateral recumbency can be gently repositioned into sternal recumbency to assess the gravity-dependent paralumbar region, if necessary (e.g. for renal pelvis diameter assessment), following evaluation of other sites.

Dorsal recumbency is often contraindicated in patients with respiratory or cardiovascular instability, and thus the authors never scan patients in dorsal recumbency.

A microconvex/curvilinear transducer is used for all abdominal POCUS scanning, with a frequency generally between 5 MHz (for patients >15 kg) and 7.5 MHz (for patients <15 kg). Gain can be set to maximize detection of anechoic fluid by using either bile in the gall bladder or urine in the urinary bladder as a reference echogenicity for fluid. Adjust the gain, depth, and focal position as needed during the scan to maximize image quality and thoroughly answer each abdominal POCUS question. Extending the depth at the subxiphoid location allows evaluation of the pleural and pericardial spaces (see below).

Abdominal Point-of-Care Ultrasound Technique

It is essential that all key organs at each site be thoroughly evaluated to ensure that the specific question being asked is answered confidently. The five sites currently evaluated during abdominal POCUS include:

1) The subxiphoid window
2) The umbilical window
3) The urinary bladder window
4) The right paralumbar window
5) The left paralumbar window (Figure 39.1).

Given that the internal organs being evaluated have expanded beyond the liver, kidneys, spleen, and bladder, the authors prefer to use external landmarks to locate the correct sonographic windows. Fanning and rocking the transducer through 45-degree angles in both longitudinal and transverse axes at all sites maximizes the chance of detecting pathology while minimizing false negative and false positive results [1].

Subxiphoid

The diaphragm, liver, gallbladder, ventral stomach wall, and areas between these structures are evaluated. The caudal vena cava, pleural space, and pericardial space can also be evaluated at the subxiphoid site by extending the depth of the transducer beyond the diaphragm (see Chapter 17).

To find the correct subxiphoid site palpate the ribs and follow them cranioventrally until the "V" at the xiphoid region is identified. To begin with, place the transducer in the point of the "V" in longitudinal axis to the body, at roughly a 45-degree angle to the spine (Figure 39.2). Several transducer movements are used to perform abdominal POCUS (Figure 39.3); details of specific transducer movements can be found in Chapter 6.

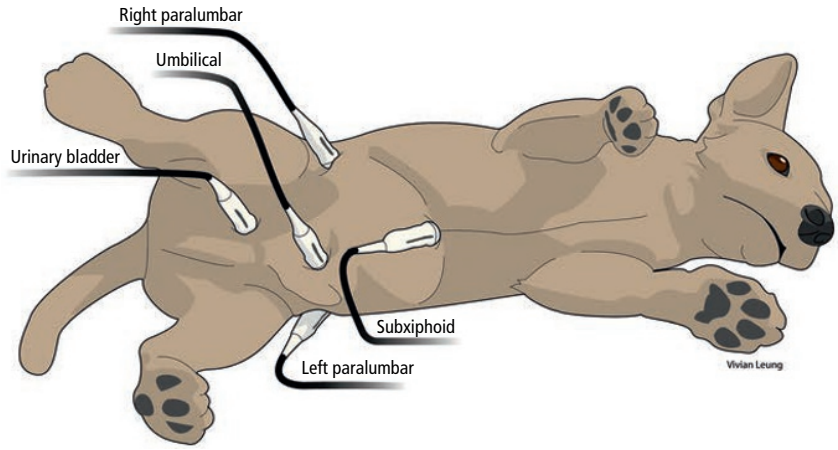


Figure 39.1 Abdominal point-of-care ultrasound windows with the patient in left-lateral recumbency: subxiphoid, umbilical, urinary bladder, right paralumbar, and left paralumbar. Each location is evaluated in longitudinal and transverse planes with rocking and fanning of the transducer to maximize the area evaluated, to ensure all target structures/sites are thoroughly evaluated, and to decrease false positive and negative results. All five sites are evaluated in all patients regardless of patient positioning; however, the area scanned can be modified slightly depending on the pathology one is attempting to rule in or out, and in light of how patient positioning will affect where pathology accumulates. For example, if the patient is in a standing position and the goal is to detect free peritoneal fluid, the transducer is placed directly on ventral midline over the umbilical and urinary bladder regions with the ultrasound beam directed toward the spine (vs. toward the tabletop when the patient is in a lateral position). With the patient in lateral recumbency, as shown, the transducer should be directed from the nongravity-dependent side of the patient toward the gravity-dependent body wall areas where fluid is most likely to accumulate. *Source:* Courtesy of Vivian Leung, 2020, with permission.

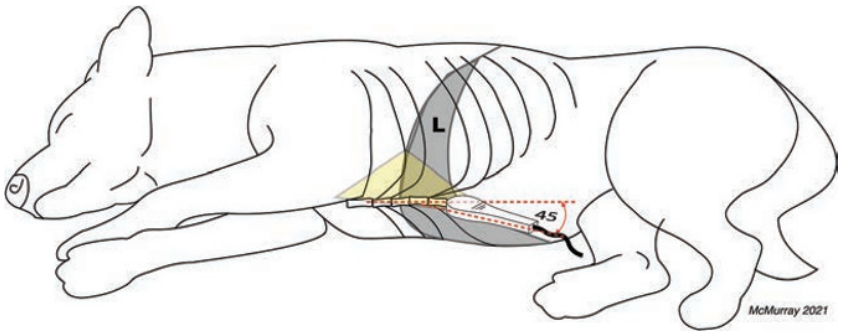


Figure 39.2 Transducer orientation and positioning at the subxiphoid view. The dog is in right-lateral recumbency. The transducer is placed on midline just caudal to the xiphoid process in the longitudinal orientation at roughly a 45-degree angle to the spine. L, liver.

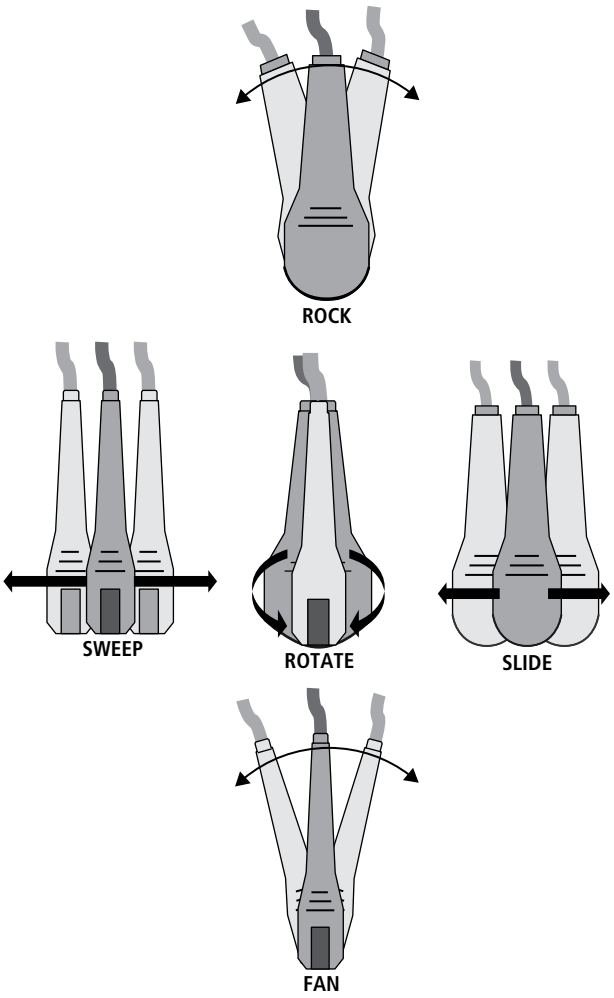


Figure 39.3 Summary of the five different probe manipulations commonly used during point-of-care ultrasound: sweep, slide, rotate, fan, rock. See Chapter 6 for further details.

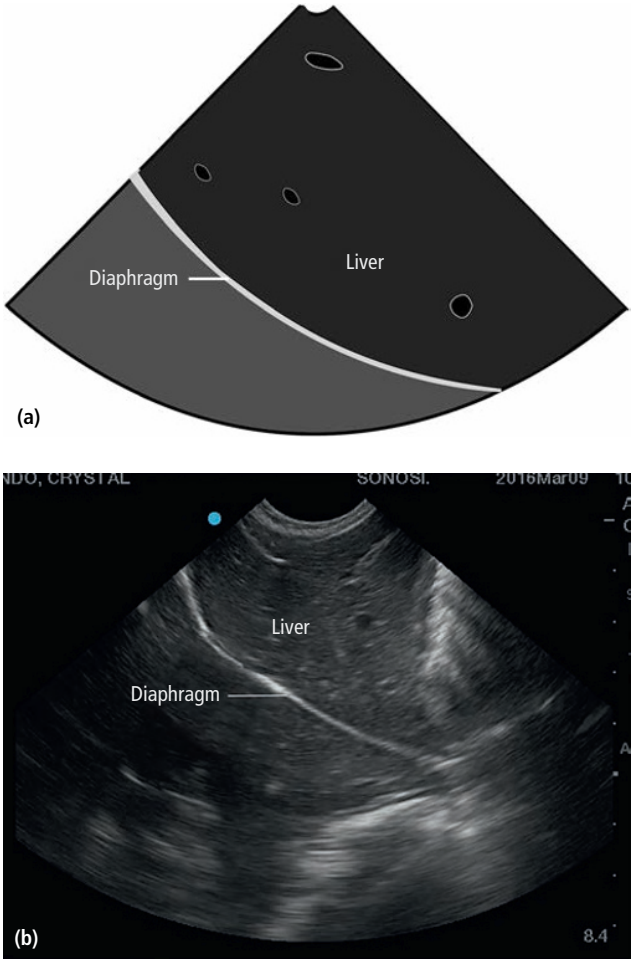
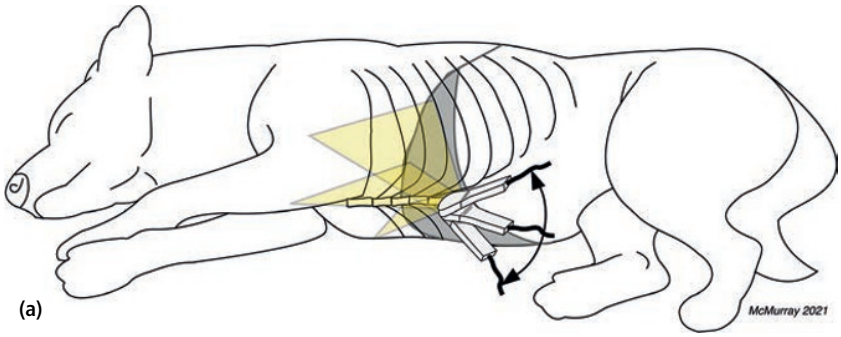


Figure 39.4 Schematic (a) and ultrasound still image (b) of liver and diaphragm obtained at the subxiphoid window with the transducer in long axis orientation.

Rock the transducer cranially and adjust the depth until the liver and diaphragm are visible within the ultrasound image (Figure 39.4). Fan the transducer through all planes of the liver and rock the transducer to assess all parts of the liver. Small fluid accumulations can collect between the liver and diaphragm and/or between liver lobes; seeing individual liver lobes is abnormal (Figure 39.5). The gallbladder is visualized to the right of midline and assessed for wall thickening, which may suggest edema or other abnormalities (see below). The transducer can be rocked caudally so it is nearly perpendicular to the spine at the subxiphoid region, which should allow the stomach to be visualized (Figure 39.6). By slowly fanning left and right, the



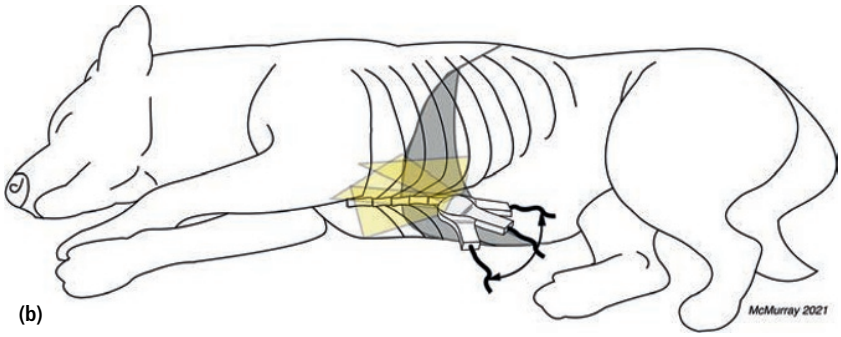


Figure 39.5 The transducer should be fanned (a) and rocked (b) through all planes of the liver and other key structures at the subxiphoid site to increase the chance of finding pathology.

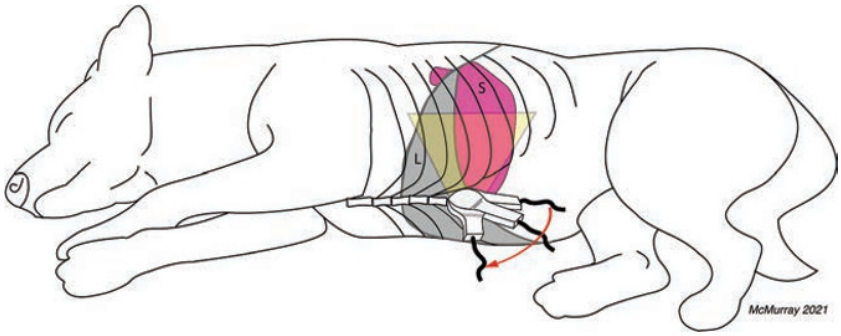


Figure 39.6 Schematic image demonstrating how the transducer, in long axis orientation, is rocked caudally at the subxiphoid site to locate the stomach. L, liver; S, stomach.

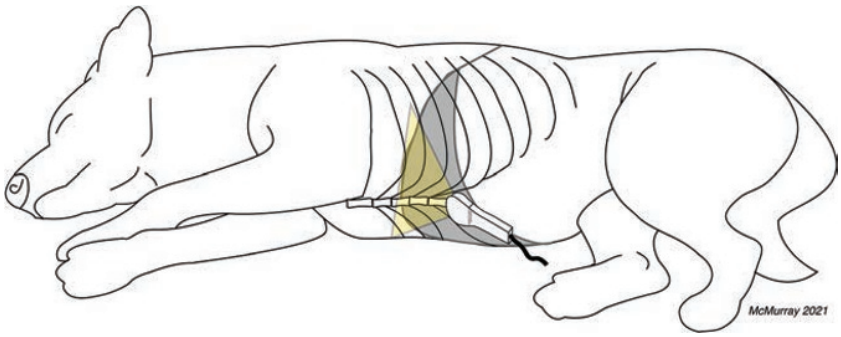


Figure 39.7 Once subxiphoid organs have been evaluated in long axis, the transducer should be rotated 90 degrees and the transducer fanned and rocked through multiple planes to allow key subxiphoid structures to be evaluated in the short axis orientation.

ventral stomach wall can be assessed for motility (see below). Following longitudinal plane scanning, the transducer is rotated into transverse axis and again fanned and rocked through all planes (Figure 39.7).

Umbilical

The gravity-dependent body wall, intestines, spleen, and regions between these structures are evaluated. To find the correct site, the transducer is placed over the umbilical region and directed toward the tabletop with the patient in lateral recumbency (Figure 39.1). The protocol is modified slightly if the patient is in sternal or standing. The transducer is then rocked and fanned through all longitudinal and transverse planes looking for specific pathology, typically free peritoneal fluid. Other pathology may be detected at the umbilical site, depending on the specific clinical question asked.

Early abdominal FAST protocols did not include the umbilical site; however, if the goal is to rule out free peritoneal fluid, the umbilical site is important to assess prior to

evaluating the gravity-dependent paralumbar site with the patient in lateral recumbency. This is because sliding the transducer under the patient to assess the gravity-dependent paralumbar window may displace gravity-dependent fluid to either side of the transducer, causing it to be missed (Video 39.1). The umbilical site is also a gravity dependent and sensitive site to identify free peritoneal fluid in the standing patient. Be sure not to apply too much pressure to the transducer when assessing the umbilical site in the standing patient as it may displace free fluid, prohibiting it from being visualized.

Urinary Bladder

The urinary bladder, gravity- and nongravity-dependent body walls, and the areas between these structures are evaluated. To find the urinary bladder, the transducer is initially placed in the longitudinal axis to the body, between the pelvic limbs. Avoid applying too much pressure to the transducer as this tends to compress and displace the urinary bladder. Adjust the depth settings to allow both the dorsal and ventral walls of the urinary bladder to be visualized.

If the patient is in lateral recumbency, slide the transducer to the nongravity-dependent side of the patient and angle the ultrasound beam through the urinary bladder, while fanning, to identify fluid that might accumulate in the deeper, gravity-dependent sites along the far body wall.

Slide the transducer cranially to locate the apex of the bladder and fan the transducer through all planes at the bladder apex. Slide the transducer caudally to evaluate the trigone and urethral areas, fanning through all planes of the trigone and visible urethra. Following thorough longitudinal scanning, rotate the transducer to a transverse axis and again scan the same sites in the orthogonal plane.

If the patient is standing or sternal, the transducer is placed on midline with the ultrasound beam directed toward the spine to ensure the most gravity-dependent area between the abdominal wall and ventral urinary bladder wall is assessed.

Urine volume, in milliliters, can be estimated and monitored serially at this site as follows (Figure 39.8) [7]:

- With the transducer in longitudinal axis to the urinary bladder, measure the length of the bladder in centimeters at its widest point (sweep the transducer through all planes in longitudinal orientation to identify the widest point). Take the measurement at this location. This is the length or "L" measurement. Be sure to keep the ultrasound beam at a 90-degree angle to the bladder once the widest point is identified to get the most accurate measurements.
- The depth of the bladder is also measured in longitudinal. This is the "DL" measurement.
- With the transducer oriented in transverse axis, sweep through all planes (apex to trigone) to find the widest point of the bladder. At the widest point a width measurement is taken in centimeters. This is "W." A depth measurement is also taken in transverse which is the "DT." Be sure to keep the ultrasound beam at a 90-degree angle to the bladder once the widest point is identified to get the most accurate measurements.
- The following formula, with measurement values entered in centimeters, is then used to calculate the urinary volume in milliliters [7]:

$$L \times W \times \left[\left(DL + DT \right) / 2 \right] \times 0.625$$

Note that, although some authors only measure the depth in a single plane, we prefer to measure it in both

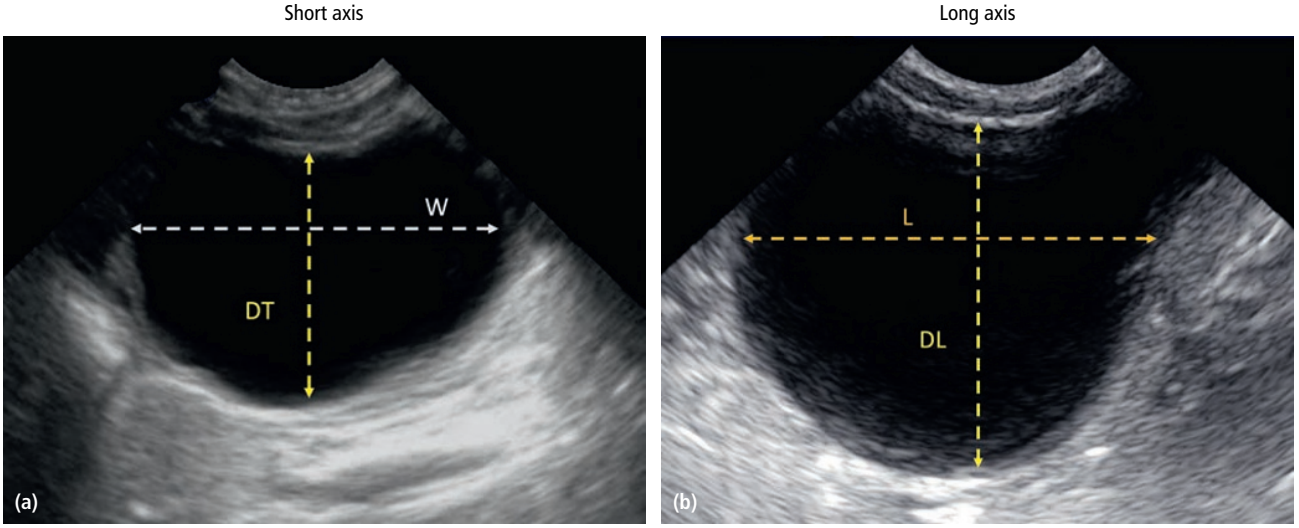


Figure 39.8 (a) Transverse (short) axis of the urinary bladder at its widest point demonstrating where the width (W) and depth (DT) are measured. (b) Longitudinal (long) axis of the urinary bladder at its widest point demonstrating where the length (L) and depth (DL) are measured. Note that the measurements are taken to best estimate the shape of a sphere.

planes and divide by two to get an average depth. This provides an easy way to check that the widest diameter of the urinary bladder was correctly identified in both planes; if the two depth measurements vary greatly (e.g. more than 10–15%), consider repeating the measurements as the transducer may be oriented obliquely to the urinary bladder, or the measurement obtained to either side of the widest urinary bladder diameter.

Right Paralumbar

The right caudal liver lobe, right kidney, body wall, duodenum, and areas between these structures are evaluated. To find the right paralumbar region in smaller patients, trace the last rib dorsally with your finger until the hypaxial/lumbar muscles are encountered. The transducer is then placed caudal to the last rib, just ventral to the hypaxial/lumbar muscles, in longitudinal axis to the body. Increase the depth setting and then fan and rock the transducer at this location until the kidney is visualized. Once found, decrease the depth until the kidney fills the proximal half of the ultrasound image. In larger dogs it may be necessary to place the transducer between the most caudal ribs (i.e. at the 11th or 12th intercostal spaces) to locate the key abdominal VPOCUS structures in this view.

In dogs, if the liver is visualized first, the transducer can be slid caudally until the kidney is identified. The kidney sits caudal to the liver in the hepato-renal fossa. Note that the right kidney is often quite lateral (relative to midline). When the correct site is identified, the transducer is fanned and rocked in both longitudinal and transverse axes to assess the right kidney and its surrounding structures. Depending on the skill of the operator the renal pelvis can also be assessed (see below).

The duodenum is located at the right paralumbar location by first locating the right kidney in longitudinal axis, and then sweeping the transducer medially toward midline until the largest small intestinal segment is found. The duodenum can be assessed for motility at this location (see below).

Left Paralumbar

The spleen, left kidney, intestines, body wall, and areas between these structures are evaluated. To find the left paralumbar region, start by finding the left kidney: use your index finger to trace the last rib from the mid-abdominal region caudally and dorsally until the last rib contacts the hypaxial/lumbar muscles. The kidney is usually located at this site. The transducer is fanned and rocked through all longitudinal and transverse axes to assess the left kidney. Depending on operator experience, the renal pelvis may be assessed at this site (see below). It may be easier to find the spleen and slide the transducer caudally until the left kidney is located. The spleen is relatively mobile, and the tip may sometimes be seen contacting the left kidney. If it is not encountered when scanning the left kidney, it can often be located cranial and lateral to the left kidney.

Specific Questions

Abdominal Effusion

Free (uncontained) abdominal fluid appears black (anechoic or hypoechoic) and forms sharp angles and triangles between organs. To be certain that fluid is free and not contained within a structure (such as stomach, urinary bladder, gallbladder), the operator should evaluate thoroughly all key organs at every site, fanning through all planes in both the longitudinal and transverse axes. Decrease the depth to highlight concerning areas if needed [1–3].

If free fluid is identified, fluid samples should be collected for urgent analysis (packed cell volume, total protein concentration, cell count, cytology, and more specific analyses such as glucose, lactate, creatinine, triglycerides, and bilirubin as indicated; see Chapter 38), which may assist with diagnosis, guide further diagnostics, and dictate immediate interventions. Ultrasound guidance is very helpful as it allows the aspiration needle to be visualized so that fluid can be collected with confidence and fewer complications. Consider sample submission to a reference laboratory for cytology and culture.

Pneumoperitoneum

Pneumoperitoneum is abnormal in patients that have not recently undergone laparotomy and should prompt consideration of hollow organ rupture (i.e. gastrointestinal perforation) [4]. Although the learning curve for detecting pneumoperitoneum using POCUS is steep, given the accuracy of ultrasound to detect free abdominal gas, and the fact that emergency surgery is indicated in positive cases, it is a valuable skill to learn. To increase the chances of detecting free abdominal gas, place the animal in lateral recumbency for several minutes to allow gas to rise to the nongravity-dependent body wall.

There are three key steps that should be followed to standardize the detection of pneumoperitoneum with abdominal POCUS (Figure 39.9) [8]. Note that abdominal POCUS is better at ruling in than ruling out the presence of free gas, as pneumoperitoneum might be missed if free abdominal gas fails to reach the nondependent peritoneal surface, and free gas is only detectable at sites scanned by

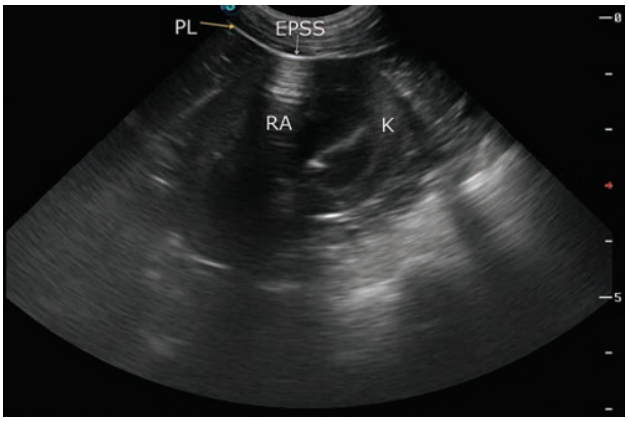


Figure 39.9 Image depicting free abdominal gas. The kidney (K) is in contact with the peritoneal lining (PL), which helps to ensure that there are no intestinal segments or other structures between the kidney and peritoneum. A segment of reverberation artifact (RA) can be seen originating from the peritoneal lining extending toward the far field of the image, obliterating the view of the normal kidney architecture. Where free gas is in contact with the peritoneal lining the peritoneal lining appears more hyperechoic, giving rise to the term enhanced peritoneal stripe sign (EPSS).

the operator. False positive results are also possible if intestinal gas is confused with free abdominal gas.

1) Identify the peritoneal lining. The peritoneal lining is important to identify because it allows gas in the abdomen to be differentiated from gas contained within the gastrointestinal tract. The peritoneal lining can be visualized directly or identified by locating structures that contact it (e.g. liver, kidney, spleen).
2) Search for the presence of reverberation artifact that originates at the peritoneal lining extending a few millimeters to several centimeters into the far field of the ultrasound image. Reverberation artifact originating from free abdominal gas in contact with the peritoneal lining passes through and obscures the structures beyond it. This can be very helpful in confirming the presence of free abdominal gas; if there is no visible intestinal wall between a solid organ (e.g. liver, spleen, or kidney) and the peritoneal lining, but there is obliteration of that solid organ by reverberation artifact arising from the peritoneal lining, it most likely indicates the presence of free abdominal gas. If the gas originates from the intestinal tract and is not free in the abdomen then the intestinal wall will be visible between the obliterated organ and the peritoneal lining (and the enhanced peritoneal stripe sign (EPSS) will be absent; see below).
3) Look for an EPSS. EPSS occurs when free abdominal gas comes in contact with the peritoneal lining, causing the peritoneal lining to become more hyperechoic (whiter) than the surrounding peritoneal lining not in contact with free abdominal gas. This finding can be subtle. Reverberation artifact resulting from free abdominal gas that reaches the peritoneal lining will be associated with an EPSS. With practice it is also possible to detect free abdominal gas in contact with the serosal surfaces of abdominal organs as it will also create a brighter enhanced region where gas contacts these serosal surfaces.

Gastrointestinal Ileus

There is insufficient evidence to state whether gastrointestinal motility can be definitively assessed with abdominal POCUS; however, it is helpful in identifying the presence of ileus, defined as a transient cessation of gastrointestinal motility or an abnormal pattern of gastrointestinal motility [5]. On average, the normal number of peristaltic contractions of the stomach and proximal duodenum that can be detected with ultrasound in dogs is four to five contractions per minute. The number of contractions is fewer and less consistent in other sections of the gastrointestinal tract. Therefore, the stomach and duodenum are often assessed for motility in postoperative and hospitalized patients, particularly if they are anorexic and regurgitation is noted. To measure the number of contractions per minute, the total number of contractions is recorded over three minutes and divided by three. If contractions are decreased or absent, a diagnosis of decreased motility/ileus should be considered.

The detection of food within the gastrointestinal tract in the absence of visible contractions is a strong indicator of ileus because luminal content is a strong stimulus for gastrointestinal contraction.

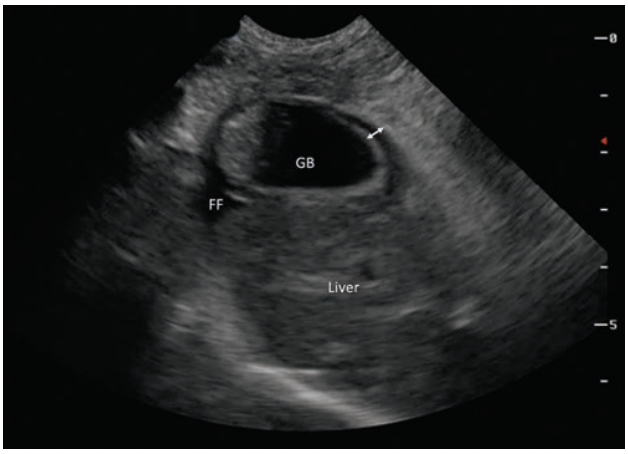


Figure 39.10 Ultrasound still image of gall bladder wall edema (aka halo sign). The gallbladder wall is thickened (white arrow) with the appearance of three distinct wall layers: an outer hyperechoic layer, a middle hypoechoic layer (edema), and an inner hyperechoic layer. When these three distinct layers are noted, it is termed the halo sign. A small volume of free fluid (FF) is also noted cranial to the gall bladder (GB).

Gall Bladder Wall Thickening (Halo Sign)

With edema of the gall bladder wall, the wall becomes thickened and may develop three distinct layers: an outer hyperechoic layer, a middle hypoechoic layer (edema), and an inner hyperechoic layer. The presence of all three layers is referred to as the halo sign (Figure 39.10). The halo sign is a nonspecific finding, being caused by several conditions [6]. However, conditions that impede right heart filling (right sided heart failure or pericardial effusion), sepsis, and anaphylaxis should be considered in unstable patients. It may be associated with sedation using dexmedetomidine [11]. If patients are serially evaluated and gall bladder wall thickening develops following fluid therapy, volume overload should be suspected. Other supporting evidence of volume overload can be assessed with POCUS (Chapter 17).

Urine Production

Although not perfectly accurate, a lack of change in bladder size (volume) despite appropriate fluid therapy in a well-hydrated patient could indicate anuria or oliguria [7].

Ureteral Obstruction

Preliminary research in companion animals suggests that assessment of the right and left kidneys at either paralumbar region allows the nonspecialist clinician to sonographically screen for pyelectasia, gross renal asymmetry, ureteral dilation, and visualization of calculi with high sensitivity and specificity when compared to consultative ultrasound [10]. Identification of nephromegaly, perirenal fluid, and hydronephrosis is also possible [9]. Together, these findings are suggestive of acute kidney injury and/or ureteral obstruction [9, 10]. For example, identifying these findings in the feline patient with acute azotemia and suspected oligoanuria may help to guide a diagnosis and further complement physical examination. In cases with confirmed ureteral obstruction in which medical management is attempted, following the renal pelvic diameter serially may assist with the decision regarding the need for and timing of surgery.

Pitfalls of Abdominal Point-of-Care Ultrasound

Hepatic Vessels

Hepatic vessels can sometimes be mistaken for fluid. It is important to remember that ultrasound is a dynamic imaging modality and therefore by fanning, rocking, sweeping, sliding, changing the depth, and rotating the transducer (longitudinal to transverse and vice versa) it is easier to confirm free abdominal fluid vs. contained fluid (blood) within vessels.

Edge Shadowing

Edge shadowing is where the ultrasound beams create dark shadows on the edges of a structure (e.g. gall bladder), which can be mistaken for fluid. This artifact can cause the wall of a structure to partially disappear, which should not be confused for rupture of an organ (e.g. do not confuse edge artifact for rupture of the urinary bladder). Edge shadowing from structures such as the gall bladder, stomach wall, or urinary bladder, for instance, should not be confused with free fluid. Edge shadowing will disappear depending on the angle the ultrasound beam strikes the structures of interest; changing the angle of insonation by manipulating the transducer to change this angle is helpful.

Intestinal and Stomach Wall and Contents

Intestinal and stomach wall and contents can also be mistaken for abnormal fluid or structures, particularly when intestinal wall edema is present. By moving the transducer and assessing multiple planes (longitudinal and transverse), it will be easier to determine whether the structure is intestine or stomach.

Mirror Image Artifact

Mirror image artifact of the gall bladder or hepatic vessels, which are often distorted, can be confused for pleural effusion, or even misinterpreted as a diaphragmatic hernia.

Acute Hemorrhage or Very Cellular Effusions

Acute hemorrhage or very cellular effusions can sometimes be so echogenic that they mimic soft tissue structures or gastric contents, making it more difficult to identify as free abdominal fluid. Transducer manipulations (e.g. applying pressure to the transducer to displace underlying free abdominal fluid) can sometimes create "swirling," which may help differentiate such cellular effusions from tissues. Fanning and rocking the transducer will also often identify "structures" within the fluid (e.g. floating omentum or intestines) and may allow sharp angles/triangles to be visualized, which helps to differentiate free abdominal fluid from fluid contained within hollow organs.

Video 39.1 This video demonstrates point-of-care ultrasound of the umbilical site to assess for gravity-dependent peritoneal fluid. This video includes audio narration.

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40

Specialized Gastrointestinal Techniques

Lisa Smart and Joyce Lau

Auscultation of Gastrointestinal Sounds

The association between abdominal auscultation and gastroenterological disease was first described in 1905 in human medicine [1]. Since then, very few studies have been conducted to validate or refute this assumption. Current gastrointestinal auscultation methods are subject to a physician's individual techniques and preferences, resulting in frequent conflicting conclusions on patient gastrointestinal status, diagnosis, and treatment recommendations [1–4]. Owing to the lack of standardized guidelines to perform and interpret abdominal auscultation, the clinical utility of this technique as part of a physical assessment has come under scrutiny.

In veterinary medicine, abdominal auscultation has been described as an adjunctive tool for the diagnosis of abdominal diseases or gastrointestinal dysfunction [5]. At least five minutes of abdominal auscultation to determine whether borborygmus is increased or decreased has been described. However, there have been no studies evaluating the diagnostic utility of borborygmus evaluation in dogs or cats, and the link between the presence of gastrointestinal sounds and normal motility is dubious. Until these studies are performed, therefore, the presence or absence of borborygmus should be interpreted cautiously.

Gastric Intubation

Introduction

There are two basic goals for intubation of the gastrointestinal tract: removing contents or administering food and medications. Although many of these techniques are used routinely in veterinary emergency and critical care medicine, all these procedures have the potential for complication, and it should always be considered whether the benefits of the procedure outweigh the risks.

Orogastric Intubation

Orogastric intubation involves inserting a tube through the oropharyngeal cavity to the stomach while the patient is under sedation or, ideally, general anesthesia with a secure airway (Protocol 40.1; Box 40.1; Figures 40.1–40.4).

Indications

The indications for orogastric intubation include decompression of gastric dilation, removal of ingested toxins via gastric lavage and administration of medications, such as activated charcoal. Complications can include damage to or perforation of the esophagus or gastric wall, regurgitation and aspiration of gastric contents, and adverse events related to sedation or general anesthesia. The risk of complications related to anesthesia may be increased if the animal is showing clinical signs of intoxication. Although in the authors' experience, these complications are uncommon if the correct technique is followed, the true prevalence of these complications has not been specifically reported. The following technique described is for conscious dogs. Cats do not tolerate this procedure sedated and are at risk of laryngospasm; therefore, general anesthesia and endotracheal intubation to secure the airway are necessary in cats. An exception is made for neonatal and newborn kittens, which are often fed by orogastric tube when they are unable to nurse (Chapter 74).

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Companion website: www.wiley.com/go/burkitt/monitoring

Protocol 40.1 Orogastric Intubation

1) Determine whether the patient is an appropriate candidate for gastric decompression, and whether sedation or anesthesia is required.
2) Choose a single-lumen orogastric tube with the largest diameter that will reasonably fit within the patient's esophagus (Figure 40.1).
3) Measure the length of tube that will be required to pass into the stomach, which is the distance from the nares to the last rib. Mark the tube with either tape or indelible marker (Figure 40.2).
4) For the conscious dog, unroll approximately 30 cm from a roll of adhesive bandage material, and leave this attached to the roll. With someone restraining the dog's head from behind, place the roll of bandage into the dog's mouth with the long plane parallel with the long axis of the muzzle, holding the mouth closed. Then wrap the free end of the bandage around the dog's mouth to hold the roll in place (Figure 40.3). If the dog starts to regurgitate or vomit, the roll can be removed quickly out of the mouth rostrally without the need to unwrap the bandage from around the muzzle.
5) Lubricate the end of the orogastric tube and slowly feed the tube through the hole in the middle of the bandage roll (Figure 40.3). Aim to push the tube through the left dorsal aspect of the dog's pharynx to avoid intubating the larynx. Try to advance the tube when the dog swallows. Leave the other (distal) end of the tube below the level of the animal in a collection container, so that any fluid within the tube will drain by gravity.
6) Location of the tube within the esophagus must be confirmed by one or more of the techniques described (Box 40.1). If there remains any doubt as to whether the tube is in the trachea as opposed to the esophagus, remove the tube and start again.
7) Once the location of the tube within the esophagus is confirmed, advance the tube gently up to the mark previously made on the tube (Figure 40.4). A rush of gas may be heard at the proximal end of the orogastric tube when passed into the stomach. In the case of GDV, if there is resistance, apply gentle pressure with a twisting motion to aid entry through the cardia. If there is still resistance, clean gauze may be placed over the end of the tube, to protect the operator's mouth from contamination, and a small amount of air blown into the tube while gently advancing it. If the tube still cannot be passed into the stomach in a patient with severe gaseous dilation, then the stomach should be trocarized percutaneously to relieve tension on the fundus (see below for technique); the tube will often then pass more easily. If there is no gastric fluid flow into the collection container below the level of the patient, gentle ballottement of the abdomen may help to dislodge any obstruction. If there is still no flow, 10–60 ml of water can be infused into the tube to flush any obstruction (see also Protocol 40.5).
8) Before removing the tube, tightly kink the tube 10–15 cm from the operator's end. Hold the tube firmly in this kinked position while removing the tube to prevent fluid leakage as it is removed from the esophagus and pharynx.

Box 40.1 Techniques to Distinguish Between Intubation of the Esophagus/Stomach and the Trachea

Indications That the Tube is Within the Esophagus or Stomach

- Seeing the presence of esophageal fluid or gastric reflux fluid in the tube. The fluid should look like saliva as well as any other substances such as ingesta, bile pigment, or blood.
- Palpation of the tube on the left side of the neck adjacent to the trachea confirms its placement in the esophagus.
- Either a rush of gas or gastric fluid will confirm its location in the stomach. Air blown into the tube by the operator (see Step 7 of technique for orogastric tube placement) with simultaneous auscultation of borborygmus within the stomach also confirms its location.

Indications That the Tube is Within the Trachea

- If humidified air is seen within the tube during each exhalation, the tube may be within the trachea.
- Coughing, though the absence of coughing does not confirm placement within the esophagus
- The tracheal rings may be felt as vibrations along the tube in a very large dog if the tube is passed down the trachea.
- A capnometer may be placed at the end of the tube. An increase in carbon dioxide while the dog exhales confirms its location in the trachea (Chapter 30, Capnography).
- If placing a nasogastric tube, intubation of the trachea is likely if a copious amount of air is aspirated via syringe from the end of the tube (ensure any side ports of the tube are sealed when aspirating).

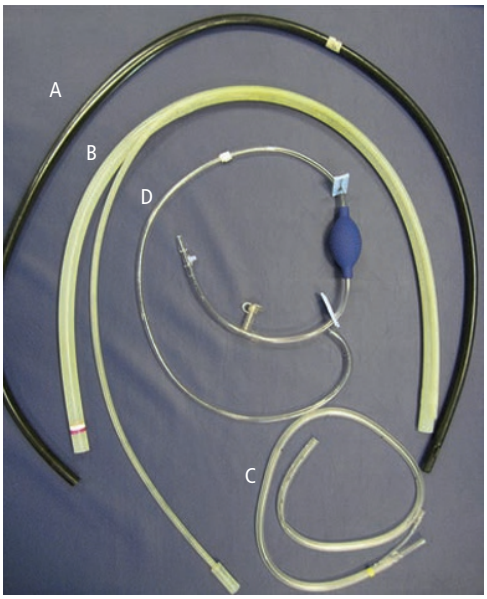


Figure 40.1 Various tubes used in gastrointestinal techniques. A: Simple orogastric tube stained with activated charcoal; B: Double lumen orogastric tube used for gastric lavage; C: Orogastric tube with fenestrations and intraluminal side port for flushing, suitable for smaller patients or for enemas; D: Enema tube with insufflation bulb to aid flushing.



Figure 40.2 Measurement of the length of tube needed for orogastric intubation in a dog. The person to the dog's left is indicating the level of the final rib with their index finger.

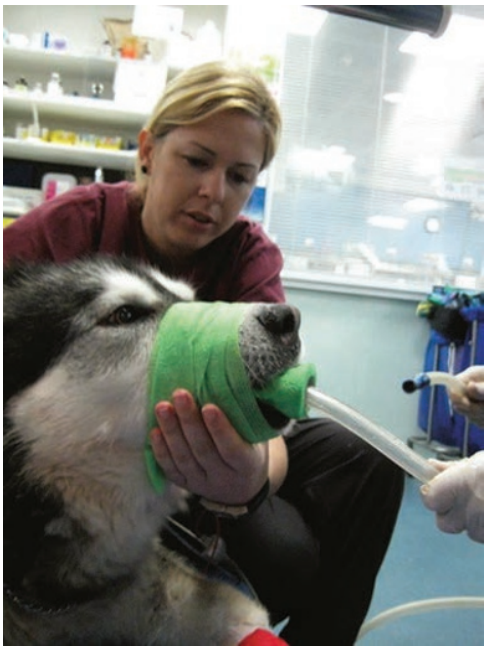


Figure 40.3 Advancement of an orogastric tube through the middle of a bandage roll in a patient with gastric dilation and volvulus. The free end of the bandage roll can be seen wrapped around the nose and mouth to keep it in place.



Figure 40.4 Passing an orogastric tube up to the premeasured mark (white medical tape) while listening for a rush of gas, indicating passage into the stomach.

Nasogastric Intubation for Gastric Decompression

Nasogastric intubation involves placing a tube through the nasal cavity down into the stomach and is best placed in a conscious animal that can swallow.

Indications

Nasogastric intubation may be useful in patients that are experiencing discomfort due to gastric dilation secondary to either aerophagia or gastric fluid accumulation (Box 40.2). This technique is not appropriate for the management of food engorgement (see below). Placement of a nasogastric tube in cats for aerophagia is not recommended, as stress will usually exacerbate the cause of aerophagia. However, if a nasogastric tube is already in place for enteral nutrition, it may be used for air evacuation in the cat. Discretion should be used as to whether or not the patient will benefit from nasogastric tube placement for the purpose of gastric decompression.

Box 40.2 Nasogastric Intubation for Gastric Decompression

Indications

Gastric dilation caused by aerophagia or gastric stasis AND one of the following:

- Increased work of breathing
- Frequent regurgitation
- Signs of nausea, such as hypersalivation and frequent swallowing
- Signs of abdominal pain on palpation

Contraindications

- Respiratory distress, whereby the stress of placing a nasogastric tube may worsen the patient's condition
- Cats with aerophagia
- Coagulopathy
- Thrombocytopenia (< 50 000/µl)

For the patient with gastric dilation secondary to gastric stasis, intermittent emptying of stomach contents every six to eight hours via a nasogastric tube may decrease vomiting and abdominal discomfort (see section on gastric residual volume monitoring). Gastric fluid pH may also be monitored by this procedure (see section on monitoring gastric fluid pH) and the nasogastric tube may be used for enteral feeding, which may stimulate some gastric motility (Chapter 44). It is unknown whether the presence of a nasogastric tube through the lower esophageal sphincter promotes reflux of gastric contents into the esophagus but, anecdotally, this does not appear to contribute to esophagitis.

Technique

The technique for placing a nasogastric tube has been covered elsewhere (Chapter 44). The nasogastric tube is most easily placed in conscious animals; however, sedation should be used in most cases of gaseous gastric dilation, as placement of the nasogastric tube can be stressful and exacerbate aerophagia. Once the nasogastric tube is in place, gentle suction should be applied to evacuate air or fluid until negative pressure is reached. Blockages of the nasogastric tube with food material or thick saliva may impede evacuation; therefore, intermittent flushing of the nasogastric tube with 3–5 ml of water or air may be necessary. Record volumes of fluid removed (see section on gastric residual volume monitoring).

Considerations for the Patient with Gastric Dilation and Volvulus

Risk factors for, presentation of, and management of shock in the patient with gastric dilation and volvulus (GDV) have been reviewed elsewhere [6–8]. Diagnosis of GDV may be confirmed by the presence of a dorsally displaced pylorus on a right lateral abdominal radiograph [9].

Analgesia/Sedation

Moderate to severe vasoconstrictive shock in animals with GDV is caused by lack of venous return from the caudal half of the body, leading to decreased cardiac output and organ ischemia, including the myocardium. Therefore, shock fluid therapy is a priority in these patients. Analgesia should also be given as soon as possible, such as a pure mu agonist opioid. Nonsteroidal anti-inflammatory drugs and corticosteroids should not be used in GDV patients.

To facilitate orogastric intubation, sedation may be given, although it is usually not needed. A combination of opioids and benzodiazepines, such as diazepam or midazolam, usually suffices. There is an increased risk of ventricular arrhythmias due to decreased myocardial perfusion [10–12]; therefore an electrocardiogram (ECG) should be performed if an arrhythmia is detected. Standard guidelines should be followed on the decision to treat ventricular arrhythmias [13], with awareness that those that occur during general anesthesia may require more aggressive treatment. Ideally, the dog's stomach should be decompressed by either orogastric intubation or trocarization (see below) before general anesthesia is induced to achieve more rapid cardiovascular stabilization.

Trocarization

Gastric trocarization (Protocol 40.2; Figure 40.5), also referred to as gastrocentesis or gastric needle decompression, is often required in the patient with GDV because orogastric intubation is difficult or impossible in the presence of severe gastric distention (see technique for orogastric intubation). Gastric trocarization often decreases the time to gastric decompression and therefore may decrease the risk of gastric necrosis. The risk of gastric tear due to trocarization is likely small, and studies that include trocarization as a part of the gastric decompression protocol do not report the presence of gastric perforation on exploratory laparotomy [14–18]. One study used trocarization alone for pre-surgical decompression, without orogastric intubation, and reported a low overall mortality rate of 10% [19].

Protocol 40.2 Gastric Trocarization

1) Provide analgesia for the patient.
2) Choose a tympanic area on the lateral abdomen, which is usually the left dorsolateral abdomen just caudal to the last rib.
3) Ideally, confirm presence of a gas interface with point-of-care ultrasonography, and the absence of a solid organ abutting the body wall (e.g. spleen; see Chapter 39).
4) Clip the area and prepare aseptically. Perform hand hygiene and don sterile gloves.
5) Insert a 16-gauge over-the-needle catheter at a right angle to the abdominal wall, with the bevel facing up, into the abdomen. Once gas flow is heard, or gastric fluid seen in the hub, advance the catheter off the stylet into the abdomen up to the hub of the catheter. Remove the stylet.
6) Hold the catheter in place with a sterile hand and allow the gas to passively escape until the abdomen becomes flaccid or noticeably reduced in size (Figure 40.5). If the catheter becomes occluded, it may be withdrawn slowly from the abdomen until gas flow resumes or the catheter comes out. Never advance the catheter back into the abdomen. Do not ballotte the abdomen with a catheter in place, as it may dislodge the catheter from the stomach and contaminate the abdomen.
7) If the flow of gas from the first catheter is insufficient, a second catheter may be placed near the first using the same technique.
8) Once the abdomen has reduced in size, orogastric intubation (OGI) may be attempted again in a gentle manner to fully evacuate the stomach. Any catheters used for trocarization should be removed before repeat OGI.
9) Alternatively, OGI may be performed under general anesthesia before surgery, if trocarization has facilitated cardiovascular stabilization.



Figure 40.5 Gastric trocarization in a dog with gastric dilation and volvulus. An 18-guage catheter (note green hub) has been placed in the region of most tympany and is facilitating gas escape from the stomach.

Conservative Management of Gastric Dilation and Volvulus

There is a low chance of a good long-term outcome after decompression only for a GDV patient. Several studies have described techniques for gastric repositioning during decompression; however, short-term survival is compromised and recurrence by one year is highly likely if gastropexy is not performed [17, 19–21]. In addition, if there is gastric necrosis present and only conservative therapy is provided, the patient is likely to suffer a painful demise. Conservative treatment should only be undertaken for gastric dilation in a stable patient with the absence of volvulus; however, gastropexy should still be considered in breeds at high risk of subsequent GDV. Immediate surgical intervention of GDV after decompression is recommended to improve the chance of survival.

Considerations for Food Engorgement

Food engorgement or "food bloat" with subsequent gastric dilation is a normal occurrence for carnivores, given their evolutionary adaptation for intermittent feeding. However, cases of engorgement associated with clinical signs are usually secondary to ingestion of large amounts of dry commercial food, which absorbs fluid and slowly expands in the stomach. This leads to gross distension of the stomach and considerable abdominal pain. A diagnosis of engorgement is confirmed by evidence of a dilated, ingesta-filled stomach in its correct anatomical position on a right-lateral abdominal radiograph. Despite the large gastric distension that can be associated with this syndrome, dogs usually have an excellent outcome with conservative medical management only, such as analgesia and fluid therapy [22]. Unlike in cases of GDV, dogs with food

engorgement do not typically display signs of shock, although they may be tachycardic, mildly hyperthermic, and appear vasodilated. These signs often resolve with analgesia. Acid–base and electrolyte abnormalities can also develop during treatment [22], so monitoring of these parameters along with hydration status is warranted. In the authors' opinion, if cardiovascular compromise is evident, then the problem may be surgical and another underlying cause should be sought, such as volvulus. Interventions such as emesis, gastric lavage, and gastrotomy are not indicated in simple food engorgement and may increase risk of adverse events. However, if the dog has ingested a substance that has solidified, such as polyurethane adhesive (Gorilla Glue®, Gorilla Glue Inc., Ohio), which does not have a chance of passing through the intestinal tract, then gastrotomy may be warranted.

Gastric Fluid Aspirate pH Monitoring

Gastric pH monitoring can be achieved by various methods: estimating pH of gastric fluid aspirate, use of an indwelling nasogastric pH monitoring probe, or placement of a gastric radiotelemetric capsule (Protocol 40.3). Although continuous pH monitoring increases accuracy for assessing gastric pH [23], the two latter methods are usually reserved for research purposes, such as measuring efficacy of antacid therapy or detecting esophageal reflux, and will not be discussed further. The use of gastric aspirate pH to guide antacid therapy has been advocated in human medicine [24]; however, there has not been widespread uptake due to pitfalls associated with this measurement and the minimal evidence for an association with improving clinical outcome [25]. However, lack of gastric aspirate pH monitoring to guide antacid dosing in human critical care has recently been challenged, and it is possible this practice may experience a revival [26].

The use of pH colorimetric indicator paper or pH strips provides a practical alternative to indwelling pH monitoring probes. pH strips with multiple test squares provide a higher degree of accuracy, and are easier to read than single square pH test strips [27, 28]. Although excellent agreement has been shown between pH measured on gastric aspirate fluid using litmus paper and gastric pH measured by a nasogastric pH probe [29], litmus paper may generate a small, although clinically relevant, positive bias when compared with a pH meter [30]. This means that pH strips could potentially give a reading up to three units higher than pH measured by a probe. However, there is minimal evidence regarding the accuracy of modern pH strips.

Maintenance of a gastric pH greater than 4.0 has been recommended in human medicine to optimize healing of gastric ulcers and reduce the risk of gastric bleeding [26, 31, 32]. A gastric pH greater than 6.0 may be important for people at risk of life-threatening gastric hemorrhage, as an acidic pH promotes blood clot dissolution [31]. Given pH strips may be inaccurate or have a positive bias, it seems prudent to aim for a gastric aspirate pH greater than 5.0 using strips if there is a concern for adverse effects of gastric acidity. Measurement of gastric aspirate pH has also been suggested to guide treatment of chemical exposure in human emergency medicine or to assist with correct placement of nasogastric tubes [25]. However, fasting gastric pH in healthy Beagles has been shown to fluctuate more widely, between pH 2.0 and 8.0 [33], than in healthy people, where fasting pH is approximately 1.0–2.0 [34, 35]. Gastric pH in cats has also shown a wide variation over 24 hours, albeit mostly staying in the acidic range [36]. Therefore, single measurements of pH should be interpreted with caution in dogs and cats, especially in regards to guiding nasogastric tube placement or attempting to use gastric pH to diagnose caustic chemical ingestion.

Protocol 40.3 pH Monitoring of Gastric Fluid Aspirates

1) Ensure that some time has lapsed since the last feeding or administration of water down the nasogastric tube (at least four hours).
2) Aspirate ≥ 1 mL of gastric fluid from the nasogastric tube with a catheter-tip syringe and place on a multi-square pH-sensitive paper or strip or single square.
3) Determine pH of the gastric aspirate by comparing a colorimetric scale to the pH strip (Figure 40.6).
4) If fluid pH is less than 5.0, and gastric healing is of concern, increase acid suppressive therapy dose or frequency, depending on drug.
5) Repeat procedure at next aspiration of the nasogastric tube after new therapy regimen instituted. Timing will depend on pharmacokinetics of antacid therapy of choice.

Indications

Owing to the lack of veterinary consensus and clinical standards on the utility and frequency of gastric aspirate pH monitoring using pH strips, we have included the preferred approach based on our experience. Clinicians should bear in mind the pitfalls of gastric pH estimation in regard to accuracy.

Gastric aspirate pH measurement is most often used for assessing efficacy of antacid dosing. In our clinical

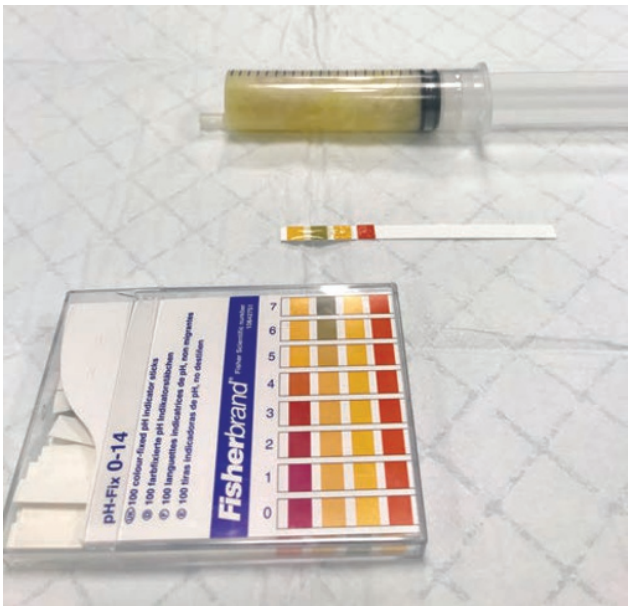


Figure 40.6 Syringe filled with scavenged gastric fluid from a nasogastric tube placed next to a multi-square pH sensitive strip (Fisherbrand pH indicator sticks, ThermoFisher Scientific, Australia). A pH of 6 is determined by comparing the colorimetric scale to the pH strip.

practice, gastric aspirate pH is measured using multisquare pH strips on fluid scavenged from nasogastric tubes (Figure 40.6) if gastric acidity poses a concern. Gastric aspirate pH estimation may be made for a patient with signs of continuing upper gastrointestinal discomfort to interrogate efficacy of antacid therapy and adjust dose or frequency if indicated.

Gastric Residual Volume Monitoring

Gastric residual volume (GRV) monitoring is the measurement of the volume of fluid that is aspirated from the stomach through a nasogastric tube. It is used to assess gastric emptying and, subjectively, the degree of dysmotility in human and veterinary patients receiving enteral nutrition.

Indications

Historically, increased GRV has been used in human critical care to trigger a pause in enteral feeding to decrease the risk of vomiting, aspiration, and ventilator-associated pneumonia. This led to measurement of GRV becoming a routine nursing care procedure in human medicine, with one survey reporting that 89% of nurses withheld enteral nutrition at GRVs greater than 300 ml [37]. However, the validity of this technique for reducing risk of aspiration has been questioned in recent times. Multiple human studies have shown the retrieved volume to range 14–136% of gastric volume [38–41]. In addition to inaccuracy of the measurement itself, interruption of enteral nutrition based on GRV has not been shown to reduce the risk of aspiration, vomiting, or ventilator-associated pneumonia [42–45]. Therefore, interruption of feeding for this reason may only lead to inadequate caloric intake. Recent human clinical guidelines have recommended against routine use of GRV monitoring in patients receiving enteral nutrition, and only withholding feeding if GRV is greater than 500 ml in combination with signs of gastrointestinal intolerance [46].

In veterinary medicine, most patients receiving enteral nutrition via a nasogastric tube are not anesthetized or intubated. However, given that conscious veterinary patients, particularly dogs, are still at risk of aspiration if frequently vomiting or regurgitating, GRV monitoring may still be pursued. Abdominal distension due to gastrointestinal intolerance may also cause discomfort. Increased GRV might prompt a reduction in administered enteral nutrition volume [47] or addition of prokinetic drugs. Unfortunately, there is very little evidence to support or refute the practice of GRV monitoring. A randomized controlled trial of continuous versus intermittent nasogastric tube feeding in dogs could not demonstrate a correlation between GRV and the occurrence of vomiting, regurgitation, or percentage of nutrition delivered [48]. Prospective studies are required in veterinary medicine to determine whether GRV monitoring impacts clinical outcome.

Given the lack of standard veterinary definitions of excessive GRV and lack of consensus on frequency of monitoring and return of gastric fluid, we have included our typical preferred approach (Box 40.3, Protocol 40.4). However, the clinician should determine whether GRV monitoring is necessary and how frequently it should be performed on an individual case basis.

Box 40.3 Clinical Signs that may Warrant Gastric Residual Volume Monitoring in Patients Undergoing Assisted Enteral Feeding

- Distended abdomen
- Signs of abdominal discomfort
- Restlessness after feeding
- Signs of nausea, such as ptyalism
- Retching
- Vomiting
- Regurgitation

Protocol 40.4 Monitoring Gastric Residual Volume

GRV monitoring can be performed in the conscious patient every 6–12 hours. It is performed before delivering the subsequent bolus of enteral nutrition formula.

1) Don clean examination gloves and attach a syringe to the proximal end of the nasogastric tube.
2) Apply gentle suction to evacuate gastric fluid until negative pressure is reached.
3) Measure and record aspirated gastric fluid. The clinician may order "return" of all or a portion of the GRV to the stomach in an attempt to avoid electrolyte or acid–base disturbances. Re-instill the ordered volume (0–100%) of gastric fluid slowly via the nasogastric tube.
4) Note any abnormal coloration of the fluid, such as frank blood.
5) Consider pH testing of the aspirated fluid.
6) If aspirated fluid volume is more than 50% of the previous volume of administered enteral nutrition formula, consider returning only 50% of that volume back to the patient.
7) If GRV is greater than 4–8 ml/kg, or more than 50% of the previous infused volume of enteral nutrition formula, and there are other signs of intolerance (see indications above), consider the presence of persistent gastrointestinal dysmotility.
8) Do not increase volume of enteral nutrition formula per feed (or hourly rate if continuous) until signs of gastrointestinal intolerance abate.

Gastrointestinal Decontamination

Gastrointestinal decontamination (GID) for ingested toxins includes emesis, gastric lavage, whole-bowel irrigation, enema, and activated charcoal administration. In human medicine, the routine use of GID has become controversial because of a lack of evidence that GID has a significant impact on patient outcome, except for specific indications that will be addressed below [49–52]. No studies in veterinary medicine have been conducted to assess whether or not GID changes patient outcome for routine poisoning cases; however, it still remains as the current recommendation for ingested toxins within certain guidelines [53, 54]. Veterinary poisoning cases may differ from those in human medicine, in that there may be a large volume of toxin within the gastrointestinal tract, and the time from ingestion to presentation can be short. Therefore, there still remains a role for GID in veterinary medicine.

Indications, risks, and drug choice for emesis have been covered elsewhere and will not be addressed in this chapter [53–62].

Gastric Lavage

Gastric lavage includes orogastric intubation and removal of gastric contents (Protocol 40.5). In human clinical studies, gastric lavage has shown no clear benefit over activated charcoal administration alone [49, 50]; however, there still remain some indications for this procedure in veterinary medicine.

Indications

Gastric lavage is indicated for intoxication cases where the amount of toxin ingested is potentially harmful, it was ingested within one to two hours of performing gastric lavage, and emesis cannot be achieved due to altered

Protocol 40.5 Gastric Lavage

Refer to Protocol 40.1 for guidance regarding insertion and removal of the orogastric tube.

1) The patient must always be orotracheally intubated with an appropriately inflated cuff and placed in lateral recumbency before lavage is performed.
2) With the orogastric tube in place, use room temperature water to lavage the stomach by use of a siphon. Although it is stated as common practice [63–65] to use approximately 5–10 ml/kg of water for each cycle, more is usually needed to lavage the stomach adequately and the authors commonly use up to 20–30 ml/kg/cycle to achieve mild gastric distension on abdominal palpation. After instilling a volume of water, allow the effluent to passively drain by gravity, aided by gentle ballottement of the abdomen. A double-lumen tube can also be used to aid continuous drainage of the stomach while flushing (Figures 40.7 and 40.8). Never attach a hose to the tube to instill water, nor attach suction to the end to empty the stomach. It is useful to record the volume of the effluent water to monitor the degree of water ingestion and avoid hyponatremia.
3) Check that the cuff of the endotracheal tube is still adequately inflated, then shift the patient onto the other lateral side.



Figure 40.7 Insertion of a double bore orogastric tube for gastric lavage.

4) Repeat the lavage as above. A third lavage on the first lateral side may be considered for snail pellet ingestion, where pellets can stick to the stomach wall (termed a three-sided lavage).
5) Save the contents of the effluent for toxicological analysis, if desired.
6) If activated charcoal administration is indicated, it may be instilled into the stomach before removal of the tube; however, be careful not to significantly distend the stomach, as this leads to increased risk of aspiration during anesthetic recovery. It seems reasonable to limit the dose of activated charcoal to 2–3 ml/kg while under anesthesia.
7) Kink the tube when removing.



Figure 40.8 Gastric lavage for snail pellet ingestion. A double bore tube is being used. A funnel (black) is attached to the ingress tube while water flows out of the stomach through the egress tube into a receptacle (white).

8) Keep the patient tracheally intubated with an inflated cuff until it is able to maintain sternal recumbency and is swallowing. This may reduce the risk of aspiration. It is not advised to delay extubation in cats due to risk of laryngospasm; therefore, activated charcoal should either not be given after lavage or the volume reduced in this species.

mentation or other neurologic signs (Box 40.4). If the patient has already vomited after ingestion of the toxin, gastric lavage is unlikely to recover a significant amount of toxin. One exception is where there has been a large amount ingested, such as in snail pellet ingestion. In our geographic region, snail pellet ingestion is by far the most common reason for gastric lavage; however, other reasons include neurotoxic substance ingestion such as puffer fish, sodium monofluoroacetate (1080), and strychnine.

The risks of gastric lavage include those associated with general anesthesia, aspiration, and gastrointestinal tract trauma. The incidence of esophageal or gastric perforation secondary to gastric lavage is unknown in veterinary medicine, but rarely occurs in human medicine [50] and the same is likely true for veterinary patients.

It is not necessary to perform gastric lavage for food engorgement (see section on considerations for food engorgement). It is also contraindicated to perform gastric lavage for animals that have ingested caustic or volatile substances, due to the risk of esophageal reflux and subsequent esophageal damage, and aspiration. These patients also have an increased risk of esophageal and gastric perforation.

If the animal has ingested a small volume of toxin, such as tablets or capsules, then gastric lavage is unlikely to be rewarding and activated charcoal administration alone should be considered instead. It is unusual for cats to ingest a large volume of toxin and thus this procedure is rarely performed in a cat; however, the principles remain the same.

Nasogastric Intubation for Decontamination

Nasogastric intubation may be useful for removal of large volumes of liquid toxin in which emesis is contraindicated (Box 40.4). It will only be useful within 30 minutes of ingestion unless delayed gastric emptying is present. The nasogastric tube can then be used for activated charcoal administration (see below).

Box 40.4 Gastrointestinal Decontamination

Gastric Lavage

Indications:

- Toxin ingested within one to two hours and emesis contraindicated
- Large volume of ingested toxin, such as snail bait pellets

Contraindications:

- Food engorgement
- Ingestion of caustic or volatile substances
- Ingestion of a small volume of toxin or number of tablets or capsules

Nasogastric Intubation

Indications:

- Large volume of liquid toxin
- Ingestion of caustic substances

Contraindications:

- Respiratory distress
- Cats with aerophagia
- Coagulopathy
- Thrombocytopenia (<50 000/µl)

In humans with caustic substance ingestion, endoscopy for identification of perforations and placement of a nasogastric tube is used, as the tube reduces the incidence of esophageal stricture development and allows for administration of enteral nutrition [66, 67]. A nasogastric tube may also aid in dilutional therapy of the caustic substance if the animal will not drink water or milk, by instilling small amounts down the tube. This is in contrast to gastric lavage, which, as stated above, is contraindicated in caustic substance ingestion.

Activated Charcoal Administration

Indications

The indications for activated charcoal administration have been covered elsewhere [53, 64]. Systemic absorption of many ingested toxins is decreased by activated charcoal if the charcoal is given within two hours of toxin ingestion. Activated charcoal may be beneficial greater than two hours post-ingestion for toxins that undergo enterohepatic recirculation or for delayed release capsules.

Contraindications of activated charcoal administration include oral administration in patients with abnormal mentation, hydrocarbon ingestion, and gastrointestinal tract perforation.

Techniques

Voluntary Consumption Some dogs, especially puppies, will drink activated charcoal suspension if it is mixed with canned commercial food or water. Often, though, they will not consume the required dose and one of the other techniques below may be required.

Syringe Feeding Animals may be restrained and syringe-fed activated charcoal. The risks include undue stress and aspiration, especially if the solution is administered with pressure. The solution is given by dribbling small amounts in the commissure of the animal's mouth and allowing them time to swallow. Gloves and gowns will need to be worn as it becomes quite messy. Another technique for larger dogs is to place the sleeve of a disposable gown over the dog's head, allowing only the nose and mouth to protrude, leaving the eyes covered (Figure 40.9). The person can then dribble the solution in the corner of the dog's mouth slowly and patiently. As the eyes are covered, the dog does not try to get away from the syringe and often accepts administration of the suspension. Also, the gown keeps the rest of the dog clean.

Nasogastric Tube Nasogastric intubation can be useful for slow activated charcoal administration. The technique of placing a nasogastric tube is described in Chapter 44.

Orogastric Tube In some poisoning cases, an orogastric tube has been placed for gastric lavage. Activated charcoal may be administered into the stomach via an orogastric tube at the end of the gastric lavage procedure. However, be aware that as the animal recovers from sedation/anesthesia, there is risk of aspiration, so only small volumes (2–3 ml/kg) should be instilled into the stomach at this point.



Figure 40.9 Administration of activated charcoal by syringe feeding. The disposable gown over the head prevents the dog from seeing the advancing syringe and keeps the dog clean.

Catharsis

Catharsis involves the use of an orally administered, osmotically active, nonabsorbed substance to speed transition of a toxin through the gastrointestinal tract. Cathartics, such as sorbitol, are often combined with activated charcoal in commercially available suspensions. Multiple doses are not recommended as cathartics may contribute to excessive fluid loss and electrolyte imbalances.

Enemas

Indications

Enemas are used to relieve constipation and for GID. They can also be used in the treatment of hepatic encephalopathy.

Techniques

Enemas for Constipation

An enema is required if there is distension of the colon with hard, dry feces and the animal is straining to defecate. If there are hard feces in the rectum only, a small amount of lubricant administered by syringe into the rectum may help passage. Alternatively, commercially available micro-enemas, containing osmotically active substances and lubricant, may be used to relieve mild cases of constipation. If there is gross distension of the colon with hard feces, then a complete enema will need to be performed. The animal, in some cases, will need to be sedated or anesthetized. Rehydration fluid therapy should also be included in the treatment plan.

To perform an enema, pass a lubricated soft red rubber catheter or similar tube (Figure 40.1) through the anus to the level of the mid-abdomen, or until obstruction is felt, and gently infuse 5–10 ml/kg of warm water into the colon. Allow time for the animal to defecate voluntarily [68]. Other substances can be added to the enema solution, such as water-based lubricant, lactulose (5–10 ml) or mineral oil (5–10 ml) to aid the removal of feces. The enema can be repeated if the colon is not emptied by voluntary defecation.

If a simple enema is not successful in relieving the patient's constipation, manual fecal extraction may be needed. The dog or cat should be anesthetized and orotracheally intubated to avoid pain and stress, and to reduce the risk of aspiration as manipulation of the colon may induce vomiting. Repeat the enema as above and gently palpate the colon via external abdominal palpation to help break up the feces and move it into the rectum, where it can be removed digitally. Be careful with the use of instruments to aid extraction, such as sponge forceps, as they can cause perforation of the colon. This procedure takes patience and, sometimes, multiple days of repeated enemas if obstipated.

Enema for Gastrointestinal Decontamination

Colonic enemas can be useful in the management of ingestion of large amounts of toxin. In particular, colonic lavage techniques are useful for removing snail pellets from the colon. The technique is similar to that described above, but the animal is anesthetized and the cycle is repeated until the effluent is clear. It is usually performed at the same time as gastric lavage (Figure 40.10). If there is resistance during passage of the tube, do not force the tube any further into the colon. Siphon water by gravity feed into the colon, allowing the effluent to pass around the tube. This cycle is repeated until the effluent is clear.

Whole-Bowel Irrigation

Whole-bowel irrigation, also known as a through-and-through enema, involves oral administration of large quantities of an iso-osmotic polyethylene glycol electrolyte



Figure 40.10 Colonic lavage of a patient that has ingested snail pellets. The green pellets can be seen around the perineal area. The enema tube (green) is being inserted into the rectum. Note the use of a gravity feed for flushing (metal container) and the orange bulb within the tubing, which can be used to increase pressure during flushing.

solution to irrigate the entire gastrointestinal tract to prevent absorption of a toxin. It is also used to prepare the colon for colonoscopy and surgery. The technique is to administer the solution via a nasogastric tube at approximately 100–500 ml/hour until the rectal effluent is clear. This dose has been adapted from human pediatric doses [69]. This method is rarely employed in veterinary medicine. This technique may be considered for large ingestions of sustained release drugs or iron tablets, or packets of illicit drugs, which would be a rare scenario for veterinary medicine. Although one study in dogs showed improved evacuation of paraquat with whole-bowel irrigation compared with no GID [70], there is little evidence in the literature supporting its clinical efficacy in removing toxins or improving outcome. Benefits exceeding that of activated charcoal alone are questionable, even in ingestion of sustained release drugs [71].

Contraindications include gastrointestinal perforation or gross hemorrhage, ileus, intractable vomiting, or cardiovascular instability. Risks include electrolyte and fluid losses, abdominal pain, vomiting, and aspiration.

Retention Enema

Retention enemas introduce substances such as lactulose that are not absorbed by the colon; hence, they are retained in the colon and may encourage water movement into the colon by osmosis. This method is most commonly used for treatment of hepatic encephalopathy. Lactulose reduces absorption of ammonia from the colon by lowering colonic pH and by osmotic catharsis [72].

The most common technique is to instill 5–10 ml/kg of a 30% lactulose solution into the colon, leave for 20–30 minutes and then flush out using warm water. A routine enema (such as that described for constipation) may also suffice for treatment of hepatic encephalopathy.

Rectal Catheters

Recumbent patients with liquid diarrhea pose many nursing challenges in terms of cleanliness, contamination of the environment, and wound infection. Each institution has their own technique of keeping patients and bedding clean, including absorbent products, skin barrier creams, and frequent washing of the rear end. It remains difficult, though, to keep the area clean and to prevent contamination of surgical wounds. These patients also inevitably develop perineal dermatitis and ulceration due to fecal scalding and frequent, abrasive cleaning.

In human medicine, rectal catheters or rectal tubes are used to collect feces over a short-term period [73]. These are wide-bore tubes, with or without an inflatable balloon on the distal end, connected to a collection system similar to a closed urinary collection system. The risks of using these tubes include colonic or rectal ulceration, pressure necrosis of the bowel wall, or obstruction of the tube leading to colonic distension and possibly perforation [73]. People can also develop temporary or permanent anal sphincter incompetence with prolonged use. Rectal tubes are now being replaced with fecal or bowel management systems such as the Flexi-seal Fecal Management System (ConvaTec, NJ), which can be used for up to 29 days [74]. There have been reports of rectal trauma associated with these devices [75–77].

No studies have evaluated the risk of use of rectal tubes in veterinary medicine; however, use of rectal tubes for less than 24 hours likely carries a low risk as it does in human postoperative patients (Box 40.5) [78]. In our clinical experience, rectal Foley catheters with a closed collection system are used routinely for dogs with frequent voluminous diarrhea, without appreciable adverse effects (Protocol 40.6; Figures 40.11 and 40.12). Veterinary-specific fecal management systems have recently become available that are designed specifically for this use, with an open-ended distal tip rather than a side-hole, which urinary catheters have (Fecal Management System, MILA International, Florence, KY).

Rectal Foley catheters are also used in human medicine to remove some rectal foreign bodies, such as bottles or jars [79]. Feeding the balloon past the foreign body and injecting some air through the catheter disrupts the vacuum created around the foreign body. The balloon can then be used to pull the foreign body out of the rectum.

Box 40.5 Rectal Tube Placement

Indications

- Recumbent patients with large-volume diarrhea
- Post-operative patients with liquid stool that are at high risk of infection from fecal contamination

Contraindications

- Rectal bleeding or compromised rectal/colonic mucosa
- Patients with bleeding disorders
- Patients with formed stool
- Use beyond 48 hours

Protocol 40.6 Rectal Tube Placement

1) Perform a digital rectal examination in the patient to ensure there is no obstruction or frank blood suggesting lack of rectal mucosal integrity.
2) Choose a large bore Foley catheter, for example 24–32 Fr for a medium-sized dog.
3) Inflate the balloon with saline to ensure integrity. No more than 10–20 ml should be used to inflate the balloon, depending on the size of the catheter. As a guide, the balloon should be no wider than slightly larger than the anus in the dog. For instance, this would be 3–4 cm in a medium sized dog. The goal is not to prevent the tube coming out but rather to encourage liquid material to flow down the tube rather than around it.
4) Lubricate the catheter and gently insert into the rectum so that the tip lies several centimeters beyond the anal sphincter.
5) Slowly inflate the balloon to the predetermined volume, watching the animal for signs of discomfort. Stop inflating the balloon if the patient shows signs of distress, as this may indicate over-distension or trauma of the rectal wall.
6) Attach the catheter to a closed collection system, such as a urinary closed collection system. Tape the catheter and collection system to the tail or hindlimb of the animal to reduce traction on the catheter (Figures 40.11 and 40.12).
7) The bag should be emptied intermittently, and the eliminated volume recorded to monitor fluid balance.
8) If the catheter becomes repeatedly blocked with solid fecal material, or it repeatedly comes out of the patient, it should be removed. If the catheter is not draining fecal material adequately, then colonic distension will stimulate defecation and therefore the catheter is no longer suitable.
9) The balloon should be deflated, repositioned, and reinflated every 6–12 hours to avoid pressure necrosis of the rectal wall.
10) The catheter should be removed if bleeding becomes evident.

Note: Be aware that if the balloon is residing in the colon, it is possible to palpate the balloon externally within the colon, which may be mistaken for a mass or foreign body.

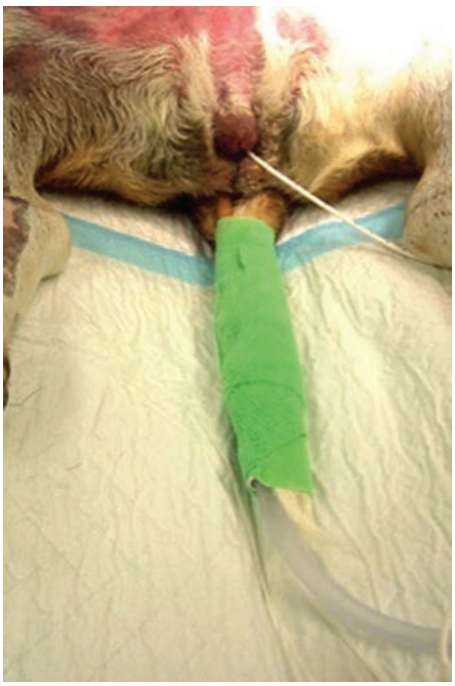


Figure 40.11 A rectal Foley catheter placed in a dog in dorsal recumbency to prevent fecal contamination of the perineal area with liquid feces. The collection tubing has been taped and bandaged (green) to the dog's tail to prevent the catheter migrating out of the anus. The dog also has an indwelling urethral catheter (white).



Figure 40.12 A rectal Foley catheter placed in a dog with watery diarrhea to facilitate cleanliness and estimate fluid losses. The catheter has been secured to the limb due to lack of availability of a tail. *Source:* Courtesy of Claire Sharp.

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41

Postoperative Peritoneal Drainage Techniques

Margo Mehl

There are numerous indications for abdominal drainage, some of which include peritonitis, uroperitoneum, increased abdominal pressure, and peritoneal dialysis. The type of peritoneal drainage chosen by the overseeing clinician depends on the primary disease process. The techniques for postoperative peritoneal drainage discussed in this chapter include maintaining an open abdomen, vacuum-assisted peritoneal drainage, closed-suction abdominal drains, and intermittent drainage using a percutaneous catheter. Several types of passive drains, such as Penrose drains or dialysis catheters, have been described for use in abdominal drainage. These passive drains rely on gravity and are less effective and associated with higher complication rates compared with other abdominal drainage techniques. I do not recommend their use for abdominal drainage.

Open Abdominal Drainage

The underlying cause of contamination must be determined and controlled, the abdominal cavity copiously lavaged, and then open peritoneal drainage techniques can be used [1]. In all patients with open abdominal drainage, it is ideal to have an indwelling urinary catheter to prevent urine soiling the abdominal bandages and to allow for the measurement of urine output (Chapter 35). The technique for open abdominal drainage involves leaving a gap in the midline abdominal wall closure. This is accomplished by loosely suturing the ventral rectus sheath in close proximity while not pulling the suture tight. The span of the gap in the midline abdominal closure depends on patient size and generally ranges from 1 to 4 cm (Figure 41.1a). This space or gap in the abdominal wall closure allows for fluid to drain easily out of the peritoneal cavity. Ideally, the suture chosen is monofilament and suture bites are close together to prevent bowel or organ entrapment and strangulation. A detailed description of the surgical technique for performing open abdominal drainage is outside the scope of this chapter but has been described in several surgical textbooks [1–3].

After the abdominal wall is loosely closed, a sterile non-adherent dressing is applied over the incision. The second layer (Figure 41.1b) is a thicker and more absorbent sterile layer, and the third layer is usually one or more sterile towels. The bandage is then secured in place with a stretchy bandage material that wraps around the patient's body. There are many good material choices for the external bandage layers, some of which include 3M Vetrap®, cling gauze, and elastic adhesive tape, such as Elastikon® (Johnson & Johnson). Additionally, per the clinician's request, a water barrier layer (Figure 41.1c) may be incorporated into the bandage layers before the final layer is placed (Figure 41.1d). The bandage should extend far enough cranially and caudally to ensure coverage of the incision even in the case of bandage migration. The abdominal bandage should be changed once or twice a day depending on fluid production and strikethrough of the bandage. The patient with open peritoneal drainage does not require an increased level of analgesics. Certainly, most patients managed with open peritoneal drainage have significant systemic disease and have recently undergone a laparotomy; these conditions are indications for pain medications, but the technique of leaving the abdominal wall slightly open does not increase the patient's level of pain.

Changing the Abdominal Bandages

In a patient with open abdominal drainage, the bandages should be monitored for strikethrough and changed once or twice in 24 hours. The bandages should be weighed before and after removal to estimate continuing fluid loss, which should be considered when calculating a patient's fluid

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Figure 41.1 (a) Open abdominal drainage. Note the gap in the midline abdominal wall closure. This is accomplished by loosely suturing the ventral rectus sheath in close proximity. The span of the gap in the midline abdominal closure generally in the range 1–4 cm. A sterile, nonadherent dressing is applied over the open incision (not shown). (b) The second layer, a thicker and more absorbent sterile layer (laparotomy pads) is applied. To secure the pads, sutures are preplaced along the full length on both sides of the incision. The pads are held in place with umbilical tape tied through the stay sutures. (c) An infant diaper is used as a water barrier layer and is incorporated into the bandage layers before the final layer is placed. (d) An elastic bandage wrap is applied as the final layer.

requirement. When weighing the bandages and converting weight into volume, the bandages should first be weighed prior to placement and then when removed the bandages should be reweighed. The weight of the wet bandages minus the weight of the dry bandage will give you the fluid weight, though some volume may be lost by evaporation if the bandage is not waterproof. One gram of fluid is equivalent to 1 ml in volume, and this quantity should be recorded as a fluid loss in the patient's daily record. Changing the bandages involves the following steps (Protocol 41.1).

First, the patient often requires sedation, the choice of which depends on its cardiovascular status. Second, any person handling the bandages should perform hand hygiene, wear sterile gloves, and use aseptic technique. The patient may have the bandage changed either while in a standing position with support or while in dorsal or lateral recumbency. Each layer is carefully removed, and then the midline incision is visually evaluated to make sure no bowel loop or intra-abdominal structure is outside the peritoneal cavity. Then the bandage is replaced in the same order as previously described: (i) sterile nonadherent bandage material over the incision; (ii) sterile, thicker more absorbent layer; (iii) sterile surgery towels; and (iv) outer nonsterile layers including gauze and dry cast padding and elastic tape to secure the bandage around the patient. Some clinicians advocate including a water-impermeable barrier in the bandage (potty training pads or infant diapers), which would decrease contamination wicking from the exterior into the peritoneal cavity. Ideally, the bandage is changed before strikethrough occurs; however, if the situation arises in which the bandage cannot be changed that often, then a water-impermeable barrier incorporated

Protocol 41.1 Open Abdominal Drainage: Changing the Bandages

Items Required

- Sterile gloves
- Sterile nonadherent bandage material
- Sterile absorbent layer
- Sterile surgery towels
- Outer nonsterile gauze (dry cast padding)
- Elastic tape
- Optional: water-impermeable barrier in the external layers of bandage (drapes, potty training pads, or infant diapers)

Procedure

1) Sedate and position the patient.
2) Perform hand hygiene.
3) Don sterile gloves using aseptic technique.
4) Carefully remove bandages and weigh them.
5) Visually evaluate midline incision to make sure no bowel loop or intra-abdominal structure is outside the peritoneal cavity.
6) Place sterile nonadherent bandage material over the incision.
7) Place second layer of sterile, thicker, absorbent bandage material such as laparotomy pads.
8) Place sterile surgery towels.
9) Incorporate water impermeable layer as needed.
10) Use outer nonsterile layers to secure bandage to the patient using stay sutures and umbilical tape as a tie-over bandage, followed by a water-resistant layer if desired.

into the bandaging layers will assist in preventing outer contamination wicking from the environment into the patient's peritoneal cavity.

When the volume of fluid produced within the abdominal cavity has significantly decreased and the cytologic characteristics of that fluid indicate the abdominal cavity is free of infection (i.e. non-degenerate neutrophils, lack of intracellular bacteria), the overseeing clinician will recommend closure of the abdominal wall.

The patient undergoes an anesthetic procedure for closure of the abdominal incision. At the time of closure, I recommend re-exploring the abdominal cavity, copiously lavaging the abdominal cavity, and obtaining aerobic and anaerobic cultures.

Complications of open abdominal drainage predominantly include hospital-acquired infections and loss of plasma proteins [4, 5]. The risk of hospital-acquired infections can be reduced if aseptic techniques are always instituted for bandage changes and during abdominal wall closure. The clinician should expect loss of plasma proteins such as albumin that maintain oncotic pressure; fluid therapy that supports oncotic pressure should be used as needed. Additionally, patients with continuing protein loss may benefit from enteral feeding and thus may have an esophageal or nasogastric feeding tube in place (Chapter 44).

Vacuum-Assisted Drainage

The technique of vacuum-assisted peritoneal drainage begins in a similar manner to the open peritoneal drainage, in which after the abdominal cavity is explored, a portion of the linea alba is closed with a 1–6 cm gap between the edges. The region of the linea alba with the gap in closure is covered with a foam sponge connected to fenestrated evacuation tubing and covered with an adhesive draping material. The evacuation tubing is connected to a vacuum pump to provide continuous negative pressure. Bandage changes or changing the foam is typically done every 24–48 hours, which is less frequently than that with open abdominal drainage. After peritoneal drainage is complete, the patient requires general anesthesia for removal of the vacuum-assisted drainage system and abdominal wall closure. As with open abdominal drainage techniques, we recommend re-exploring the abdomen at the time of closing the abdominal cavity and obtaining cultures. Complications with this technique are similar to those seen with open abdominal drainage, including hypoproteinemia and bacterial peritonitis [6].

Active Closed-Suction Drains

The use of closed-suction drains allows the clinician to close the abdominal cavity and may be associated with less protein loss than open abdominal drainage. Closed-suction drains may still be associated with hospital-acquired infections, although potentially to a lesser degree than with open peritoneal drainage [7]. The disadvantage of the closed-suction drainage technique is it does not allow the clinician to re-explore or re-evaluate the abdominal cavity. Closed-suction drains permit measurement of fluid production from the peritoneal cavity and also allow for cytologic evaluation. The presence of a drain in a body cavity will stimulate fluid production; therefore, waiting for cessation of fluid production before drain removal is unnecessary. The time at which to remove the abdominal drains is guided

by when the cytology demonstrates the abdominal cavity is free of infection and when the fluid production has decreased to less than 2.2–5 ml/kg/day [8, 9].

A number of closed-suction drains are available. All these drains aim to increase the surface area for drainage and reduce the incidence of drain obstruction by omentum, other tissue, or cellular debris. Closed-suction drains can be placed in a closed manner (see later), but I advocate placing the drains surgically. Placing the drains surgically allows for evaluation of the abdominal cavity and treatment of the primary disease. Also, closed placement of abdominal drains can cause damage to intra-abdominal organs. After the abdominal cavity has been explored and the cause of contamination controlled, the drains are placed away from the midline incision, either laterally or cranial or caudal to the incision. The drains should never exit through the midline abdominal incision. The size of the drain and the number of drains placed is determined by the weight of the patient. I advocate using one to two drains in a cat or small dog (<10 kg) and two to four drains in larger patients. Once the drains are placed and exiting through the abdominal cavity, they are secured with an anchoring suture and a finger trap pattern (Figure 41.2) up the tubing of the drain, not so tight as to occlude the drain. Once the abdominal cavity is closed, the drain tubing should be attached to a vacuum-assisted chamber that has an anti-reflux valve, to collect fluid and reduce the risk of ascending infection (Figure 41.3). In the absence of a commercial product, an active drainage system can be constructed by connecting the intra-abdominal drain to an extension set with a needle attached, or a butterfly needle, which is then inserted into a large vacuum blood collection tube to generate negative drain pressure. This technique is further described elsewhere [10]. Once drains are secured in place, the drain exit holes are covered with a sterile bandage and an abdominal bandage to reduce hospital contamination.



Figure 41.2 Once the drains are placed and exiting through the abdominal cavity, they are secured with an anchoring suture pattern, such as a finger trap pattern.



Figure 41.3 Jackson-Pratt drains, a closed drainage system, used in a cat following abdominal surgery.

Managing Closed-Suction Drains

The vacuum created by the reservoir does not pull as effectively once the drain reservoir expands with fluid. Therefore, it is recommended to empty the drain reservoir when it is full two-thirds or more. Wear examination gloves when handling the drains. The reservoir bulb is disconnected from the drain tubing, and both openings/surfaces are wiped with isopropyl alcohol prior to collecting or emptying fluid. All fluid collected is measured and cytology is performed every 24–48 hours. The drain exit sites from the abdominal cavity should be evaluated for infection or fluid accumulation and addressed as indicated. These drain exit sites should also be cleaned daily with an antimicrobial scrub, wiped with sterile saline, and rebandaged with new clean dressings (Protocol 41.2). As previously stated, all drains incite production of some amount of fluid; therefore, the drains are removed when the patient's clinical status improves, the fluid production decreases to an acceptable level, and the cytology of the collected fluid shows no evidence of infection. The patient does not often require additional analgesics for drain removal. The drains are gently pulled from the abdominal cavity and the exit holes are evaluated; if no evidence of infection is seen, the holes can be covered with a sterile dressing and allowed to heal by second intention.

Percutaneous Catheter Drainage

Abdominal drainage can be achieved by percutaneous drain placement. This technique can be used to assist with abdominal fluid collection, rapid decompression of large

Protocol 41.2 Closed-Suction Drain Management

Items Required

- Closed-suction drain (Jackson-Pratt) in place
- Examination gloves
- Alcohol-soaked gauze pad
- Laboratory specimen tubes and culturette (optional)
- Graduated cylinder
- Surgical scrub
- Sterile saline
- Materials for light dressing

Procedure

1) Gather materials.
2) Empty drain reservoir when the bulb is no more than two-thirds full.
3) Perform hand hygiene and don examination gloves prior to handling the drains.
4) Disconnect the reservoir bulb from drain tubing and wipe both openings with isopropyl alcohol prior to emptying or collecting fluid.
5) Measure all fluid collected; cytology may be performed.
6) Evaluate drain exit sites from the abdominal cavity; notify clinician of excessive redness, heat, swelling, apparent pain, or discharge from insertion site(s).
7) Clean drain exit sites daily with an antimicrobial scrub, wipe with sterile saline, and keep bandaged with light, clean dressings.
8) When closed suction drains are no longer needed, drains can be removed by gently pulling drain tubing from the abdominal cavity.
9) Once drains are removed, evaluate exit holes and apply dressings as needed.

abdominal effusions, or to stabilize a patient prior to surgical intervention such as in the case of a uroabdomen. Peritoneal dialysis catheters can also be placed in this manner. This therapy is described in Chapter 36. Percutaneous catheter drainage is not indicated for treatment of septic peritonitis because continuing contamination of the abdomen would not be controlled. There are a number of catheters available on the market that could be placed in a closed manner or via a mini laparotomy. A fenestrated catheter is likely to be more effective than a non-fenestrated catheter because it is less likely to become occluded by omentum or other tissue. There are commercially available fenestrated catheters (Cook peritoneal dialysis catheters), or alternatively a regular over-the-needle catheter or red rubber tube can be fenestrated for this purpose. It is essential to be very careful when manually fenestrating these catheters to avoid weakening the integrity of the catheter and to maintain sterility. Fenestrations are made with a scalpel blade and should be staggered and each hole less than the diameter of the catheter.

A closed method of placement can be used with any over-the-needle type catheter, and local anesthetic can be infused into the site prior to placement. The catheter is placed through the abdominal wall, with careful technique to avoid damage to intra-abdominal structures. The site of placement is best identified with ultrasound guidance. In the absence of ultrasound, the catheter is usually placed caudal to the umbilicus in an effort to minimize the chance of puncturing the liver or spleen. A mini-laparotomy technique can also be used for placement of catheters, and it allows use of catheters without a trocar, such as a red rubber tube. The mini-laparotomy technique requires strict asepsis, and a small incision is made on the ventral midline. Through the skin incision the linea alba is accessed, and a stab incision is made through a tented portion of the linea alba. The catheter is introduced through a separate skin opening and associated subcutaneous tunnel and then passed into the previously made stab incision through the linea alba. The small midline approach incision is closed, and then the skin incision is closed in a routine manner. The catheter is secured with a pursestring suture and finger trap suture pattern, making sure it is not occluding the catheter lumen. Lastly, a sterile dressing is applied over the catheter exit site.

Managing a Fenestrated Catheter

The management of a percutaneously placed catheter is similar to those techniques previously described. Intermittent or continuous drainage can be used. The person handling the catheter should always wear examination gloves and use aseptic techniques. Wipe the opening to the catheter with isopropyl alcohol before aspirating and use sterile syringes and tubing.

Conclusion

Managing peritoneal drainage is a key step in treating patients with abdominal disease. Effective nursing care with abdominal drainage techniques will maximize the therapeutic benefits of abdominal drainage and minimize complications.

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Section Five

Nutrition

42

Nutritional Requirements in Critical Illness

Daniel L. Chan

Basic Physiology of Malnutrition in Critical Illness

Appropriate nutritional support has long been considered essential for the recovery of critically ill people. Although there is convincing evidence of the deleterious effects of malnutrition in people, the optimal nutritional strategies for critically ill animals remain largely unknown. Despite the lack of definitive answers, recommendations for nutritional support of critically ill animals are based on having a good understanding about the disease processes involved and the limited information available. The fact that there is limited information available should not discourage the implementation of nutritional support for critically ill animals. In fact, with proper patient selection, sound nutritional planning, and careful monitoring, nutritional support can be an integral part in the successful recovery of many critically ill animals.

The metabolic responses to illness or severe injury are complex and place critically ill animals at high risk for malnutrition and its deleterious effects. These effects, which are likely to have a negative impact on overall survival, include alterations in energy and substrate metabolism, compromised immune function, and impaired wound healing [1–3]. Although generalizations tend to oversimplify these complex systems, the concept of "ebb and flow" offers a basic description of the metabolic response to critical illness or severe injury. According to this model, there is an initial hypometabolic response ("ebb phase"), followed by a period of a more prolonged course of hypermetabolism ("flow phase"). The ebb phase refers to a period of hemodynamic instability associated with decreased energy expenditure, hypothermia, mild protein catabolism, decreased cardiac output, and poor tissue perfusion. Without intervention, this instability may progress to a state of refractory or irreversible shock characterized by severe lactic acidosis, decreased tissue perfusion, multiple organ failure, and death. Nutritional intervention during the ebb phase is associated with a high risk for complications such as electrolyte abnormalities, which may result in further detrimental effects to some critically ill animals [4–7]. Following successful resuscitation, patients enter the flow phase, during which profound metabolic alterations occur. Increases in energy expenditure, glucose production, insulin and glucagon concentrations, cardiac output, and profound protein catabolism are the hallmarks of this response [1, 4, 8]. Provision of nutritional support during this stage of illness can attenuate and sometimes reverse the detrimental effects of malnutrition [9].

One of the major metabolic alterations associated with critical illness involves body protein catabolism, in which protein turnover rates may become markedly elevated. Whereas healthy animals primarily lose fat when deprived of sufficient calories (simple starvation), sick or traumatized patients catabolize lean body mass when they are not provided with sufficient calories (stressed starvation) [8, 9]. During the initial stages of fasting in the healthy state, glycogen stores are used as the primary source of energy. Within days, a metabolic shift occurs toward the preferential use of fat deposits, sparing catabolic effects on lean muscle tissue.

In diseased states, the inflammatory response triggers alterations in cytokines and hormone concentrations, which rapidly shift metabolism toward a catabolic state. Glycogen stores are quickly depleted, especially in strict carnivores such as the cat, and this leads to an early mobilization of amino acids from muscle stores. Because cats undergo continuous gluconeogenesis, the mobilization of amino acids from muscles is more pronounced than that observed in other species. In both cats and dogs, continued

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inadequate food intake leads to further changes in metabolism where the predominant energy source is derived from accelerated proteolysis, which in itself is an energy-consuming process. Muscle catabolism that occurs during stress provides the liver with glucogenic precursors and other amino acids for glucose and acute-phase protein production [8, 9]. The resultant negative nitrogen balance or net protein loss has been documented in critically ill dogs and cats [10, 11]. One study in particular estimated that 73% of hospitalized dogs across four different veterinary referral centers were deemed to be in a negative energy balance [12]. A more recent study documented that 84% of hospitalized dogs in a veterinary teaching hospital consumed less that 25% of their daily resting energy requirements (RER) [13]. The consequences of continued lean body mass losses include negative effects on wound healing, immune function, strength (both skeletal and respiratory), and ultimately on overall prognosis [9, 13, 14].

It is becoming increasingly evident that the metabolic consequences of critical illness are part of an integrated response to the initial insult, driven in part by cytokines and counterregulatory hormones [6, 8]. These mediators may result in changes in energy expenditure, substrate metabolism, and body composition. The most well-known cytokine to affect metabolism is perhaps tumor necrosis factor-α, which is responsible for increased energy expenditure, hepatic gluconeogenesis, protein breakdown, activation of the hypothalamic–pituitary–adrenal axis, lipolysis, and peripheral insulin resistance [6].

Owing to the metabolic alterations associated with critical illness, and in part to an inability or reluctance of many critically ill dogs and cats to ingest sufficient calories, this patient population is at increased risk for rapid development of malnutrition. Given the serious sequelae of malnutrition, preservation of lean body mass or reversal of deteriorating nutritional status via nutritional support is paramount.

Rationale for Providing Nutrition in Critical Illness

Identification of overt malnutrition in animals can be challenging because there are no established criteria of malnutrition in companion animals. In dogs, a period as short as three days of anorexia has been documented to produce metabolic changes consistent with starvation in people [15]. In cats, detectable impairment of immune function can be demonstrated in healthy individuals subjected to acute starvation by day four, and therefore, nutritional support should be also considered in any ill cat with inadequate food intake for more than three days [16]. The need to implement a nutritional intervention becomes more urgent when a dog or cat has not eaten for more than five days because by this time there are detectable changes in metabolism and immune function directly related to the prolonged period of poor food intake [15, 16].

However, even in patients with severe malnutrition, the immediate therapy of critically ill patients should focus on resuscitation, stabilization, and identification of the primary disease process. As steps are made to address the primary disease, formulation of a nutritional plan should strive to prevent (or correct) overt nutritional deficiencies and imbalances. By providing adequate energy substrates, protein, essential fatty acids, and micronutrients, the body can support wound healing, immune function, and tissue repair. A major goal of nutritional support is to minimize metabolic derangements and catabolism of lean body tissue. During hospitalization, repletion of body weight is not the top priority because regaining body weight is best achieved once the animal has recovered from the primary disease. Therefore, there is little justification to feed excessive calories to critically ill animals.

Nutritional Assessment

The formal assessment of patients in terms of their nutritional needs and what precautions need to be considered in formulating a plan is referred to as "nutritional assessment." Because all nutritional support techniques carry some risk of complications, appropriate patient selection is crucial in ensuring the full benefits of nutritional support. Careful patient selection is the first step in performing nutritional assessment. This subjective clinical assessment process remains the predominant method of identifying malnourished patients that require immediate nutritional support as well as identifying patients that are not currently malnourished but where nutritional support is aimed at preventing the development of malnutrition [17–19]. The next step in nutritional assessment involves assessing how urgently nutritional support is required. This involves consideration of various risk factors for developing malnutrition including periods with inadequate food intake, unintended weight loss, loss of body condition, evidence of muscle wasting, presence of gastrointestinal dysfunction, hypoalbuminemia, and estimation of when the patient may recover [18, 19]. Table 42.1 outlines a nutritional assessment scheme to help to identify patients in need of immediate nutritional support.

Nutritional assessment also allows identification of factors that can have an impact on the nutritional plan, such as electrolyte abnormalities, hyperglycemia, hypertriglyceridemia, hyperammonemia, or comorbid illnesses (e.g. kidney or liver disease) that should lead to alterations of the composition of the diet used. In summary, nutritional

Table 42.1 Process for identifying patients in need of urgent nutritional support

	Risk		
Parameter	**Low**	**Moderate**	**High**
Food intake:			
< 80% RER for < 3 days	✓		
< 80% RER for 3–5 days		✓	
< 80% RER for > 5 days			✓
Presence of weight loss		✓	
Severe vomiting/diarrhea			✓
Body condition score < 4/9			✓
Muscle condition score of moderate to severe			✓
Hypoalbuminemia		✓	
Expected course of illness:			
< 3 days	✓		
2–3 days		✓	
> 3 days			✓

A patient with two or more high-risk factors should receive nutritional support as soon as they are stabilized. Patients with fewer than two high-risk factors should be closely monitored and reassessed every few days.

assessment involves performing a risk assessment of a patient in terms of its nutritional status and the considerations required in providing safe and effective nutritional support.

Nutritional Plan

The key to successful nutritional management of critically ill patients lies in the proper diagnosis and treatment of the underlying disease. Another crucial factor is the selection of the appropriate route for nutritional support. Providing nutrition via a functional digestive system is the preferred route of feeding, and so particular care should be taken to evaluate whether the patient can tolerate enteral feedings. Even if the patient can only tolerate small amounts of enteral nutrition, this route of feeding should be pursued and supplemented with parenteral nutrition as necessary to meet the patient's nutritional needs. In some circumstances feeding patients enterally may seem to be contraindicated (e.g. animal anesthetized for mechanical ventilation, animals with upper airway dysfunction); however, there is a strong argument that in such a situation there should be some consideration for feeding enterally as long as the compromised area is avoided. Ventilated human patients are commonly fed via nasogastric tubes, and it would be reasonable to consider a similar approach in companion animals receiving mechanical ventilation. Patients with severe laryngeal or esophageal dysfunction could be fed via gastrostomy or enteric feeding tubes. On the basis of the nutritional assessment, the anticipated duration of nutritional support, and the appropriate route of delivery (i.e. enteral or parenteral), a nutritional plan is formulated to meet the patient's nutritional needs.

The first steps of instituting nutritional support include achieving hemodynamic stability, restoring proper hydration status, and correction of electrolyte or acid–base disturbances [18]. Beginning nutritional support before these abnormalities are addressed can increase the risk of complications (e.g. regurgitation, vomiting, hypotension) and, in some cases, further compromise the patient [4–7]. For example, feeding leads to mesenteric vasodilation, which could compromise systematic mean arterial pressure in some patients. It should be emphasized that this strategy is *not* counter to the concept of "early nutritional support," which has been documented to result in positive effects in several animal and human studies [20–24]. Early nutritional support advocates feeding as soon as possible after achieving hemodynamic stability rather than delaying nutritional intervention for several days [21, 22]. Implementation of the nutritional plan should be gradual, with the goal of reaching target level of nutrient delivery in 48–72 hours.

Rapid feeding has several potential complications. First, animals that have not eaten for several days have significant delays in gastric emptying and compromised overall intestinal motility. In some patients, this situation leads to significant ileus. Feeding these patients rapidly leads to abdominal pain, distension, and potentially vomiting and regurgitation. Second, because of various hormonal disturbances associated with poor food intake (e.g. low insulin, high glucagon, high cortisol), there is a potential for severe metabolic derangements such as refeeding syndrome whereby a sudden spike in insulin concentrations leads to life-threatening electrolyte abnormalities.

Nutritional Requirements

Whereas the protein requirements of critically ill people have been determined based on nitrogen balance studies, this information is not readily available in critically ill animals. One method of estimating the extent of amino acid catabolism is to measure urinary urea nitrogen content. Although measurement of urinary urea nitrogen in critically ill dogs has been shown to be a feasible tool in assessing nitrogen balance in an experimental setting, further studies are warranted to better characterize the protein requirements of critically ill animals seen in practice

[10, 11]. A study [25] demonstrated significant changes in amino acid status in critically ill dogs; however, further studies are required to determine whether correction of these amino acid changes is beneficial.

Currently, it is generally accepted that hospitalized dogs should be supported with 4–6 g protein/100 kcal (15–25% of total energy requirements); cats are usually supported with 6–8 g protein/100 kcal (25–35% of total energy requirements) [26]. Patients with protein intolerance (e.g. hepatic encephalopathy, severe azotemia) should receive reduced amounts of protein. Similarly, patients with hyperglycemia or hyperlipidemia may require decreased amounts of these nutrients. Other nutritional requirements depend on the patient's underlying disease, clinical signs, and laboratory parameters.

When an animal is unable to synthesize adequate amounts of a nutrient and must rely on dietary sources, that nutrient is qualified as *essential*. However, during certain conditions including critical illness, nutrients usually considered nonessential become in short supply due to increased demands. These nutrients have been termed *conditionally essential*, and glutamine is one such example. A number of recent human studies have evaluated the modulation of disease with these so-called immuno-modulating nutrients such as glutamine, arginine, omega-3 fatty acids, and nucleotides [7, 10, 27–31]. Although study results have been mixed, glutamine supplementation has been associated with the most beneficial results [7, 27, 28, 30–32].

Glutamine is the primary energy source for enterocytes and cells of the immune system, and its supplementation may attenuate gastrointestinal permeability and improve overall immune function [28–31]. In select populations of critically ill people, supplementation with either enteral or parenteral glutamine has been shown to reduce infectious complications and improve survival [30, 31]. Studies in dogs and cats have failed to demonstrate clear benefits of glutamine supplementation [32, 33]. Nevertheless, it is increasingly evident that the requirements of specific nutrients during critical illness may be considerably different than those in health. Future studies are warranted to evaluate whether "critical care diets" designed for dogs and cats should be enriched with these conditionally essential nutrients.

Calculation of Nutritional Requirements

Ideally, nutritional support should provide ample substrates for gluconeogenesis, protein synthesis, and adenosine triphosphate (ATP) production necessary to maintain homeostasis. Ensuring that enough calories are being provided to sustain critical physiologic processes such as immune function, wound repair, and cell division and growth would necessitate the actual measurement of the patient's total energy expenditure. However, precise measurements of energy expenditure (i.e. calorimetry) in clinical veterinary patients are still in the developmental phases. The basic premise of calorimetry is to measure the total heat lost by an animal, as a reflection of total energy produced by metabolism. With direct calorimetry, the animal is placed in an airtight insulated chamber, and precise thermal measurements are made of the chamber. This method is only suitable for experimental models because clinical cases would not be able to be managed in this environment. Indirect calorimetry, in contrast, is more commonly used in human hospitals and by veterinary clinical researchers to extrapolate energy requirements. This method provides relatively noninvasive means of estimating energy expenditure by measuring the rate of oxygen consumption and the rate of carbon dioxide production and applying the obtained values to a mathematical equation known as the Weir formula [34, 35]. Because consumption of oxygen and production of carbon dioxide can directly be related to glucose, protein, and fat metabolism, energy expenditure can be calculated from the measured variables. However, indirect calorimetry also requires specialized equipment, so-called metabolic carts, making the technique available only in a few select sites. Oxygen and carbon dioxide exchange is measured with a hood, canopy, or expiratory collection device. These systems are portable and easier to use in clinical situations than previous calorimetry units where the patient needed to be confined within the device. A few studies have used indirect calorimetry to estimate energy expenditure in select populations of clinical veterinary patients, but the use of mathematical formulas currently remains the most practical means of estimating a patient's energy requirement (Box 42.1) [35, 36].

Results of indirect calorimetry studies in dogs support the recent trend of formulating nutritional support to meet RER as a starting point, rather than more generous illness energy requirements, which require multiplying resting or even maintenance energy requirements by an illness

Box 42.1 Estimating Total Daily Energy Requirements in Dogs and Cats

$$\text{RER}(\text{kcal/day}) = 70 \times (\text{current body weight in kg})^{0.75}$$

For animals weighing 2–20 kg, the following linear formula may be used as an estimate:

$$\text{RER}(\text{kcal/day}) = (30 \times \text{current body weight in kg}) + 70$$

RER, resting energy requirement.

factor [37]. RER is defined as the number of calories required for maintaining homeostasis at rest in a thermoneutral environment while the animal is in a postabsorptive state [38, 39]. Although several formulas are proposed to calculate the RER, a widely used allometric formula can be applied to both dogs and cats of all weights. For animals weighing between 2 and 20 kg, there is also a linear formula that provides reasonable estimation of RER (Box 42.1).

Until recently, it was recommended to multiply the RER by an illness factor between 1.0 and 2.0 to yield an illness energy requirement to account for increases in metabolism associated with different diseases and injuries. However, less emphasis is now being placed on such subjective and extrapolated factors, and the current recommendation is to use more conservative energy estimates (i.e. start with the animal's RER) to avoid overfeeding. Overfeeding can result in metabolic and gastrointestinal complications, hepatic dysfunction, and increased carbon dioxide production [4, 7].

It should be emphasized that these general guidelines should be used as starting points, and animals receiving nutritional support should be closely monitored for tolerance of nutritional interventions (see the section on complications of providing nutrition in the critically ill). Continual decline in body weight or body condition should prompt the clinician to reassess and perhaps modify the nutritional plan (e.g. increasing the number of calories provided by 25%).

Nutritional Requirements in Special Cases

Much remains unclear regarding the nutritional requirements of critically ill animals in general. In certain circumstances assumptions are made that nutritional requirements in animals are similar to people afflicted with similar diseases. However, it is important to recognize that there may be significant species and disease differences that make direct comparisons or extrapolations less applicable. For example, pancreatitis in people is often related to gallstones or alcoholism. Because pancreatitis in animals is often related to high-fat diets or is idiopathic, the nutritional requirements for treatment of this disease in each species are likely different.

Burns

Experimental data suggest dramatic changes in energy requirements in animals with thermal burns; however, there is a paucity of clinical data in animals in this regard [40]. In experimental models, dogs with thermal burns experienced increased energy requirements, accelerated gluconeogenesis, glucose oxidation, lipolysis, and increased amino acid oxidation [39, 41]. In light of limited clinical data, current recommendations are to start nutritional support as soon as it is deemed safe and initially target RER, and then to continually reassess the patient as energy requirements are likely to exceed three times the RER. The goal of aggressive nutritional support is to optimize protein synthesis and preserve lean body mass. Feeding at least 6–8 g protein/100 kcal (25–35% of total energy) may be necessary, even in dogs. It is unknown whether nutrients such as glutamine and arginine would provide extra benefits in this patient population.

Tetanus

Another population that may merit more vigilant attention to energy requirements is dogs with tetanus. A recent study demonstrated that despite feeding a median of 1.4 times RER, dogs lost a median of 5% body weight during the course of hospitalization [42]. It is proposed that increased muscle activity in this disorder increased energy requirements despite the fact that dogs were mostly recumbent.

Sepsis

Animals with sepsis are perhaps another population in which nutritional requirements may be altered. The intense inflammatory response, coupled with changes in substrate handling, likely alters the metabolic rate and nutrient requirements. Experimental data in dogs suggest that during the early phase of sepsis, energy expenditure may increase by 25%, which appears to be accompanied by an increase in oxidation of free fatty acids and triglycerides [43]. However, it is also recognized that energy expenditure can be quite variable in sepsis and may even decrease in septic shock [44]. Depending on the etiology of sepsis (e.g. septic peritonitis, pyothorax), protein requirements may also dramatically increase, and therefore general nutritional recommendations for animals with septic peritonitis may involve initially feeding at RER with 35% of total calories derived from protein, 40% from fats, and 25% from carbohydrates [26]. Further studies are warranted to determine whether these recommendations are optimal for clinical veterinary patients with sepsis.

Complications of Providing Nutrition in the Critically Ill

Body weights should be monitored daily in critically ill animals. However, the clinician should consider fluid shifts in evaluating changes in body weight. For this reason, assessing body condition scores is very important.

The use of the RER as the patient's caloric requirement is merely a starting point; however, there is growing evidence that this conservative approach or even feeding less than RER may be preferable in the critically ill patient [45]. The number of calories provided may need to be increased to keep up with the patient's changing needs, typically by 25% if well tolerated. In patients unable to tolerate the prescribed amounts (e.g. start to vomit), the clinician should consider reducing volumes of enteral feedings and supplementing the nutritional plan with some form of parenteral nutrition.

Possible complications of enteral nutrition include mechanical complications such as clogging of the feeding tube or premature tube removal by the patient. Metabolic complications of enteral or parenteral feeding include electrolyte disturbances, hyperglycemia, fluid overload, and gastrointestinal signs (e.g. vomiting, diarrhea, cramping, bloating).

In rare instances, a condition known as refeeding syndrome may develop [46, 47]. This syndrome occurs when severely malnourished patients (particularly cats) are fed perhaps too aggressively, and the sudden increase in insulin concentrations results in severe hypophosphatemia and hypokalemia [46]. During prolonged fasting, cells become metabolic inactive, and sudden reintroduction of substrates leads to rapid synthesis of ATP, leading to consumption of phosphate, which causes severe hypophosphatemia. Other electrolytes such as potassium and magnesium also translocate intracellularly in response to insulin [46]. More recently, the development of hyperglycemia has also been documented in cats with refeeding syndrome, though the mechanism of glucose dysregulation remains unknown [47]. Recommendations for reducing the risk for the developing refeeding syndrome include gradually introducing feeding, limiting the proportion of calories derived from carbohydrates, and preemptively supplementing patients with phosphorus, potassium, magnesium, and thiamine [46, 47].

In critically ill patients receiving enteral nutritional support, the clinician must also be vigilant for the development of aspiration pneumonia. Monitoring parameters recommended for patients receiving enteral nutrition include daily checks of body weight, serum electrolytes, feeding tube patency, the appearance of the feeding tube stoma site, gastrointestinal signs (e.g. vomiting, regurgitation, diarrhea), and signs of volume overload or aspiration pneumonia.

Possible complications associated with parenteral nutrition include sepsis (such as seen with catheter infections, contamination of the parenteral solution bag), mechanical complications associated with the catheter and lines (obstructed catheters, line breakage), thrombophlebitis, and metabolic disturbances related to the composition of the parenteral nutrition solution (hyperglycemia, electrolyte shifts, hyperammonemia, and hypertriglyceridemia). Avoiding serious consequences of complications associated with parenteral nutrition requires early identification of problems and prompt action. Frequent monitoring of vital signs, catheter exit sites, and routine biochemistry panels may alert the clinician to developing problems. The evolution of persistent hyperglycemia during nutritional support may require adjustment to the nutritional plan (e.g. decreasing dextrose content in parenteral nutrition) or administration of regular insulin. This obviously necessitates more vigilant monitoring.

With continual reassessment, the clinician can determine when to transition the patient from assisted feeding to voluntary consumption of food. The discontinuation of nutritional support should only begin when the patient can consume approximately its RER without much coaxing.

Summary

- Nutritional support of the critically ill patient is an essential part of the overall treatment plan.
- Metabolic responses to illness or severe injury place critically ill patients at high risk for development of malnutrition.
- Consequences of malnutrition include altered substrate metabolism, compromised immune function, impaired wound healing, and potentially increased mortality.
- Energy expenditure in critically ill animals may vary considerably depending on the patient, underlying disease, and illness severity; therefore, initial nutritional support should target RER.
- Specific nutritional requirements for critically ill dogs and cats have not been determined, but recommended levels of protein provision include feeding 4–8 g protein/100 kcal or 15–35% of total calories derived from protein.
- Before implementation of nutritional support, patients must be cardiovascularly stable and have hydration, acid–base, and electrolyte abnormalities addressed first.
- Monitoring of patients receiving nutritional support is extremely important because this population is prone to various metabolic complications.
- Upon reassessment, nutritional support may be increased, decreased, or discontinued depending on patient response and disease progression.
- With appropriate patient selection, accurate nutritional assessment, and careful execution of the nutritional plan, nutrition can play an instrumental role in the successful recovery of many critically ill patients.

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Enteral Diets for Critically Ill Patients

Sally C. Perea

When selecting an enteral diet, there are several factors to consider, including route of delivery, nutritional status, and underlying disease. Because veterinary specific enteral formulas are limited, other options such as blended canned slurries, home-cooked formulations, and enteral products designed for people can be used to help tailor the diet to the patient's individual needs.

Nutrient Considerations for Critical Care Diets

Energy

One of the primary goals of nutrition support in critically ill patients is to provide energy to limit the loss of lean body mass and to sustain critical physiologic processes such as immune function and wound healing. Because individual energy needs vary, the most accurate method of determining energy expenditure in hospitalized patients is by indirect calorimetry [1]. However, indirect calorimetry is costly and unavailable in most veterinary hospitals; therefore, calculated requirements remain the most practical tool to estimate energy needs. During hospitalization, patients' energy needs are estimated to be equivalent to their calculated resting energy requirement (RER). Multiple RER equations have been recommended, but exponential equations (which estimate the patient's metabolically active body mass from its body weight in kg) are the most accurate, such as:

$$\text{RER} = 70 \times \left(\text{body weight in kg}\right)^{0.75}$$

The use of illness energy factors is no longer recommended, as multiplying RER by these factors generally results in overestimation of true energy needs in hospitalized patients and may lead to complications associated with overfeeding [2].

Protein

Like energy needs, protein needs in critically ill patients should be focused on minimizing muscle catabolism and maintaining lean body mass. A healthy animal under conditions of starvation adapts by decreasing muscle breakdown and converting to the use of fatty acids and ketones for energy. However, in critically ill patients, muscle catabolism is not appropriately downregulated, and elevations in endogenous corticosteroids, catecholamines, and inflammatory cytokines promote a hypercatabolic state [3]. In critically ill dogs, urinary nitrogen excretion has been shown to be two to six times the obligatory nitrogen excretion reported in healthy dogs, demonstrating the significant protein catabolism occurring in these patients [4]. Furthermore, the amino acids generated from muscle breakdown are primarily used for gluconeogenesis and production of acute-phase proteins, whereas synthesis of other selected proteins (such as albumin, transferrin, prealbumin, retinol-binding protein, and fibronectin) is decreased [5].

Common adult maintenance dog and cat foods provide 20–25% and 30–35% protein on a metabolizable energy (ME) basis, respectively. For the critical care patient, a protein level on the higher end of this range is commonly recommended; however, the appropriate level of dietary protein also depends on individual patient's needs and underlying disease. For example, animals with advanced kidney disease or hepatic encephalopathy should be provided with reduced protein levels, whereas growing animals and patients with significant protein losses (e.g., patients suffering from burns) may require increased protein levels.

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Fat

Foods designed for critically ill patients are commonly high in fat content to help increase energy density. Because some patients have difficulty tolerating food volumes when initiating enteral feeding, increased fat and energy density can help to reduce the required daily volume.

Typical adult maintenance dog and cat dry foods range from 20% to 35% fat ME, while critical care enteral foods range typically from 40% to 58% fat ME. Animals that have been without food for more than three days are using primarily endogenous fat for energy, making the transition to a higher fat food a fairly smooth transition for most patients. Because of the high fat content of many critical care diets, caution should be used when refeeding a patient with a condition associated with fat intolerance, such as hyperlipidemia, pancreatitis, or lymphangiectasia. For patients such as these, a lower fat enteral diet, canned food slurry, or home-cooked diet slurry may be required.

Carbohydrates

Dietary carbohydrates help to provide needed energy and spare the use of protein for gluconeogenesis. Carbohydrates also help to reduce the proportion of calories coming from fat, which can be helpful in patients with fat intolerance. However, carbohydrates are not a required nutrient, and for some patients, a lower carbohydrate diet may provide some benefits. Patients experiencing hyperglycemia are some in which limiting dietary carbohydrates may be helpful. Increased risk of mortality has been associated with hyperglycemia in human, canine, and feline critically ill patients [6–10]. Although hyperglycemia is more commonly seen as a metabolic complication in patients receiving parenteral nutrition, hyperglycemia has been reported in a feline patient with pancreatitis following implementation of enteral nutrition [11].

Hyperglycemia in critically ill cats is also commonly seen prior to nutritional intervention; therefore, careful monitoring and adjustment of treatment protocols is recommended as nutrition support is implemented in these patients. Critically ill cats have been shown to have significantly higher glucose, lactate, cortisol, glucagon, and norepinephrine concentrations, and significantly lower insulin concentrations, when compared with controls [12]. These findings are consistent with those from human studies, showing higher concentrations of counterregulatory hormones and insulin resistance in critically ill patients [13, 14]. Further research in this area is needed to determine the most appropriate management strategies for hyperglycemic veterinary patients. However, maintaining tighter glycemic control in patients receiving nutritional support may help to improve patient outcome.

Amino Acids

Many critical care foods are enriched with select amino acids, such as branched chain amino acids, arginine, and glutamine. The branched chain amino acids valine, leucine, and isoleucine are metabolized by skeletal muscle, whereas other amino acids are metabolized by the liver [15]. For this reason, some foods designed for critically ill patients are supplemented with branched chain amino acids to supply energy for lean body tissue and help maintain lean body mass. Additionally, recent studies have demonstrated benefits of branched chain amino acids to protect against ischemia/reperfusion injury [16, 17]. One study in critically ill dogs showed that concentrations of branched chain amino acids were significantly higher in survivors compared with nonsurvivors [18]. Similarly, the ratio of branched chain amino acids to aromatic amino acids was also significantly higher in surviving dogs. This study supports the nutritional philosophy of supplementing with branched chain amino acids in critically ill patients; however, no studies have been conducted to date evaluating the efficacy of supplementation and its impact on patient outcome in dogs or cats.

Because branched chain amino acids are not metabolized in the liver, they also have a theoretical benefit in patients with liver disease and hepatic encephalopathy. Like in critically ill dogs, it is known that the ratio of plasma branched chain amino acids to aromatic amino acids is decreased with declining liver function [19]. However, although branched chain amino acids have many theoretical benefits, studies evaluating their use in dogs with hepatic encephalopathy have failed to demonstrate measurable benefits [20].

Glutamine is another amino acid commonly incorporated into critical care diets. Glutamine is preferentially used by enterocytes as an energy source, and it may help to promote the health of the small intestine and reduce the risk of bacterial translocation. In addition, glutamine is also used as a fuel source by immune cells and may aid in improving the patient's immune response. Despite the numerous theoretical benefits of glutamine supplementation, clinical studies evaluating its impact on outcome have reported mixed results.

Studies evaluating the use of glutamine-supplemented enteral diets in veterinary patients are limited. One study evaluating a glutamine-enriched enteral diet for cats with experimentally induced gastrointestinal disease did not find a significant clinical benefit [21]. Another experimental study evaluating radiation injury in dogs also failed to show a benefit of glutamine supplementation [22].

Studies in experimental animals have demonstrated that glutamine supplementation helps to maintain mucosal barrier function, but other studies evaluating hospitalized surgery and trauma patients have failed to demonstrate a

benefit on outcome [23–25]. Human studies evaluating free glutamine versus glutamine-rich protein supplementation have shown that feeding glutamine from complete protein sources is more efficacious in increasing mucosal glutamine concentrations [26]. Further studies are needed to determine how to use glutamine most effectively in enteral diets for critical care veterinary patients [27].

Arginine is another amino acid that may provide benefits in critically ill patients [27–29]. Arginine is an essential amino acid for dogs and cats but is not normally essential in people. In recent years, arginine has gained attention in human medicine as "conditionally essential" during periods of stress, due to its important roles in wound healing, immune function, and nitric oxide synthesis [30]. Studies in people and rodents have demonstrated that arginine-supplemented enteral support can help to improve wound healing, decrease length of hospital stay, and change cytokine expression from a pro- to an anti-inflammatory profile [31, 32]. Arginine supplementation has not been evaluated in veterinary critical care patients, but studies showing lower plasma arginine levels in critically ill dogs and dogs with early chronic valvular disease suggest that arginine supplementation may be beneficial [18, 33].

Other Nutrients

Like supplemental amino acids, many of the other enhanced nutrients in critical care diets have theoretical benefits but have not been evaluated in critically ill dogs or cats. Antioxidants may benefit by counteracting the generation of free radicals associated with inflammation or reperfusion injury. Increased levels of long-chain omega-3 polyunsaturated fatty acids can aid in modulating inflammatory reactions associated with underlying inflammatory processes. Finally, the addition of prebiotics, such as fermentable fibers and oligopolysaccharides, may be beneficial in critically ill animals with a compromised gastrointestinal tract by promoting production of short-chain fatty acids and energy for colonocytes.

Enteral Diets

Liquid Enteral Diets

Liquid enteral diets are required when feeding through a nasoesophageal or jejunostomy feeding tube. Liquid enteral diets can be categorized as polymeric or elemental formulations. Polymeric formulations are composed of proteins, carbohydrates, and fats in a high molecular weight form. Technically, a true "elemental" formulation would be comprised of free amino acids, monosaccharides, and fatty acids; however, true elemental formulations are rare, and many human formulations are "semi-elemental," containing hydrolyzed proteins, di- and tripeptides, disaccharides, oligosaccharides, and/or dextrin. Elemental diets have the advantage of requiring little to no digestion, which is ideal for patients with severe gastrointestinal disease, short bowel syndrome, or those being fed at the level of the jejunum. The potential disadvantage of elemental formulations is that some may have a higher osmolality when compared with polymeric formulations.

Studies in human patients with pancreatitis receiving jejunal enteral nutrition have shown that both polymeric and semi-elemental formulations are well tolerated; however, some advantages were seen with semi-elemental formulations including reduced length of hospital stay and less marked weight loss [34]. Currently available liquid canine and feline enteral foods are limited to polymeric formulations (Table 43.1).

Although the selection of liquid enteral foods designed for dogs and cats is limited, those available generally meet the needs of most hospitalized patients. For those patients that may require semi-elemental or hydrolyzed ingredients, a human liquid diet or enteral formulation may be used. It should be noted that human liquid products often contain vitamins and minerals but are not necessarily complete and balanced to meet the nutrient needs of dogs and cats. This is especially true in cats, in which protein and amino acid supplementation is generally required to meet protein and essential amino acid needs. For these more challenging cases, consultation with a board-certified veterinary nutritionist should be considered.

Canned Enteral Diets

Canned foods for critical care veterinary patients are designed to be highly digestible, energy dense, and provide a moderate to high protein content. Diets may be enriched with additional nutrients such as antioxidants, specific amino acids (arginine, glutamine, branched chain amino acids), omega-3 fatty acids, prebiotics, and soluble or insoluble fibers. These canned diets are also designed with a smooth consistency that facilitates syringe feeding through a feeding tube. These products can generally be delivered through a 12–14 Fr feeding tube without additional water dilution; however, they may need to be slightly warmed and mixed well to facilitate delivery. Current commercially available canned enteral foods for dogs and cats are outlined in Table 43.1.

Blended Commercial Diet Slurries

Blended commercial dry (presoaked) or canned foods can be used when feeding through an esophagostomy or gastrostomy tube. Food provision through feeding tubes is

Table 43.1 Veterinary canned and liquid enteral foods[a].

	Caloric distribution			Energy density	Ca	P	Na	K
Veterinary Foods	**Protein (%)**	**Fat (%)**	**Carb. (%)**	**kcal/g**	**g/Mcal**			
Canned								
Hill's Prescription Diet® a/d® (canine/feline)	33	55	12	1.2	2.6	2.4	1.8	2.4
Royal Canin Recovery® ultra-soft mousse (canine/feline)	38	58	4	1.6	3.2	2.7	3.0	2.3
Liquid								
Royal Canin Recovery Liquid(canine/feline)	32	48	20	0.9	2.3	1.8	1.0	2.3
Royal Canin Low Fat™ Liquid (canine)	35	19	46	0.9	2.3	2.0	1.1	2.4
Royal Canin Renal Support Liquid (canine)	13	41	46	1.3	1.4	0.9	1.0	1.6
Royal Canin Renal Support Liquid (feline)	26	50	24	0.9	1.3	0.8	0.9	1.8

Ca, calcium; Carb., carbohydrate; K, potassium; kcal, kilocalories; Mcal, megacalories (1000 kcal); Na, sodium; P, phosphorus.
[a] Nutrient and caloric composition of products as of March 2021.

discussed in detail in Chapter 44. The use of blended slurries helps immensely to expand the dietary options and allows the diet to be more easily tailored to the pet's specific dietary needs. Canned foods are used the most commonly, but dry food can also be used if presoaked in water to allow softening for blending. To achieve the highest possible energy density, it is recommended to add water slowly to the slurry while blending until a smooth consistency that can be delivered through the tube is achieved (Figures 43.1–43.3). The amount of water required to achieve a consistency for delivery through a 12 or 14 Fr feeding tube will range from diet to diet depending on the ingredient composition. Typical water amounts are 8–15 ml/oz canned diet for canine formulas, and 5–10 ml/oz canned food for feline formulas. Once an appropriate consistency has been achieved, the energy density and moisture level of the slurry can be determined and feeding volumes can be calculated (Box 43.1).



Figure 43.1 Blended food slurries can be used for esophagostomy and gastrostomy enteral feedings. Slowly add water to the canned food and blend mixture for four to five minutes until a smooth texture is achieved.

Home-Cooked Diet Blended Slurries

Home-cooked (or hospital-prepared) blended slurry formulations can be helpful when commercially available products do not meet patient needs (Figure 43.4). Dietary fat intolerance is one of the most common complications that leads to the need for a home-cooked formulation. Highly digestible/low fat, uncommon ingredient/low fat, and renal disease/low fat canine and feline formulations are outlined in Tables 43.2–43.7. The ingredients in these formulations are designed to be simple and require minimal preparation in a hospital setting.

When preparing these slurries, the water should be slowly added to the mixture during the blending step,



Figure 43.2 Once the slurry has been adequately blended, measure the final slurry volume for energy density calculations. For blenders that do not provide volume designations, volume guidelines can be premeasured and marked on the side of the blender using known volumes of water.

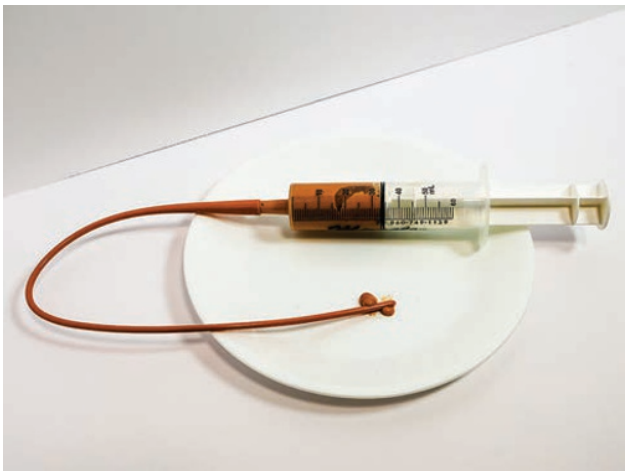


Figure 43.3 Test the final slurry to ensure that it can be easily delivered through the appropriate feeding tube size.

Box 43.1 Enteral Feeding Worksheet

Step 1: Calculate patient's resting energy requirement

Resting energy requirement (RER) kcal/day = 70 × body weight (kg)$^{0.75}$

Example

10 kg adult dog:
RER = 70 × $10^{0.75}$ = 393 kcals/day

Step 2: Calculate energy density of blended slurry

Energy per can food (kcal)/final slurry volume (ml) = kcal/ml

Example

Veterinary intestinal formula 400 kcal/can
Added 200 ml water to reach desired consistency
Total volume of final slurry (canned food + water) = 600 ml slurry
400 kcal/600 ml = 0.67 kcal/ml

Step 3: Calculate feeding daily volume

RER (kcal/day)/energy density of slurry (kcal/ml) = ml slurry/day
Day 1 = 25–33% × total ml slurry/day = total day 1 volume (ml)
Day 2 = 50–66% × total ml slurry/day = total day 2 volume (ml)
Day 3 = 75–99% × total ml slurry/day = total day 3 volume (ml)
Day 4 = 100% × total ml slurry/day = total day 4 volume (ml)

Example

393 kcal/day/0.67 kcal/ml = 587 ml/day
Day 1 = 25% × 587 ml = 147 ml
Day 2 = 50% × 587 ml = 294 ml
Day 3 = 75% × 587 ml = 440 ml
Day 4 = 100% × 587 ml = 587 ml

Step 4: Calculate water contribution from blended slurry

Moisture of canned food (%) × g/can = water from food (ml)
Water from food (ml) + water added to slurry (ml) = total water volume (ml)
Total water volume (ml)/slurry volume (ml) = moisture of final slurry (%)
ml daily slurry × moisture of final slurry (%) = total water delivered daily (ml)

Example

Veterinary intestinal formula = 78% moisture; 396 g/can
0.78 × 396 = 309 ml water from food
309 ml water from food + 200 ml water added = 509 ml total water
509 ml water/600 ml total slurry = 84.8% moisture
587 ml daily slurry × 0.848 = 498 ml total water deliver daily

Step 5: Calculate additional water needs

Patient's water requirement (ml) minus water delivered from blended slurry (ml) = additional water needed daily (ml)

Example

Patient's water requirement = 132 ml × $10^{.75}$ = 742 ml
742 ml water – 498 ml from slurry = 244 ml additional water needed



Figure 43.4 Highly digestible low-fat ingredients can be used to create low-fat recipes, and supplemental ingredients can be used to improve energy density of the diet.

Table 43.2 Feline low-fat recipe[a].

	Quantity	Common Measures
Hormel[b] canned chicken breast in water, no salt added (g)	142	1 can (not drained)
White rice, long grain, regular, cooked (g)	118.5	¾ cup
Canola oil (g)	3.4	¾ teaspoon
Nordic Naturals[c] pet cod liver oil (g)	1.2	¼ teaspoon
Balance IT[d] feline (g)	4.9	1 red and 7 white scoops
Water (ml)	150	
Total recipe kcal	330	
Total slurry volume (ml)	400	
Total slurry moisture (%)	82	
Energy density of slurry (kcal/ml)	0.83	
Caloric distribution (g):		
Protein (% ME)	37.4	
Fat (% ME)	20.6	
Carbohydrate (% ME)	42.0	

kcal, kilocalories; ME, metabolizable energy.
[a] Guidelines developed by Dr. Sally Perea, Mason, OH.
[b] Hormel Foods Corporation, Austin, MN.
[c] Nordic Naturals, Inc., Watsonville, CA.
[d] Davis Veterinary Medical Consulting, Inc., Woodland, CA.

Table 43.3 Feline low-fat uncommon ingredient recipe[a].

Ingredient	Quantity	Common Measures
Bumble Bee[b] canned white crab meat (g)	120	1 × 6-oz can, drained
Betty Crocker[c] mashed potato buds, dried potato flakes (g)	23	⅓ cup dry amount mixed with equal parts hot water
Canola oil (g)	3.4	¾ teaspoon
Balance IT[d] feline (g)	2.9	1 red scoop
Water (ml)	80	
Total recipe kcal	211	
Total slurry volume (ml)	280	
Total slurry moisture (%)	82	
Energy density of slurry (kcal/ml)	0.75	
Caloric distribution (g):		
Protein (% ME)	45.9	
Fat (% ME)	18.5	
Carbohydrate	35.6	

kcal, kilocalories; ME = metabolizable energy; oz, ounces.
[a] Guidelines developed by Dr. Sally Perea, Mason, OH.
[b] Bumble Bee Foods, LLC, San Diego, CA.
[c] General Mills, Inc., Minneapolis, MN.
[d] Davis Veterinary Medical Consulting, Inc., Woodland, CA.

as adding all of the water at the beginning step can result in a soupy blend, leaving fragments of rice or other ingredients that are not well blended. A slightly longer blending time than required for the canned food slurries is also recommended for the home-cooked formulations (minimum of five minutes). Allowing ample time for blending helps ensure that the ingredients are broken down and smoothly distributed throughout the mixture.

Table 43.4 Feline low-fat renal recipe[a].

Ingredient	Quantity	Common Measures
Hormel[b] canned chicken breast in water, no salt added (g)	71	½ can (not drained)
White rice, long grain, regular, cooked (g)	118.5	¾ cup
Canola oil (g)	3.4	¾ teaspoon
Nordic Naturals[c] pet cod liver oil (g)	1.2	¼ teaspoon
Balance IT[d] feline-K (g)	3.9	1 yellow and 1 white scoop
Water (ml)	170	
Total recipe (kcal)	262	
Total slurry volume (ml)	315	
Total slurry moisture (%)	83	
Energy density of slurry (kcal/ml)	0.83	
Caloric distribution (g):		
Protein (% ME)	25.9	
Fat (% ME)	21.2	
Carbohydrate	52.9	

kcal, kilocalories; ME, metabolizable energy.
[a] Guidelines developed by Dr. Sally Perea, Mason, OH.
[b] Hormel Foods Corporation, Austin, MN.
[c] Nordic Naturals, Inc., Watsonville, CA.
[d] Davis Veterinary Medical Consulting, Inc., Woodland, CA.

Table 43.5 Canine low-fat recipe[a].

Ingredient	Quantity	Common Measures
Cottage cheese, 2% milk fat (g)	339	1½ cups
White rice, long grain, regular, cooked (g)	158	1 cup
Light corn syrup (g)	88	4 tablespoons
Corn oil (g)	4.5	1 teaspoon
Nordic Naturals[b] pet cod liver oil (g)	1.2	¼ teaspoon
Balance IT[c] canine (g)	10.6	4¼ teaspoons
Water (ml)	20	
Total recipe (kcal)	808	
Total slurry volume (ml)	525	
Total slurry moisture (%)	67	
Energy density of slurry (kcal/ml)	1.54	
Caloric distribution (g):		
Protein (% ME)	26.6	
Fat (% ME)	13.9	
Carbohydrate	59.5	

kcal, kilocalories; ME, metabolizable energy.
[a] Guidelines developed by Dr. Sally Perea, Mason, OH.
[b] Nordic Naturals, Inc., Watsonville, CA.
[c] Davis Veterinary Medical Consulting, Inc., Woodland, CA.

Table 43.6 Canine low-fat uncommon ingredient recipe[a].

Ingredient	Quantity	Common Measures
Bumble Bee[b] canned crab meat (g)	120	1 × 6-oz can, drained
Betty Crocker[c] potato buds (dried potatoes) (g)	52	¾ cup dry amount mixed with equal parts hot water
Canola oil (g)	4.5	1 teaspoon
Balance IT[d] canine (g)	3.75	1½ teaspoons
Water (ml)	150	
Total recipe kcal	324	
Total slurry volume (ml)	460	
Total slurry moisture (%)	83	
Energy density of slurry (kcal/ml)	0.70	
Caloric distribution (g):		
Protein (% ME)	32.0	
Fat (% ME)	15.5	
Carbohydrate	52.5	

kcal, kilocalories; ME = metabolizable energy; oz, ounces.
[a] Guidelines developed by Dr. Sally Perea, Mason, OH.
[b] Bumble Bee Foods, LLC, San Diego, CA.
[c] General Mills, Inc., Minneapolis, MN.
[d] Davis Veterinary Medical Consulting, Inc., Woodland, CA.

Table 43.7 Canine low-fat renal recipe[a].

Ingredient	Quantity	Common Measures
Hormel[b] canned chicken breast in water, no salt added (g)	71	½ can (not drained)
White rice, long grain, regular, cooked (g)	237	1½ cups
Light corn syrup (g)	66	3 tablespoons
Corn oil (g)	6.8	1½ teaspoons
Nordic Naturals[c] pet cod liver oil (g)	2.5	½ teaspoon
Balance IT[d] canine-K (g)	6.5	1 blue and 8 white scoops
Water (ml)	250	
Total recipe kcal	633	
Total slurry volume (ml)	550	
Total slurry moisture (%)	77	
Energy density of slurry (kcal/ml)	1.15	
Caloric distribution (g):		
Protein (% ME)	12.6	
Fat (% ME)	14.0	
Carbohydrate	73.3	

kcal, kilocalories; ME = metabolizable energy; oz, ounces.
[a] Guidelines developed by Dr. Sally Perea, Mason, OH.
[b] Hormel Foods Corporation, Austin, MN.
[c] Nordic Naturals, Inc., Watsonville, CA.
[d] Davis Veterinary Medical Consulting, Inc., Woodland, CA.

Preparation Instructions for Blended Slurries

Measure out the ingredient and add to a blender. Add one quarter of the estimated water volume and begin to blend the mixture, adding an additional quarter of estimated water every 30 seconds until the full volume has been added. Blend the mixture for an additional four to five minutes and test the final slurry to ensure that it can easily be delivered through the appropriate feeding tube size. If needed, additional water can be added to create a more dilute mixture for easier delivery. If additional water is added, slurry energy density and moisture content should be recalculated to reflect the new slurry volume. Unused slurry may be tightly covered and stored in the refrigerator for a later feeding; however, the slurry may require reblending and heating to room temperature to regain a smooth and fluid consistency prior to feeding. Slurry should be discarded after three days to prevent spoilage.

Supplements

When using or adding human foods or supplemental ingredients, it is important to consider the impact on the overall balance of the diet. When adding more than 10% of the total calories from a supplemental ingredient to a commercial food, additional nutrients such as vitamins and/or minerals may be required to ensure that minimum nutrient requirements are met. For short-term in-hospital management (less than one to two weeks), adding additional vitamins and minerals to create a complete and balanced formulation may not be necessary. However, for patients that require long-term enteral nutrition support, consultation with a board-certified veterinary nutritionist is recommended to ensure that the diet formulation is complete and balanced for long-term feeding.

Fat Supplements

Oils can be added to solutions to increase fat and energy density, as well as to provide specific fatty acids of interest. Vegetable oils, such as corn and canola oil, can be used to increase both total dietary fat and linoleic acid levels of the diet; fish oils can be used to provide long-chain omega-3 polyunsaturated fatty acids.

Carbohydrate Supplements

Light corn syrup is an energy-dense, highly digestible carbohydrate ingredient that can be added to formulations to boost energy density without increasing the fat level. The addition of light corn syrup can be very helpful to boost calories for canine formulations but is not recommended for feline formulations due to the fructose content. As an alternative, glucose or dextrose powders or solutions could be used in cat formulations.

Considerations for Specific Underlying Conditions

When providing assisted enteral feeding to patients with underlying conditions, the nutrient modifications and recommendations mirror those of oral diets. Thus, efforts should be made to select a food that first addresses the patient's underlying illness, and then consider how to prepare the food for enteral delivery.

Gastrointestinal Disease

Highly digestible diets are recommended for patients with underlying gastrointestinal diseases. There is a variety of highly digestible veterinary diets available that serve as excellent choices for blended canned food slurries. These highly digestible foods generally provide moderate protein and fat levels, also making them good choices when looking for a more equally distributed caloric composition of protein, fat, and carbohydrates compared with some of the critical care formulas.

Adverse Reactions to Food

For patients with adverse reactions to food such as food allergy dermatitis or inflammatory bowel disease, a hydrolyzed protein or uncommon/limited ingredient canned food may be used to create a blended slurry for esophagostomy or gastrostomy feedings. If the patient is currently being managed on an uncommon ingredient food, then a canned food with the same ingredients or hydrolyzed protein source should be selected. For patients recently diagnosed with food allergies, or with suspected food allergies, a food with ingredients that the pet has not previously been exposed to should be selected.

Fat Intolerance

It should be noted that although canned diets designed for intestinal disease and food allergies generally provide less fat than those designed for critical care, many are still relatively high in fat, especially when compared with their dry food equivalent. For patients with disease conditions related to dietary fat, such as hyperlipidemia, pancreatitis, and lymphangiectasia, a lower fat formulation should be selected. Patients with decreased gastrointestinal motility and chylothorax may also benefit from a low-fat diet.

While common commercial canned foods typically run higher in fat, there are a limited number of low-fat canned food options available that can be used in a blended slurry. Low-fat home-cooked recipes provided in this chapter can also serve as low-fat dietary options when commercial options are unavailable.

Kidney Disease

For patients with kidney disease, a canned kidney food can be used to create a blended slurry for esophagostomy and gastrostomy feedings. If a liquid food is required, liquid kidney canine and feline formulas are also commercially available. These liquid formulas may be fed alone or blended with the canned diet to help increase energy density of the blended slurry. Some human liquid diets, such as Ensure® (Abbott Nutrition), are lower in protein but are not phosphorus restricted, making them less than ideal for longer-term management.

Liver Disease

For patients with hepatic encephalopathy, a canned protein-restricted liver disease veterinary food can be used to create a blended slurry for esophagostomy and gastrostomy feedings. Although kidney failure canned foods are also protein restricted, they may contain higher inclusions of meat and organ-based protein sources compared with liver-disease diets, and these are not as ideal for patients with hepatic encephalopathy compared with plant and dairy based proteins [35]. Care should therefore be taken when selecting a diet for hepatic encephalopathy to ensure that both the protein level and source are appropriate.

For liver disease patients that are not suffering from hepatic encephalopathy, protein restriction is not necessary or recommended. A standard critical care diet or a highly digestible canned food blended slurry are both acceptable options for these patients.

Initiating Nutritional Support

Patients should be slowly introduced to full energy needs, starting at approximately 25–33% of RER, followed by 25–33% increases every 12–24 hours until full RER is reached [36, 37]. Patients that have been without food for an extended period of time are at increased risk of developing hyperglycemia or electrolyte abnormalities upon refeeding (i.e. hypokalemia, hypophosphatemia, and hypomagnesemia, as well as low thiamine) and may require a slower introduction and increased frequency of monitoring. Once full RER is reached, the patient may be reassessed to determine whether increased caloric levels are needed to maintain body weight.

Enteral formulas can be fed via continuous infusion or via intermittent bolus feeding. Esophagostomy and gastrostomy feedings are generally given by intermittent bolus feedings. One clinical study evaluating continuous versus intermittent bolus feedings in dogs with gastrostomy tubes showed no differences in weight maintenance, gastrointestinal adverse effects, glucose tolerance, nitrogen balance, or feed digestibility [38]. Continuous infusions may be better tolerated for nasoesophageal feedings if large volumes of liquid formulas are required to meet daily energy needs, and they are recommended for jejunal feedings to help minimize malabsorption and diarrhea associated with feeding large volumes of nutrients directly into the jejunum.

When feeding via intermittent bolus, four or more feedings per day are generally required. The amount fed per feeding should not exceed 5–10 ml/kg of body weight during initial introduction of feedings [39]. Maximum gastric capacities for dogs and cats are reported as high as 45–90 ml/kg body weight [28]. However, meeting the patient's RER should be achievable at volumes far below these maximum capacities. Feeding boluses should be given slowly to allow for gastric expansion. Patients should be monitored for signs of nausea such as salivating, gulping, or retching during the feeding. If any of these signs develop, the feeding should be temporarily discontinued or stopped. Further details and guidelines for enteral feeding are provided in Chapter 44.

Monitoring

Patients receiving enteral nutrition should be monitored daily for potential complications and to ensure that the nutritional plan is continuing to meet the patient's needs. Daily physical examinations should include assessment of body condition and measurement of body weight. Because of the risk of aspiration and fluid overload, care should be taken to assess respiratory parameters, and thoracic radiographs should be made if respiratory distress or fever develops. The stoma site of enteral feeding tubes should be cleaned with dilute antiseptic (e.g. chlorhexidine or povidone-iodine) and carefully examined daily for signs of infection (see Chapter 63 for more information).

Serum potassium, magnesium, and phosphorus concentrations should be measured within 12–24 hours of starting enteral nutrition. Daily monitoring of electrolytes should be continued during the weaning on period, and no less than once every 48 hours once at goal rate of infusion for critically ill hospitalized patients. A complete blood count and full serum biochemistry panel should be measured within 24 hours of instituting enteral nutrition. Continued monitoring of a complete blood count and chemistry panel every two to three days is recommended for critically ill patients.

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44

Assisted Enteral Feeding

Avalene. W. K. Tan

Introduction

Adequate nutritional intake is vital to support the immune system, for synthesis of important biological molecules, for wound healing, and to maintain intestinal mucosa integrity. As such, animals with decreased nutritional intake are at a higher risk of infection, increased catabolic states, wound dehiscence, and intestinal villous atrophy [1].

Uncertainty persists about the best method of nutritional support in critically ill patients. While there is insufficient evidence to say that enteral nutrition alone is superior to parenteral nutrition in improving mortality rates, enteral nutrition is associated with reduced sepsis, and the combination of both routes appears to be associated with reduced mortality [2, 3]. Dogs with septic peritonitis that receive enteral nutrition are more likely to survive than dogs receiving parenteral nutrition alone [4]. Enteral nutrition remains the preferred route, as it is more physiologic than parenteral nutrition and directly maintains gastrointestinal motility, hormone secretion, and barrier function [5, 6]. Human critical care guidelines suggest commencing enteral over parenteral nutrition if there are no contraindications (Box 44.1) [7, 8]. There are currently no formal veterinary guidelines. Furthermore, there is growing support for early enteral nutrition in both human and veterinary medicine alike [6–11]. While more high-quality studies are required, the current evidence does not suggest harm from early enteral feeding within 48–72 hours [7, 10, 12–14].

Detailed nutritional assessment of critically ill patients is discussed in Chapter 42 and involves the use of historical and physical parameters. Checklists have also been proposed to help determine a patient's requirement for nutritional support (Table 44.1) [1]. Resting energy requirement (RER) is the recommended target for critical patients, but continuing reassessment is still required, as some patients may have demands exceeding this baseline [16, 17]. Gradually increasing enteral feeding in increments of 25% RER daily, over four days is typical (Box 44.2). This chapter focuses on techniques for assisted enteral feeding.

Enticing Voluntary Eating

Improving the palatability of the diet and/or offering a variety of different foods are common first steps, in the hope that the animal will consume sufficient calories voluntarily. Force-feeding, including syringe feeding or placing small boluses of food in the mouth, is discouraged, as many animals find the procedure stressful. There is also a risk for food aspiration with force-feeding. This practice may increase the risk of learned food aversions, making it challenging to feed the patient when it has recovered or to introduce therapeutic diets for disease states [18, 19]. A more strategic approach is to entice voluntary intake with a single diet initially and to use supplemental assisted enteral and parenteral techniques as needed.

There are many causes of anorexia, such as disease, nausea, pain, or stress, and addressing these causes may be sufficient to encourage partial voluntary intake (summarized in Figure 44.1) [20]. Medications are also a common cause of anorexia, nausea, and vomiting and clinicians should take them into account when treating an anorexic dog or cat (Box 44.3).

Improving diet palatability by modifying the moisture, fat, protein, sugar, or salt content may also be helpful [22]. Feeding canned diets with 70–85% moisture, rather than dry diets with 7–10% moisture, increases food palatability. However, canned diets often contain higher protein and fat, which may be contraindicated in some patients. Dry kibble can be soaked to increase the moisture content.

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Box 44.1 Indications and Contraindications for Enteral Feeding [1, 15]

Indications

- 3 days of anorexia/withheld food (sooner for puppies, kittens, and overweight cats)
- Inability to voluntarily consume food
- Low body condition score (dogs ≤3, Cats ≤4 on a 9-point scale)
- Weight loss of ≥10%
- Hypoalbuminemia
- Requirements for post-pyloric feeding

Contraindications

- Prolonged recumbency
- Regurgitation
- Risks of aspiration (e.g. poor gag reflex, protracted vomiting)[a]
- Intolerance to enteral nutrition
- Risks of cardiovascular decompensation during sedation/anesthesia
- Coagulopathy limiting tube placement
- Concerns for intra-cranial hypertension[a]
- Facial trauma[a]
- Ascites[b]
- Infiltrative disease of gastric/abdominal wall[#]

[a] Nasoesophageal or nasogastric tube.
[b] Percutaneous feeding tubes.

Box 44.2 Calculations and Steps for Enteral Feeding Plans

1) Calculate resting energy requirement (RER):

$$\text{RER in kcal / day} = 70 \times (\text{body weight in kilograms})0.75$$

2) Calculate energy density of diet:

$$\text{Energy density}(\text{kcal / ml}) = \frac{\text{Total kcal}(\text{diet})}{\text{Total ml}(\text{diet} + \text{water added})}$$

3) Calculate amount of feed per day:

$$\text{Amount to feed}(\text{ml/day}) = \frac{RER(kcal/day)}{Energy\ \ density(kcal/ml)}$$

Feeding plan		Day 1	Day 2	Day 3	Day 4
RER	Option 1	⅓	⅔	Full	
	Option 2	¼	½	¾	Full

4) Calculate amount to feed per meal:

$$\text{Amount per meal} = \frac{\text{Volume/day}}{\text{Number of meals/day}}$$

$$\text{Constant rate infusion}(\text{ml/hour}) = \frac{\text{Volume/day}}{\text{Hours of infusion}}$$

Table 44.1 Assessment for need of nutritional support [1].
Source: Adapted from Chan 2020.

Parameter	Low risk	Moderate risk	High risk
Food intake <80% resting energy requirements			
<3 days	✓		
3–5 days		✓	
>3 days			✓
Weight loss		✓	
Severe vomiting/diarrhea			✓
Body condition score <4/9			✓
Muscle mass score <2			✓
Hypoalbuminemia		✓	
Expected course of illness			
<3 days	✓		
2–3 days		✓	
>3 days			✓

Patients with ≥ 2 high risk factors should receive nutritional support as soon as they are stabilized.
Patients with high risks factors should be monitored closely and reassessed every few days.

Increasing the fat content of the diet also improves palatability, and additionally improves the energy density, so that less volume is required to meet caloric targets. However, higher fat content is contraindicated in dogs with pancreatitis and animals with impaired gastrointestinal motility. Amino acid receptors are the most common taste receptors in cats and dogs, and these receptors are particularly responsive to amino acids L-proline and L-cysteine [23, 24]. Increasing protein may therefore enhance palatability but may not be appropriate for animals with hepatic encephalopathy or renal disease. Dogs show a preference for sugars, such as lactose, sucrose, and fructose, and adding sugars or syrups to foods can augment food palatability [25, 26]. Cats lack sweet receptors and are indifferent to sweetened foods [23, 27]. Any added sugar or syrup should not constitute more than 10% of the total calories to avoid the risk of creating an imbalance in the balanced base diet [22]. Sweetened diets with rapidly digestible and absorbed carbohydrates should be avoided in diabetic patients, and artificial sweeteners, such as xylitol, should be avoided due to risks of toxicosis. Salt receptors are absent in cats and dogs, but interestingly, the amino acid receptors are stimulated by high

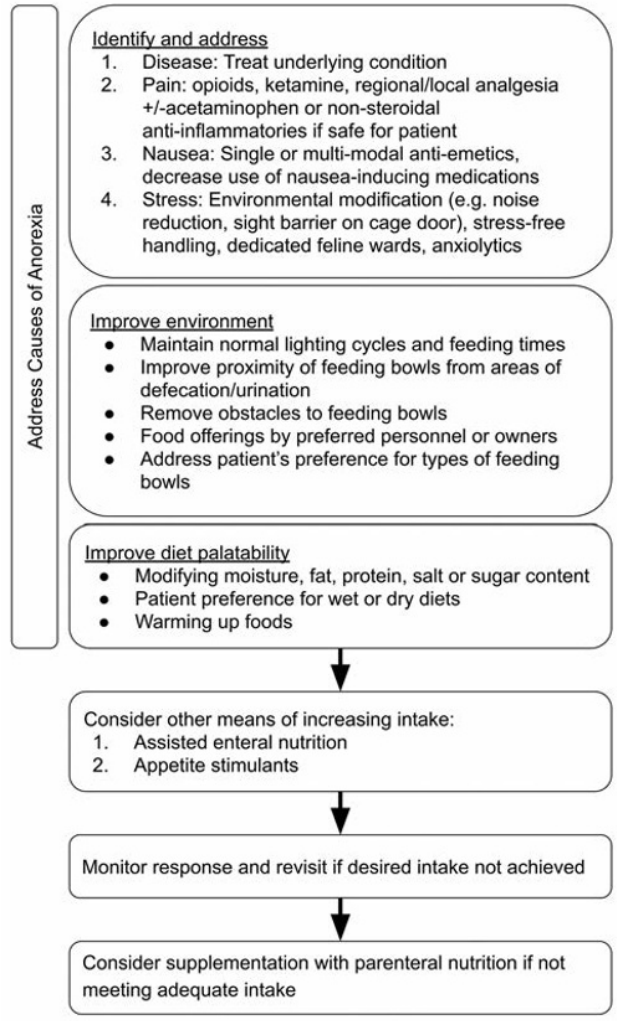


Figure 44.1 Considerations or options for addressing anorexia.

Box 44.3 Medications that May Cause Anorexia, Nausea, and Vomiting in Dogs and Cats [21]

- Aminocaproic acid
- Amoxicillin
- Amoxicillin/clavulanate
- Cardiac glycosides
- Cephalexin
- Chloramphenicol
- Erythromycin
- Most chemotherapeutic agents
- Most narcotic analgesics
- Nonsteroidal anti-inflammatory drugs
- N-Acetylcysteine
- Tetracyclines
- Trimethoprim/sulphadiazine
- Tranexamic acid

concentrations of sodium chloride [23]. Salt may also increase taste responses to sugars in dogs [24]. Therefore, adding salt or using diets with a higher sodium content may improve food palatability. However, caution is required with patients suffering from renal disease, cardiac disease, ascites, or hypertension [22].

Other factors, such as texture, "mouth feel," and temperature preference, are recognized but have not been studied to the same degree as flavor preference in small animals. Cats prefer foods near body temperature (101.5°F; 38.5°C) and warming food can increase its olfactory stimulus [22]. The "mouth feel" of the food is also important for cats, and they may reject foods with a powdery or greasy texture or a different kibble shape [21].

If voluntary enteral intake is still not adequate, then other means of increasing intake should be considered. Appetite stimulants may be helpful as an adjunctive to improve intake, but can have unpredictable efficacy, and over-reliance on pharmacological stimulation may delay the institution of assisted nutrition methods or addressing of the underlying disease process. A list of appetite stimulants currently used, and their potential adverse effects, is provided in Table 44.2 [28–32]. Enteral nutrition with feeding tube placement or use of parenteral methods should be considered in the persistently hyporexic or anorexic patient or individuals with severe illness.

Tube Route Selection

The indications, concerns, and contraindications for each assisted enteral feeding tube route are shown in Table 44.3, which can be used to determine which technique to use [1, 33–35].

If the tube is likely to be required long term, polyurethane or silicone tubes should be selected in preference to the red polyvinyl chloride (PVC) tubes, because they do not become brittle or disintegrate in situ when exposed to digestive juices [33]. The internal diameter of the polyurethane tubes is slightly larger than the silicone tubes of the same French size, because they are stronger with thinner walls. A French unit is equivalent to 0.33 mm and measures the external diameter of the tube [33].

Nasoesophageal and Nasogastric Tubes

Placement and Verification

Placement equipment and technique of nasoesophageal (NE) and nasogastric (NG) tubes are described in Protocol 44.1 and shown in Figure 44.2 [36]. NE and NG tube types include weighted and non-weighted feeding

Table 44.2 Appetite stimulants [28–31].

Drug	Dogs	Cats	Drug class	Adverse effects
Diazepam Oxazepam Flurazepam		✓	Benzodiazepine	Non-purposeful eating, sedation, worsening hepatic encephalopathy, idiosyncratic hepatic necrosis
Mirtazapine	✓	✓	Tetracyclic antidepressant	Sedation
Cyproheptadine	✓	✓	Serotonin antagonist Antihistamine	Hyperexcitability, aggression, vomiting
Prednisolone Prednisone	✓	✓	Glucocorticoid	Polyuria/polydipsia, impaired wound healing
Capromorelin	✓	✓	Ghrelin-receptor agonist	Vomiting, diarrhea, hypersalivation, hyperglycemia, bradycardia and hypotension in cats
Nandrolone			Anabolic steroid	Uncommon
Megestrol acetate	✓	✓	Synthetic progestin	Polyuria/polydipsia, diabetes mellitus, hepatotoxicity, adrenocortical suppression
Cannabidiol oil			Cannabinoid receptors	Sedation, hyperexcitability
Cyanocobalamin			Vitamin	

Table 44.3 Indications, concerns, and contraindications of feeding tubes [1, 33–35].

Type	Indications	Concerns	Contraindications	Time to use
NE/NG	Short-term tube feeding (< 10 days). Functional nasal cavity, pharynx, esophagus, stomach, and intestines. High anesthesia risk	Unable to evaluate gastric residual volumes with NE tube; liquid diets only due to tube size	Vomiting, comatose/ laterally recumbent, respiratory disease, lack of gag reflex, facial trauma involving nose/nasal cavity	Immediate
E	Medium-term feeding (1–20 weeks). Facial/oral trauma, surgery, chronic illness, cancer	Unable to evaluate gastric residual volumes; anesthesia required	Vomiting, respiratory disease, megaesophagus esophageal stricture, impaired wound healing[a]	Immediate
G	Long-term feeding (months to years). Esophageal disorders. Oral surgery/trauma. Pancreatitis without vomiting. Hepatic lipidosis. Specific dietary need	Cost; complications from early inadvertent removal of tube	Anesthesia risk Impaired wound healing[a], persistent vomiting	Wait 24 hours
J	Long-term feeding (weeks to months). Resting upper gastrointestinal tract (e.g. pancreatitis, recent gastric surgery). Intestinal anastomosis. Coma	Cost; limited diet selection; complications from early inadvertent removal of tube; can only use in hospital	Anesthesia risk, impaired wound healing[a]	Within 24 hours from placement (i.e. 2–6 hours)

NE, nasoesophageal; NG, nasogastric; E, esophageal; G, gastrotomy; J, jejunal.
[a] Skin and alimentary tract incisions are required to place these tube types.

tubes (Figure 44.3a,b, respectively). Weighted feeding tubes with guidewires are preferred in patients with esophageal disease at the author's institution (William R. Pritchard Veterinary Medical Teaching Hospital, University of California). NG tubes have the added function of gastric decompression, while NE tubes are used mainly for feeding.

It is essential to confirm that the tube placement is correct before commencing tube feedings, to avoid inadvertent feeding into the airways. A lateral thoracic radiograph is considered the gold standard for verification of NG and NE tube positioning and is the most common method (Figure 44.4a) [37]. The available methods of checking tube placement are detailed and described in Box 44.4 [20, 37–42].

Protocol 44.1 Nasogastric and Nasoesophageal Tube Placement [36]

Items Required

- 5–8 Fr, 22–43 inch (55.9–109 cm) tube for dogs < 15 kg and cats
- 8–10 Fr, 43 inch (109 cm) tube for dogs > 15 kg
- 2% lidocaine or 0.5% proparacaine
- Water-soluble lubricant or 5% lidocaine ointment
- Nylon suture material
- Luer slip catheter plug
- Elizabethan collar
- Tape or marker

Procedure

- Gather the equipment needed for placement (Figure 44.2a).
- Measure the tube from the nasal meatus to the last rib (region of the stomach) for nasogastric tubes and from the nasal meatus to the seventh to ninth rib space for nasoesophageal tubes (Figure 44.2b).
- Mark the tube at the measured point with tape or a permanent marker.
- Drip a few drops of local anesthetic into the nostril and allow time for the anesthetic to take effect.
- Generously lubricate the tube.
- With the animal's head held in a normal static position, insert the tube in a caudoventral medial direction into the ventromedial aspect of the nasal cavity (Figure 44.2c).
  - In dogs, the external nares are pushed dorsally after the tube has been introduced into the nose (approximately 0.80–1.2 inches; 2.0–3.0 cm) to open the ventral meatus and aid the tube's passage into the oropharynx (Figure 44.2d).
- Brace the introducing hand against the animal's maxilla and introduce the tube in short, well-controlled insertions up to the premeasured mark (Figure 44.2e).
  - Positioning the head in a slightly flexed position makes swallowing easier to help with passage of the tube into the esophagus.
- Secure the tube to the lateral portion of the nares with a stay suture and then a finger-trap suture around the tube. Then secure the tube lateral or medial to the eye with a stay suture to the skin, that will then be tied around a taped portion of the tube (Figure 44.2f,g).
- Place an Elizabethan collar to prevent removal of the tube by the animal.
- Check correct placement (Box 44.4)

Feeding

Feeding through NE and NG tubes may begin as soon as the tube position has been confirmed, unless the animal has been sedated. The tube should be flushed with warm water before feeding (3–5 ml) to ensure patency and tube location [29]. Flushing the tube with 5–10 ml of warm water after each meal is also essential to prevent a blockage [29]. It is advisable to monitor total fluid intake in because excessive water administration may lead to hyponatremia.

Only liquid enteral diets can be used for NE and NG feeding because of the small diameter of these tubes [33]. The amount of the chosen diet to be administered can be calculated using Box 44.2. Feeding may be given as planned boluses or as a constant rate infusion (CRI). There does not appear to be any differences in gastrointestinal complications, like vomiting or regurgitation, between the two methods [43–45]. The chosen option is often dependent on clinician preference, hospitalization set-up, and nursing care. For bolus administration, the daily diet is divided up into three to six meals that are given slowly over 15–20 minutes [1]. It is recommended that the volume of meal boluses (including flushes) are less than the gastric volume, which is approximately 10–12 ml/kg [1, 29]. For a CRI, the volume is given over a set period of time (e.g. 4–6 hours, 24 hours) via a syringe pump. Highly mobile patients are more likely to cause a line disconnection and may be less amenable to a continuous feeding plan.

Food should be warmed to between room and body temperature prior to feeding, for patient comfort. The patient should also be monitored during the feed for salivation, retching, or vomiting. After feeding, flush the tube as previously described, and close the port at the end of the tube to prevent ingestion of air, which could cause distension and discomfort in the stomach [33].



Figure 44.2 (a) Equipment for nasogastric/nasoesophageal tube placement. (b) Measuring the length of tube required. (c) Insert tube in a caudoventral medial direction into ventromedial aspect of the nasal cavity. (d) Pushing nares dorsally in the dog. (e) Tube inserted up to desired mark. (f) Stay suture placed at lateral nares, then finger-trap suture tied around tube. (g) Secure tube to face with stay suture and tape.

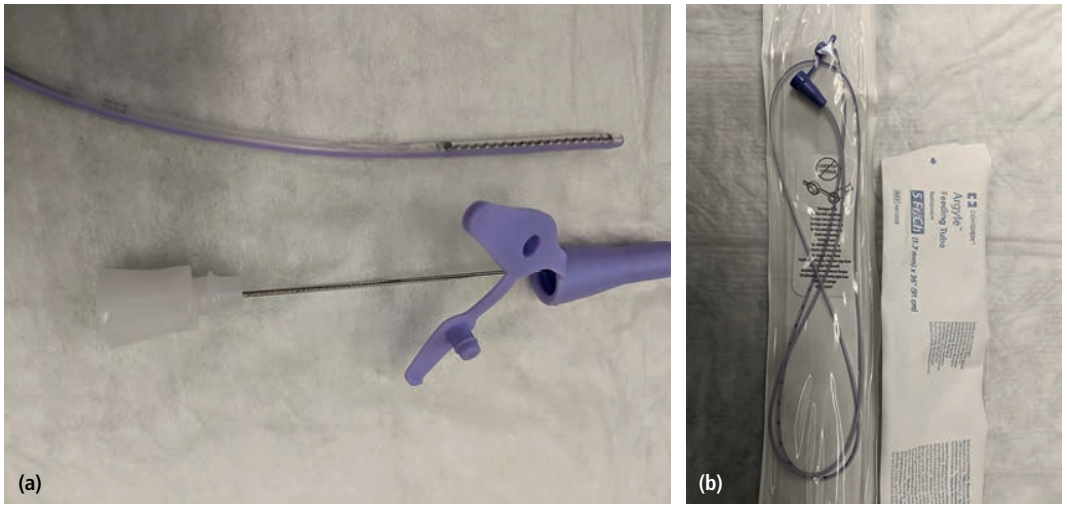


Figure 44.3 Weighted (a) and non-weighted (b) nasogastric/nasoesophageal tubes.

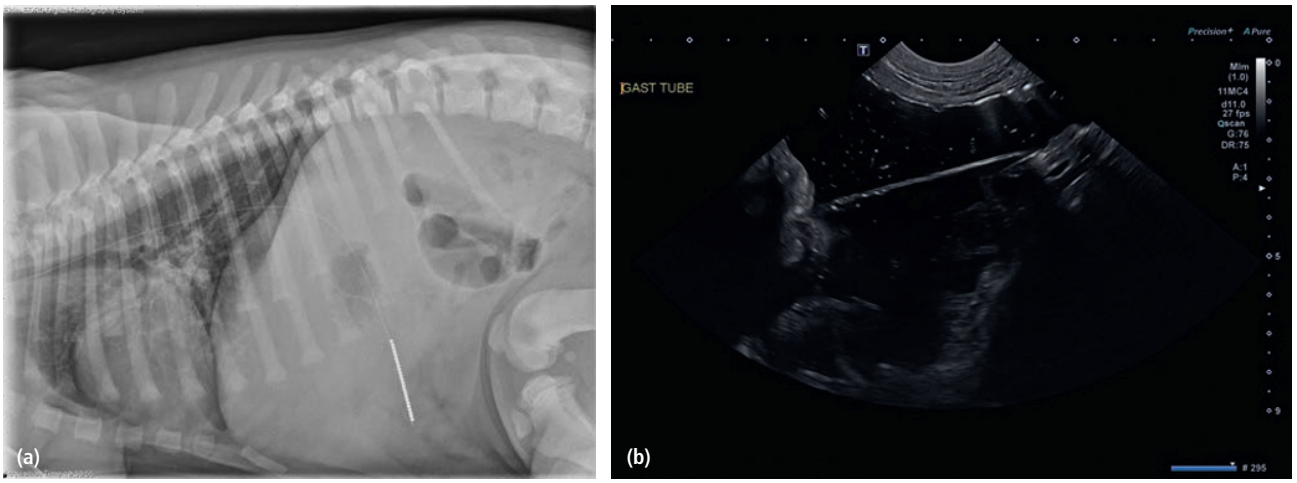


Figure 44.4 (a) Radiograph showing course of nasogastric tube into fundus. (b) Ultrasound confirmation of nasogastric tube in stomach.

Box 44.4 Methods to Verify Correct Nasoesophageal/Nasogastric Tube Positioning

1) A lateral thoracic radiograph looking for the outline of the feeding tube or radiopaque weight, is considered gold standard [37]:
   - A nasogastric tube should end in the fundus of the stomach (Figure 44.4a).
   - A nasoesophageal tube should be course dorsally to the carina and end between the seventh to ninth rib space.
   - If the location is not definitive, then thoracic radiographs in an orthogonal view should be taken to attain further information about the course of the tube.
2) Ultrasound to identify the tip of the tube within the fundus (Figure 44.4b) [38, 39].
3) Capnography which should have a zero to low carbon dioxide reading [40–42].
4) Fluoroscopy if already available and convenient.
5) Visual confirmation during concurrent exploratory laparotomy.
6) Injecting 3–15 ml sterile water and assessing for coughing.
7) Injecting 5–10 ml air and auscultating the cranial abdomen for borborygmus (nasogastric tubes only).
8) Measurement of pH of aspirated fluid which should be < 5.

Complications and Troubleshooting

The risks associated with NE and NG tubes are generally considered to be low. Commonly reported complications are vomiting, regurgitation, diarrhea, and inadvertent tube removal [46, 47]. The use of NG tubes does not appear to have increased risks of such complications compared with NE tubes [46]. The tube can be dislodged by vomiting or sneezing, and if vomiting has been witnessed, the position of the tube should be checked [34]. The tube may be visible inside the mouth or protruding from the mouth if it has been dislodged. The mark made during placement may also be checked, or another radiograph may be taken. If the tube has been dislodged, removal and replacement is indicated. Tube removal by the animal is common if appropriate steps are not taken, and may be prevented by the placement of an Elizabethan collar.

Using the correct diet, avoiding administration of bulky medications via the NE and NG tube, and flushing after each meal will reduce the risk of tube blockage. Implementation of a continuous feeding method with a syringe pump may also decrease the risk of a blockage. If a tube has become blocked, flushing and suctioning with warm water may dislodge the obstruction. Other alternatives are listed in Box 44.5 [48]. If all fails, the blocked tube can be removed and replaced with a new tube. Epistaxis may occur during tube placement and is self-resolving unless a coagulopathy is

Box 44.5 Methods to Unclog Feeding Tubes [48]

- 5 ml warm water
- ¼ teaspoon of pancreatic enzymes with 325 mg sodium bicarbonate in 5 ml water
- 5 ml cranberry juice
- 5 ml carbonated beverages

Solutions may be injected with mild pressure and suctioned, or left in-situ for 5–120 minutes before flushing again.

present. Animals may also develop rhinitis or sinusitis. Patients with repeated removal of gastric fluid may trend toward a hypochloremic metabolic alkalosis. If frequent gastric emptying is performed via NG aspirations, daily acid–base evaluation should be considered [49].

Placement of the tube in the trachea is the most serious complication and can be life threatening. It is essential to confirm correct placement before initiating feeding, as described in Box 44.4. If there is coughing or discomfort during the pre-feed water flush or feeding, the planned feeding should not be continued, and a repeat lateral thoracic radiograph should be taken [34]. Once the proper tube position is confirmed, feeding can then be restarted at a smaller volume and slower rate of infusion. Other fatal complications include induced pneumothorax, intrapleural placement, and accidental intravenous infusions of enteral diets, but these are rare [50–54].

Esophageal Tubes

Placement and Verification

The equipment and placement technique of an esophagostomy (E) tube are shown in Protocol 44.2 and Figure 44.5 [29, 34, 55]. A modified technique has also been described whereby the distal tip of the tube is immediately fed into the caudal esophagus with the forceps, rather than being guided rostrally out of the mouth [56]. Radiographic confirmation of tube positioning is most common, but endoscopic visualization is also a viable method if it is conveniently available [34].

Protocol 44.2 E Tube Placement [29, 34, 55]

Items Required

- 8–14 Fr tube in cats and small dogs
- 14–20 Fr tube in medium to large dogs
- Mouth gag
- Mayo scissors
- Right-angled/curved forceps (Carmalt)
- #10 or 11 scalpel blade
- Nylon/polypropylene suture material
- Marker
- Luer slip catheter plug
- Conforming bandage/light bandage material

Procedure

1) Gather the equipment for E-tube placement (Figure 44.5a,b).
2) Anesthetize, endotracheally intubate, and position the animal in right lateral recumbency. Measure the tube from the center of the neck to the eighth to ninth intercostal space and mark with a permanent marker. Clip the left side of the neck and aseptically prepare the area (Figure 44.5c).
3) You may trim off the distal end of a red rubber tube to create a distal opening with the Mayo scissors (Figure 44.5d). This allows for future rewiring of the E tube with a weasel wire for replacements. Round and smoothen the cut edges of the distal opening with the Mayo scissors.
4) Insert the curved forceps (Figure 44.5e) into the esophagus until the midpoint in the cervical esophagus, and then push outward to tent the skin. Ensure that the jugular vein, carotid artery, and trachea are not overlying the incision site. Make an incision through the skin over the tip of the forceps and extend the incision through the subcutaneous connective tissues and wall of the esophagus. The incision should only be large enough to allow the tips of the forceps to be pushed through.
5) Grasp the distal end of the tube in the forceps, taking care not to catch the tissues in the mouth in the hinge of the forceps (Figure 44.5f).
6) Pull the distal end of the tube grasped in the forceps rostrally out through the mouth (Figure 44.5g).
7) Unclamp the distal end of the tube and gently push the distal end caudally with gentle digital manipulation. The forceps can be used to direct the tube into the esophagus. The proximal end of the tube can be manipulated to facilitate the progression of the distal end of the tube into the esophagus to prevent the tube from forming hard kinks that could remain during tube advancement (Figure 44.5h).
8) Once the tube is in place, the proximal end will flip from caudal to cranial. Check that enough of the tube has been inserted to the premeasured mark.
9) Confirm correct tube placement by radiographs. The outline of the tube should be visible and show the tube coursing dorsally to the carina and ending between the seventh to ninth intercoastal space (Figure 44.5i).
10) Place a loose purse-string suture around the tube entrance site (Figure 44.5j).
11) Place a finger trap suture around the base of the tube where it enters the skin to secure it in place (Figure 44.3k). At the author's institution, a second finger-trap suture is commonly placed on top of the first for added security (Figure 44.3l).
12) Wrap the E-tube comfortably around neck (Figure 44.5m).

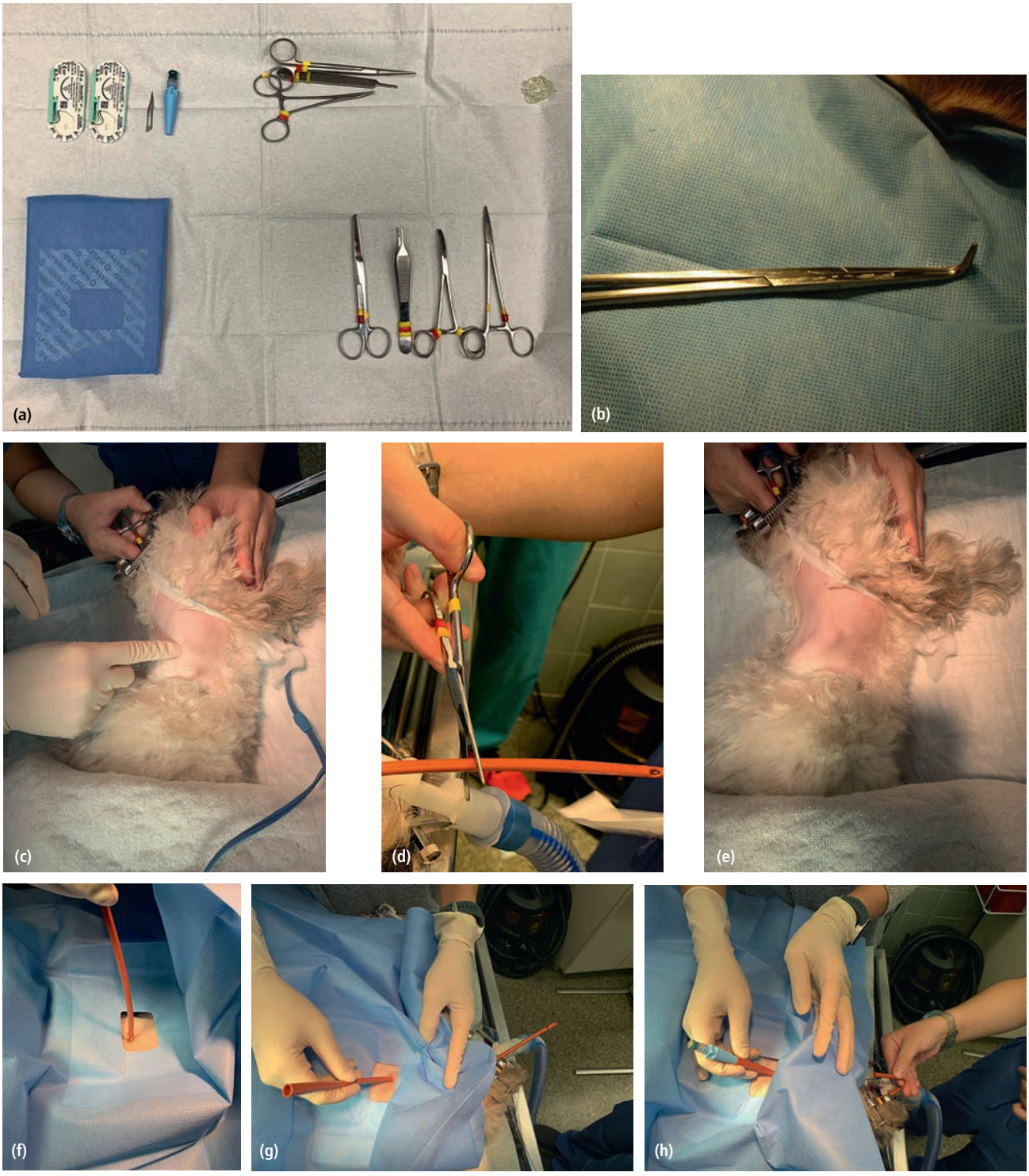


Figure 44.5 (a) Equipment for esophagostomy tube placement. (b) Curved Carmalt forceps. (c) Animal in right lateral recumbency and aseptic preparation of left neck. (d) Trimming of distal feeding tube. (e) Insert forceps into esophagus and push outwards to tent the skin. An incision is made over the tip of the forceps. (f) Distal end of tube grasped in the forceps. (g) Distal end of tube pulled rostrally out through the mouth. (h) Distal end of tube being pushed caudally with fingers while manipulating the proximal end. (i) Radiographic confirmation of correct positioning of esophageal tube dorsal to the carina and at seventh to ninth intercostal space. (j) Purse-string suture placement in skin. (k) Finger-trap suture placement on esophagostomy tube. (l) Appearance of double finger-trap suture placement. (m) Temporary esophagostomy tube wrapping with conforming bandage.

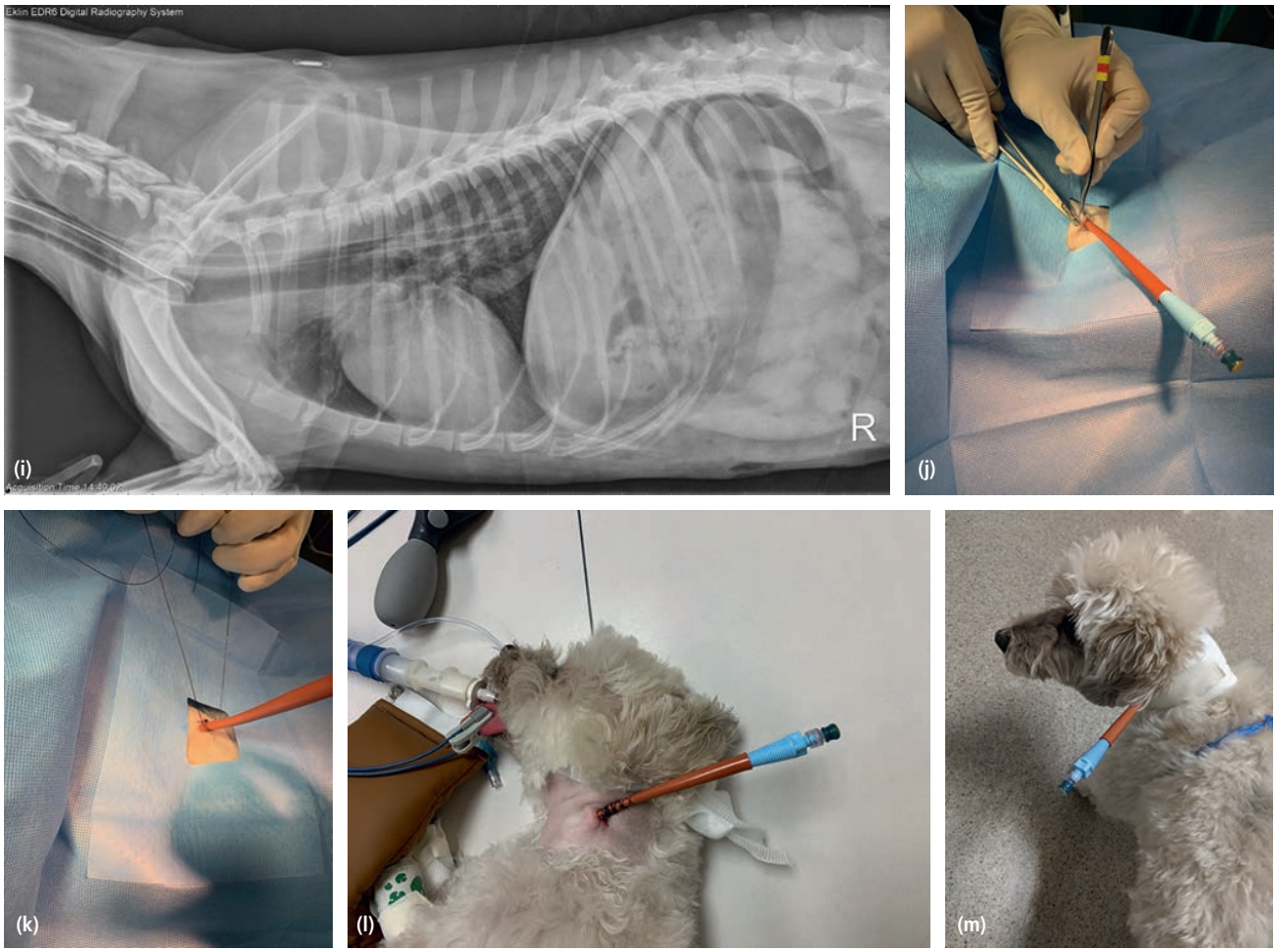


Figure 44.5 (Continued)

Feeding

Feeding may be initiated when the position of the tube has been confirmed and the animal has recovered from the anesthetic. The larger diameter of these tubes compared with the NE or NG tubes allows the use of blended or liquid diets [29]. The amount of water that needs to be added to the diet will depend on the tube size and the initial composition of the diet; however, enough water should be added to achieve a consistency that allows passage through a syringe tip. See Box 44.2 for calculating feeding amounts. As for NE and NG tubes, the tube should be flushed before and after feedings with 5–10 ml of warm water to prevent blockage. Warm the food to between room and body temperature prior to feeding and infuse slowly. Close the port at the end of the tube after feeding to prevent ingestion of air that may cause distension and discomfort in the stomach.

Stoma Site Care

The stoma site should be kept as clean and dry as possible at all times. Routine daily cleaning with sterile saline or a weak (0.05%) chlorohexidine solution should be performed. The site should be thoroughly dried after cleaning. Checking the stoma site for redness, swelling, heat, and discharge at least daily is recommended. For more information on the care of the stoma site, see Chapter 63. A light bandage (e.g. a conforming bandage or commercial fabric collar) may be placed, taking care not to occlude the airway.

Complications and Troubleshooting

Complications rates relating to E tubes are reported to be between 35% and 45% in dogs and cats and are mostly minor [57, 58]. The most frequent complications are tube dislodgements by patients and stoma site infections [57, 58]. Marking the skin exit point of a tube with a permanent marker when the position is initially radiographed can help to detect subsequent movement of the tube [59]. Minor dislodgements may only require readjustments and resuturing but could also require full replacement with a rewiring procedure under sedation or anesthesia. Stoma

site infections can be prevented by daily hygiene routines and monitoring. If serious cellulitis or abscessation develop, antibiotics are required, and surgical debridement may be indicated [57]. Changing the material of the tube can also be considered to reduce inflammation and risks for future infections [29].

Vomiting is another complication. If vomiting is witnessed, the oral cavity should be inspected for a possible tube dislodgement. If the tube has been dislodged, it should be removed and another placed. If the patient vomits, regurgitates, salivates, or shows discomfort during feeding, feeding should be stopped and repeat radiographs should be taken to ensure that the tube has not migrated caudally toward the lower esophageal sphincter or dislodged elsewhere. When feeding is resumed, feed a smaller amount at a slower rate, and ensure that food is freshly prepared and warmed prior to feeding.

Tube blockages can be prevented and addressed similarly to NE and NG tubes as mentioned earlier (Box 44.5). Only liquid medications should be delivered through the tube. Where tablets are required, these should be crushed to a fine powder, where possible, before mixing with water [59].

Severe complications, such as inadvertent placement in the trachea or periesophageal space, can be avoided with careful placement technique and verification of the location with radiographs (Figure 44.5i) [34].

Gastrostomy Tubes

Placement

There are two forms of gastrostomy (G) tubes: standard-length and low-profile. The standard tubes have a length of tubing extending from the skin when placed (Figure 44.6), while low-profile devices are designed to sit flush with the skin (Figure 44.7). The advantages and disadvantages of the two types of tubes are shown in Table 44.4 [60]. Low-profile devices are generally used to replace a standard percutaneous endoscopic gastrostomy (PEG) tube after three months when a permanent stoma has formed, but it is also an option to use one-step low-profile gastrostomy devices as the initial G tube [60–63].

G tubes may be placed surgically or percutaneously, with no differences in complication rates [64]. Percutaneous placement can be performed blindly or with the use of an endoscope [29, 34]. The equipment required for percutaneous endoscopic placement is shown in Box 44.6. Surgical placement of a G tube is often done when the animal is undergoing abdominal surgery for another reason and is described in surgical texts [65]. A modified surgical procedure has also been recently reported [66].



Figure 44.6 Standard gastrostomy tube.

Feeding

Water may be given within 6–12 hours of tube placement but feeding should not be initiated until 12–24 hours after placement to allow formation of a fibrin seal at the stoma [29, 34]. A permanent stoma will form in 10–14 days at the gastrocutaneous junction, after which time the tube may be removed [29, 33].

Diets of gruel consistency can be fed due to the larger caliber of the G tubes. To calculate the feeding amount, see Box 44.2. Warm food before feeding, infuse food slowly, and flush with 5–10 ml of warm water before and after feeding as with other enteral feeding tubes. Close the port at the end of the tube after feeding.

Stoma Site Care

G tube stoma site care is the same as E tube stoma site care, described earlier.

Complications and Troubleshooting

Complication rates vary widely between studies and are reported to be up to 46%, but are usually minor in nature [60, 64, 67, 68]. Complications that may occur during placement include gastric hemorrhage, splenic laceration, and pneumoperitoneum. Complications that may occur after tube placement include vomiting, aspiration pneumonia, inadvertent tube removal, peritonitis, gastric

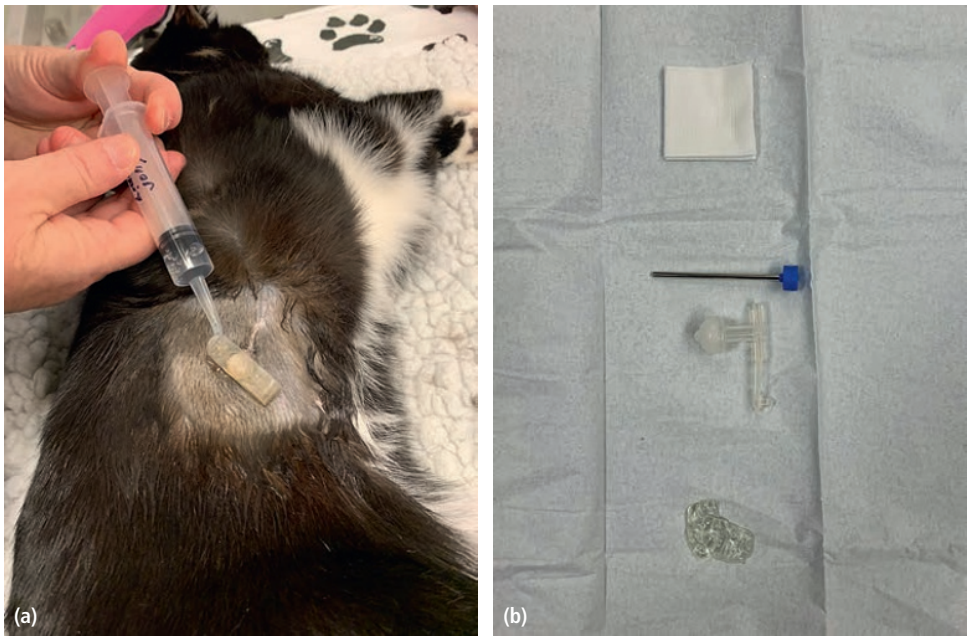


Figure 44.7 (a) Low-profile gastrostomy tube in a cat. (b) Low-profile gastrostomy tube before placement.

Table 44.4 Advantages and disadvantages of standard-length and low-profile gastrotomy devices [60].

	Standard length	Low profile
Positives	• Lower initial cost • Lower cost for short-term use • Well tolerated • Readily available in most clinics	• Less frequent replacement and reduced overall cost for long-term use • More esthetically pleasing to owners • Fewer complications (blockage, inadvertent removal)
Negatives	Higher risk of blockage and inadvertent removal	Higher initial cost

Box 44.6 PEG Tube Placement Equipment [29, 34]

- 20–24 Fr tube
- Endoscope
- Endoscope grasping instrument
- Scalpel blade
- 14- to 16-gauge needle or catheter
- 2-0 nylon suture material
- Catheter guide
- Sterile lubricant
- Luer slip catheter plug

pressure necrosis, tube migration, and infection of the tube site [15, 60, 64, 67–70].

Hesitations exist with the use of G tubes with specific groups of patients. Retrospective studies evaluating tube placement in dogs with septic peritonitis have shown minimal development of major complications and conclude that G tubes can be safely considered for this population of patients [68, 71]. One study suggests that patients on concurrent corticosteroids may have a higher risk of developing severe complications and advises careful consideration of tube placement in this group of animals [72].

Pressure necrosis can be prevented by ensuring that the tube can be rotated after it has been placed, with a 5-mm space between the skin and the external flange [73]. Marking the tube at the level of the skin on initial placement with a pen or tape can help to detect subsequent movement of the tube. An Elizabethan collar can be placed to help prevent tube removal by the patient. Where the tube is being removed by another pet in the household, a low-profile device should be considered.

If inadvertent removal or dislodgement occurs before a stoma has formed, surgical intervention is advised. 5–15 ml of iodinated contrast can be administered via the tube to confirm displacement [29]. The presence of contrast in the peritoneal cavity is suggestive of a gastric leak due to tube displacement. Interrogation with ultrasound is also an alternate option.

Tube obstruction may occur, although it is less likely in larger-diameter tubes, and it can be prevented by flushing before and after feeding, blending the diet with sufficient water, only using elixir medications, or thoroughly crushing medications and flushing well after administration. Use of sucralfate and antacids via enteral feeding tubes should be avoided, as they commonly precipitate and cause

blockages [29]. Methods to address a tube blockage are described in Box 44.5.

Most minor complications relate to irritation or infection of the stoma site. These can be minimized by routine cleaning and monitoring as described for E tubes. Systemic prophylactic antibiotics are not recommended but may be required with infections.

Jejunostomy Tubes

Placement

Jejunostomy (J) tubes are most commonly placed surgically, often in animals that are identified as requiring post-pyloric feeding and require surgery for other reasons. Surgical placement of J tubes is described in surgical texts [65]. The equipment used for surgical placement of J tubes is shown in Box 44.7. Laparoscopic-assisted placement of J tubes has also been used and is an option for J tube placement when the animal does not require a celiotomy for another purpose [74]. Other options include placements of a percutaneous endoscopic gastrojejunal tube, percutaneous radiologic gastrojejunostomy tube, endoscopically or fluoroscopically guided nasojejunal tubes, and esophagojejunal tubes [75–79]. Low-profile jejunostomy devices are also available and may be a feasible option for long-term nutritional support [80]. J tubes are recommended to be left in place for at least 7–10 days to allow for adhesions to form around the tube site, which help prevent leakage into the abdomen [29, 33].

Feeding

Feeding may be initiated within 24 hours after tube placement and are often started within two hours after anesthetic recovery [29, 75]. Owing to the small diameter of these feeding tubes, only a liquid enteral diet can be used. A CRI is recommended because bolus feeding may cause cramping and diarrhea [29]. See Box 44.2 to calculate the feeding amount to be provided each day (step 3), and then divide this amount by 24 (hours/day).

Box 44.7 J Tube Placement Equipment [65]

- 3.5 Fr for animals < 4 kg
- 5 Fr for animals 4–10-kg
- 8 Fr for animals > 10 kg
- #11 scalpel blade
- 4–0 absorbable suture material
- 2–0 nonabsorbable suture material
- Hemostat
- Luer slip catheter plug

Tube and Site Care

Flush the tube with water every four hours and after any disruption in CRI feeding [59]. The syringe and tubing (or other delivery equipment) through which the diet is delivered should be replaced every 24 hours to minimize bacterial growth [33]. J tube exit site care is the same as described earlier for other enteral tubes.

Complications and Troubleshooting

Common complications are vomiting, osmotic diarrhea, tube migrations, tube kinking, stoma erythema or cellulitis, accidental dislodgement, and tube obstruction [33, 81–83]. Gastrointestinal complications may be alleviated by decreasing the administration rate or by adding fiber to the liquid diet for diarrhea. Tube clogging can be minimized by using suitable diets, and by flushing the tube well every four hours and after any interruption in CRI feeding [33, 83]. Previous methods described for unblocking tubes may also be employed to relieve the obstruction. Alternatively, the tube may be replaced surgically or left in place until a stoma has formed while using parenteral nutrition.

Peritonitis is a serious complication that may occur from leakage of small intestinal contents at the enterostomy site from dislodgement or premature removal of the tube. In non-surgically placed tubes, retrograde movement may occur, which is best prevented by placing the tube as far into the small intestine as possible on initial placement [84].

Summary

- Adequate nutritional intake is required to support the immune system, wound healing, and intestinal structure and function.
- Enteral nutrition is the preferred route of nutrition if there are no contraindications.
- It is recommended to initiate early enteral feeding within 48–72 hours if there are no contraindications.
- RER is the minimum caloric goal for animals in the hospital.
- Use a strategic approach to entice voluntary consumption and reduce the risk of learned taste aversions.
- Tube selection is based on multiple factors, including the disease or condition being treated, functionality of gastrointestinal tract, anticipated length of feeding assistance, and cost of administration.

- Tube position should always be verified prior to initiating feeding.
- Tubes should be flushed with enough warm water to fill the tube prior to and after feeding to maintain tube patency.
- Ensure that an appropriate diet consistency is used to reduce the risk of tube blockage.
- Delivery of medications through E tubes and G tubes should be done with caution.
- Patients should be monitored closely during enteral meals for adverse reactions.

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45

Parenteral Nutrition

Jennifer Larsen

Malnutrition in hospitalized patients is an ongoing problem in veterinary medicine and is associated with poor outcomes. One study showed that hospitalized dogs and cats that received less than one third of their target energy requirements had a higher rate of poor outcomes [1]. Another study reported lower odds of dying in dogs that consumed their calorie requirements [2]. It is difficult to separate the impacts of more severe or chronic disease from the effect of nutritional support; however, overall the data in both humans and animals strongly suggest that early and adequate feeding promotes better outcomes. It is well known that illness and other physiologic stressors are associated with a hypermetabolic state characterized by increases in circulating cytokines, catecholamines, and other stress mediators, which then result in an inflammatory response with undesirable effects including increased protein catabolism and impaired healing ability [3, 4]. The preferential catabolism of lean body mass over glycogen and fat stores in animals that are critically ill has a profoundly negative impact on healing, immune function, and recovery. As such, timely intervention and provision of appropriate and adequate nutritional support is indicated.

Indications for Parenteral Nutrition

Feeding by the enteral route is the preferred method of providing energy and nutrients to maintain the functional integrity of the gastrointestinal tract and prevent dysfunction of the immune barrier [5–7]. The evidence from the veterinary literature shows early enteral nutrition is well tolerated [8–10] and is associated with improved outcomes [11, 12]. However, enteral feeding may not be possible in patients with an increased risk of aspiration or that are not candidates for feeding tube placement. Contraindications of gastrointestinal feeding may include protracted vomiting or regurgitation, decreased consciousness, and a decreased or absent gag reflex. Likewise, many patients with head trauma, those that need ventilator-assisted respiratory support, those requiring medications that impair consciousness, or those with severe pancreatic or malabsorptive gastrointestinal diseases sometimes cannot safely be fed enterally; however, nutritional support is still essential. In such patients, parenteral nutrition (PN) can be the only way to administer calories and nutrients (Figure 45.1).

When to Initiate Support

The ideal time to initiate nutritional support varies by individual and depends on nutritional status (current and serial body weights and when the patient last consumed adequate energy and nutrients), the disease process, and prognosis for voluntary intake. Assessment tools for assigning and monitoring body and muscle condition scores should be used routinely in the clinic for healthy pets as well as hospitalized patients (Figures 45.2, 45.3). For acutely ill or injured patients in good condition, nutritional support should be implemented within three to five days of anorexia. Longer periods of starvation are certainly of no benefit and carry the risk of negatively impacting immune function, healing, and overall condition [1, 2, 13]. For patients that are more debilitated, are growing, have inadequate muscle mass or adipose stores, have recent or continuing weight loss, or that are not expected to consume food voluntarily within two or three days, intervention should be more immediate. Feline patients that are at risk of hepatic lipidosis from inadequate energy consumption should also have their nutritional needs addressed within a shorter time frame. Please refer to Chapter 42 for specific guidelines. For all hospitalized cases, the initial medical management plan, as well as the owner's cost estimate, should consider the need for nutritional support.

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Figure 45.1 Mechanically ventilated patients are at higher risk of complications when fed enterally; parenteral nutrition can be used to provide support.

How Much to Feed

For all hospitalized patients, regardless of feeding method, the ultimate goal is the provision of resting energy requirement (RER) calculated for the current weight regardless of body condition. RER is the amount of energy needed by a resting, awake animal that is lying down in a thermoneutral environment. The RER is estimated by this equation (Box 45.1):

$$70 \times \text{body weight}(\text{kg})^{0.75}$$

This amount is adequate to maintain weight in a majority of hospitalized patients but may vary due to underlying disease and nutritional status. Regardless, overfeeding has risks and should be avoided. An initial target of RER allows assessment of patient tolerance during incremental increases in the PN infusion rate. Adjustments based on the individual patient's response are necessary.

Overfeeding can result in hyperglycemia and hyperlipidemia, which are also the most commonly recognized adverse effects associated with use of PN in veterinary patients [14–18]. Hyperglycemia has been identified as a risk factor for poor outcome in both human and feline patients [17, 19]; however, a causal relationship remains unclear and some evidence suggests that tight glycemic control is not ideal [20]. In addition, increases in blood glucose concentration that develop after PN is initiated typically normalize within one to four days without insulin therapy, especially in dogs [15, 18]. Hyperglycemia is a common feature of critical illness in both dogs and cats even without PN [18, 21]. In addition, insulin does not always achieve normoglycemia in canine and feline patients that develop hyperglycemia after initiation of PN [18], which suggests other mechanisms may be contributing.

Regardless, since hyperglycemia may be more likely with shorter times to increase PN to maximal rate [18], this may be at least partially controlled by starting with an initial infusion rate of 25% of RER (calculated to be administered over 24 hours). If this is well tolerated, the rate should be increased in 25% increments every 4–12 hours depending on patient response, until full RER is successfully reached (Box 45.2). Body weight should be assessed at least once daily, and if PN is used for a prolonged period without complications and weight loss is noted, slowly increasing the rate by 10–20% increments is reasonable.

Central and Peripheral Nutrition

Parenteral nutrition is sometimes called "total" or "partial." These terms refer to the completeness of the diet with respect to required nutrients or calories. In human medicine, PN is often used for prolonged periods, and the solutions include all required nutrients, including trace elements. In contrast, the average length of PN administration in veterinary patients is between three and five days [14–18], and veterinary PN formulations do not typically include the full complement of required nutrients. As such, the veterinary nomenclature is more accurate in reference to the route of administration, with central parenteral nutrition (CPN) being administered through catheters that terminate in the caudal or cranial vena cava, and peripheral PN (PPN) being administered through standard short catheters that terminate in peripheral vessels.

Both types of PN are appropriate for delivery of full caloric requirements, despite differences in energy density and osmolarity (Table 45.1). This can be achieved with PPN because of its higher fat content. Fat solutions contribute more calories per milliliter than protein and carbohydrate, but the osmolarity is much lower. Recommendations for maximum osmolarity values are 750 mOsm/l for PPN and 1400 mOsm/l for CPN [22]. This upper limit for PPN is intended to reduce the risk of phlebitis; however, other characteristics of the formula and its administration (especially pH and flow rate), as well as patient factors, also contribute to the risk of phlebitis. The higher limit for CPN formula osmolarity reflects the delivery into a large, high-volume and high-flow vessel, which results in rapid dilution of the solution.

Composition of the Solution

PN solutions are composed primarily of three base elements: amino acid solution, fat emulsion solution, and dextrose solution. The most commonly used amino acid solutions have concentrations of 8.5% or 10% and include



Figure 45.2 Canine and feline body condition score charts. *Source:* Courtesy of the World Small Animal Veterinary Association (WSAVA), https://wsava.org/global-guidelines/global-nutrition-guidelines (accessed 20 September 2022).



Figure 45.2 (Continued)

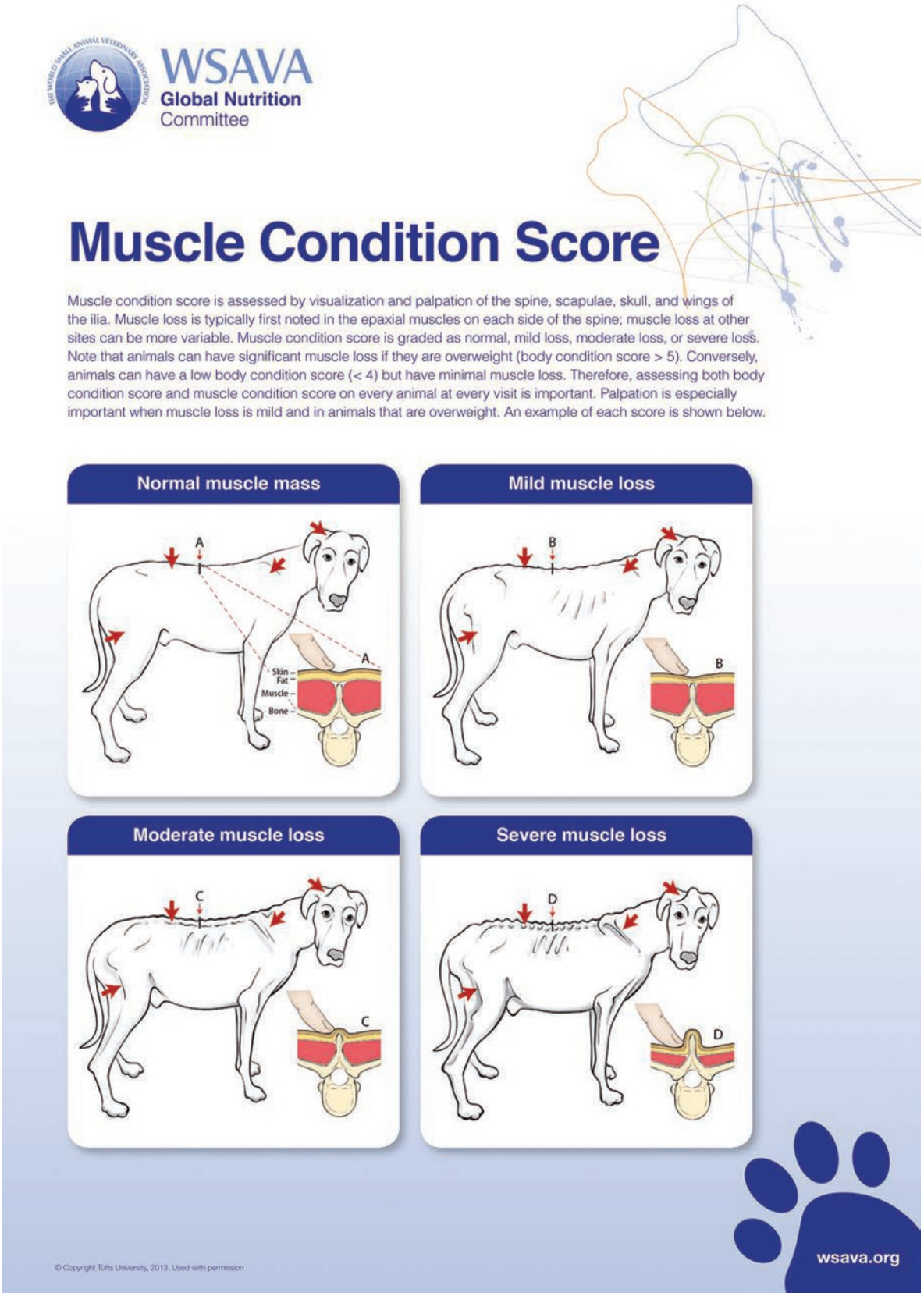


Figure 45.3 Canine and feline muscle condition score charts. *Source:* Courtesy of the World Small Animal Veterinary Association (WSAVA), https://wsava.org/global-guidelines/global-nutrition-guidelines (accessed 20 September 2022). © Tufts University, 2014.

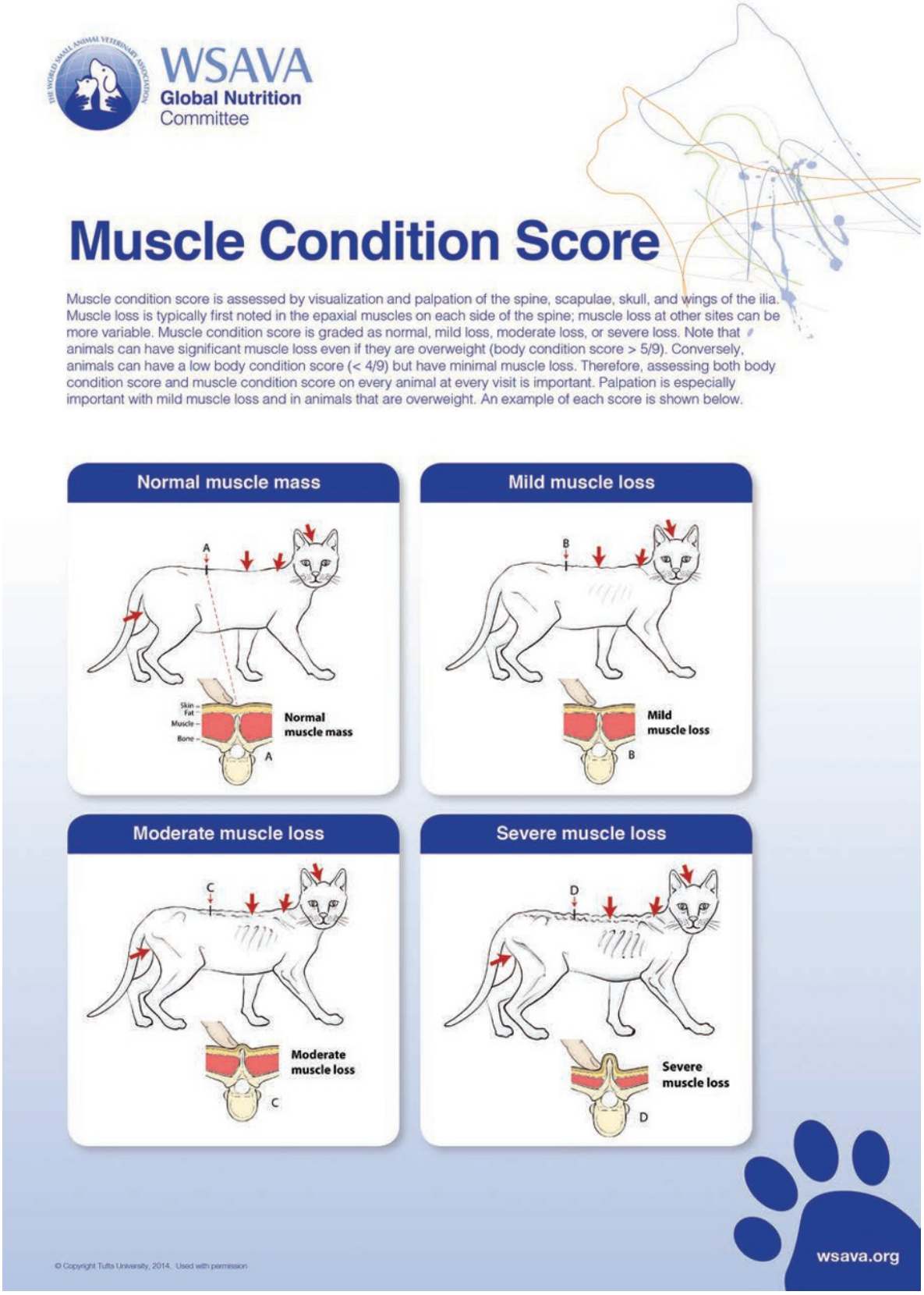


Figure 45.3 (Continued)

Box 45.1 Examples of Calculations for Meeting Energy Requirements with Parenteral Nutrition

Equation for resting energy requirement (RER):

$$70 \times (\text{body weight in kg})^{0.75} = \text{RER in kcal / day}$$

For a 35-pound (15.9 kg) canine patient, calculate RER:

$$70 \times 15.9\,\text{kg}^{3/4} = 557\,\text{kcal / day}$$

$$557\,\text{kcal / day} / 1.15\,\text{kcal / ml}$$

$$= 484\,\text{ml / day}\,484\,\text{ml / day / 24 hours}$$

$$= 20\,\text{ml / hour}$$

If the parenteral solution provides 1.15 kcal/ml, the calculations above demonstrate that the patient needs approximately 484 ml/day or 20 ml/hour to achieve full RER. This final RER goal is generally attained by starting at a fraction of the final goal infusion rate and increasing it over 2–4 days to ensure tolerance.

Box 45.2 Guidelines for Increasing to Goal Rate of Parenteral Nutrition Infusion

Adapted from Campbell et al. [22].

When increasing the infusion rate up to full resting energy requirement, start with 25%–33% of the rate, and increase by 25–33% increments every 4–24 hours if tolerated, with blood glucose within target ranges (between 100 and 250 mg/dl).

Assess blood lipids, phosphorus, potassium, magnesium, and hematocrit within 24 hours of initiation of parenteral nutrition.

After full infusion rate is achieved, assess at least once daily:

- Body weight
- Catheter insertion site
- Plasma lipemia index
- Blood glucose concentration
- Magnesium concentration
- Thoracic auscultation
- Rectal temperature
- Hematocrit

Assess at least every other day:

- Phosphorus concentration
- Potassium concentration
- Blood urea nitrogen concentration
- Albumin concentration

Table 45.1 General characteristics of central and peripheral parenteral nutrition (PN)

Characteristic	Central PN	Peripheral PN
Catheter termination	In vena cava	In peripheral vessel
Osmolarity	<1400/l	<750/l
Energy density	1.0–1.4 kcal/ml	0.7–1.0 kcal/ml
Fat content	~50% of calories	~70% of calories
Delivery of energy requirement	Yes	Yes

all amino acids required by dogs and cats except taurine. Lipid emulsion products are primarily composed of long-chain polyunsaturated fatty acids from plant oils. This ingredient is iso-osmolar and can be used in high concentrations in PN while contributing little to the osmolarity of the overall solution. There is some interest in the use of omega-3 fatty acid-containing infusions in veterinary patients, but these are not clearly beneficial and not commonly in use at this time [23]. Dextrose solutions are 5% or 50% concentration products typically found in veterinary pharmacies. In this context, dextrose is the injectable form of the 6-carbon monosaccharide glucose with an attached water molecule (D-glucose monohydrate).

Additives providing electrolytes, B vitamins, and trace minerals may also be included in the PN formulation. The inclusion of B vitamins in particular is important. With the exception of a small amount of cobalamin in the liver, there is no storage of B vitamins in the body. These are important nutrients for metabolism and efficient use of energy, protein, fat, and glucose. Further, B vitamins can be lost in the urine due to their water-soluble nature; this is an important consideration for patients with polyuria secondary to their underlying disease or due to parenteral fluid administration. Other micronutrients are also critical, and some amino acid solutions include electrolytes; however, many clinicians prefer to adjust these more precisely for individual patients by using additives such as potassium phosphate, potassium chloride, magnesium sulfate, and sodium chloride. Any of these can be added to the PN solution or to a crystalloid fluid solution. Other components including medications should not be introduced into the PN solution unless compatibility can be assured to avoid adverse effects; consultation with an experienced pharmacist is appropriate.

Formulations can also be customized in other ways, with modification of the macronutrient energy distribution of the solution the most common. For example, low-carbohydrate solutions may be useful for patients with compromised pulmonary function and hypercapnia, and

low-protein solutions are used for patients with hepatic encephalopathy or kidney disease. For some patients with severe hepatic disease, fat may also be poorly tolerated. Provided that blood lipid clearance is normal, parenteral fat infusions are generally safe for patients with pancreatitis because exocrine pancreatic stimulation results from nutrients in the small intestine. To some extent, the osmolarity and energy density of the solution can also be adjusted to address issues such as phlebitis or volume intolerance.

Compounding Parenteral Nutrition Solutions

Aseptic procedures are required for safe compounding of PN solutions (Protocol 45.1). To ensure a safe and quality product, the solution must remain both sterile and stable. Because several ingredient components are added to the solution, multiple needlesticks are used and contamination is possible. Further, the solution itself is a good medium for bacterial growth. The stability of the solution may be impacted not only by microbial contamination but also by storage or transport conditions. Many nutrients are sensitive to temperature, light, and oxygen (Box 45.3). Degradation or destruction of the solution constituents is possible, for example fatty acid oxidation.

Solution stability may also be impacted due to mixing procedures. Some components of PN solutions may interact with others, or react with additives, which may result in instability of the product. For example, the dextrose and amino acid components should be mixed to allow for pH equilibrium and dilution of cations prior to adding the lipid solution. Alternatively, all three components can be added simultaneously, with gentle agitation to ensure proper homogeneity of the solution. Care should be taken to avoid

Box 45.3 Factors Influencing Stability of Parenteral Nutrition Solutions

- Microbial contamination
- Temperature
- pH
- Light
- Oxygen exposure
- Continuous agitation of solution
- Lipid emulsion stability

Protocol 45.1 Protocol for Compounding Parenteral Nutrition

Items Required

- Isolation chamber or laminar-flow hood (Figure 45.4)
- Empty, sterile, and unused intravenous (IV) solution bags with attached transfer tubing set and tubing clamps
- Alcohol swabs for cleansing injection ports
- Needles and syringes for additives
- Amino acid solution, lipid emulsion solution, dextrose solution, vitamin B complex, electrolyte additives (potassium chloride or potassium phosphate, magnesium sulfate)
- Sterile gloves

Procedure

1) Gather supplies.
2) Always use aseptic technique.
3) Perform hand hygiene and don sterile gloves.
4) Assemble all necessary components and supplies in the chamber or hood (Figure 45.4).
5) Hang the dextrose and amino acid solutions, swab the ports with isopropyl alcohol and allow to dry completely, and connect the tubing from the empty IV bag to each container.
6) Open the tubing to mix the specified volumes of dextrose and amino acid solution in the empty bag, and then add the lipid emulsion (or add all three simultaneously with gentle agitation to ensure thorough mixing; do not mix dextrose and lipid together).
7) Clamp and remove the transfer tubing from the IV solution bag.
8) Add any desired additives (potassium phosphate or potassium chloride, B vitamin complex).
9) Limit needlesticks as much as possible.
10) If solution will not be used immediately, omit the additives for refrigerated storage and add them just prior to use, using aseptic technique.

the mixing of dextrose and lipid solutions together. The amino acid solution will buffer the low pH of the dextrose solution and protect the lipid component, which is sensitive to excessive acidity as well as higher concentrations of reactive cations such as ionized calcium and magnesium.

PN solutions without additives can be premade and stored in the refrigerator for convenience, with the addition of a vitamin B complex injection to the solution just before beginning the infusion. Premade bags without additives may be stable for up to 28 days when stored at refrigerated temperature [24]. If the lipid becomes unstable (referred to as "breaking" or "oiling out"), visible separation, yellow streaking, and/or precipitation of particulate matter can be noted in the solution. PN solutions should always be visually examined prior to infusion and at regular intervals thereafter to assess for breaking and other stability problems. If signs of instability are noted (Box 45.4), the animal is at risk of adverse events related to lipid emboli, and the solution should be discontinued and discarded.

There are currently no formal practice guidelines regarding the use of PN from the American Veterinary Medical Association or many state veterinary medical boards. However, as of 2008 the United States Pharmacopeia (USP) Chapter 797 is enforceable by the US Food and Drug Administration and has been adopted by most state pharmacy boards [25]. This statute describes the procedures and requirements for compounding sterile preparations and applies to all settings, veterinary or otherwise. The stringency of enforcement may vary; however, these procedures and guidelines should be followed in all veterinary practices and pharmacies to ensure patient safety.

Among other restrictions and guidelines, USP Chapter 797 guidelines specify the use of a clean room or isolation chamber (such as a laminar flow hood) to prepare sterile parenteral products (Figure 45.4). For most veterinary practices, the feasibility of compliance is challenging if not impossible, and options for in-house preparation are limited. Large practices or academic institutions typically use isolation chambers or laminar flow hoods for hand mixing or automatic compounding machines to mix parenteral solutions (Figure 45.5). Automatic compounders reduce human error, increase efficiency, and improve accuracy of the measurements of each component, but these are not cost effective for most veterinary practices. However, many human home healthcare pharmacies and hospitals can provide parenteral mixtures for use in almost any veterinary practice. A prescription can be submitted that specifies the type and amounts of each component to create an appropriate parenteral solution. The product can be sent the same day or overnight to the veterinary practice, making this option more practical, albeit still potentially costly.

Box 45.4 Signs of Lipid Instability Indicating an Unsafe Parenteral Nutrition Solution

- Any change in color of solution from the typical white or off-white resulting from addition of B vitamins
- Yellow steaks in solution
- Visible oil layer in solution
- Appearance of particulate matter or "clumping" in solution
- Any loss of appearance of homogeneity of solution



Figure 45.4 To avoid contamination, parenteral nutrition solutions should be mixed in a sterile environment such as an isolation chamber or laminar-flow hood.



Figure 45.5 Hand mixing of PN solution in a laminar flow hood.

Maintenance of the Infusion and Catheter

Peripherally inserted central catheters are often used in veterinary patients for the administration of CPN; these are inserted in the limbs but are long enough to terminate in

the caudal or cranial vena cava. These devices may be more difficult to keep clean compared with those in other locations, especially if the bandaging is exposed to urine or feces. Shorter CPN catheters inserted into the jugular vein and that terminate in the cranial vena cava are also common; these typically have multiple lumens so that a dedicated line is possible. Dedicated lines are strongly recommended for PN administration to avoid incompatibility issues with other infused substances, minimize contamination, and maintain line integrity.

Catheters intended for PN infusion use should be placed aseptically, using standard techniques for skin preparation including clipping and proper cleansing. Procedures should also include the use of appropriate barriers (drapes, gloves), as well as proper handling of all equipment (see Chapter 7). Catheter maintenance during the infusion period should follow accepted protocols for cleansing and bandaging to avoid contamination and infection (see Chapter 63). The insertion site should be visually inspected for signs of inflammation and cleansed every 12–24 hours. Catheter removal may be necessary if swelling, redness, or discharge is present.

Owing to the sensitivity of some nutrients to ultraviolet light, the solution bag and infusion lines should be kept covered. Amino acids and B vitamins are most susceptible to such degradation. After the addition of B vitamins and gentle mixing of the solution, the solution is stable at room temperature during the infusion period for at least 48 hours; continuous agitation is not necessary and may destabilize the solution [26]. The bag should be labeled with the date and time it was hung to ensure that it is discarded after 48 hours along with the entire infusion tubing set. Once the infusion has started, any disconnection of the line should be prevented to maintain sterility (Figure 45.6). In all cases this means that the bag and line must accompany the patient on walks outside, trips to undergo diagnostic procedures, and to visits with owners. If the line is disconnected anywhere between the bag and the patient's intravenous catheter, the administration set and solution are no longer considered sterile and must be discarded.



Figure 45.6 The catheter should be wrapped to protect it from contamination or damage and should only be disconnected to replace the infusion lines and bag.

Contraindications and Complications

PN should be used cautiously in patients that are volume intolerant. The energy density of the solution is the primary determinant of the infusion rate; if the rate must be decreased to address volume intolerance, full RER may not be delivered. Regardless, the provision of partial energy requirements is preferable to none. Patients with vasculitis or that are septic or hypercoagulable are not ideal candidates for PN. If PN is instituted is such a patient, additional monitoring and precautions may be necessary to reduce complications. All patients should be monitored for metabolic, mechanical, and septic complications (Box 45.5).

Metabolic Complications

Commonly Reported Metabolic Complications

Metabolic complications of PN are commonly recognized in veterinary patients, amounting up to 70% of total recorded PN-associated complications in dogs in one study [15]. Although hyperglycemia is common in dogs and cats receiving PN, and has been associated with poor outcome in cats in one study [17], other studies did not show an association between hyperglycemia and mortality

Box 45.5 Examples of Complications Associated with Parenteral Nutrition Administration

- Metabolic:
  - Abnormalities in serum biochemical values (e.g. hyperglycemia, hyperbicarbonatemia)
  - Lipemic serum (or elevated serum triglyceride concentration)
  - Refeeding syndrome
- Mechanical:
  - Catheter dysfunction (dislodgement, occlusion, kinking)
  - Disconnection or leakage of the infusion line
  - Inadvertent removal by the patient
  - Equipment failure (pump dysfunction, etc.)
- Septic:
  - Inflammation at catheter insertion site
  - Fever
  - Elevated white blood cell count
  - Positive culture of blood or catheter

in either dogs or cats [16, 18]. As previously discussed, critically ill cats show abnormalities in carbohydrate metabolism associated with hyperglycemia, hyperlactatemia, and hypoinsulinemia [21], which may contribute to intolerance of dextrose-containing infusions of PN. Despite the consistently high incidence of hyperglycemia reported in veterinary patients both prior to and during PN infusion, the importance of this remains unclear.

Intolerance of Parenteral Nutrition

Patients that do not appear to tolerate PN (Box 45.6) due to hyperlipidemia, hyperglycemia, or hyperammonemia should be assessed for new or progressing underlying conditions; however, the first steps should include confirmation of proper formulation and mixing of the solution, given that even minor errors may result in a drastically different nutritional profile than intended. The formulation calculations should be rechecked, as well as the volume and specifications of the individual components. Once the composition of the solution has been confirmed, measures can be taken to reduce adverse effects. In all cases the infusion rate should be decreased or the infusion discontinued if the adverse effect is severe. In some cases, insulin can be used to control hyperglycemia, and unfractionated heparin can be used to induce lipoprotein lipase, which increases peripheral uptake of lipids to address hyperlipidemia.

Refeeding Syndrome

Another potential metabolic complication is refeeding syndrome, which can be seen in any animal fed enterally or parenterally after a period of reduced food intake. The syndrome is characterized by hypophosphatemia, hypokalemia, hypomagnesemia, thiamine deficiency, and fluid imbalances. During starvation and critical illness, reserves of metabolically important compounds are depleted, and the main energy sources are fatty acids, ketone bodies, and amino acids. Despite typically normal serum concentrations, the whole-body pool of nutrients such as phosphorus, potassium, magnesium, and thiamine is significantly reduced. When calories are delivered by any route, especially from digestible carbohydrate including glucose, the pancreas responds by releasing insulin, and there is an abrupt shift in the substrates used for energy production as glucose becomes available.

Box 45.6 Signs of Parenteral Nutrition Intolerance

- Hyperglycemia
- Hyperammonemia
- Hyperlipidemia
- Fluid overload without other identifiable cause

The binding of insulin to its receptors in peripheral tissues results in an intracellular phosphorylation cascade. This in addition to the upregulation of glycolysis and subsequent production of adenosine triphosphate (ATP; the main energy source for cellular functions) creates a sink such that phosphorus moves from the extracellular into the intracellular compartment, which depletes the extracellular (interstitium, plasma) compartment; this can result in clinical signs of phosphorus deficiency, including hemolysis and anemia.

Insulin also promotes a dramatic shift of potassium from the extracellular to the intracellular compartment, primarily due to the transmembrane sodium–potassium ATPase pump, the action of which is essential for nerve impulse transmission and muscle function as well as the action of sodium-coupled glucose transport proteins. The sodium gradient across the cellular membrane enables inward movement of glucose and is maintained with intracellular movement of potassium. As such, insulin stimulation of sodium-potassium ATPase can reveal poor whole-body potassium status in many malnourished patients. This can result in clinically significant hypokalemia manifested by hypotension, neuromuscular dysfunction including weakness and intestinal ileus, cardiac arrhythmias, and cardiac arrest.

Likewise, magnesium is an important cofactor for the first steps in glycolysis, so that when glucose is being used to produce ATP, there is a sink for magnesium in the intracellular space. Deficiency results in cardiac arrhythmias and neuromuscular signs. Assessment of magnesium status in veterinary patients remains a challenge. There is no consensus regarding the most accurate yet practical method, given that whole-body magnesium status is not reliably reflected by serum measurements. Current recommendations suggest that either serum ionized or total magnesium concentrations are most useful if low; however, when values are normal, clinical suspicion may still support diagnosis of deficiency [27]. Hypokalemia may be refractory to treatment with parenteral supplementation unless adequate magnesium concentrations are restored because magnesium closes passive potassium channels in cellular membranes.

Lastly, thiamine is important for the metabolism and use of carbohydrates for energy, and many patients have suboptimal thiamine status from decreased intake and/or increased loss. When demand increases during refeeding, subclinical or overt thiamine deficiency may result, including severe neurologic abnormalities.

Unlike in people, refeeding syndrome appears to be uncommon in dogs and cats, and usually causes mild to moderate adverse events in veterinary patients. However, in some cases the problem can result in catastrophic complications. Anemia requiring transfusion in 7 cats and acute

kidney injury affecting 6 cats was recently reported in a case series of 11 cats with refeeding syndrome; only 8 cats survived to discharge and only after being hospitalized for a mean of 14 days [28]. Given the potentially serious sequalae, monitoring and ideally preventing such occurrences should be implemented for any at risk cases. Patients with preexisting metabolic derangements such as diabetic ketoacidosis or those with a prolonged history of malnourishment are likely at higher risk and should be assessed closely. Initiation of either enteral or parenteral nutrition should be done conservatively, with very gradual increases in the amount provided to assess tolerance and address any potential problems very early (no more than 25% of RER for at least six to eight hours, in this author's opinion). When identified and promptly addressed, actions to correct these issues may improve outcome and reduce morbidity and mortality associated with this useful treatment modality.

Mechanical Complications

Mechanical complications include catheter dysfunction (dislodgement, occlusion, kinking), disconnections in the infusion line, and inadvertent catheter removal by the patient. Such situations occur in 9–26% patients receiving PN [15–18]. Preventing patient access to the catheter site, maintaining appropriate bandaging, and instituting procedures to maintain line integrity during patient care can help reduce the incidence of mechanical complications. Although infusion line obstructions are not commonly encountered, administration sets are available that include inline filters. Occlusions may be due to coalescing fat globules that would indicate an unstable or broken solution. The PN solution should be carefully inspected in the event of an occlusion because significant adverse effects may occur if a broken lipid solution is infused.

Septic Complications

Owing to the nature of the parenteral solution and the need for direct intravenous access, patients receiving PN are at higher risk of infectious complications. However, septic complication rates in clinical canine and feline patients receiving PN solutions range from 0% to 8% and are typically the least common types of complication identified [14–18]. In addition, septic complications are either not clearly associated, or are statistically not associated with mortality [14–18]. Regardless, all patients receiving PN solutions should be monitored for catheter insertion site infection, fever, and leukogram abnormalities. If catheter-related sepsis is suspected, the catheter should be removed, and blood or other appropriate culture techniques performed to institute appropriate antimicrobial therapy.

Conclusion

PN can be a useful modality to provide nutritional support to critically ill patients unable to tolerate enteral feeding. Proper formulation, compounding, administration, and patient monitoring are necessary to ensure the delivery of a safe and effective product. Metabolic complications are the most frequently documented adverse effects; septic complications are uncommon. Nutritional support using PN solutions can be safely and effectively implemented in most 24-hour veterinary practice settings.

Summary

- Critically ill patients are often malnourished or are consuming inadequate diets.
- When enteral feeding is impossible or impractical, provision of nutritional support via the parenteral route is indicated.
- Parenteral feeding is an excellent option for providing adequate amounts of energy to patients with a wide range of needs.
- Parenteral feeding can be easily employed in most practice settings with 24-hour monitoring.

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Section Six

Analgesia and Anesthesia

46

Drug Administration

Damion Asselin and Jane Quandt

The patient that undergoes treatment and monitoring in the intensive care unit (ICU) setting receives numerous drug therapies. How these therapies are administered is crucial to the success of the treatment and the wellbeing of the animal.

Treatment Sheet Orders

All patients in intensive and intermediate care should have an order sheet (Figure 46.1) delineating the treatment requested, the dose, the route of treatment administration, and frequency of administration. The order sheet is also the place to note any potential interaction that may occur between drugs. For example, a dog on a constant rate infusion (CRI) of diazepam should not receive an intravenous (IV) antibiotic through the same IV line as the diazepam. Diazepam is incompatible with other drugs and would form precipitates (small particles) in the line. The written order for the treatment drugs should match the label from the pharmacy in that it is the same name, be it generic or a trade name; for example, Torbugesic® (Zoetis) is the trade name of butorphanol. The name on the order sheet and the name on the drug when it is received from the pharmacy should be one or the other, or both names, to avoid confusion. When a treatment is administered to the animal, the initials of the person giving the medication should be placed next to the treatment time. When questions arise, initialized treatments allow for tracking of the individual responsible for the action.

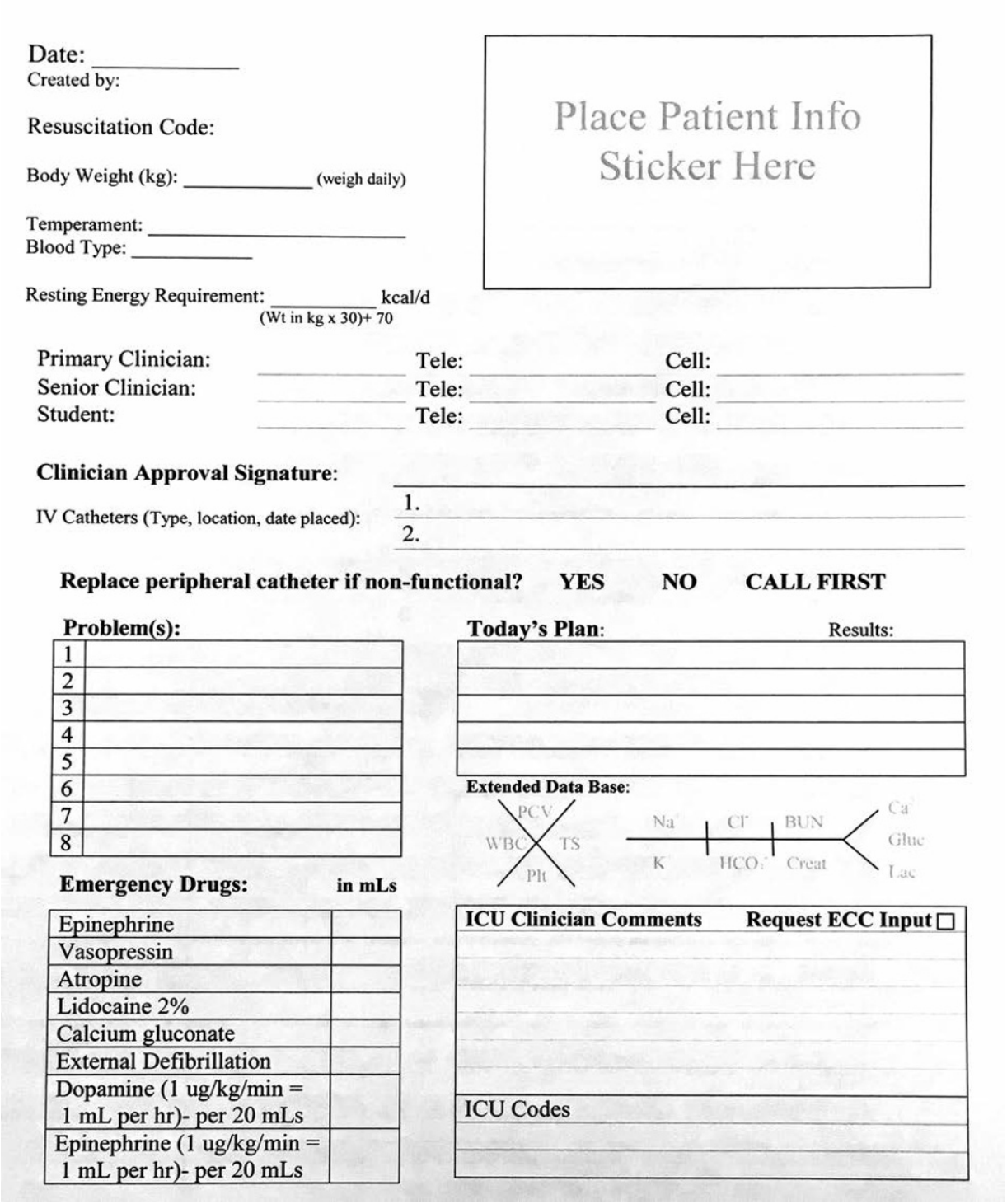
Medical Records

All administered medications should be clearly noted in the medical record, whether it is paper or electronic. Any known allergies or adverse drug reactions should be flagged with either a red sticker on the outside of the paper record or an electronic warning on computer records. This indicator is vital to prevent the administration of a drug or a blood product to which the patient has reacted in the past. Either a prominent note should be made on the treatment sheet or a sign should be posted on the patient's cage so that everyone in contact with that patient is made aware of the patient's drug sensitivities.

The pharmacy should also be a source of safeguards against drug administration error. Pharmacy personnel should check medication requests for accuracy of dosing and administration route. To prevent accidental overdose, single drug doses should be dispensed, each in its own labeled syringe, unless the medication comes in a multiuse vial or container.

It is common in veterinary medicine to use extra-label drugs. This is the use of an approved drug in a way that is not in accordance with the manufacturer's approved instructions. Animals have diseases that require treatment with agents that have been registered for use only in human patients. When an animal-approved drug product to treat a condition is not available, extra-label use may be authorized when the health of the animal is threatened, the patient is suffering, or death may occur without the treatment. The Animal Medicinal Drug Use Clarification Act explains extra-label drug use. It states that a veterinarian must be involved; only Food and Drug Authority-approved drugs are to be used; a client–veterinarian relationship must exist; the drug must be for therapeutic use only; and there are to be no residues that may present a risk to public health. When using an extra-label drug, it is important to fully document the dose, route, administration times, disease being treated, and any withdrawal times. It is not a requirement that the client be told it is an extra-label use of a drug.

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Companion website: www.wiley.com/go/burkitt/monitoring



Date: _____
Created by:

Resuscitation Code:

Body Weight (kg): _____ (weigh daily)

Temperament: _____
Blood Type: _____

Resting Energy Requirement: _____ kcal/d
(Wt in kg x 30)+ 70

Place Patient Info Sticker Here

Primary Clinician: _____ Tele: _____ Cell: _____
Senior Clinician: _____ Tele: _____ Cell: _____
Student: _____ Tele: _____ Cell: _____

Clinician Approval Signature: _____

IV Catheters (Type, location, date placed): 1. _____ 2. _____

Replace peripheral catheter if non-functional? YES NO CALL FIRST

Problem(s):

1	
2	
3	
4	
5	
6	
7	
8	

Today's Plan: **Results:**

Extended Data Base:

PCV, WBC, TS, Plt

Na, Cl, BUN, Ca, K, HCO_3, Creat, Gluc, Lac

Emergency Drugs: **in mLs**

Epinephrine	
Vasopressin	
Atropine	
Lidocaine 2%	
Calcium gluconate	
External Defibrillation	
Dopamine (1 ug/kg/min = 1 mL per hr)- per 20 mLs	
Epinephrine (1 ug/kg/min = 1 mL per hr)- per 20 mLs	

ICU Clinician Comments **Request ECC Input** ☐

ICU Codes

Figure 46.1 Intensive/intermediate care unit order-treatment sheet examples. *Source:* Reproduced with permission from the University of Georgia Veterinary Medical Center.

Routes of Drug Administration

The route of drug administration must be appropriate to prevent serious adverse effects or even death. Drugs can be given by various routes, but deviation from an established safe route could lead to severe complications, including death. The route of administration should be specified in the order sheet, and the drug must be verified as having been administered in the manner requested. When using a multi-use vial, the top of the vial should be cleaned with an alcohol swab prior to insertion of the needle to prevent possible contamination of the bottle. In the same regard, prior to drug

Patient name:_____

	Order	Parameter	S O	8:00	9:00	10:00	11:00	12:00	1:00	2:00	3:00	4:00
VITALS	q h	Temperature, Fahrenheit call if: > <										
	q h	Pulse rate/quality (Strong, Fair, Poor) call if: >___ <___										
	q h	Respiration rate/quality (Eupnea, Dyspnea, Tachypnea, Shallow) call if :										
	q h	CRT & m memb (Red, Pink, pAle, White, Cyanotic) call if:										
	q h	Mentation (Alert, Depressed, disOriented, Stuporous, Comatose) call if:										
MONITORING	Each shift	Flush catheter and palpate vein										
	q 24 h	Unwrap and inspect catheter insertion site										
	q 2 h	Patient check and update										
		Urine output (mL this hr/total) or 1-4+		0/0								
		Defecation (Normal, Diarrhea, Melena, 1-4+ for quantity)										
FLUID rate	mL/hr			0/0								
				0/0								
				0/0								
				0/0								
TREATMENTS		Drug, Dose (mg/kg), Total dose (mg), mLs (or tabs), route										
DIET		Water:										
		Food (type, how much, location)										
		Appetite (Good, Fair, Poor, None)										
LABDATA		PCV call if:										
		TS call if:										
		Glucose call if:										
		I-stat / NOVA call if:										
		Other:										
		Other:										
	Comments: number chronologically and write on back											
	SIGNATURE: First initial & last name of person doing treatments											

Figure 46.1 (Continued)

administration into an IV catheter port, the site should be cleaned with an alcohol pad. For convenience, the swabs should be kept in multiple locations throughout the ICU.

The most common route of drug and fluid administration in the ICU setting is via an indwelling IV catheter. Many of the medications given in the ICU can be delivered through the Y-port of the IV fluid line. Compatibility with the fluid and additives must be established prior to drug administration through any IV port. When using the same fluid line for incompatible drugs, the line must be flushed between the administrations. The flushing can be done with 0.9% NaCl or the fluid pump on the line can be allowed to run for 10 minutes before administering the second drug, to allow time for the first drug to be moved through the line. Incompatibilities may range from immediate precipitation (formation of particles) to an undetectable but potentially dangerous pH change. A compatibility chart of commonly used drugs in the ICU can be found in Table 46.1.

Table 46.1 Compatibility chart for commonly used drugs in the ICU. The following are the sources for this chart:

Compatibility chart	Aminophylline	Ampicillin	Atropine	Buprenorphine	Butorphanol	Calcium gluconate	Cefazolin sodium	Dexamethasone SP	Diazepam	Diltiazem	Diphenhydramine	Dobutamine	Dopamine	Doxycycline	Epinephrine	Famotidine	Fentanyl	Furosemide	Heparin
Aminophylline	c	c	c			c	c	c	x	?	x	x	c		x	c		c	c
Ampicillin	c	c				?			x	?	x	x	x	x	x	c		c	?
Atropine	c		c		c				x		c	c				c	c	c	
Buprenorphine				c															
Butorphanol			c		c				x		c	c				c	c	x	c
Calcium gluconate	c	?				c	?	?				x			c	c		c	c
Cefazolin sodium	c					?	c		x	c	x	x	x	x		?		?	c
Dexamethasone SP	c					?		c	x	c	x	x	c	x		c		c	c
Diazepam	x	x	x		x		x	x	c	x	x	x	x	x		x		x	x
Diltiazem	?	?					c	c	x	c		c	c	c	c		c	x	?
Diphenhydramine	x	x	c		c		x	?	x		c					c	c	x	?
Dobutamine	x	x	c		c	x	x	x	x	c		c	c		c	c	c	x	x
Dopamine	c	x					x	c	x	c		c	c		c	c	c	x	c
Doxycycline		x					x	x	x	c				c				c	c
Epinephrine	x	x				c				c		c	c		c	c	c	c	c
Famotidine	c	c	c		c	c	?	c	x		c	c	c		c	c		x	c
Fentanyl			c		c					c	c	c	c		c		c	c	c
Furosemide	c	c	c			c		c	x	x	x	x	x	c	c	x	c	c	c
Heparin	c	c			c	c	c	c	x	?	?	x	c	c	c	c	c	c	c
Hydromorphone	c	?	c		c		?	?	x	c	c			c	c	c	c	c	c
Insulin	x	c							x	?	x	x	x			?			?
Ketamine		x		c				?	c								c		x
LRS	c	c				c	c		?		c	c	c	?	c		c	c	?
Lidocaine HCl	c	x				c	x	c	x	c	c	c	c		x	c	c	c	c
Magnesium sulfate		c	x			c	?	x	x			x		c		c	c		c
Mannitol									x				c					c	
Maropitant																			
Metoclopramide HCl	c	x	c		c	x		?	x	c	c		?			c	c	x	c
Metronidazole	c	?					c		x	c			x						c
Midazolam	c	x	c	?	c	c	c	x	x	c	c	x			c	c	c	x	c
Morphine	x	c	c		c		c	c	?	c	c	c	c	c	c	c	c	x	x
Ondansetron	x	x	c				c	?	?	c	c		c	c		c	c	x	c
Pantoprazole	c	c	x	x	?	?	?	x	x	x	x	?	?	c	?	x	?	?	?
Pentobarbital sodium	c		c		x		x		x	x	x	x		x			x		
Phenobarbitol	c		c			c			x	x	x	x		x			x		c
Potassium chloride	c	c	c		c	c	c	c	x	c	c	?	c		c	c	c	c	c
Potassium phosphate					x	?				c	x	x				c			
Propofol	c	c	?		c	c	c	c	x		c	?	?	c	c	c	c	c	c
Ranitidine	c	?	c				x	c	?	c	c	c	c	c	c		c	c	c
Sodium bicarbonate	c	x	c	x		x		c		?	x	x	x	x	x	?	c	c	c
Sodium chloride	c	c			c	c	c	c	x	c	c	c	c	c	c	c	c	c	c
Trimethoprim–sulfamethoxazole					x		x	x	x	c	x		x	x			x		c
Unasyn	c	c							x	x		x	x	x		c			c

c, compatible; x, incompatible; ?, conditional; LRS, lactated Ringer's solution.Sources: Plumb (2015) [1]; Trissel (2013) [2]; Lexi-Comp (2019) [3].

Hydromorphone	Insulin	Ketamine	Lactated Ringers soln	Lidocaine HCl	Magnesium sulfate	Mannitol	Maropitant	Metoclopramide HCl	Metronidazole	Midazolam	Morphine	Ondansetron	Pantoprazole	Pentobarbital sodium	Phenobarbitol	Potassium chloride	Potassium phosphate	Propofol	Ranitidine	Sodium bicarbonate	Sodium chloride	Trimethoprim - sulfamethoxazole	Unasyn
c	x	?	c	c	x	c		c	c	x	x	x	c	c	c	c		c	c	c	c	x	?
?	c	x	x	?	x			x	?	x	c	x	c			c		c	?	x	c	x	c
c		c						c		c	c	c	?	c	c	c		?	c	c			
													x										
c	x	c						c		?	c		?	x		c		c		x	c		
			c	c	?			x		c			?		c	c	x	c		x		x	
?			c	x	?				c	c	c	c	?	x		c		c	x	c	c	x	
c		?		c	x			?		?	c	?	x			c		c	c	c	c	x	
x	x	c	?	x	x	x		x	x	x	?	?	x	x	x	x		x	x	x	?	x	x
c	?			c				c	c	c	c	c	x	x	x	c	c		c	?	c	c	x
c	x		c	c				?		c	c	c	x	x	x	c	x	c	c	x	c	x	
	x		c	c	x	c				x	c		?	x	x	?	x	?	c	x	c	x	x
	x		c	c		c		?	x		c	c	?			c		?	c	x	c	x	x
c			?		c						c	c	c	x	x			c	c	x	c	x	x
c			c	x						c	c		?			c		c	c	x	c		
c	?			c	c			c		c	c	c	x			c	c	c		?	c	x	c
c		c	c	c	c			c		c	c	c	?	x	x	c		c	c	c	c	x	
c	?	x	c	c	?	c		x		?	?	x	?			c		c	c	c	c	x	
c	?	x	?	c	c			c	c	x	x	c	?		c	c		c	c	c	c	?	c
c		c	c		c			c	c	c	c	c	x	c	x	c		c	c	x	c	c	c
	c	x		c	c			c		?	?	?	?	x	x				?	c	x	x	c
c	?	c		c						c	c		?			x	x	c		x	c	x	
c			c	c	c			c	c	x	c	c	c	c	c	c			c	?		c	c
	c	c	c	c				c			c		x	x	?	c		?	c	x	c	x	
c	c		c		c			c	c		c	c	?			c	c	?		x	c	?	
						c		c				c	x			x		c		c	c	x	
							c						x										
c	c		c	c	c	c		c		c	c	c	x			c	c	x	c	x	c	x	
c			c		c				c	c	c		?					?			c		
c	?	c	x					c	c	c	c	c	?	x	x	?		?	c	x	c	x	x
c	?	c	c	c	c			c	c	c	c	c	?	x	x	c		?	c	x	c	?	c
c	?		c		c	c		c		c	c	c	x	x	x	c			c	x	c	x	x
x	?	?	c	x	?	x	x	x	?	?	?	x	c		x	c		x	x	?	c		c
c	x		c	x						x	x	x		c				c	x	c		x	
x	x		c	?							x	x	x		c			c	x	c	c	x	
c			c	c	c	x		c		c	c	c	c			c		c	c	c	c	x	
		x			c			c									c					?	
c		c		?	?	c		x	?	?	?		x	c	c	c		c	c	c	c	x	
c		?	c	c				c		x	c	c	x	x	x	c		c	c		c	x	
x	c	x	?	x	x	c		x		x	x	x	?	x	c	c		c		c	c	c	x
c	c	c		c	c	c		c	c	c	c	c	c		c	c		c	c	c	c	?	c
c		x	c	x	?	x		x		x	?	x		x	x	x		?	x	x	?	c	x
	c		c							x	c	x	c							x	c	x	c

To minimize errors, one person should be responsible for setting up the fluids. When a patient is initially set up on IV fluids, the tubing should be purged of air by filling the line with fluid; this is done to prevent air embolus. Only one person should be responsible for the addition of fluid additives to avoid the potential of exceeding the ordered concentration.

Certain drugs, such as mannitol or blood products, may require the use of an in-line filter to prevent the infusion of crystals, particulate matter, or clots. Microfilters of 150–170 μm pore size are commonly used with blood products to remove clots, larger red blood cells, and platelet aggregates. Finer filters with 18–40 μm pore size can be used with drugs such as mannitol to remove microaggregates and crystals. Crystals can be seen as small white precipitates or cloudiness in the IV line. The use of filters with blood products prevents the possible infusion and formation of emboli. Drug crystals can be irritating to the vein. Filters should be changed if they become occluded or with each new syringe drug or bag of blood product. Frequent changing of the filter will assure its proper filtering function.

Drug Delivery: Venous Access

When an IV catheter is used for fluid or drug administration, the catheter must be verified to be in the vein and patent. Although catheters are generally flushed each time the patient is disconnected from fluids, patency can be difficult to assess because bandages are in place for catheter protection and support. Since catheters may not spontaneously bleed back when aspirated, palpation of the vessel that is being flushed may be necessary to ensure patency. If the flush is given in a pulsatile fashion, the fluid can be felt traversing the vein. Fluid that diffuses into the surrounding tissue, resulting in tissue swelling, may be due to a catheter that is extravascular.

In the neonate or very small patient, an intraosseous (IO) catheter may be used. IO catheterization provides rapid access to the central circulatory system [6]. IO catheterization should not be performed if there are skeletal abnormalities, overlying skin or wound infections, abscess over the bone, bone fractures, or sepsis. The bony sites most commonly used include the flat medial surface of the proximal tibia, tibial tuberosity, trochanteric fossa of the femur, wing of ilium, ischium, and the greater tubercle of the humerus. If more than one hole is placed through a bone cortex, extravasation of fluid or medication into the subcutaneous (SC) tissue may occur. When administering IO fluids, observe for fluid extravasation; if it occurs, the needle should be removed and another bone site chosen. A bone should not be reused for 12–24 hours after perforation of a cortex [6]. If the needle is correctly placed and fluid does not flow freely, rotate the needle 90–180 degrees to move the beveled edge away from the inner core.

Case Study 46.1

A four-year-old 6.8 kg domestic shorthair male castrated cat was presented to the ICU for recovery following emergency surgery for a suspected liner foreign body. At surgery, a single enterotomy was performed at the distal jejunum to remove a string foreign body. The gastrointestinal tract was deemed to be healthy. Closure was done in a routine manner. The cat was stable through the surgical procedure with blood pressure, heart rate, respiratory rate, mucous membrane color, body temperature, and pulse oximetry monitored every five minutes. Fluid therapy was to continue with Plasma-Lyte 148 with potassium chloride (KCl) added at 20 ml/hour IV in the ICU. The ICU technician hung the bag of Plasma-Lyte 148 with the administration set attached into the fluid pump. A CRI dose of metoclopramide had yet to be added to the fluid bag so the line had not been filled with fluid. Unknowingly, the surgeon hooked the administration set to the cat's IV port, set the drip rate and started the fluid administration pump. Approximately 10 minutes later, the cat suffered a sudden cardiac arrest. The cat was taken from the cage and placed in left lateral recumbency on a central table; cardiopulmonary cerebral resuscitation was initiated. The cat was immediately intubated, and external cardiac compressions were instituted. An IV dose of atropine at 0.04 mg/kg was given followed by an IV dose of epinephrine at 0.01 mg/kg. Resuscitation attempts continued for 10 minutes, with ventilation at 10 breaths/minute and cardiac compressions 100 times/minute. Red fluid was seen flowing up into the endotracheal tube and resuscitation efforts were discontinued. It was determined that when the fluid therapy was begun, the administration set had not been purged of air; when the fluid pump was started; the air in the set was given as a bolus to the cat via the IV catheter. This resulted in a fatal air embolism. A standard IV administration set either 60 drops/ml or 10 drops/ml contains 10 ml air. The drip set and extension line used in this case had an air volume of 24 ml.

The actual cause of death is entrapment of air in the right ventricular outflow tract leading to outflow obstruction and increased venous pressure due to an air lock and decreased myocardial contractility [4, 5]. The lesson of this case underscores the importance of having only one person responsible for setting up and instituting fluid therapy in the patient.

A standard administration set is used to deliver fluids. The IO needle or catheter is secured by placing a tape butterfly around the hub and then suturing it to the skin. The entrance area should be covered and wrapped as with an IV catheter. The needle should be protected from breakage or bending. The fluid rate in 18–25 gauge IO needles is limited to 11 ml/minute with gravity flow and 24 ml/minute with 300 mmHg pressure [6]. It is suggested that an IO needle or catheter can remain in place for 72 hours if aseptic catheter maintenance is performed [6]. The risk factors for osteomyelitis are sepsis and catheter use that persists for several days. Substances that can be infused via an IO needle or catheter include blood and blood products, crystalloid and colloid fluids, amino acids, dextrose, aminophylline, antisera, antitoxins, atropine, aureomycin, calcium gluconate, cefoxitin, dexamethasone, diazepam, digitalis, diphenhydramine hydrochloride, dobutamine, dopamine, epinephrine, insulin, morphine, penicillin, procaine hydrochloride, radiopaque dyes, streptomycin, sulfadiazine, sulfathiazole, thiopental sodium 5%, and vitamins [6]. The use of alkaline or hypertonic solutions results in edema, pyknotic marrow nuclei, and decreased cellularity. The changes will spontaneously resolve within four to six weeks.

Drug Delivery: Enteral

Feeding Tubes

Critically ill patients often have indwelling nasogastric (NG), nasoesophageal (NE), or gastrostomy tubes. These ports can be used to administer medications that are labeled to be given orally. When dealing with a fractious patient that makes administration of oral medication impossible, most oral medications can be crushed and dissolved in water to administer through the enteral feeding tube. An oral medication that has a film coating or is a sustained release preparation should not be crushed, as this will change the rate of delivery of the drug, usually resulting in a more rapid uptake and possible overdose. Many oral medications can be administered via NE, NG, or gastrotomy tube; it is important to confirm how the oral medication is to be given prior to administration. Enteral medications should not be delivered via jejunal feeding tubes, as many of these drugs require the acidity of the stomach to allow their absorption. Oral vitamins could be given via a jejunal tube. Sucralfate is often used to coat the esophagus that has been injured by the presence of an esophageal foreign body; therefore, it must be given via the oral cavity. It is necessary to flush with a volume of water adequate to fill the feeding tube after drug administration to prevent the tube from clogging. If clogging of the tube should occur, first try to aspirate the tube with a 6- or 12-cc syringe. If aspiration does not clear the tube, it may be cleared by flushing the tube with an acidic brown soda such as cola. It is advisable to use a 1-cc syringe for the purpose.

Oral Medications

Oral medication should be given only to those patients that are capable of active swallowing. Active swallowing is necessary to prevent aspiration or possible erosion of the esophagus. A caustic medication that is not properly swallowed can remain within the lumen of the esophagus. When a patient is allowed food and is eating it is usually best to give oral medication in some type of food, which may ease administration of the medication. If a patient is not allowed food, it may be helpful to follow the oral medication with a dose of water that is delivered via a small syringe. This helps to ensure that the medication is swallowed and decreases the likelihood of its adherence to the esophagus, where it could potentially cause esophagitis or stricture. Timing of oral medication administration in relationship to feeding can be important. Certain medications should be given with food, prior to feeding, or on an empty stomach. Medication administration should be avoided with certain types of food; for example, the antibiotic doxycycline is not to be administered with dairy products.

Trazodone, an oral human-labeled product, is a serotonin type 2a antagonist/reuptake inhibitor that is commonly used to provide short-term relief of anxiety in hospitalized dogs and cats. It is contraindicated in patients receiving monoamine oxidase inhibitors such as amitraz and possibly selegiline. When trazodone is used with other serotonergic drugs it is possible to precipitate serotonin syndrome [7]. Other drugs that could also contribute to serotonin syndrome include amitriptyline, tramadol, and ondansetron [8]. This points to the importance of knowing the type of drugs a patient is on when they may be taking several medications at the same time.

Sucralfate is commonly given to treat gastrointestinal ulcers and esophagitis. It is an orally administered agent, frequently given as a slurry, as the pill will readily dissolve in a syringe of water. Sucralfate may interfere with the absorption of other oral medications such as fluroquinolones, fat-soluble vitamins, digoxin, and tetracyclines, and therefore dosing should be separated by at least two hours. Sucralfate is most effective in an acidic environment; therefore, an oral H_2 blocker should be given 30–60 minutes after the administration of sucralfate. The best effect will be seen if the sucralfate is given on an empty stomach, one hour prior to feeding or two hours after feeding [9].

Activated charcoal is used as an absorbent in the acutely poisoned patient. Charcoal is administered orally, and

some patients will eat the charcoal if it is mixed with a small amount of baby food, cheese, or canned pet food. This mixing technique makes administration easier and potentially cleaner than using a dose syringe. If the patient will not readily eat the charcoal mixture, the liquid charcoal is given via a dose syringe orally. If the animal will not tolerate an oral dosing syringe, an NG tube can be placed for administration. The NG tube is placed in the same manner as for feeding. The tube length is measured to the last rib to ensure placement in the stomach. The nostril is numbed with a drop of viscous 2% oral topical lidocaine solution. The tube is gently passed through the ventral nasal meatus; as the animal swallows it is threaded down the esophagus into the stomach. Proper tube placement is verified via a lateral radiograph of the thorax. The charcoal is then administrated through the NG tube. Owing to the viscosity of charcoal suspension, it may require dilution with water to allow passage through the NG tube. Following administration of the charcoal, the NG tube can be removed or drawn back to the appropriate NE location for continued treatment. If the charcoal is given too rapidly, it can induce emesis and increase the risk for aspiration. The administration of charcoal should be separated by at least three hours from other orally administered agents.

Buprenorphine is commonly given by a parenteral route but can be given via the oral transmucosal (OTM) route. This can be useful in patients that object to needles and do not have an IV catheter in place. The volume required makes it easier to deliver in small dogs and cats. The OTM route has improved bioavailability as compared to oral. The injectable buprenorphine is placed on or beneath the tongue or into the cheek pouches via a syringe [10].

Drug Delivery: Subcutaneous Route

The SC route is commonly used for insulin, famotidine, and metoclopramide. When giving a SC medication after entry under the skin the needle should be aspirated to ensure it is not in a vessel and still under the skin. Doses of SC medication are checked after injection to ensure that the site is not wet. If there is question as to whether the medication was administered or went through the skin, the clinician should verify and determine whether the patient should be redosed. When administering a SC medication it is best to administer the dose between the shoulder blades with the skin tented. If the area between the shoulder blades is not available due to trauma, bandage, skin infection, or scarring, the SC dose is administered where an appropriate skin tent can be obtained. When performing SC drug administration, it is best to use a small gauge needle such as a 25 gauge to avoid leakage of the agent out of a larger skin puncture. If a large volume of medication or fluid needs to be delivered, a larger gauge needle such as an 18–20 gauge may be used to deliver the volume in a timely manner. Note that some fluid may leak from the skin hole.

A new formulation of buprenorphine, Simbadol® (Zoetis) has been developed for use in the cat. It is given once daily SC, for up to three days to treat postoperative pain. It is a higher concentration than the traditional buprenorphine products. The extended duration should be taken into account when used in combination with other agents. In addition, buprenorphine can be more difficult to reverse compared with other opioid agents, so adverse effects may be more persistent due to the long duration of this agent [11].

Nocita® (Elanco US), a new bupivacaine liposome suspension is used as a single-dose infiltration into tissues at a surgical site incision closure. The slow release of the bupivacaine from the liposomes will provide local analgesia for 72 hours. This agent is not meant to be delivered by any route other than tissue infiltration. When administering other amide local anesthetics such as lidocaine or bupivacaine, the dosage may need to be reduced due to the liposomal bupivacaine [12].

Drug Delivery: Transdermal Patch

A human-labeled fentanyl transdermal patch can be used to provide long-term analgesia to dogs and cats. The patch is applied to clipped and clean dry skin. Holding the patch in place for two to three minutes with a warm hand will help the patch to adhere. People handling a patch should either wear gloves or rinse their hands with water afterwards to remove any residue. The patch has a highly variable absorption and efficacy among individual animals, this can impact the effect seen when used in conjunction with other agents. Dose of other agents may need to be decreased to avoid bradycardia and depression. Onset of action may take several hours and the duration can vary from three to five days. Patches should be dated when placed to keep track of duration of application. Patches are commonly applied to the lateral thoracic wall or lumbar areas. The patch should not be exposed to exogenous heat sources such as heating pads or forced hot air warming blankets as this may lead to increased drug release and absorption. The patch should be kept dry and clean. Patches can be repeated but monitor the skin at the patch site as rashes may develop, if this should occur a new site should be chosen for the next patch. If is it suspected that the patient has a developed severe respiratory depression or bradycardia from the fentanyl the patch should be removed, and if necessary, the adverse effects may be reversed with naxolone [13].

Drug Delivery: Intramuscular Route

Intramuscular (IM) injection can be used for the administration of agents such as analgesics and insulin. The commonly used muscles for the site of injection are the epaxial, biceps femoris, semitendinosus, or the triceps. The gauge and length of the needle are important to ensure that the medication is properly deposited in the muscle belly and not in the fat or fascial plane, which could diminish drug absorption or result in delayed adsorption.

Insulin can be given IV or IM for the treatment of the unregulated diabetic. Once the critical state resolves, long-term insulin therapy is usually initiated; long-term insulin therapy is generally administered by the SC route.

Furosemide can be given as a single IV or IM injection or as a CRI in the treatment of renal failure, heart failure, and pulmonary edema.

Constant Rate Infusion

A CRI consists of a calculated volume of medication at a specific concentration that is added to a set volume and type of fluid for continuous delivery. A CRI can increase the efficacy of a drug because it delivers a steady dose of the agent, which can help maintain steady plasma concentration and enhance safety through slower delivery. A CRI may be used to deliver antibiotics, long-term analgesics, or even sedative drugs for animals that require longer-term sedation or anesthesia. The drugs can be given either mixed in the daily fluids with the fluid bag clearly labeled with the type, amount, and rate the additive is to be administered or via a separate syringe pump or fluid bag (Figure 46.2). Critically ill patients may require more than one infusion for treatment and hemodynamic support, when using multiple syringe pumps all should be clearly labeled and facing the front of the cage for easy visualization (Figure 46.3).

The drug label on the fluid bag should be large and easy to see (Figure 46.4). The label is to alert personnel that the bag has an additive, and therefore these fluids should not be used to give a fluid bolus to the patient. A fluid bolus



Figure 46.3 Multiple syringe pumps clearly labeled and facing the front of the cage.

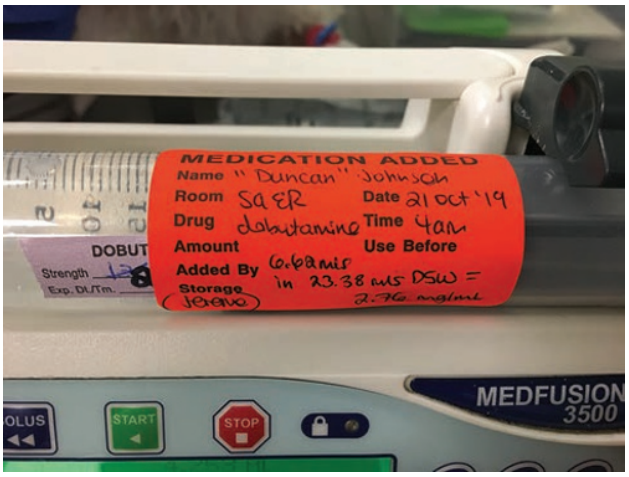


Figure 46.2 Syringe pump with a proper label.

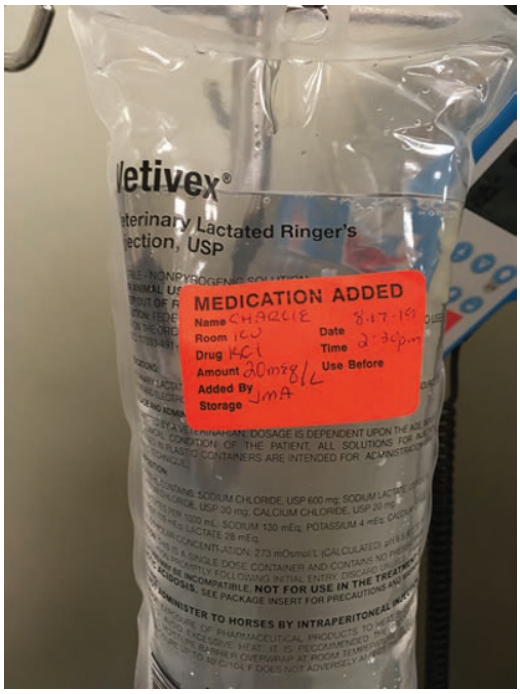


Figure 46.4 Fluid bag with additive label.

from a bag containing an additive could be unsafe and potentially lethal, such as a fluid bolus containing KCl. It may be safer for certain agents to be given via a separate fluid bag or a syringe pump. The separate bag or syringe pump will allow for a more accurate rate of delivery and easier monitoring, and avoid potential under or overdosing when fluid rates are changed. Accurate and obvious labeling is fundamental for all medications delivered by CRI. When labeling, the volume of diluent should be listed in case there is a question as to concentration; listing the quantity of diluent allows calculations to be verified.

When insulin is given as a CRI, it should be infused through an administration line separate from the fluid therapy line. This separation of insulin from the fluids allows for adjustments to be made in the insulin dose without alterations in the fluid therapy rate. Regular insulin adsorbs to the surfaces of IV infusion tubing and filters. Insulin adsorption to the administration equipment can lead to a 20–30% decrease in its potency. The adsorption process is instantaneous, with most insulin adsorption occurring within the first 30–60 minutes. To saturate binding sites and therefore deliver a more predictable dose when giving an IV infusion, run 50 ml of the insulin-containing solution through the IV tubing, discard this volume, and then begin the infusion [14].

Drugs such as diazepam are used as a CRI to treat severe seizure disorders. This light-sensitive drug may adhere to the plastic tubing of the IV line and should not be stored in plastic syringes long term. It has been found that diazepam 10 mg in a 2-ml volume can be stored in a polypropylene-polyethylene syringe for four hours; longer-term storage in a syringe results in absorption of the drug to the plastic surface, which deceases the amount of drug available [15]. The propylene glycol in diazepam is incompatible with most other fluids, and thus the drug requires a dedicated delivery system. Do not administer if a precipitate forms and does not clear [16]. Diazepam is irritating to veins and should only be given IV via a large central vein. It is suggested that high-dose dextrose, 7.5% or greater, is also best delivered by a central vein because of its hypertonicity. It is recommended that drugs or agents that have an osmolality greater than 600 mOsm/l be infused via a large or central vein to avoid the possible complication of vein irritation or phlebitis that may occur if small peripheral veins are used. Light-sensitive drugs are generally stored in brown bottles and should be protected from the light if they are being delivered as a CRI.

The hyperosmotic agent mannitol is commonly used for treatment of head trauma, renal failure, and glaucoma. Warming of the fluid line decreases the likelihood that the mannitol will recrystallize. Mannitol should be administered with a filter to prevent precipitates from being introduced intravenously. The filter should be changed with every separate dose syringe. Drawing up a one-hour volume and using a syringe pump are recommended to prevent crystallization of the mannitol and to ensure appropriate changing of the filter. Sodium or potassium chloride can cause mannitol to precipitate out of solution if the mannitol concentration is 20% or greater [17]. Owing to its hyperosmotic nature, it is best to administer mannitol through a large central vein catheter.

Metoclopramide is commonly given as a CRI within the animal's regular IV fluid therapy. Metoclopramide is light sensitive so the fluid bag should have a covering to prevent light interaction. It is compatible with 5% dextrose in water, 0.9% NaCl, 2.5% dextrose in 0.45% NaCl, Ringer's and lactated Ringer's solutions (LRS).

Intravenous Nutritional Support

Total parenteral nutrition (TPN) is administered through a central venous catheter that is specifically designated for the purpose. The catheter is placed using aseptic technique into the jugular, medial, or lateral saphenous vein and advanced to a central position. TPN is hyperosmolar, so a central vein is needed for administration. If a central catheter cannot be obtained, partial parenteral nutrition (PPN), which has a lower osmolality, can be provided by use of a peripheral venous catheter. This catheter should also be placed in a sterile manner. Once administration of PPN or TPN has begun, the administration IV line is never disconnected until the bag is empty. The bag travels everywhere with the patient to ensure sterility. If the patient is going to a visiting room the fluid pump can be placed on a mobile IV pole to allow the administration to continue. If the bag must be removed from the fluid pump, the line backflow of blood is prevented by clamping the roller clamp of the line and by clamping the T-port at the catheter. No other products should be given via this administration line or catheter to maintain sterility and decrease the potential for bacterial contamination.

Fluid Additives

Additives are commonly added to the fluid bag. They should be noted on the bag label (Figure 46.4). The label should note the amount of the product added; if it is an electrolyte, the final electrolyte concentration per liter should also be listed.

Additives commonly given to patients in the ICU include potassium salts such as KCl or potassium chloride (KPO_4). Potassium must be diluted as noted on the bottle and given slowly when administered intravenously. Undiluted and rapid IV administration may result in a fatal hyperkalemia. The rate of IV potassium chloride should not exceed

0.5 mEq/kg/hour. The potassium chloride can be supplemented, and is compatible with commonly used IV replacement fluids. The fluid bag should be clearly labeled that it contains potassium and the concentration of the potassium. It is best to have a consistent method for noting the concentration, such that all measurements of potassium are denoted as per liter volume regardless of the bag volume. For example, to a 1000 ml bag of LRS with 4 mEq already present is added 16 mEq of KCl; the final concentration would be noted as 20 mEq/l KCl total on the bag label. In some patients, there is a deficiency in potassium and phosphate, making the use of KPO_4 warranted. KPO_4 must also be diluted prior to IV administration, and is compatible with dextrose in water and 0.9% NaCl [18]. As with KCl, KPO_4 should not be delivered at a rate greater than 0.5 mEq K+/kg/hour. KCl and KPO_4 can be combined within the same fluid bag as long as the concentration of each potassium source is labeled and the appropriate rate of administration for the combined volume is listed on the order sheet.

Calcium gluconate or calcium chloride 10% are used to treat hypocalcemia. Calcium should be given slowly intravenously over a 10-minute period and the electrocardiogram monitored during the infusion to watch for the development of bradycardia. If bradycardia develops, the calcium infusion should be slowed or stopped temporarily. Calcium chloride is extremely caustic if administered extravascularly. Calcium gluconate can be given SC if diluted with an equal volume of 0.9% NaCl [19].

Sodium bicarbonate is used to treat metabolic acidosis. A calculated dose is given slowly IV; an initial bolus dose can be given over 20–30 minutes with the remainder of the dose given over the next several hours. It can be given with fluids such as 5% dextrose in water, 2.5% dextrose in 0.45% NaCl, Ringer's solution with dextrose, and 0.9% NaCl [20].

Agents Used to Treat Specific Toxicities

N-acetylcysteine (NAC) is considered the treatment of choice for acetaminophen toxicity [20]. The recommended regimen for use of NAC is an initial dose of 140 mg/kg IV up to 280 mg/kg if the toxicosis is severe, followed by 70 mg/kg every six hours for seven additional treatments. NAC may cause nausea and vomiting when given orally. Hypotension and bronchospasm can occur with rapid IV administration. Phlebitis occurs with perivascular leaks. It is best given as an IV infusion of a 5% solution, made by diluting 10–20% NAC in 5% dextrose or 0.9% NaCl, over 30–60 minutes through a 0.2-μm filter [21].

Fomepizole 4-methylpyrazole is used to treat ethylene glycol poisoning. Once the agent is reconstituted it should be used within 72 hours; it can be stored at room temperature. The reconstituted solution can be further diluted with 5% dextrose in water or 0.9% NaCl. The agent is given intravenously [22].

Vitamin K_1 is used in the treatment of anticoagulant rodenticide toxicities. Oral absorption is enhanced when given with fatty foods or by giving canned dog food with the vitamin K. IV administration is not recommended due to the potential for anaphylactoid reactions, and IM injections may result in bleeding. An initial SC dose is usually given, then long-term oral therapy is instituted. When giving the SC dose, use a small-gauge needle and administer at multiple sites [23].

Pralidoxime chloride is used to treat organophosphate poisoning. It works best as an antidote when used in combination with atropine. It can be given as IM or slow IV injection initially, with subsequent IM or SC doses [24].

Antibiotics

Antibiotics that must be diluted prior to administration should have the dilution instructions listed on the bottle. It is important to note concentration, date, and time of reconstitution, as the shelf life can vary with different concentrations; this is also important for medications that come with a vial of diluent for dilution. Not all staff in the hospital may be clear on the procedure for dilution of that particular medication and may dilute the drug to a different concentration. How the dilution was prepared and how the antibiotic was given must be recorded. The antibiotic ampicillin at a dilution of 100 mg/ml has a shelf life of 30 minutes before degradation of the product. The antibiotic ampicillin sodium/sulbactam sodium, Unasyn® (Pfizer), diluted to 30 mg/ml has a refrigerated storage time of 72 hours.

Amikacin sulfate should be used cautiously – if at all – with the loop diuretics such as furosemide or osmotic diuretics such as mannitol, as there may be in increase in the nephrotoxic or ototoxic effect of the amikacin [25]. The concern for nephrotoxic and ototoxic effects will also hold true for the other aminoglycoside, gentamicin sulfate.

Enrofloxacin is used extra-label as an IV injection. It is diluted 1 : 5 with 0.9% NaCl for slow IV administration over 10–45 minutes. The injectable form of enrofloxacin must not be mixed with or come in contact with any magnesium-containing solution such as Normosol and Plasma-Lyte, as microprecipitants may form and lodge in the patient's lung leading to morbidity and mortality. Enrofloxacin should be used cautiously in cats, as doses higher than 15 mg/kg have been associated with ocular toxicity and subsequent blindness [26].

Metronidazole is commonly used to treat anaerobic bacterial infections. Accurate dosing is important as neurologic toxicity may result in dogs that receive a high dose

either acutely or long term. An aluminum hub needle should not be used with this drug, as the aluminum can cause a reddish-brown discoloration of the solution [27].

Inotropes and Vasoactive Agents

Inotropes and vasopressor agents may be necessary for the treatment of hypotension. Dobutamine and dopamine are commonly used. If the hypotension is severe and does not respond to initial treatments, vasopressor agents such as norepinephrine, epinephrine, phenylephrine, and vasopressin may be used. Dobutamine when diluted for administration should be used within 24 hours. The diluent can be 5% dextrose in water or 0.9% NaCl. Dobutamine is compatible with 0.9% NaCl, dextrose–0.9% NaCl combinations, and LRS, and is administered as a CRI [28].

Dopamine is also administered as a CRI. It is commonly diluted with 5% dextrose in water, 0.9% NaCl, or LRS and should be used within 24 hours. Solutions that are pink, yellow, brown, or purple tinged indicate decomposition of the drug and should be discarded [29].

Phenylephrine is used treat severe hypotension via its alpha-adrenergic effects. It is given intravenously via CRI and is diluted in 0.9% saline or 5% dextrose in water. It should not be used if the solution is brown or contains a precipitate. Phenylephrine is compatible with the standard IV fluids [30].

Additional agents used to treat hypotension include norepinephrine and vasopressin. Both are compatible with standard IV crystalloid fluids. They are commonly diluted in 0.9% NaCl to ease administration. As with other inotropes and vasopressors, it is recommended that each agent be in a separate bag or syringe to allow for independent adjustment of individual doses.

Clevidipine is a dihydropyridine calcium channel blocker and is a smooth muscle vasodilator. It is used to treat fulminant heart failure or severe hypertension and must be administered as a CRI. The goal of administration is the reduction in blood pressure. It is titrated to effect by doubling the dose every 90 seconds. As the blood pressure approaches goal, the infusion rate should be increased in smaller increments and titrated less frequently. A primary concern is hypotension. Heart rate and blood pressure should be measured continually during the infusion along with perfusion parameters until the patient is stable. If the patient becomes hypotensive, the drug is reduced or turned off and its effects will dissipate within a few minutes. Aseptic technique should be used when handling this medication. Clevidipine should not be diluted and should not be administered in the same line as other medications. Once the stopper is punctured, Clevidipine should be used within 12 hours and any unused portion remaining in the vial should be discarded.

Nitroprusside sodium is used intravenously to treat severe hypertension. When using this agent, blood pressure must be monitored diligently to prevent severe hypotension. Excessive doses, prolonged therapy greater than three days, or severe hepatic or renal insufficiency may lead to profound hypotension or cyanogen or thiocyanate toxicity. Nitroprusside powder is diluted in 5% dextrose in water and protected from light by covering the fluid bag. The solution may have a slight brownish tint, which is normal. Discard the product if the fluid is blue, dark red, or green in color. Once reconstituted, the nitroprusside solution will be stable for 24 hours. Nitroprusside should be given IV in a dedicated line, and extravasation should be avoided. An infusion pump is recommended for delivery, to avoid a sudden bolus of the agent [31].

Analgesics and Anesthetics

Animals that require mechanical ventilation often need heavy sedation or light anesthesia to tolerate the endotracheal tube. Propofol is commonly used to help maintain ventilator patients or to treat refractory seizure conditions. Propofol is an IV anesthetic agent. It can be given as an IV bolus or as a CRI to maintain anesthesia. Propofol does not contain any antibacterial agent and has the potential to become bacterially contaminated. It is not recommended to be used beyond 24 hours once the bottle has been opened. Cats may develop Heinz body anemia from long-term use of propofol [32]. The product should not be used if the emulsion has separated. Propofol is compatible with the commonly used IV replacement and maintenance fluids, and can be injected into a running IV line. When used as a CRI, propofol is best delivered via syringe pump to assure an accurate delivery rate. A new formulation of propofol has recently become available, PropoFlo™ 28 (Zoetis). The preservative benzyl alcohol is added, allowing a shelf life of 28 days. This product is only labeled for IV use in the dog and should not be used in the cat. Benzyl alcohol is potentially toxic to the cat, owing to the cat's lack of adequate glucuronic acid conjugation [32].

Chemotherapeutic Agents

Many chemotherapeutics are given IV or even SC. Patients are given IV fluids to help maintain renal perfusion. Cisplatin, streptozocin, L-asparaginase, pamidronate, and dicarbizine are all commonly used chemotherapeutic

Case Study 46.2

A four-year-old domestic short-hair castrated male cat was presented for upper airway dyspnea and retropharyngeal swelling. The cat developed cyanosis. He had a right arytenoidectomy and a temporary tracheostomy tube placed to improve ventilation and oxygenation. While in ICU the cat had several episodes of difficult breathing through the tracheostomy tube; each time the cat received IV propofol to facilitate removal of the tracheostomy tube for it to be cleaned of mucous plugs. As the cat improved the tracheostomy tube was removed for short periods of time; IV propofol was used to reinsert the tube. The cat was in the ICU and received multiple doses of propofol over a seven-day period. On the seventh day a routine complete blood count showed a hematocrit of 14% and 25–50% Heinz bodies. On presentation the HCT was 40%; this was a significant anemia that required treatment with a packed red blood cell (RBC) infusion. The packed RBC infusion improved the hematocrit to 19%, and the cat recovered. It is important when sedating on multiple days to be aware of the possible consequences that sedative and anesthetic drugs may have. This may be species or age related, as older or debilitated animals will take longer to metabolize drugs, resulting in a possible prolonged effect [33].

Lidocaine is used to treat cardiac arrhythmias and as an analgesic. When used as a CRI, an initial loading dose is administered to achieve an effective plasma concentration, which is then maintained with the CRI. Lidocaine should be used cautiously in cats due to a high risk of adverse events [34]. Lidocaine has the potential to be toxic, with clinical signs of ataxia, nystagmus, depression, vomiting, seizures, bradycardia, and hypotension. Lidocaine is compatible with the commonly used IV replacement fluids.

The fluid bag should be properly labeled as containing lidocaine. The lidocaine solution containing epinephrine should not be infused IV; this product is used for local analgesia application, and IV use of this product could lead to cardiac arrhythmias [35].

agents for the treatment of osteocarcoma. Gloves should be worn when handling and administering these compounds. Gloves should also be worn when handling any bodily fluids from these patients (Figure 46.5).

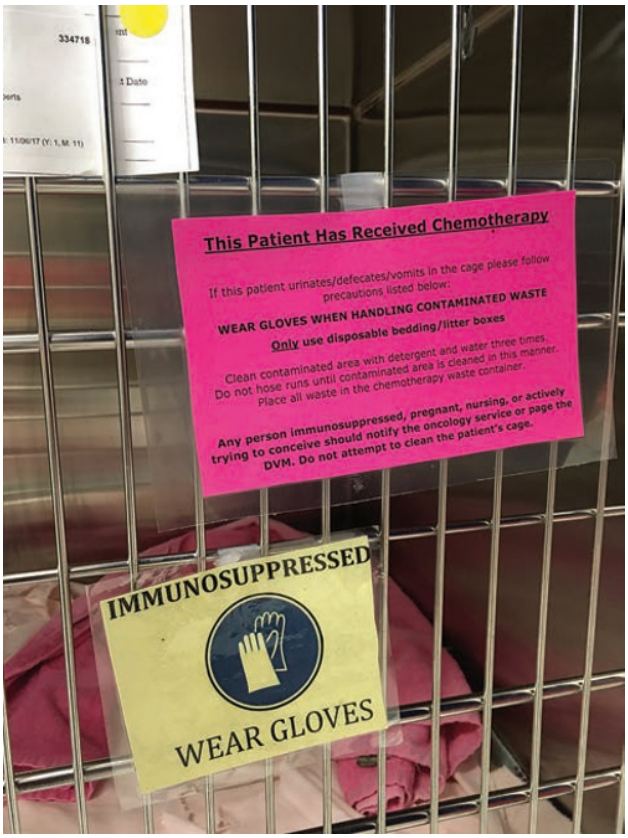


Figure 46.5 Cage label for chemotherapeutic agents and warning.

Drug Overdose

When a drug is inadvertently overdosed, an incident form should be completed and the clinician immediately notified of the event (Figure 46.6). The incident form is used not to place blame but to track errors within the hospital and to improve and refine protocols. The patient's vital signs should be assessed to gather baseline data and to evaluate patient stability in the event an overdose leads to a reaction. If the drug is capable of being reversed, the patient's clinician will determine if reversal is warranted. Reversal agents include naloxone for opioids, flumazanil for benzodiazepines, and atipamazole for the alpha-2 agonists. The possible adverse effects of the overdosed drug should be investigated. If the overdose is severe and leads to increased length of hospitalization, higher costs, or the death of the patient, the hospital director should be informed. The owners should be made aware of the situation.

University of Georgia
Veterinary Teaching Hospital
Incident Report

Patient/Client Information

Risk management is an important aspect of healthcare facilities and identification of potential risks will help create a safe and healthy hospital environment for patients, clients, and hospital employees. This incident report should be completed by a person with knowledge of an accident or incident involving a patient, client or employee at the UGA VTH. This information will be used internally to help proactively manage medical risks in the hospital.

Date/Time & Place	Date:	Time: AM or PM	Location of Incident: (Include building and specific location in building or Room #)
Type of Incident (please mark all that apply)	( ) Related to Medication: ( ) patient ( ) dose ( ) type of medication ( ) time ( ) route ( ) Related to a Procedure or Treatment ( ) Related to Equipment: ( ) damage ( ) malfunction ( ) other ( ) Related to Personnel: ( ) injury ( ) other ( ) Related to Clients: ( ) services received ( ) finances ( ) other ( ) Related to Biosafety: ( ) sharps disposal ( ) other ( ) Other (please describe):		
Incident Description	Please include only facts about what happened. Avoid speculation and assignment of blame. Attach addendums if necessary.		
Treatment Given or Action Taken Following Incident			
Adverse Effects of Incident (if any)			
Case Management	Personnel with Knowledge of Case (provide first and last name) ( ) Faculty _____ ( ) Resident _____ ( ) Intern _____ ( ) Staff _____ ( ) Other _____ Who was Notified at Time of Incident? _____ Was the Client/Owner Notified of the Incident? ( ) Yes (Please give date, time and who notified the client) ( ) No		
	What, if any, changes would you recommend to avoid the recurrence of this type of incident?		

_____ _____ _____
Print/Signature Name of Person completing Incident Report Date Phone

Please submit the completed incident report to Brooke Hogan in the Hospital Director's office within 24 – 48 hours of the occurrence. Thank you.

Figure 46.6 Incident reporting form. *Source:* Reproduced with permission from the University of Georgia Veterinary Medical Center.

Case Study 46.3

A male intact Akita was seen for a several-month history of collapse and paralysis. A neurological exam was performed. A test for myasthenia gravis was performed. The dog received IV edrophonium and the response was noted. The dog immediately improved and was able to move freely. This was considered a positive test for myasthenia gravis, and the dog then received IM neostigmine for treatment. At the time of neostigmine administration, it was noted by technicians involved with the case that the amount of drug seemed to be a large volume. The drug was given, even though there was concern about the amount of drug. The dog subsequently seizured, vomited, and defecated but seemed to recover. Vomiting persisted; treatment with IV and IM atropine was not effective. The dog developed cyanosis and dyspnea. Thoracaic radiographs reveled aspiration pneumonia. The dog required mechanical ventilation to treat hypercarbia and hypoxemia. The dog became hypotensive and developed acute respiratory distress syndrome. The dog's condition worsened and he went into cardiac arrest and died. It was later determined that the neostigmine was inadvertently administered at 10 times the recommended dosage. It is important to question drug doses that seem to be wrong either in amount, in type, in route of delivery, or for the species. If there is any doubt as to the validity of a drug administration, the clinician in charge of the case should be notified and the proper dosing information verified.

The medical therapies that are required in an ICU situation can vary with each patient. The patient treatment sheet should have clear instructions as to dosage and routes of administration. It is vital to be aware of all drug interactions and effects of those medications in that patient. Mistakes can be made by anyone; if there is a question regarding a drug, a dose, or route of administration, it is better to investigate rather than make an error. Record keeping is instrumental to the successful treatment and well-being of the patient in ICU.

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47

Pain Recognition and Management

Chiara Valtolina and Liza Lindeman

Accurate recognition and effective management of pain are vital to veterinary critical care. Optimal pain control depends on a sound understanding of the physiology of nociception, an ability to recognize pain behaviors, diligent and thorough pain assessment, and an understanding of the principles of multimodal therapy. We must also be cognizant of potential analgesic-related adverse effects to recognize and manage them appropriately.

Nociceptive Physiology

The International Association for the Study of Pain (IASP) defines pain as an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage (Table 47.1) [1].

Because the IASP definition relies mainly on the individual patient describing the pain, Molony and Kent (1997) proposed an alternative definition for animals: "animal pain is an aversive sensory and emotional experience representing an awareness by the animal of damage or threat to the integrity of its tissues; it changes the animal's physiology and behavior to reduce or avoid damage, to reduce the likelihood of recurrence, and to promote recovery" [2].

Pain may be classified as physiologic or pathologic. Nociceptive or physiologic pain is an acute pain that arises from noxious stimuli associated with the risk of tissue injury. Physiologic pain is proportional to stimulus intensity, transient, and characterized by a high stimulus threshold and narrow localization. Physiologic pain is protective because it induces withdrawal reflexes and avoidance responses but it is rare in a clinical setting [3–5].

In the clinic, noxious stimuli are persistent and perpetuated by inflammation (inflammatory pain) or nerve injury (neuropathic pain). This type of pain is pathologic and implies that tissue damage has already occurred. Pathologic pain is characterized by a low stimulus threshold and an exaggerated pain response to noxious stimuli (hyperalgesia) [3, 5]. Pathologic pain is felt at sites of injury (primary hyperalgesia) and in surrounding areas (secondary hyperalgesia or extraterritorial pain) [6–9].

Pain may also be classified as adaptive or maladaptive [10]. Adaptive pain encompasses nociceptive and inflammatory pain, and it is a normal response to tissue damage and confers tissue protection. Inadequate management of adaptive pain may alter brain and spinal cord function leading to maladaptive pain, which does not have protective properties and is challenging to control. The longer the duration of pain, the more likely this switch to maladaptive pain [5, 7, 10]. Intense or prolonged noxious stimuli can alter nervous system function peripherally by decreasing the threshold of nociceptors or centrally by increasing the spinal neuron responsiveness through altered neuronal gene expression [5, 9, 11].

Pathologic pain is pain that ceases to serve a protective function and becomes maladaptive, degrading health, and functional capabilities. Pathologic pain may be acute or chronic. In a study evaluating pain in canine and feline emergency admissions to a teaching hospital, inflammation was identified as the cause in 70% of patients [12]. By contrast, chronic pain persists beyond the time frame expected for a given disease or injury and has been arbitrarily defined as persisting for more than three to six months [1, 5, 9]. Chronic pain may arise from sustained noxious stimuli due to continuing inflammation or may be independent of tissue injury. More than 200 clinical syndromes are associated with chronic pain including cancer, osteoarthritis, and postamputation "phantom limb" syndrome [3, 5].

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Companion website: www.wiley.com/go/burkitt/monitoring

Table 47.1 Pain definitions.

Type of pain	Definition
Adaptive pain (inflammatory)	Spontaneous pain and hypersensitivity to pain in response to tissue damage and inflammation. Occurs with tissue trauma, injury, surgery. Causes suffering. Responds to treatment.
Adaptive pain (nociceptive)	Transient pain in response to a noxious stimulus. Small aches and pains that are relatively innocuous and that protect the body from the environment.
Allodynia	Pain caused by a stimulus that does not normally result in pain.
Analgesia	Absence of pain in response to stimulation that would normally be painful.
Anesthesia	Medically induced insensitivity to pain. The procedure may render the patient unconscious (general anesthesia) or merely numb a body part (local anesthesia).
Causalgia	A syndrome of sustained burning pain, allodynia, and hyperpathia after a traumatic nerve lesion, often combined with vasomotor and sudomotor dysfunction and later trophic changes.
Distress	Acute anxiety or pain.
Dysphoria	A state of anxiety or restlessness, often accompanied by vocalization.
Hyperalgesia	An increased response to a stimulus that is normally painful.
Hypoalgesia	Diminished pain in response to a normally painful stimulus.
Hyperesthesia	Increased sensitivity to stimulation, excluding the special senses.
Hyperpathia	Painful syndrome characterized by an abnormally painful reaction to a stimulus and an increased threshold.
Maladaptive pain (functional)	Hypersensitivity to pain resulting from abnormal processing of normal input.
Maladaptive pain (central)	Pain initiated or caused by a primary lesion or dysfunction in the CNS. Often called "central pain."
Maladaptive pain (neuropathic)	Spontaneous pain and hypersensitivity to pain in association with damage to or a lesion of the nervous system.
Multimodal analgesia	Use of more than one drug with different actions to produce optimal analgesia.
Neurogenic pain	Pain initiated or caused by a primary lesion, dysfunction, or transitory perturbation in the peripheral or central nervous system.
Neuropathic pain	Pain initiated or caused by a primary lesion or dysfunction in the peripheral or central nervous system.
Nociceptor	A receptor preferentially sensitive to a noxious stimulus or to a stimulus that would become noxious if prolonged.
Nociception	Physiologic component of pain consisting of the processes of transduction, transmission, and modulation of neural signals generated in response to an external noxious stimulus.
Noxious stimulus	A noxious stimulus is one that is damaging to normal tissues.
Paresthesia	An abnormal sensation, whether spontaneous or evoked.
Pain	An unpleasant sensory and emotional experience associated with actual or potential tissue damage.
Preemptive analgesia	Administration of an analgesic before painful stimulation.
Wind-up pain	Heightened sensitivity that results in altered pain thresholds both peripherally and centrally.

Nociception

Nociception involves perception of noxious stimuli at the site of injury, transduction into electrical signals, transmission of that signal to the spinal cord, signal modulation by amplification or inhibition, and finally supraspinal conduction and central integration to produce a pain experience unique to the individual (Figure 47.1). This multiple component system of transmission and integration also permits modulation of nociceptive information [5, 9].

Peripheral nociception begins with specialized free nerve endings (nociceptors) of primary afferent fibers located in cutaneous tissues, muscle, and viscera. These nociceptors transduce high-threshold stimuli into electrical activity and transmit this information to the spinal cord [5, 8, 9, 13].

Nociceptors encode the localization, intensity, and duration of noxious stimuli. Most nociceptors are nonselective ion channels gated by temperature, chemical ligands, or mechanical shearing forces [5, 9, 10]. Once activated, the channels permit sodium (Na^+) and calcium (Ca^{2+}) ion influx, producing an inward depolarizing current, which if

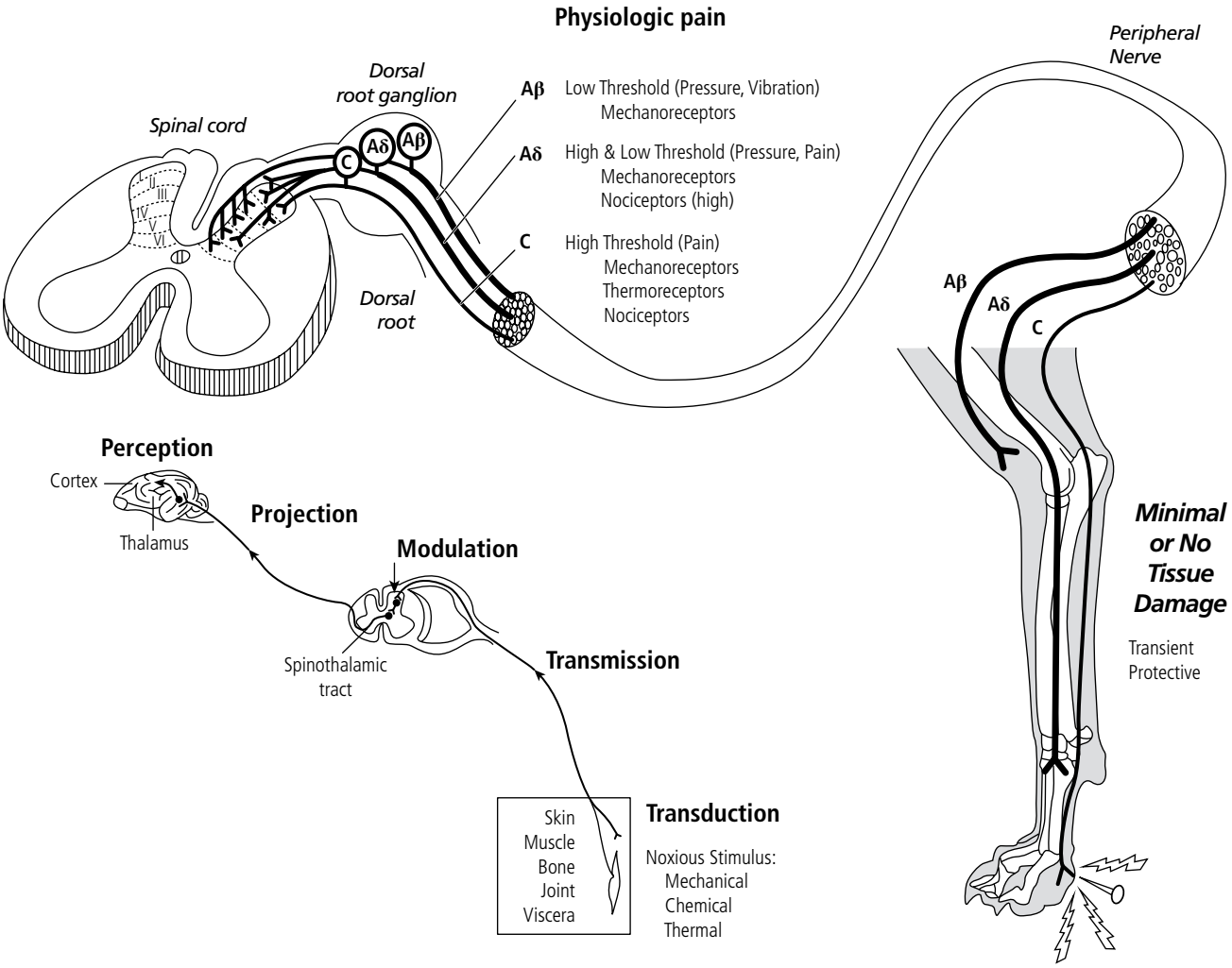


Figure 47.1 Schematic diagram of the pathways of physiologic pain sensation. Minimal or non-tissue-damaging stimuli are transduced by thermal, mechanical, and chemical peripheral nociceptors and transmitted to the dorsal horn of the spinal cord by Aδ and C fibers that release glutamate, activating dorsal horn neuron receptors that mediate reflex responses and transient pain. The pathways of nociception then involve stimulus transduction, transmission, modulation, projection, and perception. *Source:* Adapted from Muir and Woolf (2001) [6].

of sufficient magnitude, activates voltage-gated Na^+ channels, further depolarizing the membrane and initiating bursts of action potentials. These are conducted from the periphery to the central nervous system (CNS) along primary afferent nociceptive fibers [14]. Nociceptors, by virtue of their specificity and threshold, constitute the first and most important filter in nociceptive processing [8].

Sensory nerve fibers are divided into three groups (Figure 47.1). Aβ fibers are large myelinated sensory fibers activated by low-intensity stimuli; these fibers normally conduct non-noxious information (touch, vibration, pressure, and rapid movement). Aδ and C fibers are the principal nociceptive primary afferents responsible for fast and slow pain [5, 9, 13].

Aδ fibers are small (1–5 μm in diameter), myelinated, rapidly conducting (5–30 m/s) fibers responsible for the sensation of physiologic pain, fast pain, or "first pain," which is sharp, localized, and transient. Aδ fibers have small receptive fields and specific high-threshold ion channels activated by noxious thermal or mechanical input [5, 9, 13].

C fibers constitute most of the cutaneous nociceptive innervations, but they are also found extensively in muscle and viscera. They are small (0.25–1.5 μm in diameter) and unmyelinated with conduction velocities of only 0.5–2 m/s. Their receptive fields are large compared with those of Aδ fibers. These characteristics contribute to the nature of pathologic pain, slow pain, or "second pain," which is a poorly localized, dull, aching, or burning sensation that persists despite termination of the noxious stimulus. C fibers are considered polymodal because they can be activated by thermal, mechanical, or chemical stimuli [3, 5, 9, 13].

Afferent sensory nerve fibers enter the spinal cord via the dorsal nerve root and then separate to innervate second-order neurons within the gray matter. Aδ fibers terminate in laminae I, II, and IIa, whereas C fibers terminate in laminae II, IIa, and V. Sensory fibers synapse first at the level of the dorsal horn of the spinal cord where initial integration and modulation of nociceptive input occurs. Three principal paths are possible. Primary afferents may synapse with local interneurons (excitatory or inhibitory) causing modulation, with neurons involved in segmental spinal reflexes or with neurons that project to supraspinal structures [3, 5, 9].

Three types of nociceptive neurons project from the dorsal horn to supraspinal centers [3]. Wide dynamic range (WDR) neurons receive innocuous input from low-threshold Aβ fibers in addition to nociceptive input from Aδ and C fibers. WDR neurons respond in a graded manner over large receptive fields and often receive convergent inputs from visceral and nociceptive-specific (NS) neurons. NS neurons involved in stimulus localization and discrimination have small receptive fields and respond to noxious, mechanical, and thermal stimuli via Aδ and C fibers. WDR and NS fibers define the spatial and temporal qualities of pain. NS neurons may be persistently sensitized by repetitive noxious stimuli. WDR neurons exhibit prolonged after-response generated by primary afferent input, intensifying, and continuing nociceptive transmission [3, 5, 9, 13]. WDR and NS neurons project to the reticular formation and thalamus via multiple parallel pathways, including the spinothalamic and spinocervicothalamic tracts and the dorsal column [3, 5, 9].

Transmission and Modulation

Interneuronal transmission in the dorsal horn occurs via excitatory and inhibitory amino acid-mediated signaling [5, 15]. Glutamate is the principal excitatory synaptic neurotransmitter in both the spinal cord and brain. Different types of glutamate receptors exist: kainate, α-amino-3-hydroxy-5-methylisoxazole-4-proprionate (AMPA), and *N*-methyl-D-aspartate (NMDA) receptors. Glutamate initially binds to AMPA receptors inducing ligand-gated sodium and calcium channel activation and rapid depolarization, which then activates NMDA receptors. Normally, NMDA receptors are blocked by magnesium (Mg^{2+}) ions, but intense stimuli may remove this blockade. This in turn allows generation of a greater post-synaptic depolarizing response causing pain to remain after the stimulus has disappeared [16]. C fibers also release various neuropeptides, particularly those of the tachykinin family including substance P and the neurokinins. Because these neuropeptides are the mediators of pathologic pain, the development of neuropeptide antagonists offers the hope of improved therapies for chronic pain states [5, 16].

Nociceptive pathways permeate the medulla, pons, mesencephalon, diencephalon, and cerebrum. Modulation of pain responses occurs on four levels: spinal cord dorsal horn, the rostroventral medulla (RVM) of the brainstem, the periaqueductal gray matter (PAG) in the midbrain, and the thalamocortex. The thalamocortical system produces both the sensory, discriminative aspects of pain and the motivational, behavioral aspects of the pain experience. The thalamus integrates the pain experience with other brain centers. Relay nuclei exist between the thalamus; the limbic system (involved in behavior and emotion), which includes the amygdala (conditioned fear and anxiety); the prefrontal cortex; the hypothalamus (sympathetic autonomic activity); and the PAG (fight-or-flight behavior), stress-induced analgesia [5, 6, 9]. The PAG is a key structure in the endogenous analgesia system consisting of endorphin- and encephalin-containing neurons. Excitatory and inhibitory projections extend from the PAG to the brainstem. The RVM integrates and processes ascending nociceptive information and modulates descending output. Within the RVM, there are unique populations of cells referred to as facilitative and inhibitory or *on* and *off* cells involved in transmission of nociceptive stimuli, nociceptive reflexes, and behavioral responses. These cells are critical in producing hyperalgesia after peripheral tissue injury by maintaining central sensitization. At the level of the spinal cord, high concentrations of gamma-aminobutyric acid, glycine, serotonin, and the endogenous opioid peptides (encephalin, endorphin) produce inhibition of nociceptive stimulus transmission [3, 5, 6, 16].

Allodynia

Tissue inflammation may lead to an exaggerated response to noxious stimuli (hyperalgesia) or a reduction in the intensity of the stimulus necessary to induce pain (allodynia) [6]. This sensitization is caused by changes in the chemical environment of nociceptor peripheral terminals following release of adenosine triphosphate and hydrogen (H^+) and potassium (K^+) ions from damaged cells at the site of injury. Proteases, cyclooxygenase-2, and nitric oxide synthase induced by inflammation, together with cytokines, chemokines, serotonin, and histamine produced by recruited inflammatory cells, act synergistically to lower the threshold for Aδ and C fiber activation [5, 17]. This threshold reduction recruits silent nociceptors producing allodynia.

Central Sensitization/"Wind-Up"

Hypersensitivity also occurs due to dynamic modification of the receptive field properties of dorsal horn neurons in the spinal cord. Temporal summation and cumulative depolarization of dorsal horn neurons causes "wind-up," which is due to NMDA receptor disinhibition. NMDA stimulation leads to intracellular Ca^{2+} mobilization increasing responsiveness to glutamate [3, 5, 10]. Wind-up increases dorsal horn neuron excitability, which in conjunction with decreased spinal cord neuron inhibition creates central sensitization. Peripheral sensitization involves sensitized Aδ and C fibers while central sensitization allows low-threshold Aβ fibers to induce pain by increasing spinal neuron excitability and altering spinal cord sensory processing.

Glial cells, particularly those in the spinal cord, previously thought only to provide neuronal support and nutrition, are now considered key players in the creation and maintenance of pathologic pain states [18]. Glial cell activation due to nerve trauma or inflammation results in proinflammatory mediator production, which contributes to central sensitization. These cells may also play a role in reducing opioid efficacy [18, 19].

Importance of Pain Control

The word *pain* derives from Greek and Latin words meaning punishment or penalty [20]. Numerous definitions of pain exist in the human literature where terms such as *distress, suffering*, and *stress* have been associated with pain [21–23]. When left untreated, pain may have consequences beyond unnecessary suffering [23, 24]. Pain increases sympathetic tone and causes catecholamine release, resulting in tachycardia, vasoconstriction, decreased gastrointestinal blood flow, and the potential for gastrointestinal ulceration, decreased bladder tone, and increased muscle tone [3, 23, 24]. Pain-induced anxiety and fear enhance sympathetic outflow and may increase blood viscosity, prolong clotting times, and induce fibrinolysis and platelet aggregation. Intense vasoconstriction may lead to decreased tissue oxygen delivery and shock. Pain-induced tachycardia causes an increase in myocardial work and oxygen demand, and high levels of catecholamines predispose to arrhythmias.

Pain activates the renin-angiotensin-aldosterone system and induces the secretion of cortisol, glucagon, antidiuretic hormone, growth hormone, and interleukin-1; it decreases insulin secretion [3, 23, 24]. Increased counterregulatory hormone levels generate a catabolic state characterized by hyperglycemia, proteolysis, lipolysis, sodium and water retention, and decreased glomerular filtration. Such catabolism may have direct consequences for the immune system, decreasing wound healing and reducing an individual's capacity to respond to infection [3, 23, 24].

Pain also reduces appetite, mobility, and leads to postoperative weight loss. Immobility promotes urine and fecal retention [25].

An important addition to the definition of pain states that "the inability to communicate in no way negates the possibility that an individual is experiencing pain and is in need of appropriate pain relieving treatment" [26]. As caregivers, we have two important reasons to address pain: a moral and ethical obligation toward animals that are suffering and cannot speak for themselves, and a medical duty to reduce the morbidity and mortality associated with pain.

The American Animal Hospital Association published guidelines in 2015 and the World Small Animal Veterinary Association Global Pain Council guidelines for recognition, assessment, and treatment of pain highlighting advances in pain management, and both focus on the recognition and assessment of pain, pharmacological intervention, non-pharmacological interventions, and the importance of a team approach and education of the client [27, 28].

Pain Recognition

Effective pain management can only be achieved if pain can be accurately and consistently assessed to enable evaluation of therapeutic response. Pain is considered an individual experience, and the way this experience translates into observable and measurable behavior depends on numerous factors. Animals are unable to describe their pain as most humans do, so the potential for observer bias is inherent in any attempt to measure pain in animals [22].

The assessment and treatment of pain is greatly influenced by knowledge of the specific animal's normal behavior, of normal species behavior, and the caregiver's observational skills and attitude toward pain [22]. Differences in attitude toward pain and analgesia exist within the veterinary profession [29–32]. These differences appear primarily due to gender and age, with female veterinarians and those recently graduated being more proactive in the evaluation and management of pain [33]. A recent article showed a better implementation of the analgesic plan and an increase in postoperative analgesic when veterinarians are working alongside with veterinary technicians and nurses [29, 34, 35].

The difficulties inherent in assessing pain in animals may result in analgesia being withheld inappropriately.

Patients may not demonstrate *expected* signs of pain, or assessment may fail to identify these signs. Such problems have been cited by various studies as a major cause of veterinarians withholding analgesia [36–38]. Conversely, analgesia may be administered unnecessarily based on an anthropomorphic evaluation of the animal's condition, a scenario that increases the likelihood of adverse drug reactions (ADRs).

In human medicine, despite most patients being able to report their pain verbally, the ideal method of pain assessment remains unidentified. In 2000, the Joint Commission on Accreditation of Healthcare Organizations adopted pain as the fifth vital sign (after heart rate, respiratory rate, temperature, and blood pressure) [39]. In veterinary medicine, pain is considered to be the fourth vital sign, with temperature, peripheral pulse and respiratory rate, which should be assessed by all clinicians [27, 28].

In veterinary medicine, in which our patients are unable to report pain verbally, physiologic and behavioral responses used to form the basis for pain scoring scales. Physiologic responses to pain include tachycardia, tachypnea, hypertension, pyrexia, and mydriasis, reflecting increased epinephrine, norepinephrine, and cortisol concentrations. These physiologic alterations are also induced by disease processes other than pain, however, making them nonspecific and of limited discriminant value [2, 5, 22, 40].

Because our patients do not speak our observational skills are fundamental in evaluation of animal behavior and it is an essential part of the assessment of pain but is subject to limitations. Observers must be familiar with the typical behavioral changes associated with pain (Figure 47.2; Table 47.2). Animals undergoing veterinary examination in a strange environment may display altered behaviors that mask signs of pain. It is essential to gain as much information as possible from the primary caregiver regarding the animal's normal behavior prior to attempting pain assessment because an animal's character and temperament influences its response to pain [41, 42]. It is widely recognized that changes in behavior secondary to pain may be difficult to distinguish from that due to anxiety [41, 42]. Dogs especially may vocalize, whimper, or become agitated when anxious, and these signs are not pain specific [41].

Attempts to assess pain responses are complicated by the high degree of variation across species, between patients,



Figure 47.2 A young canine patient in the intensive care unit at the faculty of Veterinary Medicine, Utrecht University, The Netherlands with abdominal pain demonstrating a characteristic "prayer" position. This abnormal posture is far more common in dogs than in cats, which tend to adopt a hunched posture or become recumbent and reluctant to move.

Table 47.2 General and species-specific behavioral manifestations of pain.

Aspect of behavior	General
Temperament	A change in temperament becoming aggressive or withdrawn. Aggression in response to forceful movement of painful area. Insomnia.
Vocalization	Vocalization in response to palpation or movement of painful area.
Posture, locomotion	Guarding of the painful area. Severe abdominal pain may result in hunched posture, prayer position, falling and/or rolling. Reluctance to lie down. Trembling; increased muscle tension.
Facial expression	Dull eyes, "staring into space," drooping ears.
Grooming	Decrease of normal grooming, unkempt hair coat. Piloerection. Licking, kicking, biting, or scratching painful area. Self-mutilation if pain is severe.
Activity level	Restlessness or overall decrease in activity level. Failure to use litter box; increased or decreased urination.
Food and water consumption	Decreased.
Aspect of behavior	Species specific.
Cats	Vocalization is rare. Hissing or growling when approached or handled. Tendency to hide in enclosed space. Tendency to hide painful body parts. Decreased activity, lack of grooming, hunched posture, dissociation from the environment and lack of interaction with severe pain. Aggression if approached or when painful area is manipulated.
Dogs	Attention seeking, whimpering, whining, and howling. Vocalization often stops when animal is comforted. Hunched or "prayer" posture with abdominal pain. Shivering, panting.

and within individual patients themselves. In a recent study, thermal pain sensitivity was evaluated in three different dog breeds, the Harrier Hound, Greyhound, and New Zealand Huntaway. Their study showed that the Huntaway appeared to be less sensitive to thermal pain than the other breeds [43]. In humans and mice, an interesting correlation between hair color and pain sensitivity has been discovered [44, 45].

Critically ill animals may be unable to display classical signs of pain (Table 47.2) despite being painful. Critically ill animals may be obtunded, stuporous, or immobile, and unable to shift position in response to noxious stimuli. They may not vocalize as otherwise healthy animals would [25]. Caregivers must be aware of the potential for underrecognition of pain causing inadequate analgesia provision in our sickest patients who may be in greatest need. The absence of perceptible behavioral displays associated with trauma or illness may be a factor in undertreatment.

Pain Scales

Over recent years, there has been more attention to the treatment and evaluation of pain, especially in cats, which has led to the publication and validation of different pain scoring in this species. The number and diversity of pain scales in use demonstrates there are deficiencies in each and suggests that none has been universally adopted. In addition, scales used to evaluate pain in the acute setting may be less useful in the assessment of chronic pain.

Numerous pain assessment scales have been developed for humans, including the simple descriptive scale (SDS), visual analog scale (VAS), numerical ratings scale (NRS), and the multifactorial pain scale. All have been used and adapted for veterinary patients, with variable clinical utility.

Unfortunately, SDS, VAS, and NRS may all be unreliable in assessing acute pain in dogs in a hospital setting because they are all unidimensional. They measure only the intensity of pain rather than also encompassing its sensory and affective (behavioral) qualities [41, 46, 47].

Attempts have been made to overcome this unidimensional limitation by refining the assessed behavioral characteristics and including more objective physiologic data.

The Melbourne pain scale (MPS), developed in the late 1990s, consists of six broad categories divided into levels with associated scores [48]. The maximum possible score using this scale is 27. The MPS represents a refinement of the scales previously described but also has limitations. Again, significant interobserver variability occurs and the MPS is insensitive to non-overt behavioral displays as demonstrated by the example of a quiet, depressed, immobile, inappetent amputee suggested by Hansen [49].

The need to assess the intensity, sensory, and motivational or behavioral aspects of pain formed the basis for the development of multifactorial or composite-measure pain scales, which provide better interobserver repeatability.

The Colorado State University Veterinary Teaching Hospital pain score for cats and dogs is based on eight categories of behavioral and physiologic signs [50]. Comfort, movement, appearance, unprovoked behavior, interactive behavior, vocalization, heart rate, and respiratory rate are assessed. Each category is assigned a score of 0–4 according to predefined criteria. The total score is the sum of the category scores. Unfortunately, the detailed rationale for the selection of these categories is obscure, which has limited its use by other researchers working in this field. In a recent study the interrater reliability and convergent validity of the Colorado State University feline acute pain scale (CSU-FAPS) was assessed. The CSU-FAPS showed moderate-to good interrater reliability when used by veterinarians to assess pain in cats after ovariohysterectomy. Unfortunately, this scale showed a wide interrater reliability, which makes its use in a clinical setting not yet recommended [51].

The Glasgow composite measure pain scale (GCMPS) designed using psychometric principles (reliability, validity, standardization, freedom from bias), has been validated for assessing acute pain in dogs [52–55]. The GCMPS is a behavior-based system based on a structured questionnaire that includes clinical observation, assessment of spontaneous and evoked behavior, and animal–observer interaction. The questionnaire is structured around seven categories: posture, activity, vocalization, attention to wound/painful area, demeanor, mobility, and response to touch. To standardize scoring by multiple observers, each category contains descriptors from which those best matching the dog's behavior are selected. A rapid, easy-to-use short form was developed to facilitate use in a busy clinical setting (Figure 47.3) [56].

Assessment of pain in cats can be extremely challenging because behavioral manifestations may be subtle especially in the clinical setting [57–59]. Observation of behavior remains the best means of assessing the degree of pain in feline patients (Figure 47.4) [42, 59]. A VAS that includes physical interaction called the dynamic and interactive visual analog scale has been used in cats by several groups to improve sensitivity [47, 60]. Most information can be acquired if the animal is observed first from a distance, its response to a person's approach assessed, and finally its response to physical interactions such as stroking or palpation evaluated. Wound sensitivity and response to palpation correlates well with VAS scoring in cats. Manipulation of the affected area appears to be of value in feline pain evaluation [47, 59, 61].

SHORT FORM OF THE GLASGOW COMPOSITE PAIN SCALE

Dog's name _____

Hospital Number _____ **Date** / / **Time**

Surgery Yes/No (delete as appropriate)

Procedure or condition _____

_____

In the sections below please circle the appropriate score in each list and sum these to give the total score.

A. Look at dog in Kennel

Is the dog?

(i)		(ii)	
Quiet	0	Ignoring any wound or painful area	0
Crying or whimpering	1	Looking at wound or painful area	1
Groaning	2	Licking wound or painful area	2
Screaming	3	Rubbing wound or painful area	3
		Chewing wound or painful area	4

In the case of spinal, pelvic or multiple limb fractures, or where assistance is required to aid locomotion do not carry out section **B** and proceed to C
Please tick if this is the case ☐ then proceed to C.

B. Put lead on dog and lead out of the kennel.

When the dog rises/walks is it?

(iii)	
Normal	0
Lame	1
Slow or reluctant	2
Stiff	3
It refuses to move	4

C. If it has a wound or painful area including abdomen, apply gentle pressure 2 inches round the site.

Does it?

(*iv*)	
Do nothing	0
Look round	1
Flinch	2
Growl or guard area	3
Snap	4
Cry	5

D. Overall

Is the dog? (*v*)		*Is the dog?* (*vi*)	
Happy and content or happy and bouncy	0	Comfortable	0
Quiet	1	Unsettled	1
Indifferent or non-responsive to surroundings	2	Restless	2
Nervous or anxious or fearful	3	Hunched or tense	3
Depressed or non-responsive to stimulation	4	Rigid	4

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Total Score (i+ii+iii+iv+v+vi) = _____

Figure 47.3 Short form of the Glasgow Composite Measure Pain Scale. Copyright 2008University of Glasgow. Permission granted to reproduce for personal and educational use[HDR1] only. Commercial copying, hiring, lending is prohibited. The short form composite measure pain score (CMPS-SF) can be applied quickly and reliably in a clinical setting and has been designed as a clinical decision-making tool that was developed for dogs in acute pain. It includes 30 descriptor options within 6behavioral categories, including mobility. Within each category, the descriptors are ranked numerically according to their associated pain severity, and the person carrying out the assessment chooses the descriptor within each category that best fits the dog's behavior/condition. It is important to carryout the assessment procedure as described on the questionnaire, following the protocol closely. The pain score is the sum of the rank scores. The maximum score for the 6 categories is 24, or 20 if mobility is impossible to assess. The total CMPS-SF score has been shown to be a useful indicator of analgesic requirement, and the recommended analgesic intervention level is 6/24 or 5/20.

In recent years, three different composite pain scales have been described and validated for cats (Box 47.1) [42, 58, 59].

Future directions in the field of pain assessment may involve computerized behavior and activity monitoring [49, 67]. One study reviewed the use of monitoring parasympathetic tone activity and the bispectral index as objective tools for perioperative pain evaluation dogs and cats. Such systems have not been clinically validated [67].

The evaluation of pain with the use of a scoring system should be performed with every patient deemed to be in

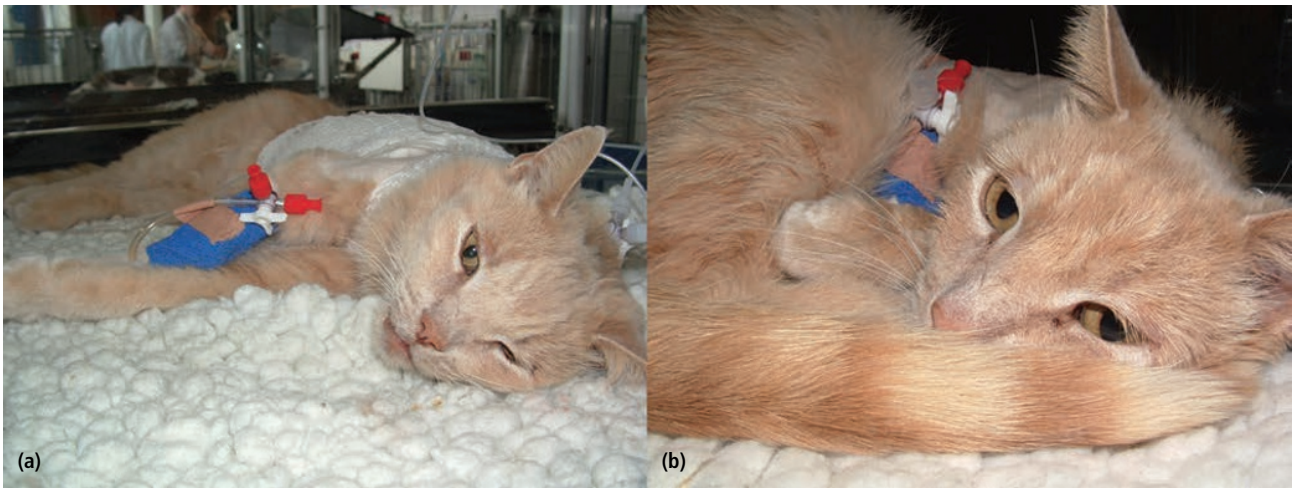


Figure 47.4 Observation of posture and behavior are key components of pain assessment in cats. (a) A feline patient recovering following a thoracotomy. The cat is laterally recumbent, inactive, and disinterested in the environment. (b) The same cat following pain assessment and augmentation of the analgesic plan. The cat appears more comfortable and is seen adopting a more typically feline curled-up pose. *Source:* Images courtesy of Dr. J. H. Robben (Intensive Care Unit, Utrecht University).

Box 47.1 Composite Pain Scales for Cats

UNESP-Botucatu Multidimensional Composite Pain Scale

Created assessing postoperative pain in cats undergoing ovariohysterectomy [62].
Link: www.animalpain.com.br/assets/upload/escala-en-us.pdf

Pain assessment
The pain assessment is divided in to three subscales:

- *Pain expression*: including miscellaneous behavior, vocalization, reaction to abdomen/flank and surgical wound.
- *Psychomotor changes*: including evaluation of the cat's attitude, posture, comfort, and activity.
- *Physiological variables*: including appetite and arterial blood pressure measurement.

Scoring
Maximum score: 30.
Analgesia is recommended with a score of 7/30 or higher.

Limitations
The limitations of this pain scale: time consuming, the inclusion of blood pressure measurement and the single surgical procedure used to develop it [59].

Glasgow Revised Composite Measure Pain Scale: Feline

Designed to be a clinical decision-making tool for use in cats in acute pain [63].
Link: https://www.newmetrica.com/acute-pain-measurement.

Pain assessment
This revised version was developed using psychometric principles (Calvo et al. [64]) and embedded a three-point facial expression (ear position and muzzle) scale within the tool [58, 64, 65]. It is a behavior-based system, structured around seven behavioral categories.

Scoring
Within each category, the descriptors are ranked numerically.
Maximum score for the seven categories: 20.
The recommended analgesic intervention level is > 4/20.

Feline Grimace Scale

The newest among pain scales [66].

Pain assessment
The scale relies on facial expressions that can be objectively assessed using a facial action coding system that measures the individual movements or "action units" of the face that comprise an expression. A feline-specific coding scheme (CatFACS) had been previously developed by studying the facial musculature of the domestic cat. Five action units were highlighted in cats that could easily discern cats in pain versus control cats: ear position, whiskers position, orbital tightening, muzzle tension, and lowering of the head.

Scoring
The scale detected response to analgesic treatment (scores after analgesia were lower than before) and a cut-off score was determined (total pain score > 0.39/1.0).

pain and the same scoring system should be used to re-evaluate and titrate the effect of the administered analgesic. The PLATTER approach (plan, anticipate, treat, evaluate and return) has been proposed to describe the continuum of care loop for pain management [27].

Multimodal Analgesia

Multimodal analgesia is an approach to pain relief provision that involves the use of multiple drug types or techniques with complementary mechanisms and sites of action within the peripheral and central nervous systems. Some authors have used the term solely in reference to systemic administration of multiple drugs with different mechanisms of action [68] whereas others refer to systemic drug administration combined with regional anesthesia [69].

The aim of this approach is to target different points in the nociceptive pathways so more complete analgesia can be provided while minimizing adverse effects. The multimodal approach uses the additive or synergistic analgesic effects of multiple drug classes, maximizing patient comfort and allowing lower doses of individual analgesic agents to be used, reducing the potential for ADRs [70]. Effective implementation of multimodal analgesia requires an understanding of physiology and pathophysiology so that complementary drugs and methods can be selected. This approach has been adopted by the medical and veterinary medical world [27, 69, 71–74].

Timing of Analgesic Administration

The motto of the Global Year Against Acute Pain was "anticipate, assess, alleviate." In humans, universal agreement that pain management should begin before surgery using an effective preemptive approach, involving multimodal analgesia is well established [75]. Preemptive analgesia is an analgesic intervention begun before the onset of noxious stimuli to block peripheral and central nociception, thereby preventing postoperative pain amplification [75, 76]. Continuing nociceptive input modulates the CNS through activation-dependent plasticity resulting in peripheral sensitization, allodynia, and hyperalgesia. Sensitization causes increased spontaneous activity and afferent responsiveness, reduced threshold, and prolonged discharge after repeated stimulation ("wind-up"). Neuronal plasticity induces "pain memory" allowing a more rapid response from the CNS following future similar stimuli. Maladaptive plasticity thereby leads to central sensitization [71, 76, 77].

The concept of preemptive analgesia has been much debated among anesthetists and pain management specialists [78, 79]. Kissin, in 1994, proposed that preemptive should mean "preventive" rather than simply "before" because ineffective afferent blockade cannot be preemptive even if administered before the noxious stimulus of surgery [77]. Preventive analgesia differs from preemptive analgesia in that it considers all three perioperative phases (preoperative, intraoperative, and postoperative) and that factors within all three phases can contribute to the development of central sensitization [76, 78]. Preventive analgesia is based on the assumption that the only way to prevent central sensitization might be to completely block any nociceptive input from the surgical wound from the time of incision until final wound healing. The blockade of nociceptive pathways must therefore be maintained from the preoperative stage, throughout surgery, and into the postoperative period approach [76, 78].

Whether preemptive analgesic interventions are more effective than conventional regimens in managing acute postoperative pain remains controversial. Some anesthesiologists now favor use of a multimodal approach to therapy with less concern about the timing of their interventions [75]. Others believe that for a preemptive technique to be effective, it must be multimodal and active before, during, and after surgery [80].

Contraindications and Complications of Pain Management

Recent shifts in veterinarian attitudes toward pain management and the use of the multimodal approach have greatly increased the frequency and diversity of analgesic prescribing [27, 28, 76]. Although concerns about the potential for adverse reactions associated with analgesic administration have been reported, it is likely these have been overemphasized [34, 37, 81–83]. Despite improvements in our understanding of feline drug disposition, such concerns have slowed progress in provision of pain relief to cats [31, 84]. There is no longer a rationale for withholding analgesics from cats because previously held misconceptions about complications associated with analgesics in the species have been largely discredited [27, 40, 42, 85–87]. But we must remain vigilant for adverse effects to manage them correctly.

An ADR is defined as any unexpected, undesirable, or harmful consequence associated with the administration of a medication. ADRs may be common, mild, and reversible or rare, severe, and permanent [88]. As with any medications, analgesic agents have the potential to cause such negative effects. Indeed, in human hospitals, analgesics are associated with a large number of adverse effects that can lead to an increase in the duration of stay in the intensive care unit

(ICU) or potentially be fatal [89–91]. A prospective study of analgesic prescribing in a veterinary ICU suggested that ADRs or concerns about adverse effects affect the prescribing of analgesic medication for small animals [92]. The authors identified that although 64% of analgesic medication was administered as prescribed, 23% of prescribed doses were reduced or not administered. Dose reductions were primarily due to concerns about levels of sedation, hypotension, and hypothermia associated with analgesic administration.

Although we must consider the potential for ADRs, such concerns should not prevent us from improving efforts to control pain in our patients. It should also be remembered that the risk of analgesia-related complications is reduced in patients in pain. When in doubt if a patient is (still) in pain, a test dose of an analgesic drug can be administered and monitored closely for any signs for ADRs. We should be vigilant for signs of adverse reactions but proactive in our provision of pain relief because pain itself reduces the likelihood of a successful outcome for our patients.

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48

Systemic Analgesia

Sarah Haldane and Angela Chapman

Pain can occur in small animals as a result of trauma, disease, or surgery. The detrimental effects of pain include tachycardia, altered respiration, aggravation of the sympathoadrenal response, and behavioral changes. Pain can also lead to alteration of glucose metabolism, increased protein catabolism, and impaired wound healing and it can severely compromise the ability of a patient to recover from severe illness or trauma [1].

Although analgesia is an essential component of therapy for many patients, analgesic medications can have adverse effects. The objective of a balanced analgesic regimen is to combine medications to provide the best pain relief with the fewest complications. To choose the most appropriate medications for the individual patient, a basic knowledge of the effects of these drugs on essential organ systems is required.

Opioids

Opioids can be used for moderate to severe pain. The range of medications within this class of drugs allows for flexibility in route and frequency of administration. Opioids exert their analgesic effects by activating opioid receptors located within the brain, spinal cord, and peripheral neurons. There are three main families of opioid receptors: mu, kappa, and delta, and each has differing clinical effects when activated (Table 48.1). Every opioid activates each of the receptors to a different degree, so the activity of each drug can vary in terms of strength of analgesia, degree of sedation, and adverse effects (Box 48.1). Opioids can be generally classified into (i) mu receptor agonists, (ii) partial mu receptor agonists, (iii) mixed agonist–antagonist drugs, and (iv) opioid antagonists.

Some common mu receptor agonists include morphine, fentanyl, oxymorphone, hydromorphone, and methadone (Table 48.2). These drugs have strong analgesic effects and can be used for patients with moderate to severe pain. Analgesia provided by mu agonists is dose related; increased dose will increase analgesia. Similarly, the incidence of adverse effects increases when higher doses are administered.

Buprenorphine is a partial mu agonist opioid with a long duration of action. It has a high affinity for the mu receptor but only moderate efficacy. It is an antagonist at kappa receptors.

Butorphanol is a mixed agonist–antagonist drug and acts as a kappa agonist and mu antagonist. This drug has limited analgesic activity and duration and it is used primarily for its sedative effects [2].

Opioid antagonists include naloxone and naltrexone. They have very high affinity for opioid receptors, so will block the effects of any of the agonist opioids.

Analgesic Effects

Opioids have a central analgesic effect that works within the brain and the spinal cord to reduce transmission of pain signals. There are also opioid receptors located on the presynaptic terminals of the peripheral nociceptors. When opioid receptors are activated, they inhibit the transmission of painful stimuli through these fibers. In addition, activation of receptors in the brain decreases the amount of the neurotransmitter gamma-amino-butyric acid (GABA) that is released from neurons. GABA is an inhibitory neurotransmitter that reduces the production and release of the stimulatory neurotransmitter dopamine. Stimulation of opioid receptors thus increases available dopamine, which is responsible for the pleasurable sensation associated with opioid administration [5].

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Table 48.1 Opioid receptors.

Receptor	Location	Analgesic effects	Central nervous system effects	Respiratory effects	Gastrointestinal effects
Mu	Brain (medial thalamus), brainstem	Supraspinal analgesia	Euphoria, sedation, dependence	Respiratory depression	Decreased gastrointestinal motility
Kappa	Brain (diencephalon, including limbic system), brainstem, spinal cord	Spinal analgesia	Sedation, dependence, dysphoria	Dyspnea, respiratory depression	
Delta	Brain	Possible pronociceptive effects. Development of tolerance to opioids	Dysphoria		

Box 48.1 How Strong Is your Opioid?

1) The *affinity* of an opioid is a measure of how strongly it interacts and binds with its receptor. For example, buprenorphine has a higher affinity for mu receptors than morphine, so it will bind more strongly to these receptors and can competitively antagonize the effects of morphine.
2) The *efficacy* is a measure of the strength of the effect the drug has after binding to its receptor. For example, morphine has a higher efficacy at mu receptors than buprenorphine, which means that when it binds to the receptor it will stimulate a stronger analgesic response.
3) The *potency* of a drug is the amount of drug required to produce an effect. For example, fentanyl is a more potent drug than morphine as a much smaller amount (micrograms rather than milligrams) is required to activate the receptor.
   - An *agonist* opioid binds strongly to the receptor and stimulates them to high activity. These drugs are considered to have both affinity and efficacy.
   - An *antagonist* opioid binds to the receptor (has high affinity) but does not stimulate the receptor (no efficacy). Its antagonist action comes from binding to the receptor as it stops other opioids from exerting their effects.
   - A *partial agonist* opioid has affinity but less efficacy than pure agonists; they bind strongly to opioid receptors but have a weaker effect on them [2].

Central Nervous System Effects

Opioids act in the central nervous system to cause sedation. They are often used as premedication agents prior to anesthesia and have an anesthetic-sparing effect in addition to their analgesic activity [6–8]. They are also effective anxiolytic agents. Chronic administration or high doses of opioids can lead to dysphoria and, in some cases, vocalization. These effects will usually resolve if the patient is changed to a different opioid medication or to a lower dose of the same medication [2].

Cardiovascular Effects

Administration of opioids at therapeutic doses has little clinical effect on the cardiovascular system [9]. Opioids are considered relatively safe drugs in cardiovascularly compromised patients. However, bradycardia can develop if these medications are administered at high doses in combination with sedative drugs or to patients not in pain [2, 9]. Opioid-induced bradycardia is vagally mediated so can be effectively treated with atropine or glycopyrrolate. In most cases, the effects of pain counteract the vagal effects, so bradycardia is an indication that the patient's degree of pain has reduced and the opioid dose can be decreased.

In feline studies, both morphine and fentanyl have been shown to cause pulmonary vasodilation [10, 11]. In a study using desflurane-anesthetized dogs, butorphanol also caused pulmonary vasodilation [12]. For this reason, opioids are often recommended for use as anxiolytic agents in patients with congestive heart failure.

Respiratory Effects

At high doses, opioids can cause respiratory depression. This effect tends to be more marked when patients are being treated with concurrent sedative or anesthetic agents. Respiratory depression occurs soon after bolus administration of opioid drugs but tolerance to this effect develops quickly, so that repeated or long-term opioid use will have little effect on the respiratory system [5].

Morphine, methadone, oxymorphone, and hydromorphone have all been associated with panting in dogs. Panting usually starts soon after administration of the medications and tends to be transitory [13, 14].

Table 48.2 Opioid agonist and antagonist drugs.

Opioid	Agonist activity	Antagonist activity	Dose in dogs	Dose in cats	Notes
Morphine	μ κ δ	–	0.1–0.5 mg/kg IM, SC every 4–6 hours; CRI 0.1–0.2 mg/kg/hour	0.1 mg/kg IV, IM, SC every 4–6 hours	Histamine release if given IV in dogs, leading to vasodilation, panting
Fentanyl	μ	–	2–5 mcg/kg IV, IM, SC then CRI 2–10 mcg/kg/hour	2–5 mcg/kg IV, IM, SC then CRI 2–10 mcg/kg/hour	
Remifentanil	μ	–	0.1–0.5 mcg/kg/minute IV	0.1–0.5 mcg/kg/minute IV	
Hydromorphone	μ (δ)	–	0.1–0.2 mg/kg IV, IM, SC every 4–6 hours	0.1 mg/kg IV, IM, SC every 4–6 hours	Panting
Oxymorphone	μ	–	0.1–0.2 mg/kg IV, IM, SC every 4–6 hours	0.1 mg/kg IV, IM, SC every 4–6 hours	Panting
Methadone	μ	NMDA receptor	0.1–0.5 mg/kg IV, IM, SC every 4–6 hours	0.1–0.2 mg/kg IV, IM, SC every 4–6 hours	Panting
Buprenorphine	μ	κ	0.01–0.03 mg/kg IV, IM, SC, TM every 6–8 hours [3]	0.01–0.03 mg/kg IV, IM, SC, TM every 6–8 hours [4]	Dysphoria, euphoria (cats), mydriasis
Butorphanol	κ	μ	0.1–0.4 mg/kg IV, IM, SC; 0.5–1 mg/kg PO every 6–8 hours	0.1–0.4 mg/kg IV, IM, SC	Sedative effects much longer duration (2–4 hours) than analgesic effects (30 minutes)
Codeine	μ	–	0.5–2 mg/kg PO every 6–12 hours	0.5–2 mg/kg PO every 6–12 hours	Care with products that contain paracetamol in dogs; do not use these products in cats
Naloxone	–	μ κ δ	0.04 mg/kg IV IM SC	0.04–0.1 mg.kg IV	Can be given via endotracheal tube during cardiopulmonary resuscitation
Naltrexone	–	μ κ δ	1–2 mg/kg PO once daily	25–50 mg/kg PO once daily	Bitter taste

CRI, intravenous constant rate infusion; IM, intramuscular; IV, intravenous; PO, per os (orally); SC, subcutaneous; TM, transmucosal.

Many opioid drugs have a cough suppressant (antitussive) effect, making them useful for bronchial disease or tracheitis [15]. They also reduce laryngeal sensitivity, increasing the ease of tracheal intubation and prolonging a patient's tolerance of endotracheal intubation after anesthesia.

Urinary Effects

Administration of mu agonist opioids has been shown to cause decreased urine production by the kidneys. Conversely, kappa agonist opioids have been shown to have a diuretic effect in rats[16]. The exact mechanism for these effects are unknown [17]. Opioid use has also been associated with urinary retention in the bladder due to an increase in urethral sphincter smooth muscle tone [5].

Gastrointestinal Effects

Two of the most common and most undesirable effects of opioid administration are nausea and vomiting. These effects occur due to stimulation of the chemoreceptor trigger zone. This zone lies outside the blood–brain barrier so it is rapidly penetrated by any drug in the plasma. The vomiting center lies inside the blood–brain barrier. Interestingly, stimulation of the opioid receptors in the vomiting center has an antiemetic effect. As opioids easily cross the barrier, they can counteract the initial vomition impulse and have an ongoing antiemetic effect [18]. Animals are more likely to vomit with administration of pure mu agonist opioids than with partial agonist or mixed agonist-antagonists.

Opioids can decrease gastric emptying and alter intestinal motility, leading to intestinal ileus and, more chronically, constipation [2, 9, 18, 19]. These effects are mediated through opioid receptors in the central nervous system [18]. Decreased intestinal motility, in combination with opioid-induced sedation, can contribute to a patient's lack of appetite which is a common complication of prolonged illness in small animal patients.

Hepatic Effects

Opioids are primarily metabolized in the liver, although some extrahepatic metabolism does occur [13]. Animals with hepatic insufficiency may require lower doses of opioids to achieve adequate analgesia. The sedative effects of opioids can be pronounced in patients with hepatic dysfunction.

Opioids that do not require hepatic metabolism (e.g. remifentanil) or with a short duration of action administered as a constant rate infusion (CRI; e.g. fentanyl, sufentanil, alfentanil) are the most appropriate choice for patients in hepatic failure so that the dose can be titrated to achieve adequate analgesia with minimal sedation.

Effects on Body Temperature

In dogs, mu agonist opioids have been shown to reduce body temperature by altering the central thermoregulatory set point. Opioid-induced panting could also contribute to a decrease in body temperature as heat loss via the respiratory system is a major factor in canine thermoregulation [13].

In cats, opioid administration has been associated with post-anesthetic hyperthermia. In one retrospective study, 47% of 125 cats became hyperthermic at least once within a 20-hour period after anesthesia and opioid administration. In 2 of 125 cats, palliative therapy did not reduce the rectal temperature and naloxone was administered. In both cats, naloxone was effective in reducing temperature. The mechanism for opioid-induced hyperthermia in cats is not known. It does not tend to be responsive to the anti-pyretic effects of nonsteroidal anti-inflammatory drugs (NSAIDs) [20].

Opioid Tolerance and Addiction

With prolonged administration opioids become less effective as analgesic agents in a phenomenon known as opioid tolerance. Doses often need to be increased markedly to maintain analgesia in patients that require analgesia for more than a few days, and in experimental trials, tolerance has been shown to begin after a single dose of opioid. The mechanisms associated with development of tolerance are complex; tolerance is mediated in part by the opioid receptors themselves. With chronic activation the mu receptors become less sensitive to opioid binding, grow less effective in reducing transmission of pain signals and in some cases, internalize within the cell membrane and become inaccessible to circulating opioids. As mu receptors become resistant to opioids, delta receptors are upregulated. It has been hypothesized that activation of delta receptors decreases the effectiveness of mu receptor stimulation. *N*-methyl-D-aspartate (NMDA) receptor activation also contributes to opioid tolerance by increasing production of pronociceptive neurotransmitters [21, 22].

Similar mechanisms lead to development of opioid dependency and withdrawal. Stimulation of delta receptors and upregulation of the NMDA receptors lead to increased pain sensation, so patients become uncomfortable and disoriented if opioid therapy is rapidly withdrawn. In people the pleasurable sensation (mediated by dopamine) associated with opioid use and the adverse sensation when opioids are withdrawn can lead to addiction [5, 21, 22]. There is no evidence that dogs and cats become addicted to opioids.

Use of a balanced analgesic regimen (multimodal analgesia) can be effective in preventing development of opioid tolerance. Abrupt cessation of opioid analgesia should be avoided to reduce symptoms of opioid withdrawal in veterinary patients. Instead, weaning the patient from a pure mu agonist drug to a partial agonist such as buprenorphine or gradually decreasing the dose prior to discontinuation is recommended.

Regulations Involved in Clinical Use of Opioids

The phrase "controlled drugs" refers to drugs of addiction or high abuse potential that are supplied to registered practitioners for medical use. Opioids are classified as controlled drugs because they have the potential to produce addiction in people. Legislative requirements for controlled drugs vary slightly from country to country and state to state. An overview of the general legislative requirements is provided here; however, reference should be made to the specific country or state's individual legislation for full details.

In the United States, limitations on production and prescription of opioids have been introduced by the Drug Enforcement Agency in an effort to reduce opioid abuse in people. This has impacted the supply of these drugs for veterinary use. Prescription drug monitoring programs have been developed, with associated laws and regulations, on a state-by-state basis. Awareness has also been raised within the veterinary industry around identification of people who visit multiple veterinary clinics with their pet to obtain drugs for personal use, and drug diversion, which is the acquisition of veterinary drugs or prescriptions for personal use rather than for the intended recipient.

Supply of Controlled Drugs

Licensed wholesalers or pharmacists must only supply drugs to an authorized or licensed person. Details of registered practitioners and/or license numbers must be recorded by the supplier.

Storage Requirements

Controlled substances must be stored in a securely locked, substantially constructed cabinet or safe, fixed to the floor

or wall. Controlled drugs should not be stored with any items other than other drugs of dependence. The receptacle should be kept locked at all times other than when in immediate use and should be accessed only by authorized personnel.

Record Keeping Requirements

A drug registry must be maintained in a form that shows the balance remaining after each dose is administered or supplied and cannot be altered without detection. A separate page is required for each drug and for each different formulation of the drug. The date, patient's name, owner's name, amount of the drug dispensed, balance, and details of prescribing veterinarian and their signature, are the minimum inclusions required on a drug register. Details of incoming stock, date, invoice number, supplier, and the balance of stock are also required. Records are to be retained for periods of two to three years in most countries and must be readily available for the regulatory authorities should an audit occur.

Disposal

The legal requirements for the disposal of controlled drugs varies; however, disposal generally needs to be witnessed and co-signed on the drug register along with the quantity of drug disposed.

Systemic Administration of Opioids

Opioids can be administered parenterally via the intravenous (IV), intramuscular (IM), and subcutaneous (SC) routes [14]. Morphine is usually administered IM or SC to dogs or very slowly via the IV route, as it has been associated with histamine release when given as a rapid IV bolus, which causes vasodilation and potentially hypotension [23, 24].

Most opioids can be administered by either intermittent bolus injections or as a CRI. Longer acting opioids (such as buprenorphine) are rarely given as a CRI, while this is the primary method of administration of very short acting opioids like fentanyl.

Transdermal patches can be used to provide long-term administration of an opioid at a constant rate. Fentanyl is often administered via this route and buprenorphine patches are also available. Onset of action can take 12–18 hours for fentanyl patches and duration of action is three to five days. As there is significant interindividual variation in the plasma concentration of fentanyl reached when transdermal patches are applied, the patient's comfort level should continue to be monitored while the patches are in place [25, 26]. Effective administration of the drug can be adversely affected by the loss of adherence of the patch. Fentanyl from the patches can also be absorbed transmucosally, so patients should not be allowed to lick or chew the patch [27].

Most opioids are relatively ineffective if given orally as they are subject to a strong "first pass" effect, meaning that they are absorbed from the gastrointestinal tract and metabolized in the liver to inactive substrates before ever entering systemic circulation. Opioids that can be used as oral medications in dogs and cats include codeine and butorphanol. An oral form of morphine is available but has varying absorption in dogs and thus is difficult to dose effectively. The doses required for oral administration are up to 10 times higher than those given parenterally. The main adverse effect of orally administered opioids is constipation.

Injectable preparations of buprenorphine are well absorbed through the oral mucosa in cats. This is an effective route of administration in this species as long as the drug is not swallowed into the digestive tract. To prevent this, the buprenorphine should be squirted under (rather than on top of) the tongue or along the gum line. This route is also effective for administration of buprenorphine to dogs. However, in one study transmucosal (TM) administration to dogs achieved approximately 30–40% of the serum plasma concentration of buprenorphine than an equivalent IV dose and had a shorter duration of analgesic action. Therefore, higher dose rates of buprenorphine are indicated for TM administration in dogs [28–31]; the cost can be prohibitive for larger dogs in the current veterinary market. Buprenorphine tablets are also available for transmucosal administration.

Morphine

Morphine is the prototypical mu agonist opioid. It is an excellent analgesic and sedative in both dogs and cats. At high doses (1–2 mg/kg) it has been associated with hyperexcitability in cats, but these effects are rarely witnessed at analgesic doses of 0.1 mg/kg. Morphine can cause vomiting after bolus administration and in some cases is used as an emetic agent. Bradycardia, gastrointestinal stasis, constipation, reduced urine output, and urinary retention have all been associated with morphine administration. Morphine is metabolized in the liver; codeine and hydromorphone are two of the metabolites produced. Intravenous morphine administration can cause histamine release in dogs (see section on systemic administration of opioids) [5, 6, 17, 18, 32–38].

Fentanyl, Remifentanil, Sufentanil, Alfentanil

Fentanyl is approximately 80 times more potent than morphine because it binds very strongly to the mu opioid receptor, so preparations of the drug have a much lower

concentration. It has a rapid onset of action and rarely causes gastrointestinal side effects such as vomiting or nausea. It is also an extremely effective sedative. Administration can lead to bradycardia and decreased temperature. Fentanyl can have respiratory depressant effects similar to morphine, [39] and although these effects are relatively minor at therapeutic doses [40], fentanyl's rapid onset can result in acute hypoventilation [41]. Fentanyl's duration of action is very short and so it is primarily used as a CRI or administered in a transdermal patch. Aside from decreased nausea, the primary advantage of using fentanyl as a CRI is the ability to rapidly change the dose and hence the analgesic and sedative effects. Fentanyl can be used during anesthesia to reduce the requirement for gaseous anesthetic agent [7] and is often used in combination with other sedatives for patients requiring mechanical ventilation.

Remifentanil is an ultra-short-acting opioid with a half-life of three to six minutes. It can be used as an anesthetic-sparing agent and analgesic in small animals. Its primary advantage is that it is broken down by cholinesterase enzymes in the plasma, so metabolism is not dependent on hepatic or renal function. Also, with such a short half-life, it is very easy to rapidly change the plasma level of the drug, which can be of benefit in animals that have cardiac or respiratory dysfunction [42–44].

Sufentanil and alfentanil are very rapid-acting derivatives of fentanyl. Both have anesthetic-sparing effects and sedative and analgesic efficacy in small animal patients, and both allow rapid recovery from anesthesia. Both drugs can cause bradycardia in dogs but have minimal effect on heart rate in cats. In clinical trials, alfentanil was associated with hypotension when administered with propofol as total IV anesthesia, but this effect was not seen when it was administered with isoflurane [45].

Oxymorphone and Hydromorphone

Oxymorphone and hydromorphone are semi-synthetic mu agonist opioids with similar analgesic efficacy to morphine [13, 14]. Although they can display a similar range of adverse effects to morphine, the incidence of occurrence is reduced. Histamine release is not associated with IV administration of either of these drugs [23]. Hydromorphone has a shorter half-life than morphine in dogs [46]. In both canine and feline clinical trials, hydromorphone appears to have a similar duration of sedative and analgesic action as oxymorphone (four to six hours) [8, 47, 48].

Methadone

Methadone is a synthetic mu agonist opioid that also has antagonist activity at NMDA receptors. Methadone has a prolonged duration of activity and good oral availability in people; the oral form is used to treat heroin addiction [5]. In dogs and cats, the duration of action of methadone is much shorter. When administered IV, the onset of action is 5–10 minutes and duration of action is four to six hours. After SC administration, absorption is variable and it can take more than an hour to reach maximal plasma concentrations. The plasma half-life is longer (10 hours as compared to four hours for IV administration) and dogs are more likely to vocalize after subcutaneous administration [49].

Pethidine (Meperidine)

Pethidine is mu agonist opioid that has excellent analgesic activity. It has a short duration of action (45-60 minutes) and is associated with a relatively high incidence of dysphoria [50]. It is rarely used in small animal clinical practice.

Buprenorphine

Buprenorphine is a partial agonist (Box 48.1) at mu receptors and an antagonist at kappa receptors. It is effective in dogs for mild to moderate pain [5]. Buprenorphine appears to have better analgesic efficacy in cats than dogs and can also cause euphoria and mydriasis in feline patients[4, 50, 51]. It has not been proven to be associated with post-anesthetic hyperthermia in cats in contrast to the pure mu agonist opioids [20].

Buprenorphine has a long duration of activity in comparison with other opioids and has a higher affinity for mu receptors than the full mu agonist opioids. This means that it can be used to wean patients from full mu agonist opioids or as a partial antagonist if mu agonists are causing adverse effects.

This can be an undesirable effect if buprenorphine is given to a patient with severe pain, as it will blunt the effects of any other opioids that are subsequently administered for the duration of its activity (6–12 hours) [52]. Buprenorphine causes minimal sedation compared with full mu agonists and has less of an inhibitory effect on the gastrointestinal tract [4].

Butorphanol

Butorphanol is a kappa agonist and provides good sedation with minimal cardiovascular or respiratory effects. It is a weak analgesic with a short duration of activity. The duration of analgesic effect is one to two hours, while its sedative effects last two to four hours [52–54]. Its analgesic and sedative activities have a "ceiling" effect, whereby increasing the dose of the medication will not increase its efficacy. Butorphanol can antagonize the effects of pure mu agonist opioids if they are used in combination [55].

Butorphanol is an excellent antitussive agent so is often used as a short-term agent to control coughing. It also has some antiemetic effects. The primary adverse effect of butorphanol is dysphoria.

Codeine

Codeine has greater oral availability than any of the other opioids. It has weak activity at mu receptors. Codeine is used in veterinary medicine for chronic pain or as an antitussive agent[15]. The main adverse effect of codeine is constipation, making it a difficult drug to use long term. It can also cause vomiting, even at low doses, through stimulation of the chemoreceptor trigger zone [5].

Naloxone and Naltrexone

These drugs have antagonist activity at mu, kappa, and delta receptors. Naloxone has a short duration of action and poor oral availability so is used primarily as a parenteral agent for opioid overdose or to rapidly reduce the effects of opioids in patients at risk of respiratory or cardiac arrest. Naltrexone has good oral availability and a long duration of action, so it is more frequently used in people recovering from opioid dependence than in veterinary medicine. It is sometimes effective for behavioral disorders such as tail chasing and excessive licking in dogs and cats.

Tramadol

Tramadol is a unique, centrally acting analgesic that has multiple mechanisms of action. It is a codeine analog and has some activity at mu opioid receptors. It also has a centrally acting GABA effect. In addition, tramadol has activity at alpha-2 adrenergic receptors and serotonin receptors, preventing norepinephrine and serotonin reuptake respectively [36, 56, 57].

Opioid Effects

Tramadol has a weak affinity for mu and delta opioid receptors [56]. Mu receptor activation provides some of its analgesic activity, as well as mild sedation and respiratory depressant effects [56–60]. It is metabolized in the liver to an active metabolite (O-desmethyl tramadol) that binds to opioid receptors with a higher affinity than the parent drug [57]. These metabolites are produced in most species of animals but in differing amounts. There are also species differences in elimination half-life and time to maximum plasma concentration, with dogs generally metabolizing and excreting the drug more rapidly than cats [57, 59, 61].

Despite its weak affinity for mu receptors, tramadol has not been associated with dependence in people; tramadol is scheduled as a controlled drug in the United States, although this is not the case in all areas of the world.

Route of Administration

Oral and IV forms of tramadol are available. Although maximum plasma concentration is reached more quickly when the drug is given IV, the oral form is rapidly absorbed from the gastrointestinal tract. The oral medication comes in tablets, capsules, and liquid form, making it easy to dose in any size cat or dog. It is generally prescribed in an intermediate-release form that can be administered two to three times a day. There is also a sustained-release form that is used for once-daily dosing in people. Dogs metabolize tramadol rapidly, so sustained-release tablets still need to be given twice a day in this species. Additionally, administration of the sustained-release tablets to dogs provides a lower maximum plasma concentration of tramadol than the intermediate-release tablets and it takes more time to achieve maximum plasma concentration [56, 59]. Tramadol has a longer elimination half-life in cats than in dogs (3.4 hours vs. 1.7 hours); however, time to maximum plasma concentration is similar [57, 59].

Clinical Efficacy of Tramadol

The analgesic efficacy of tramadol varies between individuals as well as between species. In cats it can be an effective analgesic when used in a variety of situations, including management of postoperative pain and osteoarthritis [62–65].

In dogs, the majority of published studies evaluate the effect of tramadol as a postoperative analgesic. While it can be an effective analgesic after some soft tissue surgeries [36, 66, 67], it is not as useful as NSAIDs or mu opiate agonists after orthopedic procedures or in dogs with osteoarthritis [68–72]. However, it may have some benefit as an adjunctive analgesic in this species [73–76].

Tramadol reduces the minimal alveolar concentration of sevoflurane in anesthetized cats [77]; however, it is less effective than opioid drugs as a sedative in dogs [35].

There are few adverse effects noted with clinical use of tramadol in dogs and cats. Administration of tablets and liquid can cause excessive salivation due to their bitter taste. Dispensing the liquid into a gelatin capsule can reduce this effect and allow for easier administration, though this needs to be done just prior to giving the medication or the liquid will permeate through the capsule. Seizures have been associated with the use of tramadol in people, but this has not been reported in veterinary medicine. Tramadol can be administered to both dogs and cats. The dose in dogs is 2–4 mg/kg every 8–12 hours. In cats, a dose of 1–2 mg/kg every 12 hours is used.

Nonsteroidal Anti-Inflammatory Drugs

NSAIDs are a diverse group of medications that act to decrease the production of inflammatory mediators at the site of tissue injury. Inflammatory mediators increase transmission of pain signals by stimulating previously unaffected nociceptors surrounding the area of injury.

Cyclo-Oxygenase 1 and 2

After a tissue has been damaged, arachidonic acid is released from cell membranes. The family of inflammatory mediators that are derived from arachidonic acid are known as eicosanoids. Two enzyme groups act to metabolize arachidonic acid: the lipoxygenases and the cyclo-oxygenases (COX). Eicosanoids produced from the lipoxygenase cascade include leukotrienes and chemotactic factors. Products of the COX cascade include prostaglandins, prostacyclin, and thromboxanes.

NSAIDs are potent inhibitors of COX enzymes, thereby reducing prostaglandin production. In some situations, this can have adverse consequences as many prostaglandins have protective or homeostatic functions. The prostaglandins required for normal homeostasis are continuously produced, and so are known as constitutive prostaglandins. Inducible prostaglandins are produced in response to tissue injury. Inducible prostaglandins act to escalate the inflammatory response and increase peripheral nerve sensitization; they also have protective actions and play a role in tissue repair [78, 79].

There are two main COX enzymes known as COX-1 and COX-2. A COX-3 enzyme also exists in the CNS. COX-1 is important in producing many constitutive prostaglandins, important for normal function of the gastrointestinal tract, kidneys, and primary hemostasis [78]. COX-2 produces more inducible prostaglandins that play an important role in gastric mucosal healing and systemic inflammation, as well as in regulating homeostatic mechanisms in the normal brain, kidney, and reproductive systems. COX-2 also catalyzes production of prostacyclin, and a balance exists between this anti-coagulant molecule and the pro-coagulant thromboxane A_2 regulated by COX-1 [79–81].

NSAIDs are described as being nonspecific or dual acting if they inhibit both COX-1 and COX-2. Aspirin is the classic example of a dual-acting NSAID. Others include ketoprofen and tepoxalin, which also have lipoxygenase-suppressing effects. COX-2-selective drugs, which inhibit COX-2 with relatively less suppression of COX-1, are commonly administered in veterinary medicine. It is important to realize that all COX-2-selective NSAIDs will still inhibit COX-1 to some degree. Examples include carprofen, meloxicam, deracoxib and firocoxib.

Analgesic Effects

There is considerable proof of the efficacy of analgesia in dogs and cats provided by NSAIDs. NSAIDs provide peripheral analgesia and, unlike opioids, are very effective at reducing pain during movement. They also have a mild antagonistic effect at NMDA receptors. The analgesic effects of many NSAIDs, including carprofen, ketoprofen, meloxicam, deracoxib, tepoxalin, and firocoxib (Table 48.3), have been favorably compared to opioid administration in various studies of osteoarthritic and surgical pain [50, 51, 53, 83–90].

NSAIDs are nonaddictive and nonsedating, so animals often feel better and are more alert on these medications than when being treated with opioid analgesics [50, 51, 53, 83–96]. Individual animals may respond better to one NSAID than another, so if analgesia is not adequate with one NSAID, it is often worth trialing a different nonsteroidal medication. However, different NSAIDs should never be used in combination as the risk of adverse effects greatly outweighs the potential benefits and a "washout period," where no anti-inflammatory medications are administered, of 5–7 days (or 7–10 days for aspirin) is recommended if changing from one NSAID to another [97].

Adverse Effects

In healthy dogs, most new generation NSAIDs have a wide margin of safety. Few NSAIDs are licensed for use in cats and none for long-term use. Chronic use or overdose of new generation prescription NSAIDs in small animals has been reported to cause adverse effects, but the risk is much less than with older generation NSAIDS, such as flunixin,

Table 48.3 Nonsteroidal anti-inflammatory drug doses.

Drug	Dog	Cat
Carprofen	2–4 mg/kg/day IV, SC, PO	2–4 mg/kg SC, PO once
Deracoxib	2 mg/kg PO every 24 hours	
Firocoxib	5 mg/kg PO every 24 hours	
Ketoprofen	1–2 mg/kg IV, SC then 1 mg/kg PO for no longer than 5 days	1–2 mg/kg IV, SC then 1 mg/kg PO for no longer than 5 days
Meloxicam	0.1 mg/kg IV, PO then 0.01–0.03 mg/kg PO every 24 hours	0.1 mg/kg IV, PO once, then 0.01–0.03 mg/kg PO every 1–2 days [82]
Tepoxalin	10 mg/kg PO every 24 hours	

IM, intramuscular; IV, intravenous; PO, per os (orally); SC, subcutaneous.

indomethacin, ibuprofen, or naproxen. However, in animals with perfusion deficits, gastrointestinal, renal, or hepatic disease, or with disorders of primary coagulation, NSAID administration can have significant detrimental effects.

Gastrointestinal Effects

Gastrointestinal ulceration is a common adverse effect of NSAID administration with prolonged use, high doses, or administration to hypovolemic or inappetent animals [97, 98]. COX-1 induced prostaglandins in the gastric mucosa have protective effects as they increase gastric mucous production and enhance local blood flow. COX-2 is induced once damage has occurred to the intestinal mucosa and produces prostaglandins that play an important role in mucosal healing [78, 80, 98–100]; thus, COX-2 suppression can delay ulcer healing [101]. Some NSAIDs, such as aspirin, also cause direct injury to the gastric mucosa by disruption of surface phospholipids [80].

Despite the increased safety margin of COX-2 selective NSAIDs, adverse gastrointestinal effects are still their most common adverse effects [97, 98]. In one experimental study assessing long-term NSAID use, carprofen had the lowest incidence of gastrointestinal effects when compared to meloxicam, ketoprofen, flunixin meglumine, and etodolac [53]. Multiple endoscopic and mucosal permeability studies have been performed evaluating the effects of short-term NSAID use and no significant adverse effects have been found. However, only a few dogs were assessed in each trial making it difficult to evaluate the importance of their findings [101–106].

Both deracoxib and meloxicam have been associated with gastrointestinal perforation in dogs. In most cases the drugs were administered at higher than recommended dosages (greater than 2 mg/kg for deracoxib or 0.1 mg/kg for meloxicam) or were administered concurrently with a corticosteroid or another NSAID [107–109].

The incidence of these highly undesirable adverse effects means that client education is vitally important if patients are discharged with NSAIDs for administration at home. The dose and duration of administration should be carefully explained, and clients should be advised not to administer the medications if their pet is not eating, has vomiting or diarrhea, or if they see hematochezia or melena. NSAIDs should never be administered concurrently with other NSAIDs or with corticosteroids.

Renal Effects

When perfusion to the kidney declines, glomerular filtration rate decreases accordingly. The juxtaglomerular apparatus in the kidney releases prostaglandins that act to vasodilate the afferent renal arteriole to maintain renal blood flow and glomerular filtration rate. This effect is mediated by both COX-1 and COX-2 enzymes[110]. When NSAIDs are administered to hypovolemic or dehydrated patients, the prostaglandin-mediated effect of local vasodilation is diminished or lost. Significant damage to the kidneys can result, and in severe cases this can lead to acute kidney injury. COX-2-selective medications may increase the relative production of thromboxanes, which have local vasoconstrictive effects and can exacerbate kidney damage. Ketoprofen, carprofen, and tepoxalin at therapeutic doses have all been shown to have little effect on renal perfusion in healthy anesthetized dogs [111–113]; however, overdose or accidental ingestion of large doses of NSAIDs can lead to acute kidney failure despite normal blood volume [97].

Hepatic Effects

NSAIDs are metabolized in the liver and their use has been associated with an increase in liver enzymes and hepatocellular injury in some patients. This toxicity is not dependent on dose or duration of treatment so is classified as an idiosyncratic reaction. In one report describing hepatoxicity secondary to carprofen administration, there was a marked similarity in the course of disease in Labrador Retrievers, which may indicate a possible underlying genetic basis in this breed. However, this result may have been biased by the large number of Labradors that require NSAID administration for degenerative joint disease [114]. Owing to the risk of hepatic toxicity, NSAID administration is not recommended for animals with concurrent hepatic disease.

Coagulation Effects

Prostacyclin (prostaglandin I2) is produced from epithelial cells via COX-2 synthesis and acts to inhibit platelet aggregation and cause vasodilation. Conversely, COX-1 mediates production of thromboxane A_2 (TXA_2) from platelets and increases platelet aggregation and causes vasoconstriction [115]. The balance between TXA_2 and prostacyclin is important in maintaining a functional coagulation system.

Aspirin is the most effective NSAID at reducing platelet aggregation as it binds permanently to platelets and decreases TXA_2 production for the life of the platelet. Conversely, the COX-2 inhibitor (coxib) class of drugs has been associated with increased risk of thrombosis-related cardiac events in people [116]. In veterinary medicine, ketoprofen, carprofen, tepoxalin, meloxicam, and deracoxib have been studied to assess their effects on primary haemostasias. Ketoprofen and carprofen have been associated with decreased platelet function and deracoxib with potential for increased thrombosis in experimental trials; however, there are no published reports of clinically relevant hemostatic complications in dogs or cats [113, 115, 117, 118].

Drug Interactions with Nonsteroidal Anti-Inflammatory Drugs

Concurrent administration of an NSAID with corticosteroids or other NSAIDs significantly increases the risk of gastrointestinal and kidney injury [97, 107, 119]. Administration of COX-2 inhibitors also delays healing of gastrointestinal ulcers [100, 101]. Concurrent administration of aspirin with other NSAIDs or corticosteroids increases the risk of bleeding and gastrointestinal injury due to both COX inhibition and direct mucosal injury [80, 98, 119].

NSAIDs displace other highly protein-bound drugs from plasma proteins, leading to their enhanced bioavailability and possibly toxic plasma concentrations. Such drugs include other anti-inflammatory agents, warfarin, phenytoin, penicillins, and sulfonamide antibiotics. NSAIDs may also increase plasma levels of digoxin and can decrease the efficacy of furosemide.

Use of NSAIDS with other nephrotoxic drugs (including aminoglycoside antimicrobials) and diuretics (due to risk of hypovolemia) is relatively contraindicated but studies have not been performed to prove a clinical interaction. Drugs that induce the cytochrome P450 metabolic pathway in the liver (e.g. phenobarbital) can increase NSAID metabolism, reducing their analgesic efficacy.

Nonsteroidal Anti-Inflammatory Drugs in the Critical Patient

NSAIDs are contraindicated in patients with hypovolemia or dehydration, gastrointestinal, hepatic, or renal dysfunction, increased sympathetic stimulation (such as in trauma), or cardiac disease (particularly with concurrent administration of furosemide or digoxin). When this population is further expanded to include patients with coagulation disorders, recent history of anti-inflammatory administration, or inappetence, it seems that NSAID administration is appropriate in a fairly small proportion of critically ill patients. However, NSAIDs are highly recommended for use in cardiovascularly stable patients that have a functional gastrointestinal tract and normal kidney function. This subset of patients would include stable postoperative patients and patients with musculoskeletal injuries that are well perfused, have an adequate urine output, and are eating or receiving enteral nutrition. Their excellent analgesic properties make them indispensable in the critical care pharmacopeia.

Alpha-2 Agonists

Alpha-2 agonist drugs stimulate the alpha-2 adrenoreceptors around the body. The most important clinical effects of alpha-2 agonists include profound sedation, centrally mediated analgesia, and hypotension. The clinical effects of alpha-2 agonists can be reversed by administration of yohimbine or atipamezole [120–123].

Analgesic Effects

The analgesic effects of alpha-2 agonists are mediated within both the brain and the spinal cord. Their mechanism of antinociceptive action is complex (Box 48.2). The alpha-2 adrenoreceptors also interact with opiate receptors in a synergistic fashion. The combination of alpha-2 agonists and opioids has an analgesic effect that can last several hours. Unfortunately, the sedative and cardiovascular effects of alpha-2 agonists limit their general use as analgesics, although the sedation induced makes them very effective premedication agents [120–122].

Cardiovascular Effects

Alpha-2 agonists have a two-phase effect on the cardiovascular system. During the first phase, they cause peripheral vasoconstriction and bradycardia. Vasoconstriction is mediated by activation of post-synaptic adrenoreceptors in the peripheral blood vessels. Bradycardia occurs by direct and indirect mechanisms. The direct effect is due to

Box 48.2 Alpha-2 Adrenoreceptors

- Alpha adrenergic receptors are classified into type 1 and type 2.
- The type 2 receptors are further sub-divided into 2A, 2B, and 2C.
- Stimulation of alpha-2A and B receptors in the brain and spinal cord will inhibit pain sensation and wakefulness.
- 2C receptors are more often located on peripheral nociceptors and conversely, stimulation of 2C receptors increases the transmission of painful stimuli.
- 2C receptors are upregulated after neuronal injury and may have a pro-nociceptive effect in inflammatory and neuropathic pain syndromes[124]. Alpha-2 receptors are predominantly stimulated by norepinephrine.
- Alpha-2 receptors are found in the vasculature, liver, pancreas, kidney, platelets, adipose tissue, and the eye. They have distinct physiologic function in each of these organs.
- Receptors are also found in many areas within the central nervous system, including the dorsal horn of the spinal cord, the vagus nerve, and the locus coeruleus.
- The locus coeruleus is a small nucleus within the brainstem that is responsible for controlling wakefulness and is also an important mediator of analgesia.
- Activation of alpha-2 receptors in this area inhibits impulse transmission, leading to sedation and analgesia [120].

stimulation of the vagus nerve, slowing impulse conduction through the atrioventricular node in the heart, thereby reducing the rate of ventricular contraction. Indirectly, bradycardia results from the cardiac response to increased systemic vascular resistance, which is to lower heart rate to prevent hypertension. During phase two, the initial vasoconstrictive response declines and a centrally mediated hypotensive phase predominates, with continuing bradycardia as well as peripheral vasodilation due to decreased sympathetic stimulation [120].

The cardiovascular effects of alpha-2 agonists occur even when the drugs are administered at low doses. In one study of medetomidine in dogs, cardiovascular effects were near maximal at a dose of 5 μg/kg and were not significantly different with doses ranging from 1 to 20 μg/kg [125]. Alpha-2 agonists are therefore not recommended for use in patients with an unstable cardiovascular system.

Medetomidine

Medetomidine is a highly selective alpha-2 agonist that produces profound, dose-dependent sedation and analgesia [37, 121, 122]. The analgesic effects occur 20–60 minutes after a single injection [37]. In combination with an opioid, the analgesic efficacy of medetomidine is enhanced, but there is little improvement in the cardiovascular effects [126]. It is an effective sedative, with doses as low as 1 μg/kg causing a reduction in the amount of induction agent required for anesthesia [127]. Even at low doses, bradycardia and hypotension are common in patients treated with medetomidine [128–130]. It has also been used as a CRI for prolonged sedation (doses from 1-3 μg/kg/hour) and as an adjunctive analgesic during spinal surgery in dogs for its opioid sparing effects [131]. Medetomidine has been administered in combination with opioids via the epidural route, but with minimal improvement in analgesic activity [132].

The most common use of medetomidine is as a sedative or premedication agent. Generally, a dose of 5–20 μg/kg IV or IM is effective in dogs and cats. The low end of the dose range is used if medetomidine is administered in combination with an opioid [133].

Dexmedetomidine

Medetomidine is a racemic mixture of two compounds: dexmedetomidine and levomedetomidine. Most of the analgesic and sedative effects are due to dexmedetomidine and it is thought that levomedetomidine interferes with the function of dexmedetomidine. It has been suggested that dexmedetomidine alone provides more consistent sedation and longer lasting analgesia than medetomidine [120–123]. Dexmedetomidine can also be administered as a CRI for sedation or analgesia [134].

Xylazine

Xylazine is an alpha- 2 agonist medication that is used primarily for sedation in large animal practice. It is no longer recommended for use in small animals due to its propensity to cause vomiting, severe bradycardia, and hypotension.

Adjunctive Analgesia

Adjunctive analgesics are medications that have synergistic activity with, or potentiate the effects of, the primary analgesic (usually opioids or NSAIDs). In many cases they have mild analgesic activity on their own, but this is greatly outweighed by the benefit they have in combination with other medications. As the pain pathway is affected by many different neurotransmitters, mediators, and receptors, it is logical that we need to use analgesics with differing mechanisms of action to effectively treat pain. In addition, a multimodal analgesic approach will allow lower doses of primary analgesics to be used, reducing the potential for adverse effects and complications from these drugs.

Multimodal analgesia in its simplest form would combine opioid and NSAID medications, as these drugs act at different levels of the pain pathway. For patients with acute pain, a combination of opioid with ketamine and/or lidocaine can be used as a CRI (Box 48.3). Oral adjunctive agents can be used for chronic, inflammatory, or neuropathic pain. Examples of these include gabapentin, amantadine, and acetaminophen (paracetamol).

Lidocaine

Lidocaine is a sodium channel blocker that is primarily used as a local anesthetic agent. It is also an effective antiarrhythmic for ventricular tachyarrhythmias as it acts on the fast sodium channels in the myocardial cells. When administered as a systemic analgesic, it can reduce discharge from injured nociceptors in peripheral tissues and thus decrease the transmission of pain sensation to the spinal cord [135]. Intravenous lidocaine has also been hypothesized to interact with opioid receptors [136] as well as having a systemic anti-inflammatory effect by reducing cytokine production [137, 138]. Low doses have been shown to have antagonist effects at NMDA and neurokinin receptors in rats [139].

Lidocaine is effective as an adjunctive treatment for neuropathic pain, cancer pain, and postoperative pain in people [140–142]. It has also been reported as an effective postoperative analgesic in dogs after intraocular surgery and ovariohysterectomy [143, 144]. However, it is most commonly used in combination with an opioid rather than as a sole systemic analgesic agent [67, 145, 146].

Box 48.3 Constant Rate Infusion Recipes

MLK CRI for Syringe Pump Infusion[a]

1) Make sure that each of your components is compatible with the others, then determine your dose rate for each component.
   e.g. morphine 0.1 mg/kg/hour, lidocaine 3 mg/kg/hour, ketamine 0.6 mg/kg/hour
2) Calculate the dose/hour for each component required for your patient, based on its body weight. Using the concentration of each drug, calculate the rate in ml/hour.

Example: MLK CRI for a dog, body weight 20 kg:

Morphine 0.1 × 20 = 2 mg/hour. Using morphine 10 mg/ml, rate is 0.2 ml/hour.
Lidocaine 3 × 20 = 60 mg/hour. Using lidocaine 20 mg/ml, rate is 3 ml/hour.
Ketamine 0.6 × 20 = 12 mg/hour. Using ketamine 100 mg/ml, rate is 0.12 ml/hour.

3) Determine the volume of each component required. A 12-hour infusion of this mixture would require:
   2.4 ml morphine (0.2 ml/hour × 12 hour)
   36 ml lidocaine (3 ml/hour × 12 hour)
   1.5 ml ketamine (0.12 ml/hour × 12 hour)
4) Mix components and determine the rate at which the mixture will be infused. The MLK can be mixed together in a syringe and the rate calculated as the sum of the rates for each of the three components. In this case, the MLK mixture would be administered at 3.3 ml/hour.

MLK CRI for Fluid Pump Infusion[a]

1) Make sure that each of your components is compatible with the others and with the crystalloid fluid you are using for the CRI. In most cases, 5% dextrose is the fluid of choice for drug infusions.
2) Determine the dose of each component:
   Morphine 0.1 mg/kg/hour, lidocaine 2.4 mg/kg/hour, ketamine 0.6 mg/kg/hour
3) Draw 65 ml of fluid from a 500-ml bag of 5% dextrose and discard.
4) Add 1200 mg (60 ml of 20 mg/ml) lidocaine to the bag.
5) Add 50 mg of morphine to the bag
6) Add 300 mg of ketamine to the bag.
   - The resulting mixture contains 0.1 mg/ml morphine, 2.4 mg/ml lidocaine and 0.6 mg/ml ketamine.
   - The rate of administration (ml/hour) of the mixture is equal to the body weight of the patient in kilograms (e.g. for a 20 kg patient, run this mixture at 20 ml/hour).

[a] Other opioids (usually mu agonist drugs) can be used in place of morphine in these recipes. The appropriate CRI doses for each should be used (Table 48.2).
CRI, constant rate infusion; MLK, morphine, lidocaine, ketamine.

Administration of intravenous lidocaine can reduce the duration of postoperative ileus [138, 147, 148]. This effect may be due to the combination of reduction in opioid dose, its anti-inflammatory action, and a decrease in sympathetic tone.

Bolus dosing or high CRI rates of lidocaine (rates > 5 mg/kg/hour) have been associated with drowsiness and lightheadedness in people [140]. Other adverse effects include vomiting or nausea in dogs [141]. It can also cause sedation [149]. Cats are more prone to neurologic adverse effects (including seizures) with use of lidocaine, so lower doses are used in this species (Table 48.4). Transdermal patches are also available for lidocaine administration [150].

Ketamine

Ketamine is an NMDA receptor antagonist. NMDA receptors are found in the dorsal horn of the spinal cord as well as within the brain parenchyma. When the NMDA receptors in the dorsal horn are activated by peripheral nociceptors, they affect the neurons leading to the brain, making them hyperexcitable and increasing the sensation of pain. This process is known as central sensitization or "wind up." NMDA receptor activation is also intrinsic to development of hyperalgesia, neuropathic pain, and opioid tolerance [151, 152].

Ketamine has been shown to be effective as an adjunctive analgesic and to reduce opioid requirement in human

Table 48.4 Doses for adjunctive analgesic agents.

Drug	Dog	Cat
Amantadine	1.25–4 mg/kg every 12–24 hours PO	3 mg/kg every 24 hours PO
Gabapentin	10 mg/kg every 8–12 hours PO	10 mg/kg every 8–12 hours PO
Lidocaine	1.5–3 mg/kg/hour (25–50 mcg/kg/minute) CRI IV	0.75–1.5 mg/kg/hour (12.5–25 mcg/kg/minute) CRI IV
Ketamine	0.6–1.2 mg/kg/hour (10–20 mcg/kg/minute) CRI IV	0.6–1.2 mg/kg/hour (10–20 mcg/kg/minute) CRI IV
Acetaminophen (paracetamol)	10–15 mg/kg every 8–12 hours IV, PO	DO NOT USE

CRI, intravenous constant rate infusion; IV, intravenous; PO, per os (orally).

patients after abdominal and orthopedic surgeries [153–155]. Ketamine has dissociative properties that also make it useful as an anesthetic agent in veterinary medicine, particularly in combination with a sedative agent. The analgesic effects of ketamine become apparent at doses far lower than those required for anesthesia.

Ketamine has also been evaluated for use as a perioperative and postoperative analgesic in dogs and cats and has been shown to be effective in improving comfort after both soft-tissue and orthopedic surgery [156–158]. Ketamine does not slow gastrointestinal motility, so can be useful in patients with postoperative ileus to reduce the opioid requirement [159]. It will also increase catecholamine expression so it can have a mild positive inotropic effect[160]. The primary adverse effects of ketamine infusion are dysphoria and sedation [156, 157]. In some cases, the dysphoria is severe enough to warrant discontinuation of the ketamine infusion.

Amantadine

Amantadine is an oral anti-viral agent that has some antagonist activity at NMDA receptors. It has been evaluated as an effective adjunctive postoperative analgesic in people as well as for neuropathic and refractory osteoarthritis pain in dogs [151, 161, 162].

Gabapentin

Gabapentin was first used as an anti-epileptic medication in human and veterinary patients but has subsequently been shown to be very effective in treatment of chronic and neuropathic pain syndromes [163]. Gabapentin has multiple actions within the central nervous system [164]. Structurally, gabapentin is an analog of GABA, however activation of GABA receptors is not its principal mode of action. Its primary mechanism of action as an analgesic agent is to block voltage-sensitive calcium channels in pain fibers, which are crucial to transmission of pain sensation through the nerves [124, 165, 166].

Gabapentin may also have some NMDA receptor antagonist activity in the spinal cord which can reduce hyperalgesia associated with chronic pain. In addition, it has been shown to act in the locus coeruleus similarly to alpha-2 receptor agonists to cause centrally mediated analgesia [166].

Gabapentin has been used in combination with opiates or NSAIDs for postoperative analgesia in people and has been shown to be an effective analgesic for soft-tissue and spinal surgery [167–169]. The combination of gabapentin and NSAIDs tends to provide better analgesia than NSAIDs alone and use of gabapentin in a balanced analgesic regime can also decrease the postoperative requirement for opioids [167, 168]. The main adverse effect of administration is sedation.

There are few clinical studies assessing the efficacy of gabapentin as an adjunctive analgesic in dogs and cats and results are mixed, generally due to the confounding effect of concurrent opiate administration [170–174]. The dose recommendation in both cats and dogs is 10 mg/kg every 8–12 hours [175, 176]. The anti-epileptic dose is 10–30 mg/kg every eight hours [133].

Acetaminophen

Although acetaminophen (paracetamol) is loosely classified within the NSAID class of drugs, it does not have significant anti-inflammatory effects and its analgesic activity is more effective centrally than peripherally. In dogs, acetaminophen has been shown to have some COX-3 inhibiting effects in the CNS but different mechanisms have been proposed to account for its analgesic and anti-pyretic effects. One such mechanism is as a serotonin antagonist, by inhibiting 5HT-3 receptors in the brain and inhibiting descending serotonin-dependent pain pathways. Similar to other NSAIDs, acetaminophen has an antagonist effect at NMDA receptors [177].

Acetaminophen also inhibits the metabolism of arachidonic acid to COX-2. Acetaminophen partially reduces a free radical iron cation (Fe4+) thereby making it unavailable for use as a co-factor in the metabolism of arachidonic acid. Acetaminophen is more effective in the brain than in other body tissues due to the relatively low amount of COX-2 that is produced in cerebral cells and the low iron stores in the brain. In peripheral tissues, where the arachidonic acid cascade is initiated in a rapid burst of activity and there are far greater stores of iron, the effects of acetaminophen are overwhelmed and are of little consequence to the production of inflammatory mediators [177].

Acetaminophen can be used as an adjunctive analgesic in dogs and can be of particular benefit in cases where NSAIDs are contraindicated. It is also an effective antipyretic agent. The therapeutic dose in dogs is 10–15 mg/kg every 8–12 hours. Toxicity in dogs has been seen at doses greater than 150–200 mg/kg [178]. Acetaminophen is extremely toxic to cats and should not be given under any circumstances. Intravenous and oral preparations are available. Care must be taken when prescribing over-the-counter liquid formulations that they do not contain xylitol.

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49

Local Anesthesia

Christopher L. Norkus

Local anesthetics are among the safest and most effective analgesics for the treatment of acute pain. Because local anesthetics directly block nerve impulses, their use in locoregional techniques decreases pain in a different way than systemic analgesics like opioids or nonsteroidal anti-inflammatory drugs, often with fewer potential risks and systemic adverse effects. When used preemptively, locoregional techniques decrease the likelihood of central sensitization (wind-up) of pain pathways, ultimately helping to prevent hyperalgesia and chronic pain, and aiding in a multimodal approach to patient analgesia. Despite these known benefits, local anesthetics and locoregional techniques are often underused in the emergency department and critical care unit. This chapter focuses on local anesthetics and specific locoregional techniques that have valuable application to this setting.

Pharmacology

Local anesthetics are water soluble salts of lipid soluble alkaloids. They consist of three structural components: a lipophilic aromatic group, an intermediary link, and a hydrophilic amine group (Figure 49.1). Variation to the intermediary link results in the local anesthetic being classified as either an ester or an amide. Amides are the most widely used local anesthetics in veterinary medicine. Agents such as lidocaine, mepivacaine, bupivacaine, ropivacaine, and levobupivacaine are all amide local anesthetics and are emphasized in this chapter, as they are the most widely used. Agents such as prilocaine, tetracaine, procaine, benzocaine, and proparacaine are esters. The speed of onset, potency, and duration of the local anesthetic depends on the pKa, lipid solubility, and protein binding of the agent, respectively.

Local anesthetics work by inhibiting action potential formation by blocking neuronal voltage-gated sodium channels, specifically Na_v 1.2 in a reversible and concentration-dependent fashion [1, 2]. The binding site for local anesthetics is located in domain IV, loop S6 of the sodium channel. By inhibiting action potentials in nociceptive fibers, the local anesthetic effectively blocks the transduction and transmission of pain impulses.

The absorption of a local anesthetic depends on the site of injection, rate of injection, dosage, and effect on vasomotor tone of the agent. Amide local anesthetics are highly protein bound. Tissue distribution following administration is generally proportional to the tissue/blood partition coefficient of the specific local anesthetic, as well as to the mass and degree of perfusion of the tissue.

Ester and amide local anesthetics differ in how they are biotransformed by the body. Esters are hydrolyzed in plasma by pseudocholinesterase to para-aminobenzoic acids, which can result in hypersensitivity reactions. Conversely, amide local anesthetics undergo biotransformation in the liver and therefore can accumulate in the presence of hepatic failure or reduced hepatic blood flow; however, this is rarely a clinical concern. Some metabolites of amides, such as lidocaine's metabolite monoethylglycinexylidide, may have active properties themselves. Amides have a low potential for hypersensitivity reactions; observed adverse reactions are more commonly caused by additives such as methylparaben or vasoconstricting agents (e.g. epinephrine) that were added to the amide to increase duration, rather than by the amide itself. Elimination of amides occurs via renal excretion.

Besides producing sensory blockage to numb a specific location or region of the body, local anesthetics can also cause complete loss of motor function, depending on the properties of the drug, nerve location, myelination of the

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Figure 49.1 The three structural components of all local anesthetics: a lipophilic aromatic group (green), an intermediary link (orange), and a hydrophilic amine group (blue). Changes to the intermediary link result in the local anesthetic being classified as either an ester or an amide. *Source:* Artwork by Natalia Pisano.

nerve, dose, and size of the nerve fibers. Generally, local anesthetics cause nerve blockade in a particular order by first numbing pain, then warmth, touch, deep pressure, and finally, motor function. However, large peripheral nerves are an exception to this pattern and tend to have motor blockade before sensory blockade, as well causing proximal extremity analgesia prior to distal extremity blockade.

Common Local Anesthetics

Lidocaine

Lidocaine is the most commonly used local anesthetic in veterinary medicine. It belongs to the amino amide group and is biotransformed by liver microsomal enzymes, as previously discussed. It is available in concentrations of 0.5–5% with or without epinephrine. In addition to the injectable preparation, various other forms of lidocaine can also be found, including transdermal patches, viscous gels for use on mucous membranes, topical creams to be used on intact tissue, and nasal sprays (Figure 49.2). Lidocaine has an onset of action of approximately 5 minutes and duration of action of approximately 60–120 minutes. However, these numbers can be easily influenced by the agent's proximity to a nerve, additives, and other factors such as tissue pH. Dosing of lidocaine for local infiltration is generally 1–10 mg/kg in dogs and 1–5 mg/kg in cats. Adverse drug reactions are uncommon when lidocaine is used as a local anesthetic. Most reactions are associated with accidental intravenous (IV) injections, so care should be taken to avoid this when local effects are the goal. In dogs, IV administration of lidocaine at a dose of 22 ± 6.7 mg/kg induces convulsions and other signs of central nervous system (CNS) toxicosis, such as salivation and muscle tremors [3]. In cats, IV administration of lidocaine at a dose of 11.7 ± 4.6 mg/kg induces convulsions [4]. Based on these results, dogs should not exceed a dose of 12 mg/kg IV, whereas cats should not exceed a dose of 6 mg/kg IV. Lidocaine with epinephrine should not be used in peripheral extremities (e.g. pinnae) due to vasoconstriction and potential tissue necrosis.



Figure 49.2 Various forms of lidocaine are available. This image shows a 5% transdermal patch, a 2% viscous gel for use on mucous membranes, a 4% cream for use on intact skin, and 2% lidocaine for injection.

Bupivacaine

Bupivacaine hydrochloride is a long-acting local anesthetic that also belongs to the amino amide group and is a racemate [5]. It comes in three different concentrations, 0.25%, 0.5%, and 0.75%, available with or without epinephrine. Bupivacaine has a first onset of action of around 5 minutes, but it may take up to 20 minutes for full blockade of larger nerves. Bupivacaine has a typical duration of action of 240–360 minutes and may last longer in some tissue sites. The cumulative IV dose resulting in CNS toxicosis and convulsive activity in conscious dogs was 4.3 mg/kg [6]. In cats, the mean IV convulsant dose was 3.8 ± 1 mg/kg [4]. Based upon these studies, the dose of bupivacaine should not exceed 1–2 mg/kg for either dogs or cats. Caution must be taken if repeat doses are necessary. Accidental IV injection of bupivacaine is cardiotoxic and may lead to death; therefore, syringe aspiration before injection is crucial. Further discussion on local anesthetic toxicosis is available later in this chapter. Bupivacaine with epinephrine should not be used on peripheral extremities due to its vasoconstrictive properties and the potential for tissue death.

Liposomal Encapsulated Bupivacaine

Liposomal encapsulated bupivacaine is an extended-release formulation of bupivacaine that became available for use in people in 2011 (Exparel®, Pacira Pharmaceuticals, San Francisco, CA) and for use in cats and dogs in 2016 (Nocita®, Elanco, Greenfield, IN). The suspension comprises multivesicular liposomes which, following injection, gradually degrade and slowly release bupivacaine into surrounding

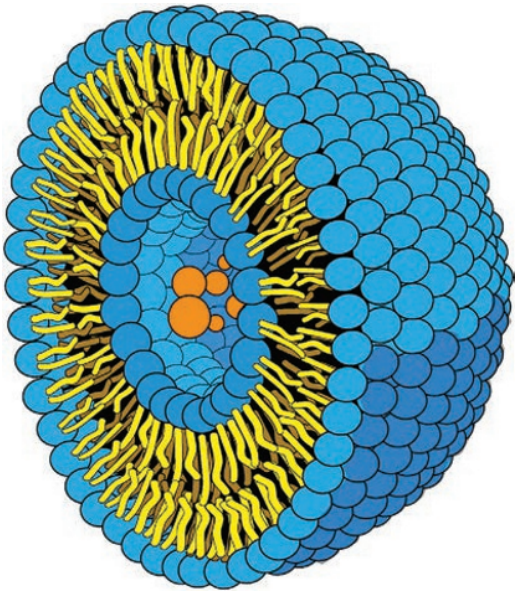


Figure 49.3 A single liposome containing a phospholipid bilayer and an aqueous core that contains bupivacaine molecules that slowly release into tissue. *Source:* Artwork by Natalia Pisano.

tissue, thereby providing local analgesia for up to 72 hours (Figure 49.3). Nocita is an off-white, preservative-free, 13.3 mg/ml solution that comes in 10-ml and 20-ml vials. The suspension should not be shaken. The onset of action in dogs and cats is not known. Because liposomes are large in size (10–30 μm), they move poorly through tissue compared with standard bupivacaine. For this reason, the product should be deposited as close to the affected site as possible. Nocita is approved as a single dose of 5.3 mg/kg into the surgical site following cranial cruciate ligament surgery. Since its release, the product is widely used off-label at lower doses and for other locoregional techniques. Volume expansion can be achieved by diluting the product with an equal volume of sterile saline if needed. An admixture with bupivacaine can be made by mixing bupivacaine and liposomal encapsulated bupivacaine, as long as one ensures the ratio of the milligram dose of bupivacaine hydrochloride to liposomal encapsulated bupivacaine does not exceed one to two (Exparel, Pacira Pharmaceuticals, Inc., Tampa, FL). The author commonly uses 0.5 mg/kg bupivacaine hydrochloride mixed with 1 mg/kg liposomal encapsulated bupivacaine for nerve blocks. Recent literature suggests that Nocita may be used as a multidose vial for up to four days as long as aseptic technique, including wearing gloves and alcohol swabbing the bottle, is used [7]. The product is always stored refrigerated. Liposomal encapsulated bupivacaine should not be used IV and is generally avoided epidurally as well. Because of its exceptionally long duration of action, Nocita has largely replaced the need for and use of soaker wound infusion catheters.

Ropivacaine

Ropivacaine hydrochloride is a long-acting local anesthetic that also belongs to the amino amide group and is a pure S(−) enantiomer, unlike bupivacaine. It comes in 0.2%, 0.5%, 0.75%, and 1% concentrations. Ropivacaine has a full onset of action of approximately 20 minutes and duration of action of 180–360 minutes. Dosing of ropivacaine is similar to bupivacaine at 1–2 mg/kg for both dogs and cats. Ropivacaine has been found to have less cardiotoxicity than bupivacaine in animal models; however, the IV route is generally still avoided.

Levobupivacaine

Levobupivacaine is the S(−) enantiomer of the drug bupivacaine hydrochloride. It was developed for use in humans, as enantioselectivity has been shown to reduce cardiotoxicity and CNS toxicity. Levobupivacaine has properties and dosages essentially identical to those of bupivacaine. Currently, the main factor limiting its use in veterinary medicine is increased cost compared to bupivacaine.

Mepivacaine

Mepivacaine is a medium duration amide local anesthetic. It has a rapid onset of action of approximately 5 minutes and a duration of 120–180 minutes. It is used at 1–6 mg/kg in the dog and 1–3 mg/kg in the cat. Cardiotoxicity and neurotoxicity can result. The IV route is contraindicated.

Local Anesthetic Adjuvants

Dexmedetomidine

The alpha-2 adrenergic agonist dexmedetomidine can be a useful adjunct when combined with amide local anesthetics [8, 9]. The addition of dexmedetomidine may result in synergistic analgesic effects due to norepinephrine inhibition at the nerve endings, as well as enhanced peripheral nerve blockade for a duration of up to 24 hours. Doses of 0.001–0.005 mg/kg are commonly selected. Systemic effects following dexmedetomidine use may include nausea, vasoconstriction, bradycardia, and decreases in cardiac output.

Opioids

Systemic opioids can be combined with amide local anesthetic agents to prolong the duration of local blockade [9, 10]. The most widely used opioid for this purpose in veterinary medicine is buprenorphine, which may extend the duration of amide local anesthetics beyond 24 hours. A dose of 0.003 mg/kg combined with a local anesthetic

is commonly used. Other opioid adjuvants appear to be less successful at extending analgesic duration of local anesthetics.

Selected Locoregional Techniques

Many locoregional techniques can be quickly, safely, and effectively performed in dogs and cats. For the purpose of this chapter, however, the author has selected a handful of techniques that he most frequently uses in the emergency and critical care setting. Many other techniques such as femoral and sciatic nerve blocks, transversus abdominis plane blocks, brachial plexus nerve blocks, paravertebral nerve blocks, and others can be useful but are beyond the scope of this chapter and are described elsewhere. Additionally, such techniques are often best achieved using a peripheral nerve locator or under ultrasound guidance, as well as under the mentorship of a board-certified veterinary anesthesiologist. A list of supplies needed for most local anesthetic blocks is available in Box 49.1.

Local Infiltration and Line Blocks

Local anesthetic infiltration, including incisional block, is simple and quick to perform (Protocol 49.1). These techniques involve the infusion of a local anesthetic in and around the site of a wound or surgical incision. Such blocks may reduce the need for other systemic drugs such as deeper sedation or general anesthesia. The most common indications for these blocks include lacerations, wounds, incisions, toe injuries and amputations, tail injuries and amputations, and surgical sites.

A strict contraindication for these blocks is a previously documented hypersensitivity reaction to a local anesthetic. Local anesthetics do not appear to hinder wound healing in dogs and cats, nor do they hinder the ability for a pathologist to perform histopathology on excised tissue [11]. Local infection may alter tissue pH and may inhibit the effectiveness of local anesthetics. Lidocaine is acidic and

Box 49.1 Supplies Needed for Most Local and Regional Anesthetic Blocks

- Clean examination gloves or sterile gloves, depending on procedure and hospital protocol
- Fur clippers
- Sterile 4 × 4-inch gauze
- Aseptic preparation (e.g. chlorhexidine scrub, 70% isopropyl alcohol)
- Sterile syringes (e.g. 3-, 6-, 12-ml sizes)
- Sterile needles of various gauges, including epidural needles if indicated
- Local anesthetic agent of choice (e.g. 2% lidocaine, 0.5% bupivacaine), dosed appropriately to body weight and diluted if so recommended for procedure
- Sterile 0.9% NaCl

Protocol 49.1 Local Infiltration and Line Blocks

Items Required

- See Box 49.1 for supplies list.

Procedure

1) Clip the affected area.
2) Aseptically prepare the area.
3) Wash hands.
4) Don clean examination gloves or sterile gloves depending on nature of procedure.
5) Select a single local anesthetic agent such as 2% lidocaine or 0.5% bupivacaine. The author commonly selects up to 5 mg/kg of lidocaine in the cat, 8 mg/kg of lidocaine in the dog, or 2 mg/kg of bupivacaine. When using lidocaine, consider making an admixture with 8.4% sodium bicarbonate to reduce pain on injection. Mix 8.4% sodium bicarbonate with 2% lidocaine in a volume ratio of 1 part to 9 parts (for a total of 10 parts).
6) With a syringe attached to either a 25-gauge, ⅝-inch needle, or a 22-gauge, 1-inch needle, insert the needle subcutaneously, aspirate to confirm the needle is not inside a blood vessel, and inject the local anesthetic while withdrawing the needle, thereby making a small fluid bleb.
7) Repeat this process around the target area in a rectangular or circular pattern. Make sure to divide the volume of drug equally throughout the area. It is best to anticipate how large the blocked area will be to avoid exceeding the maximum mg/kg dose or running out of drug before blocking the entire target.
   If your target area is a surgical incision, inject the local anesthetic along the anticipated incision, using either a 25-gauge, ⅝-inch, or a 22-gauge, 1-inch needle (size depends on the size of the animal). Alternatively, you can use a 22-gauge, 1.5–3.0-inch spinal needle (length depends on size of incision).
8) It is ideal to make these injections before any surgical procedure has begun; however, these local anesthetics are still beneficial if done after incision.

for this reason can be painful upon administration. To reduce this effect, 8.4% sodium bicarbonate can be mixed with 1% or 2% lidocaine in a volume ratio of 1 part to 9 parts (for a total of 10 parts), respectively [12, 13]. This admixture will turn cloudy and neutralize the pH to 7.4 [14]. Infection at the site of local anesthetic administration, adverse drug reactions, bleeding, and incomplete or ineffective block are possible complications. Importantly, different local anesthetics (e.g. lidocaine and bupivacaine) should not be mixed together, as this practice has consistently been shown not to produce a faster onset and actually reduces the duration of the block [15–18].

Intrapleural Local Anesthesia

Intrapleural local anesthesia administration is commonly performed through a chest tube that has already been placed (Protocol 49.2), but it can also be performed by direct injection into the pleural space. This technique is believed to enable the spread of the local anesthetic to multiple intercostal nerves via a single injection. Intrapleural local anesthesia may be used to treat acute postoperative pain that results from thoracotomy as well as pain resulting from cranial abdominal surgery or disease (e.g. liver lobectomy, pancreatitis). Multiple studies in people have found intrapleural local analgesia to be effective in addressing pain that results from cholecystectomy [19, 20]. A flail chest segment may cause local anesthetic to leak out of the pleural space and into the subcutaneous tissue, resulting in less effective intrapleural coverage. Complications in people from this technique appear low but may include transient discomfort, pneumothorax, bleeding or hemothorax, local infection, pleural effusion, and, rarely, Horner syndrome [5, 21]. The use of intrapleural lidocaine and bupivacaine has not been shown to cause hemodynamic changes or arrhythmia in dogs that have undergone pericardiectomy [22]. As the procedure may cause transient discomfort, the author prefers to perform this technique on patients that are sedated or anesthetized when possible.

Protocol 49.2 Intrapleural Local Anesthesia

Items Required

- See Box 49.1 for supplies list.

Procedure

1) Wash hands and don clean examination or sterile gloves.
2) Aseptically prepare the chest tube for injection.
3) Aspirate the chest tube and remove any fluid or air from the pleural space.
4) Slowly inject a long-acting local anesthetic such as bupivacaine or ropivacaine into the pleural space. A new, previously unopened vial of local anesthetic is recommended to ensure sterility. The total dosage should not exceed 2 mg/kg. If bilateral chest tubes are placed, divide the drug in half and administer one half of the drug into each chest tube.
5) Follow the injection with several milliliters of sterile saline or air to clear the chest tube of the local anesthetic and ensure dispersion into the thoracic cavity.
6) If a single lateral thoracotomy has been performed, the local anesthetic may be more effective if you position the patient in lateral recumbency for 10–15 minutes with the affected side down.

Intercostal Nerve Blocks

Intercostal nerve blocks (Protocol 49.3) can be performed at any intercostal nerve. This technique is most frequently performed to provide analgesia for broken ribs and flail chest segments to improve ventilation, and can also be performed for thoracotomy and cranial abdominal pain. A strict contraindication for these blocks is a previously documented hypersensitivity reaction to a local anesthetic. Intercostal

Protocol 49.3 Intercostal Nerve Blocks

Items Required

- See Box 49.1 for supplies list.

Procedure

1) Select an intercostal nerve to anesthetize.
2) Clip and aseptically prepare two adjacent intercostal spaces cranial and two adjacent intercostal spaces caudal to selected site.
3) Wash hands and don clean examination gloves.
4) Palpate the caudal border of each rib head to locate the approximate site of each intercostal nerve, artery, and vein.
5) Using a 90-degree angle, insert a 25-gauge, ⅝-inch, or a 22-gauge, 1-inch needle through the skin caudal and perpendicular to the rib head, as close to the dorsal aspect of the intervertebral foramen as possible.
6) Aspirate the syringe to confirm accidental arterial or venous puncture has not occurred. If no blood is observed, slowly inject the selected local anesthetic at each site. Lidocaine up to 5 mg/kg total in the cat and 8 mg/kg total in the dog or bupivacaine at 2 mg/kg are most commonly selected. Ensure that the volume of this total dosage is divided evenly between all sites.

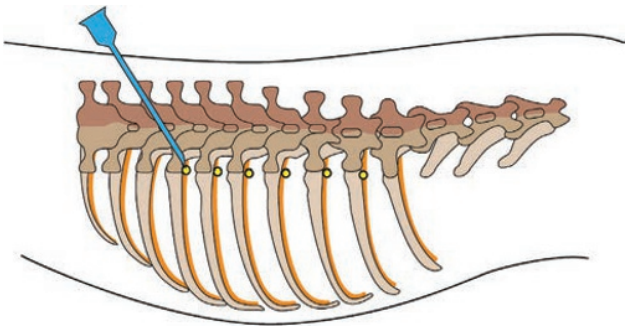


Figure 49.4 The approximate landmark at which to perform intercostal nerve blocks. The orange line signifies the intercostal vein, artery, and nerve. Note that the needle is placed along the caudal border of each rib and as dorsal on the rib as possible. *Source:* Artwork by Natalia Pisano.

nerve blocks should be avoided in patients with skin disease over the site of injection, coagulopathy, severe thrombocytopenia, or advanced thrombocytopathy. When performed correctly, complications from this technique are rare but may include transient discomfort, pneumothorax, hematoma, bleeding or hemothorax, and local infection (Figure 49.4).

Intraperitoneal Local Anesthesia

Intraperitoneal local anesthesia (Protocol 49.4) is an underused but safe and effective modality to provide analgesia in patients undergoing laparotomy or in patients with abdominal pain secondary to disease (e.g. pancreatitis) [23–27]. The practice is widely used in people and is believed to work via blockade of free afferent nerve endings in the peritoneum as well as via possible anti-inflammatory effects. It is most commonly performed by depositing local anesthetic into the abdomen during laparotomy with direct visualization prior to abdominal closure, but it can also be performed by blind injection or via ultrasound guidance through the intact abdominal wall.

Protocol 49.4 Intraperitoneal Block

Items Required

- See Box 49.1 for supplies list.

Procedure

1) Perform laparotomy, including abdominal lavage as needed.
2) Using a previously unopened vial of local anesthetic, aseptically withdraw 3–5 mg/kg of bupivacaine or ropivacaine and deposit the volume throughout the open abdominal cavity.
3) Close the abdomen as planned.

A strict contraindication for this technique is a previously documented hypersensitivity reaction to local anesthetic. If performing through a closed abdomen, intraperitoneal local anesthesia should be avoided in patients with skin disease over the site of injection, coagulopathy, severe thrombocytopenia, or advanced thrombocytopathy. The presence of abdominal effusion may render intraperitoneal local anesthesia less effective. In a closed abdomen, complications following intraperitoneal local anesthesia are rare and are similar to that of abdominocentesis; these complications may include transient discomfort, hematoma, abdominal bleeding, local infection, and entrance of local anesthetic into viscera (intestine or bladder). The author greatly prefers to perform this technique under direct visualization during laparotomy prior to closure and does so at the conclusion of most abdominal surgeries. Complications with this approach are rare but could include increased systemic toxicosis and infection. To mitigate these risks, a new, previously unopened vial of local anesthetic is recommended to ensure sterility is maintained.

Radial, Ulnar, Median, and Musculocutaneous Nerve Block

In recent years, the radial, ulnar, median, and musculocutaneous nerve block (RUMM) has widely replaced the use of the brachial plexus nerve block for providing local anesthesia in the forelimb below the elbow (Protocol 49.5) [28, 29]. This is due to the relative ease of administration and wider safety margin compared with the brachial plexus nerve block.

The RUMM block requires two separate approaches to complete (Figures 49.5, 49.6). The radial nerve is approached via the lateral aspect of the forelimb, and the ulnar, median, and musculocutaneous portions of the block are approached from the medial aspect. The radial nerve runs between the long head of the triceps and the brachial muscle on the caudolateral aspect of the humerus, along approximately one third of the bone. It feels much like a spaghetti noodle or a guitar string. The median and ulnar nerves run caudal to the brachial artery on the medial aspect of the distal humerus. The musculocutaneous nerve runs immediately cranial to the brachial artery on the medial aspect of the distal humerus.

The RUMM block is indicated for surgery or injury of the distal forelimb (e.g. the antebrachium, carpus, or paw). The block can be performed blindly by digital palpation, using a peripheral nerve finder, or via ultrasound guidance. A strict contraindication for this technique is a previously documented hypersensitivity reaction to local anesthetic. Other rare complications include accidental systemic drug administration following venous or arterial injection, hematoma, bleeding, or local infection.

Protocol 49.5 Radial, Ulnar, Median, and Musculocutaneous Nerve Block

Items Required

- See Box 49.1 for supplies list.

Procedure

1) Clip and aseptically prepare the medial and lateral aspects of the humerus over the anticipated injection sites.
2) Wash hands and don clean examination gloves.
3) Position the patient in lateral recumbency with the affected limb up. Palpate the radial nerve on the distal third of the humerus by pushing the brachialis muscle cranially so that your finger rests on the humerus between the brachialis muscle and the triceps. The radial nerve can be felt crossing the humerus at this location and feels like a piece of spaghetti or a guitar string.
4) Insert a 25-gauge, ⅝-inch, or a 22-gauge, 1-inch needle through the skin from a caudal direction perpendicular to the length of the humerus at the level of the nerve. Advance the needle until there is contact on the humerus near the nerve.
5) Aspirate. As long as no blood is aspirated, slowly inject a long-acting local anesthetic such as 0.5–1 mg/kg of bupivacaine or ropivacaine at the site. Dexmedetomidine or buprenorphine can be added to this block to extend the duration of its effects. Alternatively, Nocita with or without bupivacaine can also be used.
6) Position the patient in lateral recumbency with the affected limb down. Palpate the pulse of the brachial artery between the biceps brachialis and the medial head of the triceps, approximately one third of the way up the humerus.
7) Insert a 25-gauge, ⅝-inch, or a 22-gauge, 1-inch needle through the skin adjacent to the pulse. Advance the needle until there is contact on the humerus near the nerve.
8) Aspirate back on the syringe to check for blood. If no blood returns, slowly inject a long-acting local anesthetic such as 0.5–1 mg/kg bupivacaine or ropivacaine at the site. Dexmedetomidine or buprenorphine can be added to this block to extend the duration of its effects. Alternatively, Nocita with or without bupivacaine can also be used.



Figure 49.5 The radial, ulnar, median, and musculocutaneous nerve block requires two approaches to complete the technique. The lateral approach to the left thoracic limb, shown here, achieves blockade of the radial nerve. The dog is drawn in right lateral recumbency with its head to the left and tail to the right of the image. *Source:* Artwork by Natalia Pisano.

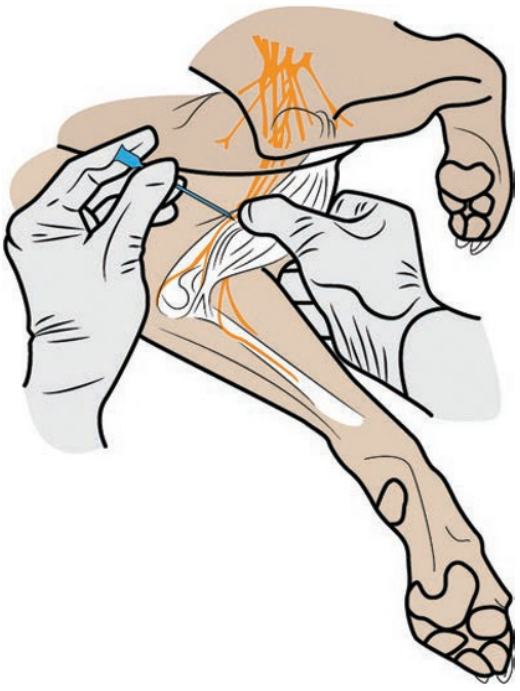


Figure 49.6 The radial, ulnar, median, and musculocutaneous nerve block requires two approaches to complete the technique. The medial approach to the left thoracic limb, shown here, achieves blockade of the ulnar, median, and musculocutaneous nerves. The dog is drawn in left lateral recumbency with its head to the right of the image and its tail to the left. *Source:* Artwork by Natalia Pisano.

Protocol 49.6 Sacrococcygeal Epidural

Items Required

- See Box 49.1 for supplies list.

Procedure

1) Sedate or anesthetize the patient.
2) Clip and aseptically prepare the sacrococcygeal area. This is easily found by palpating the space between the sacrum and the first coccygeal vertebra, which can be identified by moving the patient's tail to isolate the joint.
3) Wash hands and don clean examination gloves. Palpate the sacrococcygeal space with the index finger of the nondominant hand. Once you have located it, use your dominant hand to insert a 25-gauge, ⅝-inch, or a 22-gauge, 1-inch needle through the skin along the midline into the sacrococcygeal space. A 30–45 degree angle through the skin is often best. If blood is noted in the needle hub, the needle should be completely removed and the technique restarted. Blood suggests entrance into the ventral vertebral venous plexus. If bone is encountered, the needle may need to be partially withdrawn, redirected slightly steeper or flatter, and reinserted to enter the sacrococcygeal space.
4) Inject 0.1 ml/kg of either 2% lidocaine or 0.5% bupivacaine or ropivacaine into the sacrococcygeal space. Note that there should be no resistance to injection, and if resistance is present, the subcutaneous space or musculature may have been accidently entered.
5) If effective blockade has occurred, tail, anus, and preputial relaxation may be noted 5–10 minutes following injection.

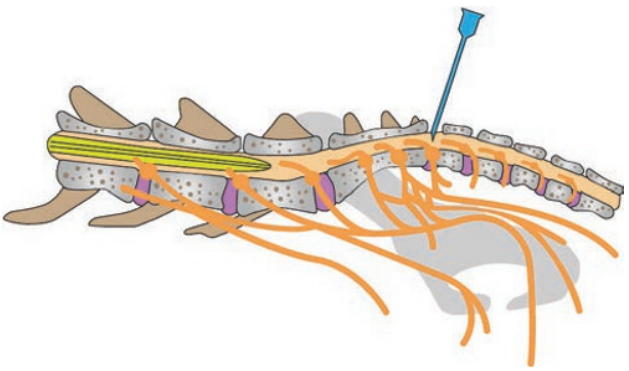


Figure 49.7 Sacrococcygeal epidurals using 0.1 ml/kg of either 2% lidocaine or 0.5% bupivacaine are commonly administered into the sacrococcygeal space for cats with urethral obstruction. The animal is drawn in right lateral recumbency with the head to the left of the image and the tail to the right. *Source:* Artwork by Natalia Pisano.

Sacrococcygeal Epidurals

Sacrococcygeal epidurals, also referred to as coccygeal nerve blocks, are safe, fast, improve analgesia, and reduce the need for systemic drugs during surgery or urethral catheterization (Protocol 49.6) [30, 31]. They can be used for a wide variety of procedures in dogs and cats including catheterization for relief of urethral obstruction, tail amputations, perineal urethrotomy, anal sac disease including sacculectomy, and other surgeries of the penis or perineal region (Figure 49.7). Contraindications for the procedure include skin infection over the sacrococcygeal space, coagulopathy, severe thrombocytopenia, and thrombocytopathy. The procedure is most safety performed in a sedated patient. Complications from the technique are uncommon but could include local infection, epidural abscess, epidural bleeding or hematoma, and injury to the cauda equina. To mitigate infection, a new, previously unopened vial of local anesthetic is recommended to ensure sterility is maintained. Because a smaller volume of local anesthetic is used and thus there is less cranial drug migration, many of the possible adverse effects of lumbosacral epidurals such as motor weakness, sympathetic block, and intrathecal injection are avoided. Most commonly, local anesthetics such as lidocaine, bupivacaine, or ropivacaine are used alone in this technique; however, opioids (e.g. morphine, buprenorphine) or other adjuncts (e.g. dexmedetomidine, ketamine) could also be included. Agents with or without preservatives can safely be used.

Lumbosacral Epidurals

Lumbosacral epidurals (Protocol 49.7) are performed in heavily sedated or anesthetized patients at the L7 and S1 vertebral space and commonly use a combination of a local anesthetic and an opioid (Figure 49.8). Once injected into the epidural space, these agents gradually diffuse across the dura into the subarachnoid (intrathecal) space. Here, local anesthetics provide sensory blockade by acting primarily on the spinal nerve roots. Conversely, opioids provide segmental analgesia by reducing neurotransmitter release (e.g. substance P, neurokinin A, glutamate) at the presynaptic level and by hyperpolarizing the membrane of the dorsal horn neurons at the post synaptic level. Injection directly into the intrathecal space is widely done in people, but because of the anatomic differences between species, this technique is far more challenging in dogs and cats, and thus epidural injections are more commonly performed. Catheterization of the epidural space can also be performed, allowing for bolus or constant rate infusions of medication to be delivered into the epidural space (Protocol 49.8).

Protocol 49.7 Lumbosacral Epidural

Items Required

- See Box 49.1 for supplies list.

Procedure

1) The patient should be heavily sedated or anesthetized.
2) Place the patient in either sternal or lateral recumbency, depending on personal preference. The patient's pelvic limbs can be positioned cranially to increase the size of the epidural space.
3) Clip a small area of fur over the lumbosacral junction and aseptically prepare the site.
4) Wash your hands and don sterile gloves.
5) With your nondominant hand, palpate the wings of the ilium with your thumb and middle finger. Using your index finger, palpate the spinous process of the seventh lumbar vertebra.
6) Slide the index finger caudally down the spinous process until the lumbosacral space is palpable. A slight divot can usually be palpated here, between L7 and S1.
7) Keeping the index finger in place to maintain positioning, insert a 20- or 22-gauge, 1.5- to 3.0-inch Quincke-type spinal needle (length and size of needle depend on patient size) perpendicular to the skin, ensuring the needle is precisely on midline. In cats and small dogs, a 25-gauge, 0.75-inch Quincke-type spinal needle can be used if available.
8) Continue to advance the spinal needle slowly, adjusting the needle angle as needed, cranially or caudally, until the needle advances smoothly and is believed to be in the epidural space (see point 9, below). The diameter of the lumbosacral epidural space is 2–4 mm in medium sized dogs and less than 3 mm in cats.
9) The epidural space sits just ventral to the ligamentum flavum. As the needle is advanced, it must pass through the skin, subcutaneous fat, supraspinous ligament, interspinous ligament, and, lastly, the ligamentum flavum. A "pop" or distinct change in resistance is often felt upon puncturing through the ligamentum flavum.
   a) Two techniques are commonly used to ensure correct epidural placement of the needle tip. The first method is the *loss of resistance* technique. Remove the stylet of the epidural needle. Fill a plastic or glass (if available) syringe with 0.5–2 ml sterile saline and a small volume of air. Invert the syringe so the small volume of air rises above the saline. Attach the syringe to the epidural needle and slowly inject some of the saline into the epidural space. There should be no resistance to the injection and no compression of the air within the syringe. Resistance or back pressure on the plunger during injection suggests that your attempt to enter the epidural space has failed. Repeat the preceding steps until no resistance or back pressure is encountered. This is the author's preferred method for epidural administration.
   b) The second technique that can be used to confirm correct epidural placement is the *hanging drop* technique. This technique must be performed with the patient in sternal recumbency. Once the needle is placed through the skin, the stylet is removed, and the hub of the epidural needle is filled with saline or local anesthetic. The needle is then slowly advanced. Once the needle enters the epidural space, the fluid within the hub of the needle drops, confirming that is has fallen into the epidural space, indicating epidural placement. Unfortunately, because the epidural space of dogs and cats is not as negatively pressured as in other species (e.g. cows), a lack of response commonly occurs, resulting in a false negative test.
10) Once epidural needle placement is verified, examine the needle for blood or cerebrospinal fluid. If blood is encountered, the needle has inadvertently entered the ventral venous plexus. No drug administration is made, and the needle is removed. The procedure is then restarted. If cerebrospinal fluid is encountered, the intrathecal space has been encountered, and the volume (dose) to be injected is reduced by 50%, regardless of drug(s) used.
11) Slowly inject lidocaine 2–5 mg/kg, bupivacaine 0.25–1.5 mg/kg, or ropivacaine 0.25–1.5 mg/kg with or without morphine 0.05–0.15 mg/kg. Within the ranges, higher doses of local anesthetic will result in more cranial drug migration and more cranial effect. For most abdominal, pelvic, or pelvic limb procedures, bupivacaine 0.5 mg/kg and morphine 0.1 mg/kg is a commonly selected combination.

Epidural drug administration is an excellent modality to provide analgesia in the emergency and critical care setting. The technique can greatly reduce systemic effects such as sedation, nausea, vomiting, and ileus that might be seen with repeated larger doses of parenteral opioids since a small dose of opioid can be targeted directly at the dorsal horn of the spinal cord. Additionally, epidural drug administration reduces inhalant anesthesia requirements, often has a long

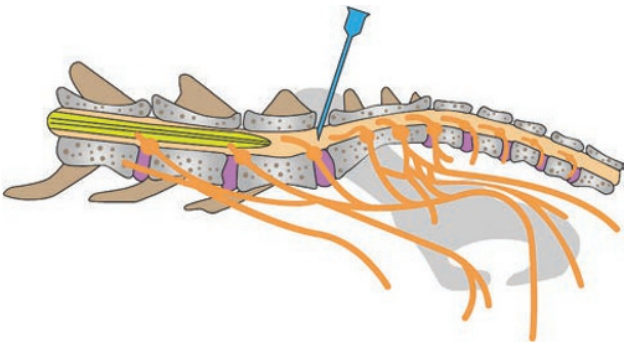


Figure 49.8 Epidural drug administration or a locoregional anesthesia technique, such as a femoral sciatic nerve block, is widely considered standard of care for invasive hindlimb orthopedic surgical procedures such as fracture repair, stifle surgery (e.g. tibial plateau leveling osteotomy), or pelvic limb amputation. The animal is drawn in right lateral recumbency with the head to the left of the image and the tail to the right. *Source:* Artwork by Natalia Pisano.

duration of action, is cost effective, and has few adverse effects. Epidural drug administration or a locoregional anesthesia technique, such as a femoral sciatic nerve block, is widely considered standard of care for invasive hindlimb orthopedic surgical procedures such as fracture repair, stifle surgery (e.g. tibial plateau leveling osteotomy), or pelvic limb amputation. Beyond hind limb orthopedic disease, epidurals are beneficial for patients with soft tissue trauma to the pelvic limb, orthopedic pelvic disease, and those with abdominal pain. If opioids are used as part of the epidural, central analgesia is produced via the dorsal horn, which also provides analgesia for those with thoracic or thoracic limb pain.

Commonly used local anesthetics for epidural administration include bupivacaine, ropivacaine, and lidocaine. Given epidurally, these agents typically have a similar onset of action to what they would have if they were administered elsewhere. Liposomal encapsulated bupivacaine is not recommended for use in the epidural space. Morphine is the mostly widely used opioid for epidural administration because it is comparatively the most hydrophobic (least lipophilic) of available opioids, thereby resulting in the longest duration of action. The onset time for morphine in the epidural space is approximately 60 minutes and it may provide analgesia for 16–24 hours. Other opioids such as buprenorphine or fentanyl can be used epidurally but with much shorter duration of action. Occasionally, other agents such as dexmedetomidine, ketamine, or corticosteroids may also be administered in the epidural space. Either

Protocol 49.8 Epidural Catheter Placement

Items Required

- Supplies listed in Box 49.1
- Commercial epidural catheter kit
- Supplies for aseptic procedure, including sterile gloves
- Light sterile dressing, such as iodophor-impregnated adhesive dressing

Procedure

1) Follow steps 1 through 8 of Protocol 49.7 for Lumbosacral Epidural.
2) The procedure for entering the epidural space is nearly identical to epidural injection, but instead of using a Quinkie-type epidural needle, a Tuohy-type epidural needle is selected. The Tuohy-type epidural needle has a curved tip, which allows a catheter to be passed through the needle and its tip directed cranially within the epidural space.
3) Once entrance into the epidural space has been confirmed, the epidural catheter is advanced through the Tuohy needle.
4) The tip of the epidural catheter should be placed just caudal to the painful area. Therefore, premeasuring the catheter prior to placement is important.
5) Remove the Tuohy needle while leaving the catheter in place. The catheter should NEVER be withdrawn through the needle; doing so could potentially lacerate the catheter, resulting in it breaking off and embolizing in the epidural space.
6) The procedure is best done under fluoroscopy. However, because epidural catheters are designed to be radiopaque, a radiograph can also be performed to verify correct placement of the epidural catheter.
7) Once the catheter is confirmed to be in the correct location, a locking mechanism is attached, together with a filter and injection port.
8) It is important to keep the injection port of the catheter sterile at all times. The insertion site should be covered with an iodophor-impregnated adhesive drape (e.g. Ioban®, 3M Healthcare, Maplewood, MN) to provide antimicrobial activity over the site. Aseptic technique should be used when delivering drugs through the injection port. The insertion site should be inspected daily for signs of inflammation or infection.
9) If an epidural catheter stops functioning, placement should be checked by fluoroscopy or radiographs. If the catheter is correctly in place, the catheter can be flushed with a small volume of sterile saline and, if necessary, slightly withdrawn until patency is reestablished. The epidural catheter should not be readvanced once any aspect of it has been withdrawn.

preservative-free or preservative-containing medications can be used for epidural injection. Only preservative-free medications should be utilized for intrathecal injection.

Strict contraindications for epidural drug administration include pyoderma over the injection site, the use of anticoagulants (e.g. low molecular weight heparins, antiplatelet drugs, anti-Factor Xa medications), coagulopathy, thrombocytopenia, or thrombocytopathy, and if local anesthetics are to be used, uncorrected hypovolemia or hypotension. Pelvic fractures may make anatomic landmarks difficult to palpate and the epidural more technically challenging to do, but they are not a strict contraindication for the procedure.

Adverse effects of epidurals may include nausea, motor weakness, delayed hair regrowth at the site of the epidural, and urinary retention if opioids are used [32–37]. Complications of epidurals may include epidural abscess, epidural hematoma, pruritus, transient Horner syndrome, hyperesthesia, myoclonus, and if local anesthetics were used in unstable patients, sympathetic blockade leading to profound vasodilation and hypotension [32–37]. Sympathetic blockade can also be seen if epidural catheters or large volumes of local anesthetic are used, thereby resulting in more cranial migration of the local anesthetic. Sympathetic blockade is addressed by crystalloid volume replacement and the use of vasoconstrictive agents such as phenylephrine or norepinephrine.

Local Anesthetic Toxicosis

Regional toxicosis from local anesthetics is uncommon. Systemic toxicosis, although also uncommon, can be life threatening. Systemic amide toxicosis can include CNS and cardiac toxicosis including tremoring, seizures, coma, arrhythmia, hypotension, and death. Bupivacaine has a greater risk for cardiac toxicosis than other drugs as it binds to cardiac sodium channels (NaV 1.5) with higher affinity and dissociates more slowly than other agents. This causes bupivacaine to accumulate during diastole, can slow cardiac conduction, and can induce reentry arrhythmias. Conversely, drugs like ropivacaine and levobupivacaine have lower risk for cardiac toxicosis as they have lower affinity for these channels.

The first step to avoiding local anesthetic toxicosis is appropriate drug dosing. Dosing of lidocaine for local infiltration is generally 1–10 mg/kg in dogs and 1–5 mg/kg in cats. Dosing of bupivacaine, ropivacaine, and levobupivacaine is lower than lidocaine at 1–2 mg/kg for both dogs and cats. Clinicians should also be mindful of drug duration and avoid premature repeated injections. When administering local anesthetics, care should be taken to aspirate prior to injection, to avoid intravascular anesthetic administration.

If early neurological toxicosis is present, supportive care including IV fluid therapy, anti-convulsant therapy, and discontinuation of the local anesthetic may be adequate. In more severe cases of toxicosis, including cardiac involvement, IV lipid emulsion should be used [38]. Although evidence-based dosing regimens are lacking, this author uses intravenous lipid emulsion at 1.5 ml/kg IV over 2–3 minutes followed by a constant rate infusion of 0.25 ml/kg/minute for 30–60 minutes.

If cardiac arrest occurs, RECOVER-based cardiopulmonary resuscitation should be started immediately, with several small adjustments. The use of IV regular insulin at 1–2 units/kg IV followed immediately by 50% dextrose at 1 g/kg IV diluted one to one with saline appears to be an effective therapy during resuscitation [39–41]. The use of epinephrine during resuscitation of local anesthetic toxicosis in dogs has been associated with worsening cardiac arrhythmia and therefore small doses are preferred (e.g. <0.001 mg/kg) [42]. Vasopressin should be avoided, as pulmonary hemorrhage has been reported in people. If ventricular arrhythmias occur during stabilization, amiodarone is the preferred antiarrhythmic in people, as lidocaine and procainamide can exacerbate the existing toxicosis. Electrolytes and blood glucose should be serially evaluated following the use of IV insulin.

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50

Monitoring the Anesthetized Patient

Benjamin M. Brainard and Jessica Perlini

Monitoring anesthesia of the critically ill patient is an important aspect of veterinary critical care. Each monitoring data point adds to a larger picture, which reflects the condition of a patient under general anesthesia. Although single values may be useful, trends of measured physical parameters are more commonly used to guide decisions for anesthetic adjustments. Critically ill patients are either at risk of, or already experiencing, compromise of the body's ability to maintain homeostasis and tissue oxygen delivery. Many critically ill animals cannot tolerate high concentrations of single anesthetic drugs (such as inhalant anesthetics), requiring multiple intravenous (IV) drugs to supplement or maintain anesthesia in a more balanced technique. The anesthetist must continually monitor the patient to ensure that anesthesia and analgesia are adequate for the duration of the procedure.

The most common perturbations expected in the anesthetized patient are hypoxemia, hypotension, hypoventilation, cardiac arrhythmias, acidosis, and volume overload.

Hypoxemia may be present prior to anesthesia in some cases and may develop during anesthesia in others. Common intraoperative events that can result in hypoxemia may be patient related (e.g. pneumothorax, pulmonary embolism, pulmonary edema) or may be machine failure (i.e. inadequate supply of oxygen, malfunctioning flow meter, or a circuit disconnect). Systemic oxygenation can be monitored with arterial blood gas analysis and estimated with pulse oximetry. Normal oxygen saturation (SpO_2) in an animal breathing 100% oxygen should not be less than 98%. An ideal partial pressure of oxygen in arterial blood (PaO_2) for a patient breathing 100% oxygen is 500 mmHg, although ventilation/perfusion (V/Q) mismatch from atelectasis and other causes (e.g. physiologic shunts) usually makes this slightly lower. An SpO_2 below 95% should spur a reevaluation of the patient for the problems listed above.

Most anesthetic drugs are potent respiratory depressants, and hypoventilation is a common occurrence in the anesthetized or sedated patient. Measurement of ventilation (partial pressure of CO_2) can be accomplished using arterial or venous blood gas analysis, and estimated with end-tidal carbon dioxide (ETCO2) monitoring. $ETCO_2$ greater than 55 mmHg is an indication to identify patient problems that may result in decreased tidal volumes (e.g. pneumothorax, unibronchial intubation) and to evaluate the machine for problems such as stuck valves or expired soda lime (this will usually result in an increased inspired CO_2 as well).

Cardiac arrhythmias can be secondary to myocardial hypoxia or hypoperfusion, or due to systemic acidosis. They can also occur in response to specific anesthetic drugs such as thiopental or ketamine, or secondary to specific myocardial injury (e.g., contusions). Electrocardiogram (ECG) monitoring will give information on both cardiac rhythm as well as any conduction disturbances. Some arrhythmias, such as ventricular tachycardia or R on T phenomenon, may require drug therapy during the anesthetic period.

Critically ill animals are frequently anesthetized prior to full resuscitation from shock. A shock state represents decreased tissue oxygen delivery and is usually accompanied by a metabolic acidosis. Resuscitation must continue into the anesthetic period, and frequent monitoring of blood gas values and blood lactate concentration will allow assessment of systemic perfusion and oxygen delivery. Keeping a record of intraoperative blood loss (which is not necessarily represented by packed cell volume/total solids measures) may indicate the need for transfusions of fresh whole blood or packed red blood cells to maintain oxygen delivery.

Although many critically ill patients will benefit from expanded intravascular volume, some risk hypervolemia from excessive crystalloid administration, and others risk the development of edema, especially if they are hypoproteinemic. In patients at risk for hypervolemia, the

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placement of a jugular catheter will allow the measurement of central venous pressure (CVP), which estimates the pressure and volume in the right side of the heart. As the CVP approaches 8 cm H_2O, the patient is approaching fluid overload and may be at risk for pulmonary edema and congestive heart failure. Hypoalbuminemic patients can have the colloid osmotic pressure measured; if it is less than 15 mmHg, colloid may be considered as part of the fluid therapy plan.

The most important tools of the anesthetist are his or her senses. Palpation of the pulse and observation of the mucous membranes give information about perfusion, oxygenation, and blood pressure. Assessment of eye position, jaw tone, and palpebral reflex give information about the patient's anesthetic depth, and auscultation of the Doppler pulse or the ECG beep can alert the anesthetist to the development of cardiac arrhythmias. Monitoring devices are available to supplement the senses; these devices allow objective evaluation of the anesthetized patient.

Anesthetists should place emphasis on physical parameters and physical assessment of the patient. Jaw tone, pulse rate and pulse quality, mucous membrane color, and other types of hands-on information can be more accurate and important than a number read from a monitoring screen. In addition, the anesthetist should perform a thorough physical examination of the patient and be familiar with the proposed procedure prior to premedication for anesthesia. Table 50.1 lists normal ranges for commonly measured physical examination parameters.

Table 50.1 Normal values for physiologic parameters in awake dogs and cats.

Parameter	Dog	Cat
Heart rate (beats/minute)	70–110	160–200
Respiratory rate (breaths/minute)	12–20	12–20
Rectal temperature	99.5–101.5°F (37.5–38.6°C)	100–102°F (38–38.9°C)
PaO_2 at sea level (mmHg)	85–100	95–115
SpO_2 (%)	97–100	97–100
$PaCO_2$ (mmHg)	35–45	30–40
$ETCO_2$ (mmHg)	30–40	30–40
Systolic blood pressure (mmHg)	90–140	90–150
Mean blood pressure (mmHg)	60–100	60–100
Diastolic blood pressure (mmHg)	50–75	60–100

When the anesthetist notes abnormal monitoring values, a series of steps should be followed. First, a brief physical examination should be performed to assess the patient. Second, the abnormal values should be verified (e.g. by readjusting the pulse oximeter probe or repeating a blood pressure). This process should take less than one minute to accomplish. With verification of abnormal values, specific therapy should be instituted. In the event of problems with oxygenation or ventilation, it should be verified that the anesthetic machine is functioning properly (i.e. that the delivery of oxygen has not been interrupted or that any valves are stuck) and that an acute change has not happened to the patient (e.g. a pneumothorax or pulmonary aspiration). If there is a problem with the machine, an Ambu bag can be used to maintain ventilation while the machine is switched, and anesthesia may be maintained with IV methods.

If hypotension is detected, in addition to verifying proper cuff placement or arterial catheter function, the patient assessment must include an evaluation of the depth of anesthesia. An assessment of the circulating blood volume of the patient should also be made. If hypovolemia is probable, the anesthetist can consider a bolus of IV fluids (either crystalloid or colloid) and should discuss with the surgeon if it is indicated to consider other fluids, such as packed red blood cells or fresh frozen plasma (or whole blood). In some cases, IV pressor or inotropic agents may be necessary for therapy of intraoperative hypotension.

Anesthesia Monitoring Record

The physiologic data gathered before, during, and after anesthesia are recorded on the anesthesia monitoring record. This record is a legal document and an important part of the medical record. Anesthesia monitoring sheets for a practice should be standardized with sections for patient name and signalment, and areas for preanesthetic history and laboratory values. This allows quick access of this information during the anesthetic period. A sample anesthetic record is shown in Figure 50.1. Separate sections of the anesthetic record should also be available to describe the type of drugs administered, dosage administered, and the effect on the patient for the premedication, induction, maintenance, and recovery phases of anesthesia. An area for notes or descriptions of events that occurred during the anesthetic episode is also necessary. Documentation should always be in ink and should be as complete as possible. Some human hospitals and veterinary practices use a computer-based anesthetic record. A carbon copy form or other mechanism to create a duplicate of the record is also useful. One copy may stay with the record, and another may be cataloged separately in an anesthesia database.

ANESTHESIA CODE
- • PULSE RATE
- V SYSTOLIC B.P.
- Λ DIASTOLIC B.P.
- — MEAN B.P.
- ○ RESPIRATORY RATE
- X START – END ANES.
- ⊙ START – END SURG.

O.R. _____
OTHER _____
DATE _____

PCV _____
T.P. _____
WBC _____
CREAT. _____
TEMP. _____
H.R. _____
R.R. _____
W.T. _____ Kg
P.S. 1 2 3 4 5 E
DRUG THERAPY:

PERTINENT HISTORY

PROCEDURE _____
CLINICIAN _____ ANESTHETIST _____
POSITION _____ BLOOD LOSS _____ mls
NUMBER SPONGES: BEFORE SURG. _____ AFTER SURG. _____

TOTAL FLUIDS
NORM-R _____ ml
N. SAL _____ ml
_____ ml

TOTAL ANESTHESIA TIME
_____ hrs. _____ mins.
RECOVERY
ARRIVAL TEMP. _____

EKG: YES _____ NO _____
I.V. CATH _____ LOC _____
I.V. CATH _____ LOC _____
ART CATH _____ LOC _____
B.P. MONITOR: DIRECT _____ INDICRECT _____

DRUG	CODE	AMT. in MLS	DISPENSER

EPIDURAL: DRUG _____
VOL. (ml) _____ INJ. SITE _____ NEEDLE
CSF YES NO BLOOD YES NO GA _____ L _____ CATHETER _____
ONSET (mins) _____ CRANIAL LIMIT _____

PRE. ANES _____ RTE _____ TIME _____ ENDO SIZE _____
EFFECT _____ H.R. _____ R.R. _____ ENDO ATTEMPTS _____
INDUCTION AGENTS: DRUG/mgs _____ DRUG/mgs _____

MAINTENANCE

CIRCLE ANESTHETIC TECHNIQUES USED: SCCS → CCS STEVENS; NRS IV; MASK BOX; LIQUID INJ. OTHER

HALOTHANE%													
ISOFLURANE%													
O_2L/min													

:00 :15 :30 :45 :00 :15 :30 :45 :00 :15 :30 :45

260 240 220 200 180 160 140 120 100 80 60 40 20 0

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D-1

Figure 50.1 Typical anesthesia record. Note provision of areas for preoperative history and blood work, American Society of Anesthesiologists classification, planned procedure, and patient identification. The anesthetist can record the blood pressure and other parameters using the grid and the legend in the upper left corner. There are spaces to note preoperative and induction medications, as well as a space to note the quality of recovery. The open area on the right side allows the anesthetist to take notes during the procedure and is referenced to numbers written on the timeline of the grid. Note also spaces available for notation of SpO_2, $ETCO_2$, and percentage inhalant administered.

A well-organized record allows veterinary technicians and veterinarians to quickly review the anesthetic record in the event of problems in the perioperative period. Accurate recording of the entire anesthetic episode also provides a reference for future anesthetics in the same patient. If a patient had a previous difficult intubation or adverse reaction to premedicant drugs, a review of the previous anesthetic record will highlight these facts.

Patient monitoring as it pertains to the anesthetic procedure begins with administration of the premedication drug or combination of drugs. The premedication regimen, route of administration (intramuscular, IV, subcutaneous), and the effect of the drugs on the patient should be recorded. Events such as emesis, defecation, bradycardia, excitation, or sedation are particularly important to note. If animals experience emesis prior to the securing of a protected airway, they may be in danger of aspiration pneumonia, and oxygenation will have to be closely monitored in the intra- and postoperative periods. Other adverse responses to the chosen anesthetic protocol can include respiratory arrest, abnormal excitation, or aggressive behavior.

Monitoring of the patient continues into induction. The type and amount of induction agent(s) used, as well as route of administration, should be recorded. Any adverse effects during induction (e.g. emesis, arrhythmias, hypotension, or excitation) are important to note, in addition to any difficulties associated with endotracheal intubation (this may also be graded on a scale of 1–4, with 1 representing a smooth intubation and 4 representing the most difficult intubation). There should be a place on the anesthetic record to note the size of endotracheal tube used for intubation.

During the maintenance phase of anesthesia, parameters such as heart rate, respiratory rate, blood pressure, temperature, end-tidal CO_2, oxygen flow rate, and end-tidal inhalant (if available) or delivered inhalant percentage should be recorded at five-minute intervals or more frequently. If acute changes occur, these should be noted on the record as well. The type and amount of fluid therapy should also be recorded, in addition to any adjunctive drugs used during anesthesia (e.g. fentanyl or norepinephrine administered as a constant rate infusion). Notes should be made in the anesthetic record about adjustments in fluid therapy, changes in anesthetic depth, changes in position or recumbency, occurrence of reflux, administration of additional drugs, surgical progress, or any other event that occurred during the anesthetic episode. The time of extubation, postoperative analgesic therapy, and other pertinent details are recorded on the recovery section of the sheet. Following extubation, patient monitoring should continue until the body temperature reaches a satisfactory level (>99°F, 37.2°C) and the patient is able to perform purposeful movement such as swallowing. During the recovery phase of anesthesia, measurement of temperature, pulse rate, and respiration rate should take place no less frequently than every 15 minutes.

Physical Parameters Under Anesthesia

Depth of anesthetia is most accurately monitored by serial physical examination of the anesthetized patient. Small animal patients progress through a predictable series of physical changes as they become more deeply anesthetized, and they encounter these signs in reverse as they become more lightly anesthetized (Figure 50.2). These stages are labeled with Roman numerals: I represents an awake animal through to V, which represents a dangerously deeply anesthetized animal (Figure 50.2). Because some physical parameters can be similar between animals that are relatively lightly anesthetized and those that are overly anesthetized (e.g. a central eye position), only serial examination will make it possible to accurately assess the patient's depth of anesthesia. For surgical anesthesia, a patient should be in a plane of light to medium stage III anesthesia. Physical parameters should be assessed no less frequently than every five minutes in the anesthetized patient.

Eye Position

The eye position of an awake animal is a central eyeball, which remains constant into light anesthesia. As the patient becomes more deeply anesthetized, the eyeball rotates to a ventromedial position and remains this way through moderate anesthesia. As the patient's anesthetic plane deepens, the eye rotates back to a central position. Thus, a central eyeball in an anesthetized animal can indicate a patient that is either not deep enough for a surgical procedure or one that is excessively deeply anesthetized. Used in combination with other physical examination findings, eye position is an important part of the anesthesia physical examination.

Jaw Tone

Jaw tone varies among different canine patients; animals with muscular jaws tend to have higher basal tone and less overall change during anesthesia. In most patients, however, high tone and a tight jaw is associated with lighter anesthetic states that gradually lessens as the animal becomes more deeply anesthetized. Jaw tone is a reliable indicator of anesthetic depth in cats.

Mucous Membranes/Capillary Refill Time

Mucous membrane color and capillary refill time (CRT) allow the anesthetist to estimate perfusion and oxygenation of the anesthetized patient. Well-perfused mucous membranes are pink with a CRT of 1.5–2 seconds. The most accessible mucous membranes to the anesthetist are the gums. Because oxygenated hemoglobin produces the pink color of mucous membranes, factors that affect hemoglobin concentration or saturation of hemoglobin with

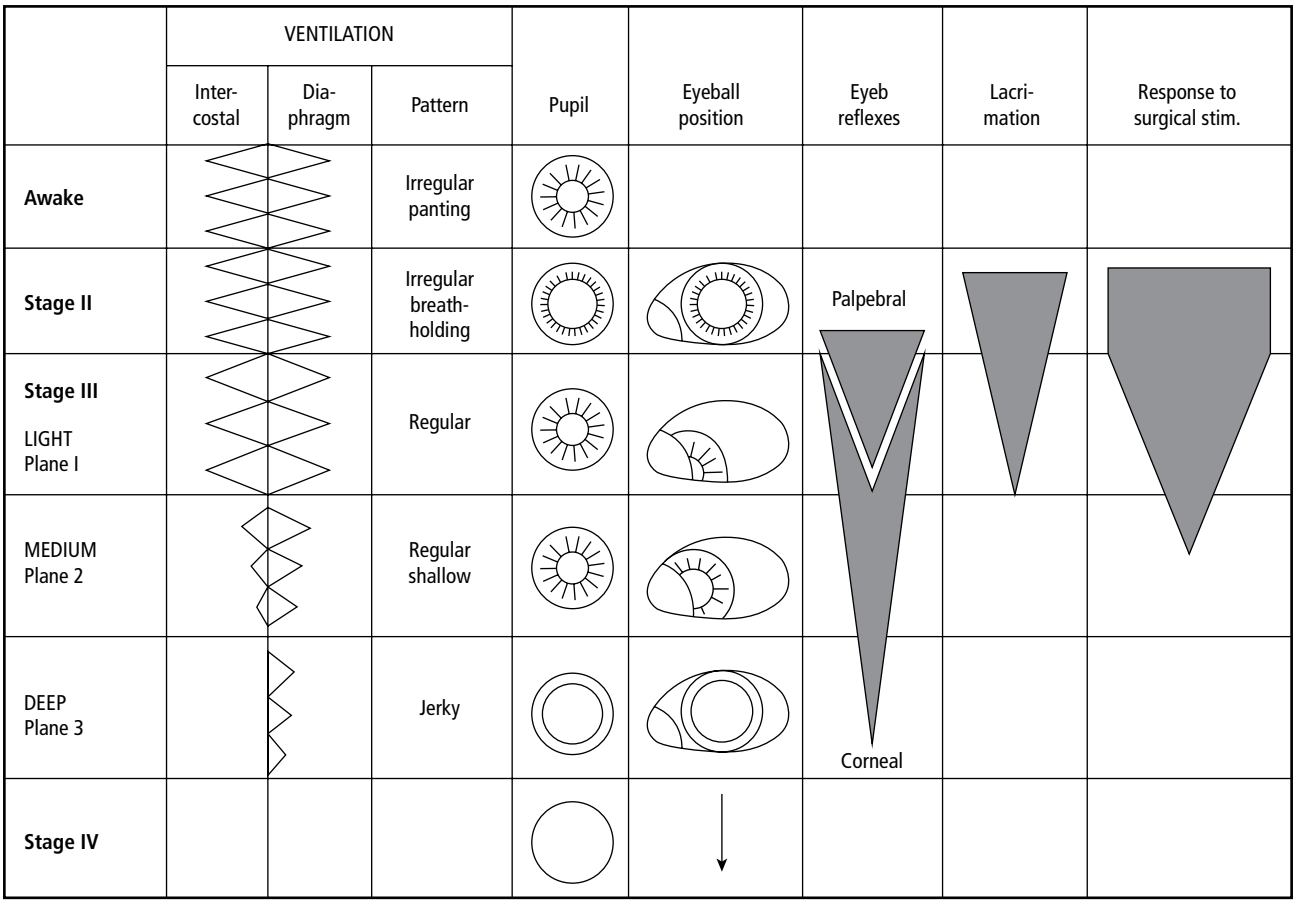


Figure 50.2 A diagram of the physiologic changes that occur in patients as they progress through the stages of anesthesia (noted in the left-hand column). *Source:* From Hall, Clarke, and Trim, *Veterinary Anaesthesiology*, 10th edn.; reproduced with permission.

oxygen can result in changes in the membrane color. In addition, if there is a disturbance that results in decreased blood flow to the gums (i.e. vasoconstriction), the mucous membrane color may be altered.

Pale mucous membranes are most associated with anemia, and the color can range from a paler pink with moderate anemia to almost white in cases of severe anemia. Other causes of pale mucous membranes include vasoconstriction or hypothermia, both of which result in decreased peripheral perfusion. Decreased perfusion may be physiologic (e.g. response to α-2 agonist drug administration) or may indicate a serious problem (e.g. hemorrhage or cardiac arrest). Membrane color and CRT should be monitored no less frequently than every five minutes in animals under anesthesia. Changes in the membrane color should be evaluated in the context of other physical examination and monitoring parameters.

A bluish tinge to the membranes, called cyanosis, indicates a severe decrease in hemoglobin saturation with oxygen. The blue color is due to the presence of deoxyhemoglobin, which is hemoglobin without oxygen molecules attached. In some cases of extremely decreased perfusion (i.e. decompensatory shock), the mucous membranes may also appear to have a blue or purple hue (sometimes described as "muddy"). Cyanosis noted during anesthesia requires immediate action to determine the cause and to restore adequate oxygenation. Possible causes of cyanosis may be due to patient problems (e.g. pneumothorax, pulmonary embolism, aspiration pneumonia) or machine failure (i.e. inadequate supply of oxygen, malfunctioning oxygen flow meter, or a circuit disconnect). Suspected abnormalities derived from mucous membrane assessment should be rapidly confirmed with other methods (e.g. pulse oximetry, arterial blood gas analysis), and the supply of oxygen to the anesthesia machine checked. The bobbin in the oxygen flowmeter will not float if gas is not traveling through the machine (unless it has become stuck). Other descriptions of mucous membrane color are covered earlier in this book.

CRT is measured by an initial blanching of the oral mucosa from pressure with a finger and followed by evaluation of the time for the color to return. Normal CRT is 1.5–2 seconds. In animals with pigmented mucous membranes, CRT may be difficult to assess. A slow return of color may indicate impaired systemic perfusion. In animals with pale mucous membranes secondary to vasoconstriction, the CRT may also be slower than normal or difficult to

assess. The converse is also true. Animals with vasodilation have bright pink to red mucous membranes. With an increase in cardiac output such as might be seen with compensatory or hyperdynamic shock, the CRT will be faster than normal, usually less than one second. The most common reason for brick red membranes and a fast CRT is septic shock.

Response to Stimulation

A patient's response to stimulation is decreased with adequate anesthesia. Responses are manifested as increases in sympathetic tone (e.g. tachycardia, hypertension) or a lightening of the anesthetic plane (e.g. an increase in jaw tone). If these responses occur in response to a surgical or other procedure, the animal may not be adequately anesthetized, and the depth of anesthesia should be verified using other physical examination or monitoring parameters. The use of local anesthetic blocks or total IV anesthesia may result in a patient that retains more tone or reflexes than animals anesthetized with inhalant anesthetic alone.

The palpebral reflex, a blink elicited by light touch over the upper or lower eyelids (or in the medial canthus of the eye), is gradually decreased as the depth of anesthesia increases. In general, for a surgical plane of anesthesia, the reflex may be sluggish but does not need to be absent. The corneal reflex, a withdrawal of the orbit when the cornea is touched, is maintained to even deeper anesthesia. If the corneal reflex is absent, the patient may be over anesthetized or near death.

Heart Rate

Cardiac output is the product of heart rate and stroke volume, indicating the amount (volume) of blood pumped from the heart each minute. Stroke volume, the amount of blood pumped with each contraction of the heart, can be difficult to measure directly; therefore, it is important to closely monitor heart rate because it is directly related to cardiac output. An acceptable heart rate under general anesthesia depends on a number of variables including species, patient size, the anesthetic regimen (especially if anticholinergic or α-2 agonist drugs are administered), and the patient's normal heart rate.

The most basic approach for monitoring heart rate is digital palpation of the pulse. The anesthetist palpates the pulse by placing two fingers (not a thumb) over an artery and applying gentle pressure. Pressing down too firmly may occlude or collapse the artery and eliminate the pulse. The most commonly used arteries for digital palpation are the femoral, dorsal pedal, caudal (ventral aspect of the tail), buccal, and lingual arteries (Figure 50.3). These areas are easily accessible during induction and surgical preparation. When patients are positioned with their head away from the anesthetist, the dorsal pedal, caudal, and femoral arteries are the best choice. However, if the head of the patient is toward the anesthetist, the lingual artery is most accessible. Other palpable pulses are the radial pulse, as well as the apex beat in the thorax. In some cases of cardiac arrhythmias (e.g. ventricular premature contractions), the palpated pulse rate will differ from the actual heart rate seen on the ECG. Arrhythmias will be palpated as weak or irregular pulses and should be verified with other monitoring equipment.

The pulse rate is calculated by counting the pulse over a set number of seconds and multiplying this time by a value to equal 60 seconds. The resulting value is the heart rate in beats/minute:

$$\text{Beats in 15 seconds} \times 4 = \text{heart rate (beats/minute)}$$

$$\text{Beats in 10 seconds} \times 6 = \text{heart rate (beats/minute)}$$

The longer the period over which the pulse is counted, the more accurate is the recorded pulse rate. It is good practice for the anesthetist to keep a hand on the pulse at all times when not otherwise occupied, to monitor pulse rate and quality.

Pulse quality is a subjective value that is not always recorded, but with experience anesthetists may gain valuable information about the patient from pulse palpation. The palpated pulse is the difference between the systolic



Figure 50.3 Commonly used arteries for digital palpation: (a) femoral; (b) dorsal pedal; and (c) sublingual.

and diastolic arterial blood pressure (DAP). Animals with a large difference in systolic and diastolic blood pressure (due to a lower diastolic, higher systolic pressure, or both) have a strong or bounding pulse. Bounding pulses may result from conditions such as normovolemic anemia or compensatory septic shock. If there is a low pulse pressure, the animal will have a weak pulse. Weak or "thready" pulses represent lower arterial blood pressure due to poor cardiac output or cardiovascular tone. If weak pulses are palpated, or if pulses become weak during a procedure, a more accurate determination of blood pressure (such as with Doppler or direct arterial pressure monitoring) and reevaluation of the patient should be performed. Weak pulses may be consistent with hypovolemia, hemorrhage, or decreased cardiac output due to anesthetic drugs or cardiac arrhythmias [1]. Pulse pressure should be evaluated in concert with mucous membrane color and CRT.

Another reliable method for monitoring heart rate is an esophageal stethoscope (Figure 50.4). This device is an esophageal probe connected to a listening device. The esophageal probe comes in a variety of sizes and lengths. The probe is connected to either a Doppler unit or standard stethoscope earpieces. After anesthetic induction and endotracheal intubation, the probe is advanced into the esophagus until the heartbeat can be heard. The probe should be advanced only to the point where the heartbeat is clearly heard; additional advancement past this point of maximal intensity may result in the placement of the probe into the stomach. Using the stethoscope, heart rate can be directly measured. An advantage of using the esophageal stethoscope is that the heart and lungs may be auscultated constantly and directly. Changes in rate, or in the quality of a heart murmur or lung sounds, may be detected as they happen. Some esophageal stethoscopes are combined with temperature probes and ECG electrodes.

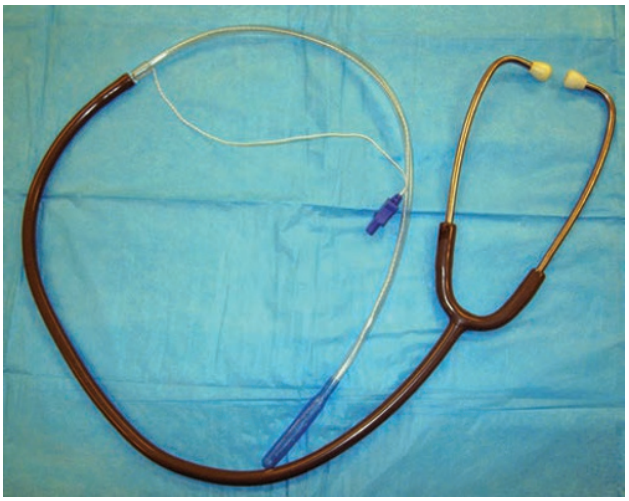


Figure 50.4 Esophageal stethoscope.

The ECG is used to monitor both the heart rate and rhythm. ECG interpretation is addressed in Chapter 11. A lead II ECG is used to monitor basic cardiac rate and rhythm in small animal patients, and leads may be attached to the patient's skin either using alligator clips or patches with snap leads. In some exotic species, the leads may be attached to the metal part of 25- to 30-gauge needles inserted through the skin. It is not necessary to clip the fur to attach alligator clips but doing so provides better contact and a better signal. Either alcohol or conductive gel must be applied to optimize electrical conduction from the patient's skin to the electrode. Hair must be clipped when using patches, and the area should be wiped with alcohol and allowed to dry before patches are applied to optimize their adherence. Patches may also be placed directly on the paw pads. Owing to evaporation of alcohol or drying of conductive gel, leads may need to be moistened periodically to maintain optimum conductance.

The ECG lead placement is similar to that used for other purposes; however, the standard arrangement may not be possible for some procedures. In the case where the procedure interferes with lead placement (e.g. forelimb amputation), ECG leads should be placed as close to the appropriate area as possible, to maintain the principles of Einthoven's triangle and surgical sterility (see Chapter 10 for more details). It is most important to keep the leads on the correct side of the body and in the correct plane with the heart. Thoracic limb leads can be moved cranially, and pelvic limb leads may be moved further caudal on the limb.

Respiratory Rate

The respiratory rate can be calculated by observation of the patient or the rebreathing bag. Adequate respiration is essential to a safe and smooth anesthetic episode because anesthesia is most frequently maintained using volatile inhalant gas anesthetics, which can suppress respiratory drive. If a patient is not taking regular breaths with an adequate tidal volume, anesthetic will not be inhaled, and the patient may not become anesthetized. Respiratory minute volume (RMV) is the amount of gas exhaled by the lungs over one minute and is the product of respiratory rate and respiratory tidal volume. The actual RMV can be measured by a respirometer (Figure 50.5), such as the Wright respirometer, inserted into the anesthetic circuit. Some machines that measure exhaled CO_2 also contain a respirometer feature. The anesthetized patient should take deep breaths at regular intervals. RMV is related to ventilation (and thus blood CO_2 concentration); an increase in RMV results in a decreased CO_2 concentration, and vice versa. Normal RMV in awake dogs and cats is approximately 200 ml/kg/minute.



Figure 50.5 A Wright respirometer (black dial) placed in the expiratory limb of the anesthesia circuit. The respirometer measures tidal volume and respiratory minute volume.



Figure 50.6 The NICO 2 Respiratory Profile Monitor (Phillips-Respironics, Murraysville, PA) a multiparameter monitor that continuously measures and reports inspired and expired PCO_2, respiratory rate, and oxygen saturation. Source: Photo courtesy of Kate Hopper BVSc, PhD, DACVECC.

Another way to monitor respiratory rate is to use an airway monitor. This device produces a humming sound as the patient exhales, which signals to the anesthetist that a respiratory cycle has taken place (i.e. the animal has inhaled and exhaled once). The monitor's sensor is placed in the anesthetic circuit between the endotracheal tube and the Y-piece. The sensor is connected to a small speaker, which amplifies the sound of the patient's exhalation. The produced sound does not reflect the quality of ventilation, just the fact that exhalation is taking place. The monitor does not remove any gas samples from the circuit. Modifications to these monitors can make them apnea monitors as well, and some will alarm if the patient has not taken a breath in a specified amount of time. Other monitors, such as the capnograph, also provide an apnea alarm.

The most comprehensive realtime bedside ventilatory monitor is the capnograph. It continuously measures and reports respiratory rate along with inspired and expired PCO_2. Some capnographs (e.g. the NiCO 2, Phillips-Respironics, Murraysville, PA) also function as a respirometer (Figure 50.6); these machines measure tidal volume and can estimate pulmonary compliance (see Chapter 31). The $ETCO_2$ is a practical estimation of arterial CO_2. Capnography is covered in depth in Chapter 30.

Hypoventilation (decreased RMV) occurs due to a decrease in respiratory rate, tidal volume, or both. Hypoventilation leads to hypercarbia (also called hypercapnia), which is an increase in dissolved carbon dioxide in the blood ($PaCO_2$ or $PvCO_2$). Hypercarbia results in a respiratory acidosis, and as the blood pH decreases, deleterious effects can be seen on vascular tone and cardiac function. High blood levels of CO_2 can also cause narcosis and may result in increased intracranial pressure, cardiac arrhythmias, hypoxemia, and ultimately cardiac arrest.

Decreased RMV may be due to a decreased respiratory rate (or complete apnea), seen with patients with an irregular breathing pattern or breath holding, or it may be due to a decrease in tidal volume, despite a normal respiratory rate. If hypercarbia or hypoventilation is identified, the anesthetist must increase the RMV by manually or mechanically ventilating the patient. Assisted ventilation may be used to increase the respiratory rate, tidal volume, or both. Assisted ventilation also increases the inhalant anesthetic delivered to the patient.

When hypoventilation is identified, the anesthetist should first evaluate the patient (Table 50.2), as well as the positioning of the endotracheal tube. Unibronchial intubation occurs when the endotracheal tube is advanced beyond the carina into the bronchus of one of the lung lobes. V/Q mismatch ensues because an entire lung is without effective ventilation. Ideally, the endotracheal tube should be placed so that the distal end of the tube is located in the caudal extrathoracic trachea. If there is a possibility that the tube has been introduced too far into the trachea (resulting in one lung intubation), it may be incrementally removed until the cuff of the endotracheal tube is palpated in the caudal extrathoracic trachea. The tube cuff should be deflated before readjusting the tube, should adjustment be necessary.

The patient with elevated RMV is hyperventilating, which causes a decreased PCO_2, called hypocarbia (or hypocapnia). Hyperventilation in the anesthetized patient is most commonly a result of an inadequate anesthetic plane or an elevated body temperature. In some cases, where the patient exhibits extreme hypoxia, hyperventilation may be a reflection of hypoxic drive because the need

Table 50.2 Assessment and approach to common abnormalities of ventilation in the anesthetized patient.

Problem	Assessment steps	Actions
Hypoventilation ($PaCO_2$ or $ETCO_2$ > 65 mmHg)	Is the patient unable to generate an adequate tidal volume by spontaneous ventilation or mechanical ventilation?	Auscultate the thorax and rule out pulmonary dysfunction such as a pneumothorax. If tidal volume is limited, consider repositioning the patient or increasing the respiratory rate. Ventilators that deliver a breath to a certain airway pressure will deliver a smaller breath if there is increased pressure in the thorax. Rule out endotracheal tube obstruction.
	Is the patient unable to generate an adequate tidal volume by spontaneous ventilation?	Consider the use of a mechanical ventilator for maintenance of anesthesia, or increase the number of assisted breaths delivered to the patient by the anesthetist. If neuromuscular blocking agents (e.g. atracurium) have been used during the procedure, consider reversal.
	Is the patient's level of anesthesia excessive? (Figure 50.2)	Efforts should be made to lighten the plane of anesthesia, and if the patient is breathing spontaneously, manual ventilation may be necessary.
	Have opiate medications been administered recently?	If anesthetized, increase either tidal volume, respiratory rate, or both. If awake, consider reversal of the opiate medication using naloxone (which also removes any opioid-related analgesia).
	Is there a kink or obstruction in the breathing system?	Inspect the system (including endotracheal tube) for kinks or obstructions; address as necessary.
	Is there a leak in the anesthetic system?	Leaks will prevent the delivery of a full tidal volume breath to the patient; if a leak is suspected, it may be necessary to change the tubing, the ventilator bellows, the rebreathing bag, or the entire system. If a leak cannot be readily detected, breathing for the patient with an Ambu bag will allow time to switch out the system and to correct the hypoventilation. It should also be verified that the cuff on the endotracheal tube is adequately inflated.
	Is there a leak in the ventilator bellows?	Leaks in the bellows of a mechanical ventilator will prevent the bellows from returning completely to the full position between breaths. The patient may be hand ventilated using the rebreathing bag or an Ambu bag while the bellows are changed.
	Is the soda lime expired?	Check the soda lime canister for heat and color, as well as the last time the soda lime was changed. Expired or used soda lime will not remove adequate CO_2 from the system and may cause it to build up. Switch machines or breathe for the patient with an Ambu bag while the soda lime is changed. Also verify that the soda lime canister is filled correctly; if it is packed too tightly or above the fill line, it may not remove CO_2 appropriately. These patients will also have a high inspired CO_2 as measured by the capnograph.
	Is there a leak in the valves of the anesthesia machine?	Incompetent inspiratory or expiratory valves will not allow air to travel in a circle system; these patients will show a high inspired CO_2 level.
Hyperventilation ($PaCO_2$ or $ETCO_2$ < 25 mmHg)	Is the respiratory rate or tidal volume excessive (spontaneous ventilation)?	Verify that the patient is adequately anesthetized for the procedure. Verify that the patient's temperature is not elevated.
	Is the respiratory rate or tidal volume excessive (mechanical ventilation)?	If a mechanical ventilator is in use, adjust either the rate or tidal volume to decrease the respiratory minute volume.
	Is the patient hypoxemic?	Assess SpO_2 and/or an arterial blood gas to verify patient oxygenation.
Very low $ETCO_2$	Was intubation successful?	Verify that the endotracheal tube is not in the esophagus.
Sudden drop in $ETCO_2$	Any acute changes in patient condition?	Severe drops in $ETCO_2$ may reflect circulatory collapse or the occurrence of a pulmonary thromboembolism. The patient should be assessed for stability immediately and additional diagnostics or CPR performed as necessary.
	Is the anesthetic circuit intact?	Evaluate the circuit for any disconnections.

for oxygen overwhelms any depressant effects of anesthetics on the respiratory center. Hyperventilation due to inadequate anesthesia may be accompanied by tachycardia and hypertension. The anesthetist may address this either by increasing ventilation or administered inhalant percentage (both of which provide more anesthetic to the patient), or by administering analgesic drugs (e.g. opioids) to provide additional analgesia.

The normal physiologic response to elevated arterial PCO_2 is an increase in RMV (either by increasing the respiratory rate or tidal volume). This normal response to high $PaCO_2$ is blunted by anesthesia, but an elevated respiratory rate may be seen in patients with a $PaCO_2$ above 65 mmHg. Owing to the effects of anesthesia on the normal responses to PCO_2 as well as effects on RMV (e.g. due to smaller tidal volumes), it is difficult to determine the patient's $PaCO_2$ based solely on observation of the respiratory rate; the only true way to assess ventilation is to measure the arterial or venous CO_2 partial pressure.

Malignant hyperthermia causes increased respiratory rate accompanied by hyperthermia and hypercapnia. Some anesthetic drugs, such as halothane and succinylcholine, have been linked as triggers for malignant hyperthermia in susceptible animals. If malignant hyperthermia is suspected, anesthesia should be discontinued as soon as possible, the anesthetic lines detached from the endotracheal tube, flushed thoroughly with oxygen, and reattached to the patient. It may be easier to switch to an unused anesthetic machine. The patient's $ETCO_2$, rectal or esophageal temperature, and oxygenation should be closely monitored. External warming and heat support should be discontinued and if the temperature continues to rise, the patient should be made wet, and a fan used to encourage convective cooling. Dantrolene may help to reverse the signs of malignant hyperthermia by altering intracellular calcium kinetics. This drug may be given at a dose of 1–10 mg/kg, IV. At-risk patients should be treated with dantrolene orally prior to elective surgeries [2].

$ETCO_2$ is directly proportional to cardiac output and tissue perfusion, except in rare conditions of mitochondrial dysfunction (usually as a result of septic shock). If the respiratory rate and tidal volume have remained the same but the $ETCO_2$ has decreased, the patient is experiencing either a decreased metabolic rate (e.g. due to hypothermia) or a decrease in cardiac output (e.g. due to hemorrhage or arrhythmias). If a sudden decrease in $ETCO_2$ is seen, the anesthetist should evaluate the patient's vital signs immediately to verify adequate perfusion because a precipitous drop in $ETCO_2$ is seen with cardiac arrest. Once it is clear that the patient is not in distress, additional evaluation of the equipment for leaks or disconnections may take place [3].

Blood Pressure

Chapters 12–14 provide an in-depth analysis of the various methods for blood pressure measurement. For the anesthetized patient, the same types of monitors and criteria for evaluation may be used. Although blood pressure is not a direct measure of perfusion in the anesthetized patient, it is the closest and most convenient surrogate measure that we have to determine that organ perfusion is appropriate. Actual perfusion of individual organs is determined by a host of local mechanisms and is extremely difficult to measure, even under experimental circumstances.

In general, mean arterial blood pressure (MAP) of 70–100 mmHg is acceptable for the anesthetized patient. Systolic arterial blood pressure should be 100–140 mmHg. DAP should be 40–65 mmHg. A MAP below 60 mmHg may be associated with decreased perfusion of and oxygen delivery to the kidney and brain [4]. A DAP less than 40 mmHg may result in poor coronary artery perfusion (the heart is only perfused during diastole), and decreased oxygen delivery to the heart [5]. In patients who are chronically hypertensive prior to anesthesia (e.g. those with chronic kidney disease), an effort should be made to maintain MAP at levels toward the higher end of the range (i.e. toward the normal values for that patient).

Low blood pressure in critically ill patients may be caused by hypovolemia, hemorrhage, a gas-filled viscus (e.g. the stomach in patients with gastric dilatation and volvulus), anesthetic drugs, heart disease, cardiac arrhythmias, or vasodilation (e.g. as a result of vasoplegic shock). Individual patients have individual reasons for hypotension, and the anesthetist's knowledge of the disease state and the patient's physical examination will help to determine therapy and plan a safe anesthetic. As an example, patients with heart disease such as dilated cardiomyopathy may need a positive inotropic drug to support blood pressure by supporting cardiac output. By contrast, a patient that is hypotensive due to hypovolemia may require IV fluid therapy to resolve low blood pressure.

Hypotension associated with anesthetic drugs may have components of myocardial depression and vasodilation, depending on the drug. When hypotension is detected, the anesthetic plane of the patient should be reevaluated to ensure that they are not too deeply anesthetized (if they are, the anesthetic plane may be lightened). An assessment of the degree of blood or fluid loss should also be undertaken, and if the animal is hypovolemic, an IV bolus of fluids (crystalloid or colloid) may help to restore adequate blood pressures. In cases of excessive vasodilation causing hypotension, pressor drugs such as phenylephrine, dopamine, norepinephrine, or vasopressin may be indicated to cause vasoconstriction.

Although hypertension is uncommon in veterinary patients, it may also have consequences such as retinal detachment or cerebral hemorrhage. Anesthetized animals with a pheochromocytoma may have paroxysmal tachycardias and hypertensive episodes that may merit therapy.

Doppler Blood Pressure Measurement

The most widely used blood pressure monitor is the Doppler flow probe. The Doppler uses a probe containing a piezoelectric crystal which emits sound waves that are reflected by the red cells in pulsatile arterial blood. When the sound is reflected from the cells, the shift in the frequency of the sound is transduced into an audible signal. The placement of a pneumatic blood pressure cuff and sphygmomanometer proximal to the probe allows the measurement of blood pressure (see Chapter 14). Common areas used for Doppler probe placement in the anesthetized animal are the palmar common digital artery on the palmar surface of the thoracic limb below the carpus; the dorsal pedal artery on the plantar surface of the pelvic limb; the plantar metatarsal artery on the plantar surface of the pelvic limb, proximal to the foot; and the coccygeal artery located on the ventral surface of the tail.

For measurement of blood pressure, an appropriate size blood pressure cuff (ideally with a width that is 40% of the circumference of the limb) is placed proximal to the probe and inflated with the sphygmomanometer until the artery is occluded; this will result in a cessation of the Doppler sound. As the pressure in the cuff is slowly released, the audible signal will return as soon as the cuff pressure is below that of the systolic blood pressure. The anesthetist can repeat this reading as often as desired. Changes in the intensity of sound on the Doppler may be consistent with arrhythmias or a decreased pulse strength (as may occur from sudden hypotension). The placement of the probe and cuff should be checked to verify that nothing has changed after a patient assessment.

An advantage of the Doppler is early detection of decreased flow, along with the ability to hear changes in heart rhythm and measure blood pressure despite arrhythmias. A disadvantage is that the signal may become difficult to hear with noise in the operating room, although most Doppler units allow the use of headphones to improve focus on the pulse.

Oscillometric Blood Pressure Measurement

Oscillometric noninvasive blood pressure monitors (e.g. the Cardell, Midmark Corp., Versailles, OH, or Dinamap, GE Healthcare, Wauwatosa, WI) are excellent tools for monitoring trends in blood pressure in the anesthetized patient. The theory behind these machines is discussed in Chapter 14. Oscillometric monitors are more automated than the Doppler devices. They can be programmed to cycle at various intervals and to give a regular readout of systolic, diastolic, and mean blood pressure. Because these monitors rely on a machine-based interpretation of the blood pressure oscillations, they may be less accurate than the Doppler probe, especially for critically ill patients who may be hypotensive (thus diminishing the strength of oscillations) or in animals experiencing cardiac arrhythmias (the machines require a regular heartbeat to accurately sense oscillations). Unlike the Doppler probe, these machines do not produce an audible signal to reflect heart rate (and in fact may continue to give readings even in the absence of a good pulse). It is thus imperative to compare the heart rate displayed on the machine to a heart rate obtained by another source (e.g. auscultation or ECG). If the heart rate does not match that of the patient, the blood pressure reading should not be trusted. Despite these caveats, the oscillometric blood pressure monitor is a useful tool for monitoring blood pressure in most anesthetized patients.

Direct Arterial Blood Pressure Measurement

Direct arterial blood pressure is covered in Chapter 13 and considered the gold standard for monitoring arterial blood pressure. Critically ill or emergent patients under anesthesia can experience fluctuations in blood pressure that may not be detectable using intermittent measurement. In these cases, or in cases where it is necessary to use vasoactive drugs to support blood pressure, a direct arterial pressure monitor is ideal. Direct arterial blood pressure also allows the anesthetist to directly observe the effects of arrhythmias that occur during anesthesia, and it provides a simple way to obtain samples for blood gas analysis. Direct arterial catheters for use during anesthesia may be placed in the dorsal pedal, caudal, or auricular arteries. The femoral artery may also be used but is associated with more complications on catheter removal.

Oxygen Saturation

Oxygen saturation is a measure of how much oxygen the hemoglobin in the blood is carrying, described as a percentage of the maximum it could carry, and it is an important component of oxygen content. Adequate oxygen content in the arterial blood, in combination with cardiac output, determines oxygen delivery to tissues.

Oxygen saturation is routinely monitored with a pulse oximeter. Pulse oximetry is covered extensively in Chapter 25. The most common site for placement of the pulse oximeter probe in the anesthetized patient is the

tongue or lips. If the tongue cannot be used, the probe can be placed on an area of thin skin such as the skin between the toes, the abdomen, or on the vulva or prepuce. Using the transmission (clip) probe, the tissue must be thin enough to allow the probe to pass light through the tissue to the sensor on the other side of the clip. Reflectance probes may be used rectally or on thin areas of skin to obtain a saturation reading without using the clip.

The pulse oximeter provides an oxygen saturation reading given as a percentage, a pulse waveform, and a heart rate. The oxygen saturation is related to the dissolved oxygen content (PaO_2) by the oxyhemoglobin dissociation curve (Figure 50.7; see also Chapter 25). Normal hemoglobin is expected to be 100% saturated with oxygen at a PaO_2 of 95–100 mmHg (most normal animals breathing room air have a PaO_2 of 80–100 mmHg and an SpO_2 of 96–100%). Under anesthesia and breathing 100% oxygen, an animal's PaO_2 is expected to be greater than 500 mmHg. Consequently, an anesthetized animal with normal lung function is expected to have a SpO_2 of 99–100% (hemoglobin cannot be saturated more than 100% with oxygen).

If SpO_2 readings below 98% are obtained in the anesthetized patient breathing 100% oxygen, there is a problem with oxygenation in the patient. In the event of low SpO_2, the patient and subsequently the anesthesia circuit should be inspected for causes of the abnormal reading.

Because the SpO_2 only reflects a percent saturation, a patient breathing 100% oxygen that has a PaO_2 of 200 mmHg will have the same SpO_2 as a patient with a PaO_2 of 500 mmHg. The first patient may have some significant lung pathology (preventing a normal PaO_2), which may become relevant during anesthesia recovery when the patient transitions to breathing room air (21% oxygen). For this reason, the pulse oximeter does not replace arterial blood gas analysis but is useful for a continuous estimate of oxygen saturation.

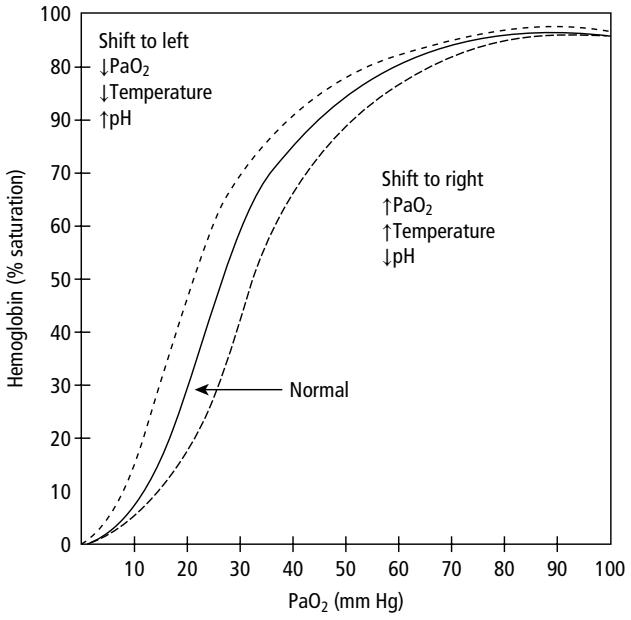


Figure 50.7 Oxyhemoglobin dissociation curve.

Many critically ill patients may have compromised lung function, whether it is due to primary lung pathology such as pneumonia or due to decreased lung function from pleural effusion or ascites (which decrease the ability to fully expand the lungs). Animals that have been recumbent frequently develop atelectasis, or lung collapse, on the dependent side. Atelectasis causes a ventilation/perfusion mismatch and decreases the ability of the animal to oxygenate the blood. Atelectasis may also develop during surgery when the patient is immobile for a period. If atelectasis occurs during anesthesia, oxygenation during the recovery period may be affected. The pulse oximeter may be used for serial evaluation of patients during anesthesia recovery.

Significant changes in oxygen saturation rarely occur acutely. It is thus important to watch the overall trend of the readings. A gradual decrease in saturation will be clear on the anesthesia monitoring sheet. Changes in saturation may be accompanied by changes in other parameters, such as respiratory rate or $ETCO_2$, and can help the anesthetist to determine the possible cause and treatment. A common cause of hypoxemia may be endotracheal tube obstruction with blood or mucus during anesthesia, especially in patients undergoing thoracic surgery, or in those with pneumonia. Other causes of a low intraoperative SpO_2 include the development of a pneumothorax (which will be accompanied by hypotension and tachycardia) and the development of atelectasis (which will cause a slower decrease in SpO_2).

When a low SpO_2 is detected, it is important to check the patient (Table 50.3) and to verify that the anesthesia machine is providing adequate oxygen. If surgery is occurring, verify that there is not a chance of an iatrogenic pneumothorax or excessive hemorrhage. If a mechanical ventilator is in use, switch to manual breaths; this will allow an increase in respiratory rate and tidal volume, and it will allow the anesthetist to gauge the pulmonary compliance. If it is very difficult to inflate the lungs when squeezing the rebreathing bag, a pneumothorax may be present. If atelectasis has caused the low SpO_2, administration of a slightly larger tidal volume (15–20 ml/kg) for two to three breaths or held for 10–15 seconds may help to open the collapsed alveoli (this is also called a recruitment maneuver). If these actions do not improve oxygenation, other diagnoses, such as pulmonary thromboembolic disease, should be considered. If it is possible, confirmation of a low SpO_2 with an arterial blood gas is indicated because this will give an actual measure of PaO_2.

Table 50.3 Assessments and actions for patients with a low pulse oximeter reading (SpO2 < 95%) under anesthesia.

Problem origin	Assessment[a]	Action
Respiratory system	Has the patient experienced an adverse pulmonary event?	Auscultate the chest to rule out presence of pneumothorax, congestive heart failure, or other lung/pleural space disease. If pneumomediastinum is suspected, radiographs may be necessary for diagnosis. Thoracic ultrasound may identify pneumothorax, pleural effusion.
	Is the patient intubated properly? Verify endotracheal intubation using a laryngoscope. $ETCO_2$ will not read if esophageal intubation	Reintubate if necessary.
	Is the airway patent? Are there kinks or obstructions in the breathing circuit? Obstruction in the endotracheal tube?	Address as necessary, potentially switching machines, endotracheal tube, or breathing circuit.
	Is oxygen still being supplied to the circuit (check that the flowmeter bobbin is still floating)?	If oxygen supply has been depleted, use Ambu bag, or other device to breathe for the patient while a new source is established.
	Has unibronchial intubation occurred?	If the endotracheal tube has been inserted too far into the bronchial tree, slowly back it out until breath sounds can be auscultated bilaterally.
Circulatory system	Does the patient have a pulse?	Palpate pulse, or verify heart beat using stethoscope, esophageal stethoscope, or preplaced Doppler flow probe.
	Is the patient hypotensive?	Measure blood pressure and adjust anesthetic protocol to alleviate hypotension.
	Is the patient peripherally vasoconstricted?	If the vasoconstriction is related to administered drugs, consider alternative protocols or alternative placement of the pulse oximeter probe. If high doses of pressors are used, it may be difficult to achieve an accurate pulse oximeter reading.
Other patient factors	Is the patient hypothermic?	Vasoconstriction that may accompany hypothermia may result in a variable pulse oximeter reading; the patient should be aggressively rewarmed if the temperature is < 96°F.
	Is the tongue (or other probe site) dry?	Moistening the tongue with warm water or saline may allow the pulse oximeter to generate a more accurate reading.
	Is the patient moving/seizuring/shivering?	Calming the patient and limiting movement will allow the pulse oximeter to focus on the arterial pulse.
	Has the probe been placed on pigmented skin?	Attempt to locate a probe site that is not pigmented.
Environmental factors	Is the probe in direct line of the surgical or fluorescent lights?	Try to cover the probe site or redirect the lights to eliminate interference from room lighting.

[a] Factors are generally listed in order of importance, with emphasis on an immediate physical examination and patient assessment, and secondarily addressing possible machine malfunction.

The pulse oximeter is an indirect indicator of peripheral perfusion; when vasoconstriction results in decreased peripheral perfusion, the probe may be unable to sense a strong enough pulse, and the monitor may display errors, noise, or incorrect values. This may also happen in the context of severe hemorrhage; the compensatory vasoconstriction and decreased perfusion to the periphery will impair the pulse available for the pulse oximeter to monitor. It is imperative to always compare the pulse reported by the pulse oximeter with another reliable measure of the patient heart rate to ensure that the machine is focusing on the correct pulse. Whereas vasoconstriction will affect the pulse oximeter reading, anemia will not, unless it is very severe (PCV < 10%). In addition, fluorescent surgical or overhead lights may affect the ability of the pulse oximeter to register an accurate pulse. If the probe is positioned on the tongue, the signal may become attenuated as the tongue becomes dry from exposure during anesthesia. For a more in-depth discussion of possible artifacts and errors in pulse oximetry, refer to Chapter 25.

Plethysmographic Variability Index

The continuous indirect information that the pulse oximeter gives us about perfusion and specifically the peripheral arterial pulse may be used to the advantage of the anesthetist. The plethysmographic variability index (PVI) is a non-invasive way of predicting fluid responsiveness, which may be superior to CVP (see Chapter 15) in monitoring for volume responsiveness under anesthesia [6]. Using a pulse oximeter, a PVI value is calculated by monitoring changes in the amplitude of the SpO_2 waveform during the respiratory cycle. The perfusion index is a measurement of the pulse oximeter signal strength, and thus overall perfusion in the capillaries over which the pulse oximeter is placed. Generally, hypovolemic patients are more likely to have changes in cardiac output caused by increased intrathoracic pressure (i.e. positive pressure breaths), which causes a dampening of the perfusion index due to cyclic decreases in perfusion of peripheral tissues. The PVI calculation is:

$$PVI = \left(PI_{max} - PI_{min}\right) / PI_{max} \times 100$$

where PI_{min} is the lowest perfusion index and PI_{max} is the highest for a given respiratory cycle. Higher PVI is associated with more variability, and thus hypovolemia.

A similar concept can be used in evaluation of the direct arterial blood pressure tracing, where pulse pressure variation can indicate hypovolemia [7]. Although most accurate when used in patients receiving positive pressure ventilation (identical tidal volumes for each regular breath), PVI may also be useful in spontaneously breathing humans [8].

In clinical human use, PVI values above 20% suggest hypovolemia and predict positive fluid responsiveness. Not all animals, especially those with reduced cardiac function, are able to increase cardiac output via increasing preload (this depends on the patient's position on the Starling curve) [7], and will not show a decrease in PVI following fluid therapy. The patient's cardiac function and clinical exam should be considered when managing patients using PVI.

Body Temperature

The body temperature of an anesthetized patient affects heart rate, respiratory rate, blood pressure, and oxygen saturation. Patients should be kept as close to normothermic as possible during anesthesia. This not only helps maintain normal physiology during anesthesia but it also correlates to a decreased recovery time [9]. It is also easier to maintain a patient's temperature in the normal range than it is to warm the animal up after it has become severely hypothermic. Monitoring temperature is done using either a rectal thermometer or an esophageal temperature probe.

Steps that can be taken to minimize loss of body heat during surgery and anesthesia include the use of warm water circulating blankets, forced-air warming units (e.g. Bair Hugger, 3M Co, Minneapolis, MN), and the administration of warmed IV fluids. Some procedures make patient warming difficult. For prolonged radiologic procedures, such as myelography or computed tomography, forced-air warming units and water blankets can interfere with imaging. In magnetic resonance imaging units, both metal monitoring devices and metal probes are contraindicated due to interference with the magnet. In these cases, blankets placed over the patient can help to minimize heat loss but are not as effective as active warming devices. Whenever active warming devices of any kind are used, patient temperature *must* be closely monitored to ensure the patient is not overheated. If a patient is to undergo many procedures during which it is exposed to room temperature for long periods of time (e.g. wound debridement, central line placement), a radiant heat source may be used; these ceramic heaters warm the environment around the patient and limit radiant and convective heat loss. Because they are located over the patient, they are not suitable for use during surgery that involves an open body cavity. These devices usually are required to be placed a certain distance (usually about 90 cm) above the patient to prevent burns. Some (e.g. Radiant Heater, Fisher-Paykel, Auckland, NZ) are also equipped with a thermometer feedback system that adjusts the radiant temperature based on the skin temperature of the patient.

Hyperthermia may cause complications as well. When using active warming devices, the temperature setting should be lowered or use discontinued altogether when a patient is at or near normal temperature because the animal's rectal temperature may continue to rise after cessation of active warming. Should a patient become hyperthermic and the cause is determined to be other than malignant hyperthermia (by evaluation of $PaCO_2$ and blood lactate), cool the patient slowly. The forced-air warming unit can be switched to a cool setting, and the warm water blanket should be turned off. If the patient is already recovered from anesthesia, it may be placed on a cool surface like a metal cage floor, and all blankets should be removed. Continue to monitor temperature every 15 minutes until the patient's temperature has normalized. Opioids are associated with the development of postoperative hyperthermia in cats. This hyperthermia may be treated in a similar manner to other causes of hyperthermia, and it may also respond to low doses of naloxone (this therapy may also reverse the analgesia) but is usually self-limiting [10].

End-Tidal Inhalant Concentrations

The partial pressure (usually expressed as a percentage of alveolar gasses) of inhalant anesthetic circulating in a patient is essentially equivalent to the amount that is exhaled, once the anesthesia has reached steady state. By comparing the exhaled percentage of inhalant anesthetic with the dose required for anesthesia (usually a multiple of the minimum alveolar concentration, or MAC), objective data can be obtained as to the dosage of anesthesia provided by inhalant drugs at any given time. This is a similar concept to the measurement of plasma drug levels after administration of an injectable drug. The MAC values for the commonly used anesthetic agents are given in Table 50.4.

Machines that measure end-tidal inhalant concentrations (Figure 50.8a) are attached to the anesthetic circuit by tubing that will constantly aspirate small amounts (50–150 ml/minute) of gas from the system into the machine (Figure 50.8b). Once in the machine, the percentage of inhalant in the aspirated gas is determined and reported. Many different companies manufacture machines to determine end-tidal inhalant concentrations, and these monitors frequently measure $ETCO_2$ as well. Manufacturers include Datex-Ohmeda (GE Healthcare, Wauwatosa, WI), SurgiVet (Waukesha, WI), and Criticare Systems (Waukesha, WI).

Table 50.4 Minimum alveolar concentration values for veterinary species.

Species	Isoflurane (%)	Sevoflurane (%)	Desflurane (%)
Cat	1.7	3.1	10.3
Dog	1.3	2.1	7.2

The use of analgesic drugs such as the opioids and α-2 agonists decrease the amount of inhalant anesthetic necessary to maintain anesthesia. In this context, monitoring of the endotracheal inhalant can show the effects of premedicant drugs and analgesics given during anesthesia. If animals have been adequately premedicated and have intraoperative analgesia provided by other drugs, the overall requirement for inhalant anesthetic is decreased (i.e. the MAC is effectively lowered). Because of this, it is recommended that the endotracheal inhalant be used as a guide, but it is more important to rely on the patient's physical examination signs of anesthesia (discussed earlier), rather than trying to target an actual MAC value for each patient.

The inhalant concentration is directly related to the appearance of adverse effects from inhalant anesthetics. As the inspired concentration of inhalant increases, hypotension and hypoventilation become more pronounced. From this perspective, the endotracheal inhalant concentration can help avoid overdoses of inhalant anesthetic and at the same time predict that adequate anesthesia is present. In critically ill patients, the adverse effects of inhalant anesthetics may be magnified, and sometimes only a fraction of the MAC dose (if any at all) may be used safely. In addition, patients with diseases such as septic shock, severe hypovolemia, and severe hypothermia have lower MAC values than similar healthy patients. The endotracheal inhalant can help to document the overall dose of anesthetic as well as any residual anesthetic in the patient if administration is discontinued.

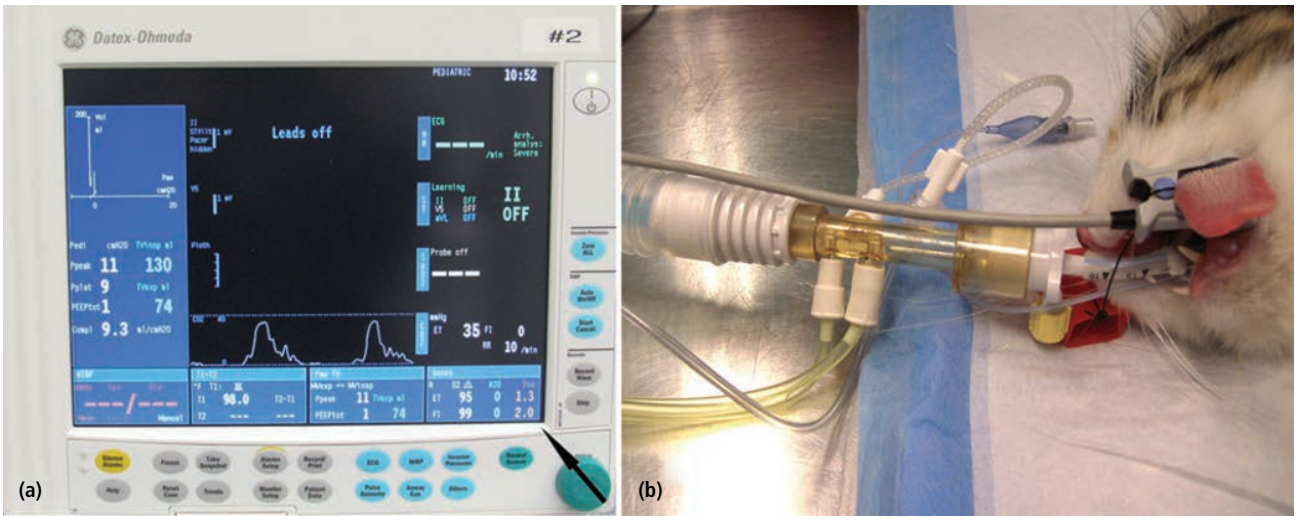


Figure 50.8 (a) Multiparameter patient monitor that has the capabilities of displaying ECG, SPO_2, end-tidal CO_2, and anesthetic inhalant concentrations. The percentage of inhaled anesthetic is displayed (black arrow). (b) The adapter placed in the anesthetic circuit that continually aspirates small amounts of gas from the system. Once in the machine, the percentage of inhalant in the aspirated gas is determined and reported.

Anesthesia in the critically ill patient can be challenging due to the sensitive nature of the cardiorespiratory systems and the possibility for decompensation. With appropriate monitoring equipment and regular attention paid to physical examination parameters, changes may be recognized early, and interventions may be subsequently taken to restore homeostasis.

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51

Nursing Care of the Long-Term Anesthetized Patient

Yekaterina Buriko and Bridget M. Lyons

Basic Indications for Long-Term Anesthesia

A small number of veterinary patients require long-term anesthesia during their hospitalization. The three major groups of patients in this category are patients that require positive pressure ventilation due an to inability to oxygenate or ventilate appropriately, endotracheal intubation for large airway disease (e.g. tracheal collapse), and those that require general anesthesia for control of refractory seizures. Typical indications for positive pressure ventilation include partial pressure of arterial oxygen (PaO_2) less than 60 mmHg with oxygen supplementation, hypoventilation with partial pressure of arterial carbon dioxide ($PaCO_2$) higher than 60 mmHg, and an unacceptable degree of dyspnea associated with increased work of breathing.

Long-term anesthesia and immobility are associated with a number of complications, affecting nearly all body systems of a patient. Recognizing these effects is of particular importance to a veterinary technician because much of requisite nursing care is focused on preventing and addressing the complications particular to the anesthetized patient. This chapter focuses on the specifics of management of this patient population and describes the complications associated with the key body systems, methods to prevent the complications, and treatment modalities, should a problem arise.

All anesthetized patients require an endotracheal or a tracheostomy tube, as well as indwelling intravenous access as a part of their care. Please refer to Chapters 31 and 63, respectively, for details on artificial airway management and care of indwelling devices.

Physical Complications Associated with Long-Term Anesthesia and Immobility

Immobility Complications

An inevitable consequence of long-term anesthesia is an extended period of immobility. Prolonged recumbency can result in a plethora of complications, including decubitus ulcers, tissue necrosis, peripheral limb edema, contracture and stiffening of muscles and ligaments, muscle atrophy, nerve damage, atelectasis, and the accumulation of airway secretions in dependent lung regions.

Neuromuscular Weakness

Neuromuscular weakness is a condition that is recognized in critically ill human patients. Depending on the method of diagnosis, it occurs in 25–100% of human patients in intensive care [1]. During the first week of immobilization, up to 40% of muscle strength may be lost [1]. The pathophysiology of critical care neuromuscular dysfunction is complex and likely multifactorial. Several factors contribute, such as disuse due to immobility, microvascular dysfunction, and a catabolic state that can be caused by malnutrition, systemic inflammation, and metabolic derangements [1–3]. Prolonged immobility is associated with a proinflammatory state that increases the production of reactive oxygen species, together with simultaneous decrease in antioxidant defense mechanisms [1, 3]. This may result in contractile dysfunction and atrophy, as well as protein loss [1, 3]. In mechanically ventilated patients, the neurologic and muscular dysfunction may increase the duration of ventilation, extend the length of stay in the

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hospital, prolong recovery time, and contribute to mortality [1–3]. Although this condition has not been formally recognized in dogs and cats, it is likely that a similar disorder exists in veterinary patients as well.

Atelectasis

Atelectasis is a major complication of both prolonged immobility and anesthesia [2]. Atelectasis refers to the collapse of the small airways and alveoli, resulting in portions of lung that are perfused but not ventilated. This may occur secondary to mechanical compression of the lung from the weight of the nondependent lung, airway obstruction or collapse, loss of surfactant, and improperly positioned endotracheal tubes. Atelectasis, when severe enough, may cause hypoxemia by causing intrapulmonary shunt [2]. If the patient is mechanically ventilated, atelectasis may increase the possibility of ventilator-associated lung injury because of both overdistention of aerated lung and shear injury.

Venous Stasis

Immobility promotes venous stasis, which may lead to many deleterious effects [2]. Human patients who are immobile are at increased risk of venous thrombosis, which, in turn, increases their risk of pulmonary thromboembolism [2, 4]. In addition, direct compression of the venous vasculature may occur from prolonged contact of extremities with bedding, which may contribute to stasis, as well as result in vascular endothelial damage [2]. Vascular compression, in turn, may result in diminished blood supply to the skin and puts immobilized patients at risk of impairment in skin integrity [5]. Skin damage and ulceration are important consequences of prolonged immobility, and they can cause significant morbidity. The etiology of skin damage is multifactorial and, in addition to impaired blood supply, includes malnutrition, as well as direct pressure to points of contact during prolonged immobility [2, 6]. Skin ulceration may create severe soft-tissue damage, cause osteomyelitis of adjacent bones, and may result in systemic infection and sepsis [2].

Ocular Complications

Patients that are anesthetized lose the ability to protect their eyes and have a significantly higher incidence of ocular surface disorders, such as exposure keratopathy and corneal ulceration [7–9]. Peak incidence occurs during the first two to seven days of hospitalization in humans [10]. Decreased tear production has been documented in dogs undergoing anesthesia, and one veterinary study found that 5% of mechanically ventilated dogs develop corneal ulcers [11–13].

Decreased tear production and loss of blink function during long-term anesthesia leave the eye vulnerable. Tears are essential for ocular health and have many functions, including lubrication of the ocular surface, provision of oxygen to the cornea, and prevention of bacterial colonization [7, 14]. Blinking spreads lacrimal secretions over the ocular surface, moistening the eye [8]. The evaporation of these tears changes the temperature of the surface, making it unfavorable for bacterial growth [8]. A loss of blink also results in incomplete closure of the eye and thus prolonged exposure of the cornea and conjunctiva to the environment. When the ocular surface is dry, small corneal defects develop that can lead to exposure keratopathy [8].

Complications Associated with the Oral Cavity

An anesthetized patient has decreased saliva production and loses the ability to swallow, clean, and protect oral structures from mechanical damage. Healthy animals rely on antimicrobial peptides such as lysozymes, hydrogen peroxide, mucins, cathelicidins, and lactoferrin in their saliva and on dental surfaces to help protect the oropharynx from colonization by pathogenic bacteria [15]. Critically ill patients with decreased saliva production are consequently predisposed to bacterial and fungal overgrowth in the oral cavity. Additionally, these patients cannot swallow appropriately and lack oral cavity hygiene, further predisposing them to colonization with Gram-negative bacteria and the formation of biofilm on the tooth surface [15–17]. The change in oral flora from Gram-positive to Gram-negative occurs in mechanically ventilated human patients within 48–72 hours of admission to intensive care [18]. These bacteria are frequently the causative agents of ventilator-associated pneumonia (VAP). Migration of bacteria from the oropharynx to the respiratory tract, as well as microaspiration of the contents of the oropharynx are leading causes of VAP [17, 19]. VAP increases both morbidity and the length of intensive care required, as well as the cost of hospitalization [20].

Ranulas and oral ulcers are potential complications of long-term sedation and intubation (Figure 51.1). Ranula refers to the accumulation of salivary gland contents, such as mucin, in the soft tissues surrounding the gland, because of trauma or obstruction to the gland. Oral ulcers are often caused by mechanical injury to the structures of the mouth; ulcers have been documented in 8% of dogs and cats undergoing mechanical ventilation [11, 18]. Persistent pressure from pulse oximeter probes, mouth gags, the endotracheal tube and its tie applied to the tongue and other soft-tissue structures leads to ulceration [18].

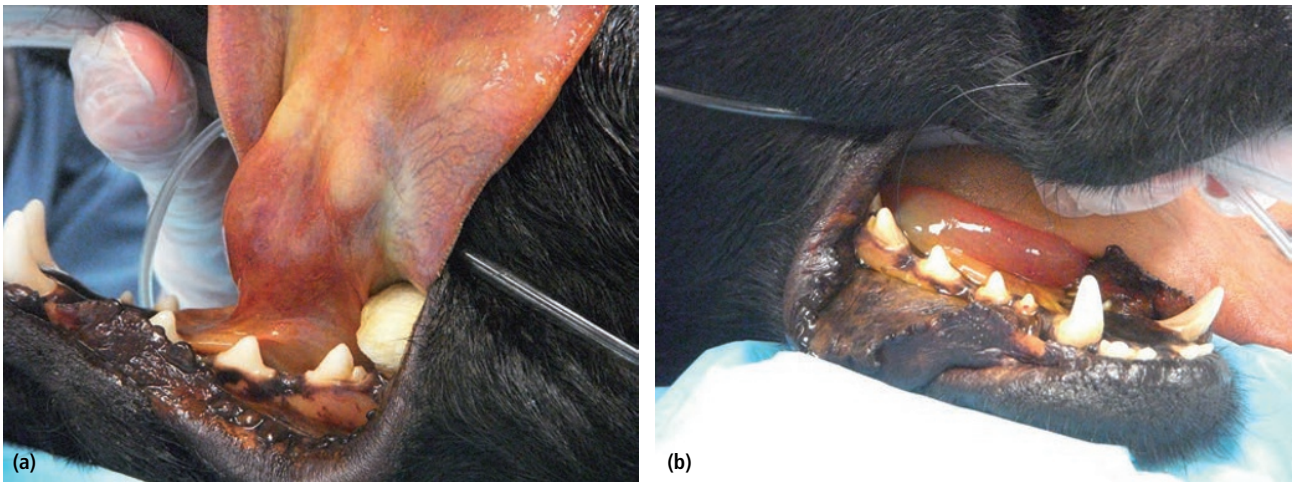


Figure 51.1 Ranulas may form during general anesthesia as a result of trauma to the salivary glands. This may be secondary to equipment in the oral cavity or manipulation of the tongue and the mandible.

Complications Associated with the Urogenital Tract

Anesthetized patients typically do not voluntarily void their bladders on a regular basis. Moreover, if patients under anesthesia urinate, they do not always empty their urinary bladder completely. Urine retention increases the risk of urinary tract infection (UTI) and detrusor atony [21]. Additionally, urine overflow secondary to incomplete emptying may result in soaking of the skin and fur with urine. Urine is irritating to the skin and can cause significant inflammation and scald, if not removed promptly and completely, which can be difficult to accomplish in larger patients.

Gastrointestinal Complications

Gastrointestinal (GI) complications of prolonged anesthesia can be significant, especially during mechanical ventilation. They include splanchnic hypoperfusion and vasoconstriction, as well as diminished GI motility. Patients undergoing mechanical ventilation, particularly those with high levels of positive end-expiratory pressure, can experience poor venous return, which can lead to decreased cardiac output and poor splanchnic perfusion [22]. Suboptimal splanchnic perfusion impairs GI tract and pancreatic function. Mechanical ventilation has also been associated with increased levels of endogenous catecholamines, which can result in splanchnic vasoconstriction and ischemia, making this population of patients at increased risk of GI ulceration and bleeding [22]. Moreover, sedatives, including narcotic agents, further diminish GI motility and contribute to ileus [2, 22]. Gut barrier function may be impeded by hypoperfusion, malnutrition, and systemic inflammation. The break in normal gut barrier may result in intestinal bacterial translocation [22].

Recumbent Patient Care

The long-term anesthetized patient requires recumbent patient care (Protocol 51.1). Three components contribute to recumbent patient care: proper patient positioning, decubital ulcer prevention and management, and early mobility.

Protocol 51.1 Recumbent Patient Care

Procedure

To be performed every four hours:

1) Perform hand hygiene.
2) Ensure that the bedding is clean, dry, and appropriate for the patient; elevate the head slightly compared with the rest of the body.
3) Perform passive range of motion exercises and massage on the nondependent limbs.
4) If the patient is in sternal recumbency, turn hips and reposition the rest of the body slightly; if in lateral recumbency, turn the patient onto the other side.
5) Check for potential sites of ulceration (e.g. bony protuberances).
6) If pressure ulcers exist, inspect all dressings to ensure they are clean and cover the wound appropriately.[a]
7) Perform passive range of motion exercises and massage on the nondependent (previously dependent) limbs.

[a] The pressure ulcer dressing may be changed once to twice daily, depending on the extent of the wound and the amount of exudate produced. Every time the dressing is changed, the size, depth, and extent should be evaluated and recorded.

Patient Positioning

Patients undergoing long-term anesthesia should be provided with clean, dry, and well-padded bedding [14, 19]. All eliminations should be cleaned immediately to maintain dry skin at all times; use of absorbent pads may assist with nursing care. Any bony protuberances should be carefully padded [23]. The extremities should be completely supported at all times and should have contact with a padded surface. A turning schedule should be established early in the patient's care [24]. It is recommended that the animal be turned every two to four hours [14, 19, 25]. Potential sites for ulcer development should be checked every time a patient is turned [23]. Regular turning is also a key component to the prevention of atelectasis [19]. Compression of dependent lung leads to airway collapse and subsequent perfusion of alveoli that are not ventilated, creating decreased gas exchange and contributing to hypoxemia. Turning the patient shifts the pressure and allows the airways to remain open.

Animals that are anesthetized for long periods of time are most commonly those being mechanically ventilated. In many patients, the need for mechanical ventilation is due to lung pathology and severe hypoxemia. Patients with pulmonary parenchymal disease may not be able to tolerate certain positions, such as lateral recumbency, for prolonged periods of time. Positioning and turning in these patients may be complicated by their disease process. In dogs, there is evidence to support sternal positioning as the optimal position for best oxygenation [26]. If an animal is in sternal recumbency, the hips should be turned every two to four hours and the position of the rest of the body slightly altered to shift pressure points. Oxygenation should be closely monitored after a turn because decompensation may occur during position shifts. Preoxygenating the patient with higher fraction of inspired oxygen prior to manipulation is advisable, in case decompensation occurs.

Decubitus Ulcer Prevention and Management

One of the primary goals of nursing a recumbent patient is the prevention of decubitus ulcers. Decubitus ulcers occur over bony prominences and are the result of soft tissue compression between the bone and a hard surface, impaired circulation, malnutrition, and humidity [2]. In dogs laying in lateral recumbency, the skin over the scapulohumeral joint, greater trochanter, and 13th rib are at greatest risk, although any bony protuberance may be prone to ulceration [27]. Animals that are naturally thin or have a poor body condition are at greater risk for ulcer development [27]. However, any animal that is recumbent for an extended period should be considered at risk. In a small canine cadaver study, memory foam mats appeared to reduce pressure compared with sheets or a polyester mattress in dogs placed in lateral recumbency [27]. If available, memory foam mats could be considered in patients undergoing long-term anesthesia to potentially reduce risk of pressure ulcer formation; however, a nonporous cover is recommended if used with multiple patients to reduce the risk of infection transmission. A number of specially designed mattress and support surfaces have been developed in human medicine to aid with recumbency care, and these surfaces may become available to veterinary practitioners in the near future. Alternating pressure mattresses and special soft surfaces that spread the pressure over a larger area may help to decrease formation of decubitus ulcers.

Should a decubitus ulcer develop, it is important that an effort be made to reduce pressure on the site. Ring devices placed around the wound are contraindicated because they contribute to venous congestion and local edema, which may prolong healing [24]. The ulcer should be cleaned with a pH-neutral, nonirritating, nontoxic solution, such as saline, and any necrotic tissue debrided [24]. Unless bacterial infection is suspected, chlorhexidine and other antimicrobial solutions should be avoided [24]. These agents are toxic to granulomatous tissue and may delay wound healing [24]. Dressings that provide a moist wound environment, keep the skin around the wound dry, control exudate, and eliminate dead space should be chosen [28]. Some examples include moist gauze, hydrocolloid, or calcium alginate dressings [28]. Wet-to-dry dressings are not recommended because they are not continuously moist and therefore not appropriate for wound dressing [24]. Negative pressure therapy could be considered for select types of wounds that are deep, have a large surface area, or are infected. The wound should be reassessed with each dressing change to determine whether amendments are needed in the treatment plan as the wound heals or deteriorates. The size, depth, whether debridement is required, and amount of exudate produced by all ulcers should be documented in the medical record each time the wound is evaluated [24]. A pressure ulcer grading system could be implemented to allow for a more objective evaluation of the degree and severity of ulceration when multiple people care for the animal [29]. Adequate nutrient intake and hydration status should be ensured to maximize the potential for wound healing [30].

Early Mobility

A number of serious complications arise from prolonged immobility, such as muscle and ligament contracture, muscle disuse atrophy, and limb edema due to venous stasis. The importance of early mobilization and physical exercise has been highlighted in humans, where trends toward lower

mortality, decreased duration of mechanical ventilation, and decreased days in intensive care have been documented if early mobility is engaged [3]. Feasibility of early mobilization in veterinary patients is of concern, especially in those that are endotracheally intubated and cannot voluntarily move on their own. In these patients, a physical exercise regimen should be established and instituted early in the hospitalization, as there is evidence that neuromuscular dysfunction happens rapidly after onset of mechanical ventilation and recumbency [31]. If a physical therapy service is available, collaboration with the service may be useful to establish a tailored protocol for the particular patient.

At minimum, the physical exercise regimen should include passive range of motion exercises and massage. These exercises should be performed every 4 hours for 10–15 minutes and should ideally involve every joint of the extremities and all the possible dimensions of movement for the particular joint [32, 33]. The joints may be moved separately or concurrently in a motion resembling walking or running [33]. Stretching may also be used. Body massage is useful and may include gentle to deeper stroking, kneading, or skin rolling parallel to the muscle bellies [32, 33]. There is evidence to suggest that even simple passive exercises are beneficial in preserving muscle mass; therefore, they should not be overlooked [34]. It is evident, however, that exercises that actively engage the muscles are required for better return to function [33]. In a select group of patients, active range of motion exercises may be used, if the pet's condition permits. These patients may include animals that are sedated, but not anesthetized, such as those mechanically ventilated via a tracheostomy tube. A wide variety of exercises, from resisted withdrawal to sitting and standing exercises, may be performed, depending on the patient's status [32, 33]. In addition, other modalities, such as electrical stimulation, cryotherapy, ultrasound, and heat therapy, may be used, if available [33].

Early and adequate nutrition (Chapter 42) is paramount in mitigating the effects of prolonged immobility, weakness, and muscle loss. Many patients that undergo prolonged anesthesia suffer from a catabolic state and specific disease processes that may require careful evaluation of their nutritional needs. A nutrition plan should be devised early in the course of hospitalization and evaluated regularly to accommodate patient needs.

Eye Care

The anesthetized patient is unable to produce and spread tear film adequately; thus, protecting the patient's eyes is an essential aspect of nursing care. Eye care (Protocol 51.2) should occur every two hours [10, 14, 19, 25]. Every two hours, the eyes should be cleaned with saline-soaked gauze, and artificial tear ointment should be placed in the eyes [10, 19, 25]. The ointment may be alternated with artificial tear drops [1]. Use of a dedicated ointment tube for each eye is recommended, particularly in patients with suspected ocular infections [8]. In humans, use of moisture chambers have proven significantly more effective for the prevention of corneal ulceration compared with lubricating ointments [9]. The varied skull anatomy of dogs and cats makes getting a good seal using goggles or covers difficult; however, if a patient under long-term anesthesia has a skull conformation amenable to moisture chamber use, these may be used to reduce corneal exposure.

Protocol 51.2 Eye Care

Procedure

To be performed every two hours:

1) Perform hand hygiene.
2) Clean periocular surfaces with saline-soaked gauze.
3) Check eyes for chemosis or inflammation.
4) Ensure that the endotracheal tube tie and any neck wraps are not tight.
5) Place artificial tear ointment in each eye, ideally using a separate designated tube of ointment for each eye. The ointment may be alternated with artificial tear drops.

To be performed every 24 hours:

1) Perform hand hygiene.
2) Fluorescein stain each eye to check for corneal ulceration.
3) If corneal ulcers are visualized, note their location, size, and depth in the progressives section of the medical record.

Aerosolization of bacteria occurs during suctioning of the airway and oral cavity [14, 35]. Care should be taken not to withdraw the suction catheter near or over the eye, as contamination of the eye with the same pathogen causing pneumonia in mechanically ventilated patients has been documented [8, 35].

Fluorescein staining of the corneas should be performed once daily to check for ulceration [10, 14, 19, 25]. If detected, a broad-spectrum antibiotic ointment can be used to prevent corneal infection [14, 19, 36]. Topical antibiotics containing polymyxin B, neomycin, bacitracin, or oxytetracycline should not be used in cats due to the risk of anaphylaxis [37]. If the ulceration does not resolve or improve within 48–72 hours, or if there is evidence of progression, such as increased area of stain uptake or depth of the defect, cytology, as well as culture and sensitivity testing, are indicated to determine the infectious agent [36].

Severe infection may require the hourly application of an antimicrobial agent [36]. Topical atropine is also indicated in the treatment of deep corneal ulcers [36]. Atropine provides analgesia and prevents synechiae formation and barrier [36]. All corneal ulcers and their progression should be documented daily by noting the location, size, and depth of the ulcer in the record.

Chemosis may occur in any patient that is immobilized for prolonged periods of time, and it may be in part responsible for incomplete eye closure and undesired exposure of the cornea and conjunctiva. Decreased drainage, increased vascular permeability, and increased venous pressure contribute to this condition [8]. Incidence is increased in patients who are maintained at a positive end-expiratory pressure greater than 5 cm H_2O [35]. Periorbital edema and chemosis may also be the result of an overly tight endotracheal tube tie, if tied behind the ears, or snug neck wraps covering central catheters or feeding tubes [35]. Precautions should be taken to avoid edema formation, and steps to resolution of edema should be taken once observed. These include carefully monitoring the patient's fluid balance, frequently checking for tightness of the endotracheal tube tie, and positioning the patient's head in such a way as to optimize venous return (head slightly higher than the rest of the body, taking care not to occlude the jugular veins).

Oral Care

The importance of oral care (Protocol 51.3) in the nursing regimen of patients anesthetized for extended periods of time cannot be stressed enough. Oral care has been documented to decrease the incidence of VAP and is thus of crucial importance in the management of patients undergoing long-term anesthesia [17, 20, 38, 39].

Despite a cuffed endotracheal tube, microaspiration of oral secretions may occur [20]. For this reason, any fluid that pools in the mouth or pharynx should be suctioned every four hours [11, 16, 19, 25]. The oral cavity may also be rinsed by instilling small amounts (5–20 ml, depending on size of the patient) of sterile saline with a syringe prior to suctioning [14, 19]. Care has to be taken to make sure the endotracheal tube is appropriately inflated during rinsing. The oral cavity should also be suctioned prior to adjustment of the endotracheal or tracheostomy tube, reintubation, or a patient position change [39]. It has also been shown that continuous subglottic suctioning, as well as intermittent, regular subglottic suctioning, leads to a reduction in the incidence of VAP in humans [20, 25, 39]. New-generation endotracheal and tracheostomy tubes, some equipped with subglottic suctioning lines, are available and could be incorporated into care of veterinary patients. No evidence exists for their use in veterinary medicine.

Protocol 51.3 Oral Care

Procedure

To be performed every four hours:

1) Perform hand hygiene and don examination gloves.
2) Remove any pulse oximeter probes, mouth gags, or gauze.
3) Inspect oral cavity for pooling oral secretions, ranulas, and ulcers.
4) Note the approximate size and location of any ulcers or ranulas in the medical record.
5) Check the endotracheal cuff for appropriate inflation.
6) Gently suction the mouth and oropharynx.[a]
7) Adjust the position of the endotracheal tube tie if needed.
8) Wipe the mucous membranes and tongue with gauze soaked with 0.05% chlorhexidine solution; flush the back of the oral cavity with 0.05% chlorhexidine solution; thoroughly suctioning afterward.
9) Reposition the tongue, avoiding draping the tongue over the teeth, and wrap in dilute glycerin-soaked gauze.
10) Replace mouth gag and pad teeth with dilute glycerin-soaked gauze.
11) Replace pulse oximeter probe, changing the position of the probe.

[a] Patients with copious oral secretions and/or persistent regurgitation may require hourly suctioning and/or the placement of an orogastric or nasogastric tube.

The oral cavity should be decontaminated with a dilute chlorhexidine solution or gel (0.05%) every four hours, concentrating on any oral ulceration that may be present [14, 16, 18, 19]. Oral ulcers provide a route for bacteria to enter the systemic circulation, and early and thorough treatment is essential. Chlorhexidine is bacteriostatic and bactericidal and has been shown to promote gingival healing [18]. Chlorhexidine use should be instituted regardless of the presence of ulceration, as a method of selective oral decontamination. Chlorhexidine has been shown to reduce the incidence of VAP in human patients undergoing mechanical ventilation when compared to saline, and there is stronger evidence for its use compared with other oral solutions [17].

Although an inflated endotracheal tube cuff may not prevent aspiration, an effort should be made to protect the airway as much as possible by checking that the cuff is appropriately inflated. A Posey Cufflator® (J. T. Posey Company, Arcadia, CA) may be used to ensure that the correct pressure is achieved (Figure 51.2). This device attaches to the endotracheal tube cuff, which allows one to



Figure 51.2 A Posey Cufflator® (J. T. Posey Company, Arcadia, CA) may be valuable in ensuring appropriate pressure of the endotracheal or tracheostomy tube cuff.



Figure 51.3 Endotracheal tube tie made out of nonporous material, such as intravenous tubing, reduces the possibility of bacterial contamination.

measure the pressure in the cuff while it is being inflated by the bulb of the device. The recommended intracuff pressure is 20–30 cm H_2O. Alternatively, administering a breath while listening for a leak as the cuff is being inflated by an air syringe can be performed. This ensures that the least amount of air to inflate the cuff sufficiently is instilled, minimizing cuff overinflation. The presence of an air leak may also be observed on the displayed ventilator scalars and loops. If the cuff pressure can be monitored, it should be checked every four hours. There is no evidence that repositioning of the endotracheal tube is necessary if the cuff pressure is optimized. In fact, some evidence exists in human patients that repositioning of the tube during mechanical ventilation may potentiate complications such as pneumonia and tracheal and airway damage [40–42]. No information is available on the benefits and adverse effects of tube repositioning in veterinary medicine. It is currently accepted that if the intracuff pressure cannot be measured, the endotracheal tube cuff should be deflated and the tube's position adjusted every four hours to prevent damage to the trachea [19]. When securing the tube, a tie made of a nonporous material is ideal [19]. Cloth that is moistened by oral secretions provides an excellent medium for bacterial growth. As an alternative to muzzle gauze, a tie may be fashioned from IV tubing (Figure 51.3) [19].

Monitoring equipment, such as the pulse oximeter probe, and the endotracheal tube and its tie should be moved every four hours [11, 14, 18, 19]. A cushion made of saline- or glycerin-moistened gauze should be placed between the tie and the soft tissues it comes into contact with [11, 18, 19]. If possible, placement of the tongue directly over the teeth should be avoided [11, 18, 19]. If this is not possible, the teeth should be packed with



Figure 51.4 Packing of the oral cavity with saline- or dilute glycerin-moistened gauze diminishes desiccation of the mucous membranes and minimizes soft tissue damage that occurs secondary to the equipment, such as endotracheal tube and pulse oximetry monitor.

saline- or glycerin-moistened gauze to reduce trauma (Figure 51.4) [11]. This is especially important in pediatric patients because the primary teeth are sharper than adult teeth [11]. The tongue and other soft tissue structures should be kept moist with gauze soaked in dilute glycerin [11, 14, 18, 19]. Any pressure that may have contributed to a lesion should be relieved (Figure 51.5).

Persistent regurgitation may further complicate oral care. Aspiration of gastric contents is a cause of VAP [11]. Additionally, regurgitation of gastric contents decreases the pH of the mouth and increases the severity of oral ulcers [18]. A nasogastric or orogastric tube may be passed to relieve the stomach of its contents and prevent further regurgitation.



Figure 51.5 A part of a 1-cc syringe may be used as a mouth gag to diminish pressure of the teeth on the tongue.

Bladder Care

The urinary bladder should be palpated every four to six hours and expressed as needed. Urinary catheterization should be considered if effective expression cannot be achieved due to patient's size or bladder dysfunction, or if measurement of exact urinary output is important.

Urinary bladder catheterization may be intermittent or indwelling. The decision as to which method is used depends on the needs of each individual patient. UTI is a risk factor for urinary bladder catheterization, regardless of method [43]. Duration of catheterization in dogs has been shown to be a more important factor for development of a UTI than the method of catheterization [43]. Bladder colonization may occur via intraluminal or extraluminal bacterial migration, introduction during catheter placement, or secondary to bacteremia [44]. It is important to note that the presence of bacteriuria does not necessarily indicate infection [44].

Regardless of the method of catheterization, aseptic technique during catheter placement cannot be overemphasized. Hair should be clipped around the catheter insertion site, and the area should be disinfected with chlorhexidine scrub and rinsed with sterile saline. Sterile gloves and lubricant should be used. A 0.05% chlorhexidine solution is used to flush the vestibule or prepuce prior to the catheter insertion. In females, catheterization is achieved by blind palpation of the urethral papilla or by direct visualization of the urethra using a speculum and a light source. If a Foley catheter is used, the balloon is inflated after the catheter is advanced into the bladder. Afterward, the catheter is gently pulled back until resistance is met. If a different type of indwelling catheter is used, the catheter is sutured in place after measurement has been taken to ensure correct placement. Although antibiotic-coated urinary catheters have been shown to reduce biofilm formation, their use is not currently recommended as there is inadequate evidence to support their use for prevention of catheter-associated bacterial cystitis [45, 46]. If the urinary catheter is indwelling, a closed collection system should be used and the collection bag kept below the level of the patient. The catheter and collection system should be inspected every at least eight hours to ensure there is no gross contamination, and the exposed portion of the catheter and vulvar or preputial area leaned with 0.05% chlorhexidine solution. See Chapter 35 for more details on placement and maintenance of urinary catheters.

Gastrointestinal Tract

Patients undergoing long-term anesthesia should be evaluated frequently for abdominal distension, presence of bowel sounds, and frequency of bowel movements [2]. Bowel sounds may be auscultated over the four quadrants of the abdomen with a stethoscope. Frequency varies quite a bit between patients and the relation to a meal, but in general, bowel sounds should be auscultated approximately four to five times a minute. If a gastric feeding tube is in place, the volume and characteristics of gastric aspirates should be noted in the record. Frequency of gastric tube aspiration varies among patients but typically occurs every four to six hours. Bedside ultrasound may be used to evaluate the presence of fluid in the stomach and intestines, as well as to evaluate gastrointestinal motility by counting the number of contractions of the pyloric antrum and the small intestines per minute. Normal values have been documented in dogs but are subject to a number of variables, such as recent water or food intake, composition of ingested material, as well as many other factors [47]. Prokinetic agents may be considered if significant gastrointestinal dysmotility is highly suspected or is present. Human patients undergoing mechanical ventilation are at high risk for development of gastrointestinal ulceration, and they are routinely administered medications aimed at reducing production of gastric acid such as histamine blockers and proton pump inhibitors [48]. Whereas the same information is not available in the veterinary population, it is reasonable to believe that the veterinary patients undergoing mechanical ventilation may be at risk for gastrointestinal ulceration and therefore would benefit from ulcer prophylaxis.

Both diarrhea and constipation may be observed in the patient population under general anesthesia. Care should be taken to keep the anal and perineal area clean and dry; removal of hair may facilitate this, and may also allow monitoring of the skin for irritation. Careful abdominal palpation should be performed daily to assess for constipation. Enemas may be required to facilitate gastrointestinal tract emptying [14].

Techniques to Decrease Stimulation

Patients undergoing long-term anesthesia are frequently intubated for the duration of the event; however, some may have a tracheostomy tube placed to minimize the amount of sedative and anesthetic agents used, as well as to avoid complications associated with orotracheal intubation, such as oral ulcerations or laryngeal edema. Drugs used to maintain heavy sedation are titrated to the lowest level that is required for immobilization and patient comfort. Occasionally, patients may be rousable and responsive to stimuli. Therefore, several techniques may be used to minimize stimulation. Treatments should be grouped together in such a way that the patient is stimulated as infrequently as possible. Additional sedation may be used during periods of stimulation, which may include anesthetics, opioids, anxiolytics, or tranquilizers. However, whenever possible, the need for sedation should be critically evaluated, as heavy sedation furthers immobility, compounds neuromuscular weakness, and contributes to delirium, making it more challenging to rehabilitate these patients. Cotton balls may be placed in the external ear canals to minimize auditory stimuli. This should be noted in the record, so that the cotton can be removed when it is not needed. Ambient noise should be kept as minimal as possible. When not absolutely necessary, lights should be turned down to minimize visual stimulation. When that is not possible, placing a towel over the eyes may aid in reducing stimuli.

Summary

General anesthesia and long-term immobilization may be necessary in a subset of critically ill patients. A number of factors may negatively impact these animals, and vigilant and well-structured nursing care program is paramount in preventing and addressing commonly encountered complications for optimal patient care.

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52

Neuromuscular Blockade

Manuel Martin-Flores and Karen L. Basher

Introduction

Neuromuscular blocking agents (NMBAs), also called muscle relaxants, are used in combination with anesthetic agents as part of balanced anesthetic techniques. They differ from all other agents used in anesthesia in that they do not contribute to hypnosis or analgesia; rather, these agents produce skeletal muscular relaxation that can range from mild weakness to complete flaccid paralysis, in a dose-dependent manner. Paralysis is produced by interrupting neuromuscular transmission. Both pre- and post-synaptic effects are exerted in the neuromuscular junction, resulting in both the reduced availability and release of acetylcholine (ACh), and the competitive antagonism of nicotinic ACh receptors (nAChR) in the post-synaptic cell. As a consequence, when blockade is complete, impulses arriving to the neuromuscular junction through a motor nerve do not trigger a skeletal muscular contraction, and paralysis ensues. Because it is the neuromuscular transmission that is interrupted, monitoring is focused on evaluating this transmission. To this purpose, a motor nerve must be stimulated, and the muscular response assessed.

Indications

While the effects of NMBAs are restricted to skeletal muscle relaxation, there are clinical advantages to their use. Muscle relaxation and immobility can also be achieved with general anesthetics, but the doses required for immobilization are significantly higher than those necessary for unconsciousness [1–3]. Such an increase in the dose of general anesthetics is often accompanied with hemodynamic instability, an undesirable occurrence in ill patients. Hence, the addition of NMBAs allows to reduce the doses of general anesthetic so that hypnosis is ensured by anesthetics and muscle relaxation is provided by the neuromuscular blocker.

Aside from balanced anesthesia, NMBAs are particularly useful to prevent or treat ventilator–patient asynchrony. Asynchrony results in ineffective alveolar ventilation and increases the risks of reaching inadequately high peak airway pressures. Ventilated patients, especially during the initial ventilator setup, may have a high respiratory drive, resulting in hypercapnia, hypoxemia, or both. In this scenario, and providing the depth of anesthesia is judged to be adequate, the inclusion of a NMBA provides an efficient means for treating asynchrony. Modern NMBAs are usually devoid of substantial cardiovascular effects, making this therapy clinically useful for ill animals. A summary of different NMBAs and commonly used doses is shown in Table 52.1.

Reversal of Neuromuscular Block

The effects of NMBAs dissipate gradually as the agent is redistributed and metabolized. As the plasma levels decrease, the agents move away from the neuromuscular junction and function is restored gradually. This process can be enhanced or abbreviated by the use of anticholinesterase drugs, also called acetylcholinesterase (AChE) inhibitors, such as edrophonium or neostigmine. AChE inhibitors are not direct antagonists to NMBAs, and hence, cannot directly reverse their effects. Rather, these agents inhibit the activity of the acetylcholinesterase enzyme, resulting in increased levels of acetylcholine (ACh) at the neuromuscular junction. It is the ACh that will ultimately compete with the NMBAs for the nicotinic acetylcholine receptors (AChRs).

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Table 52.1 Different neuromuscular blocking agents and commonly used doses.

Family	Agent	Dose (mg/kg)	Duration (minutes)	Comments
Benzylisoquinolines	Atracurium	0.2–0.5	20–30	Extrahepatic metabolism resulting in predictable duration under various conditions. Atracurium has a tendency to release histamine when administered at high doses.
	Cisatracurium	0.05–0.2	20–40	
Aminosteroid	Rocuronium	0.3–0.6	20–30	Hepatic and renal biotransformation. No histamine release but might have a tendency for vagolytic effects.
	Vecuronium	0.05–0.1	20–40	
	Pancuronium	0.05–0.1	40–60	

This process carries several important clinical implications: first, the release and accumulation of ACh requires time; hence, reversal of the block is not instantaneous. Studies in anesthetized dogs using neostigmine show that complete reversal may require up to 10 ± 2 minutes, depending on the dose of neostigmine used [4]. Second, there is a limit to the amount of ACh that can be released from the presynaptic neuron; once complete AChE enzyme inhibition is reached, no further accumulation of ACh will occur. This results in a ceiling effect whereby increasing the dose of the AChE inhibitor will have no further effect on the concentration of ACh. As a consequence, deep levels of neuromuscular block, which result from high concentrations of NMBAs, may not be easily antagonized. This is the reason why it is often recommended that reversal with AChE inhibitors may not be attempted until there is evidence of continuing spontaneous return of neuromuscular function. In the clinical setting, neostigmine (0.02–0.07 mg/kg) or edrophonium (0.5–1.0 mg/kg) will act more quickly and completely as the spontaneous process of recovery progresses [5]. Lastly, it should be remembered that as the levels of ACh increase, systemic effects such as bradycardia, may ensue. Hence, it is recommended to administer atropine (0.02 mg/kg) or glycopyrrolate (0.01 mg/kg) either with or prior to the injection of AChE inhibitors.

Complications

The use of relaxants, while ordinarily devoid of important hemodynamic effects, is not without risks and potential negative consequences. The effects of these agents may extend into the early post-anesthetic period, resulting in inadvertent residual weakness. Residual block is a common complication in humans when neuromuscular monitoring and reversal are omitted, with studies showing incidences of up to 64% [6]. Residual block, even if mild, increases the incidence of hypoxemia and upper airway obstruction [7–9]. There are no data on the incidence or consequences of residual block in dogs and cats. However, it has been demonstrated that mild neuromuscular block cannot be diagnosed without the use of objective monitoring, and that even shallow residual weakness can negatively affect laryngeal function [10–12]. Hence, it is imperative to avoid residual neuromuscular block upon termination of anesthesia, particularly in animals with respiratory impairment, as the effects of residual block on laryngeal and respiratory functions could be magnified under those conditions. To date, the most effective method for detecting and avoiding residual block is the systematic use of objective monitoring of neuromuscular transmission. Routine reversal, while recommended, does not guarantee that neuromuscular function will be restored in all cases [13]. Likewise, subjective observation of spontaneous movements including ventilation, measurement of respiratory variables, or time from last dose, cannot reliably predict when complete neuromuscular transmission will return restored [10, 14, 15].

As importantly, the provider must ensure that hypnosis is adequate. In people, the use of NMBAs has been associated with higher incidences of awareness under general anesthesia; that is, inadequate depths of anesthesia were masked by the use of these agents. The presence of residual weakness during emergence of anesthesia can also lead to cases of awareness combined with immobility. In humans, it has been suggested that the combination of neuromuscular block, lack of monitoring of neuromuscular transmission, and absence of reversal contribute to the incidence of awareness under anesthesia [16].

Monitoring of Neuromuscular Function

While there are different methods to monitor neuromuscular transmission, all rely on the same principle: the stimulation of a motor nerve and the evaluation of the evoked response. Two motor nerves are routinely used for clinical monitoring in dogs and cats: the peroneal and the ulnar nerves (Figure 52.1).

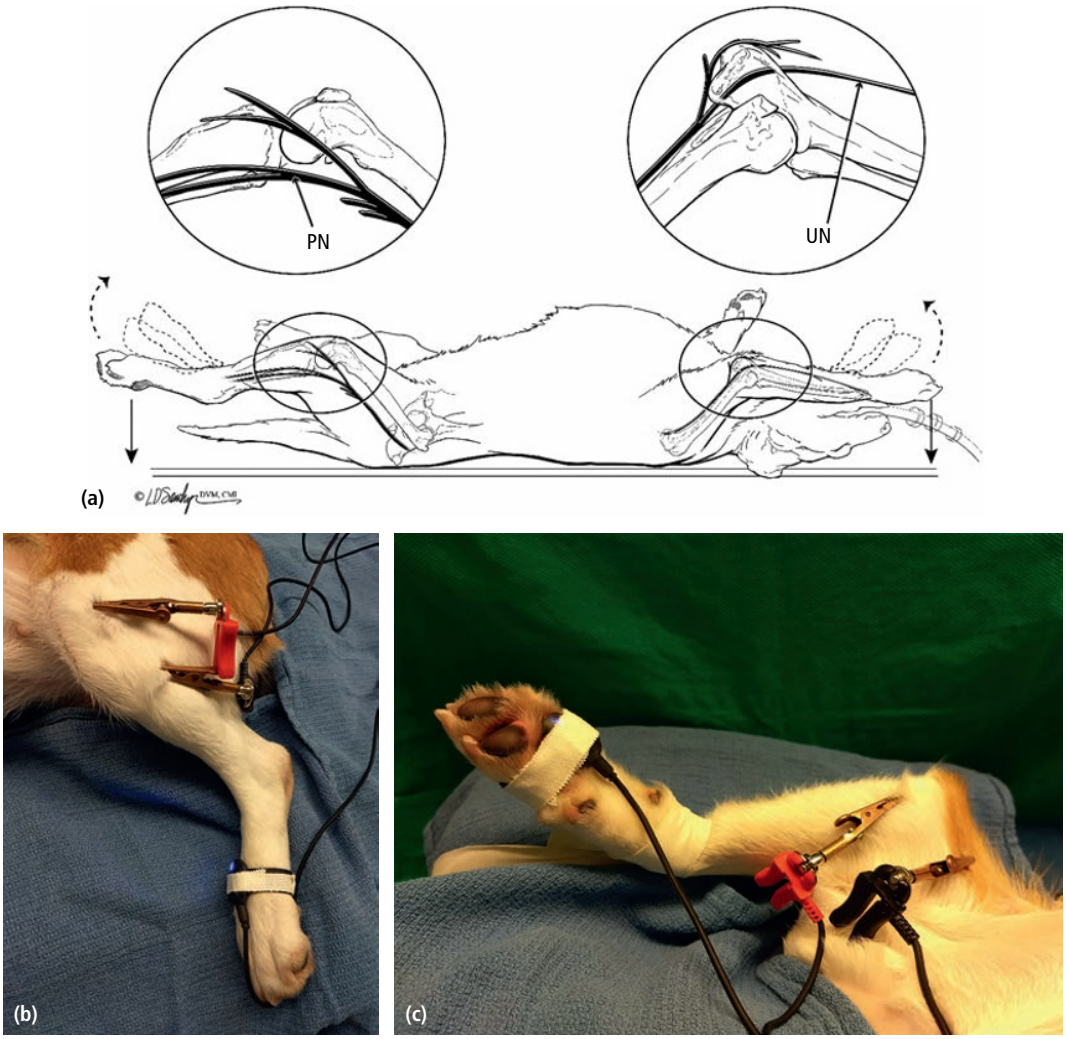


Figure 52.1 (a) Schematic representation of the peroneal (PN) and ulnar (UN) nerves in a dog on dorsal recumbency. The expected evoked responses to stimulation of those nerves (flexion of the tarsus and carpus, respectively) are indicated with dashed lines. Placement of alligator clips for nerve stimulation for both nerves are shown in (b) and (c).

Evoked responses can be judged subjectively (by observation or tact) or can be measured objectively. As mentioned above, subjective observations of evoked muscular twitches can only provide limited information. It can inform the provider on whether some response to stimulation is present or absent, but it cannot discern whether function is adequate or partially affected; that is, it cannot detect residual block. The presence of responses to stimulation is useful to determine when to redose the NMBA, or when to begin reversal with AChE inhibitors, as reversal should not be attempted during deep neuromuscular block (i.e. in the absence of evoked responses). Hence, the visual assessment of neuromuscular stimulation is not without clinical uses. There are several peripheral nerve stimulators in the veterinary market that can fill this role.

If the depth of neuromuscular block is to be accurately assessed, then an objective monitor is needed. The measurement of the isometric force of contraction (mechanomyography) has long been considered the gold standard. However, this technique is cumbersome and is not easily applied in the clinical setting. The most commonly used method, and likely the most user friendly, is the measurement of the acceleration of a free-moving limb in response to motor nerve stimulation (acceleromyography, AMG). AMG is commonly used in dogs and cats, both for research and in the clinical setting. Its accuracy has been reported in reference to mechanomyography, and it has been shown to detect residual block when visual inspection fails [10, 17]. AMG monitors are affordable and easy to use, and many devices can also be used for locoregional, electrolocation guided blocks. Examples of currently available AMG monitors are shown in Figure 52.2.

During AMG, two electrodes are placed in close proximity to either the peroneal or ulnar nerve. While adhesive



Figure 52.2 Neuromuscular transmission monitors. (a) The Stimpod® NMS450X (Xavant Technology, Pretoria, South Africa). (b) The ToFscan® (IDMED, Marseilles, France).

surface electrodes can be used, we commonly use subcutaneous needles connected to the AMG via alligator clips. An acceleration-sensitive crystal is taped to the paw of the limb being stimulated. Modern monitors measure the evoked movement in three axis and hence, it is of little relevance over which aspect of the paw the crystal is secured. It is important, however, that the carpus or tarsus is allowed to flex freely during nerve stimulation while the rest of the limb remains stable. For animals in dorsal recumbency, the front limb can be extended cranially and secured with adhesive tape, parallel to the surface, dorsally to the carpus (Figure 52.1). A similar procedure is performed for the hindlimb, with the limb secured above the tarsus. This method is also applied with animals in lateral recumbency. If the patient is resting in sternal recumbency, we prefer to displace both hindlimbs to one lateral and monitor using the peroneal nerve (Figure 52.1). A summary of how AMG is instrumented in dogs or cats is described in Protocol 52.1.

Electromyography (EMG), the measurement of the compound action potentials, can also be used to evaluate

Protocol 52.1 Items Needed for Placing Acceleromyograph

- [2] 21–25 g needles
- Acceleromyography monitor or peripheral nerve stimulator
- Alligator clamps
- Snap attachments for alligator clamps
- Medical grade tape

Procedure for Placing an Acceleromyography Monitor

1) Locate the part of the animal's body that will be stimulated (forelimb or hindlimb).
2) Extend leg to isolate the area to apply alligator clamps.
3) If using the forelimb, use medial aspect of the ulna and the epicondyle of the humerus to locate the ulnar nerve.
4) If using a pelvic limb, locate the peroneal nerve over the lateral or medial aspect of the knee.
5) Tent and place hypodermic needles through the skin so that the needle is exposed out the other side.
6) Attach the alligator clamp on the needle tip (this also prevents accidental needle stick in people and other objects).
7) Place needles approximately 1 inch apart.
8) Place the sensor over the metacarpals/metatarsals and secure with tape (or reusable hook and loop tape).
9) Select stimulating current. Current should be sufficient to elicit 4 twitches during train-of-four (TOF). If the patient is already paralyzed when the monitor is instrumented, select at least 40 mA.
10) Use the deliver button to deliver a TOF.
11) Select an interval for cycling so that TOF stimuli are delivered automatically over a desired interval. Otherwise, manual measurements can be performed.
12) If using other TOF monitors that require calibration, such as the TOF-Watch series, calibration should be performed prior to NMBA administration.
13) The patient should not be moved once calibration is obtained, to ensure adequate reversal.

neuromuscular transmission. The same principle is followed: a motor nerve is stimulated, and the response (muscular EMG) is quantified. Modern monitors quantify the amplitude of the EMG waveform and report the magnitude of evoked twitches in reference to a baseline value (in the absence of block), or as fade during train-of-four (TOF) stimulation (see below). For this purpose, a number of electrodes must be placed in particular locations over the muscle being evaluated. EMG is a practical method as, since it is cellular activation and not movement that is measured, it can be performed in any recumbency, even if the limb is not allowed to move freely. Unfortunately, this monitors are often available as part of larger multiparameter monitors with a much limited availability. The rest of this chapter therefore focuses on AMG monitoring.

Patterns of Stimulation

To make correct assessments of evoked responses, some care is necessary when stimulating the motor nerve. Square wave impulses of 0.1–0.2 msec are used, as such short stimuli do not commonly produce direct muscle stimulation. Since it is the neuromuscular transmission that is evaluated, it is imperative that the motor nerve is stimulated, and not the muscle directly. Direct muscle stimulation may result in muscular contractions, even when neuromuscular transmission is in fact impaired. The stimulating electrodes are placed over the nerve, approximately an inch apart, with the positive electrode dorsal. The electrodes may also be placed on both sides of the nerve, so that nerve courses between both electrodes.

There are several patterns of stimulation available. The most commonly used and most clinically useful is the TOF, which consists in a sequence of four stimuli, each separated by 0.5 seconds. Within each TOF, the magnitude of each twitch is assessed. In the absence of NMBAs, all four twitches are of equal magnitude (Figure 52.3). During complete neuromuscular block, all twitches disappear; that is, there is no response to nerve stimulation. As neuromuscular transmission recovers, twitches reappear in order of one to four; the "TOF count" then, can range from zero to four. Once all four twitches have return (TOF count of

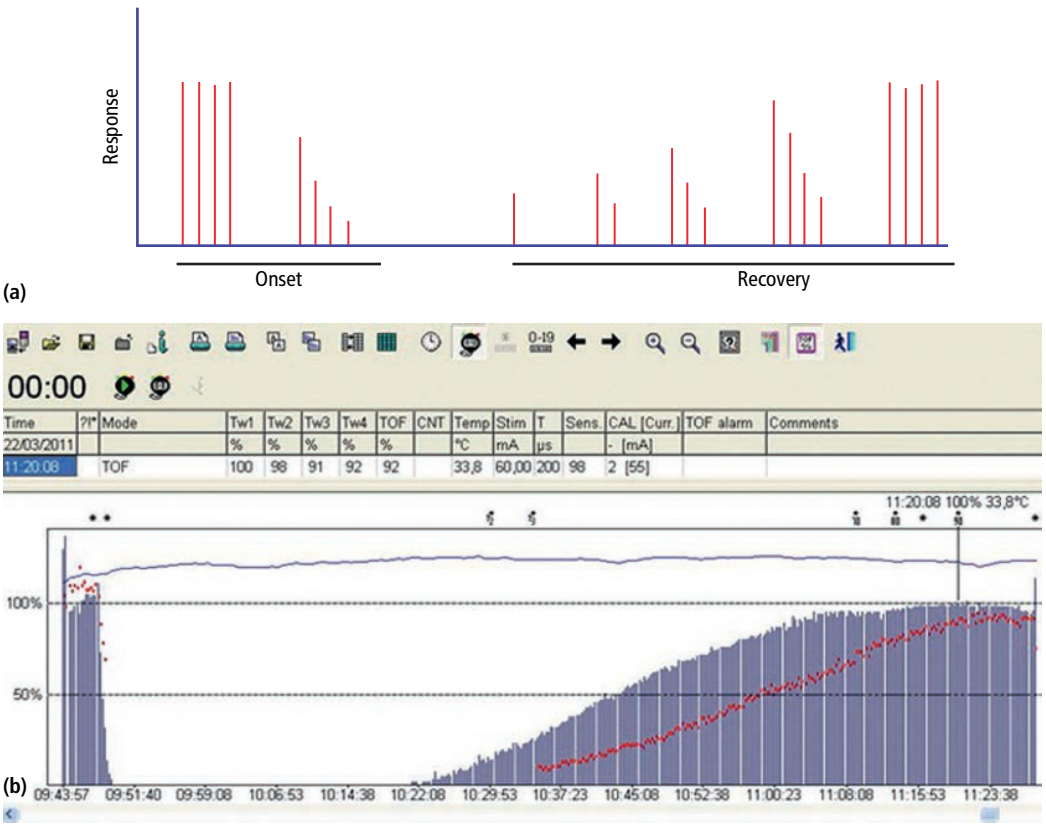


Figure 52.3 Schematic representation of train-of-four (TOF) responses. In (a), normal neuromuscular transmission is first observed (no fade during TOF). Both the magnitude of each twitch, and a fade, is observed during onset of neuromuscular block. During recovery, a progressive return of responses is observed. A trace of neuromuscular block and recovery measured with acceleromyography (AMG) obtained from an anesthetized dogs is shown in (b). The blue represent the magnitude of the first twitch in the TOF, and the superimposed red dots represent the TOF ratio (i.e. the ratio of T4 : T1). Recovery of neuromuscular function is adequate when the TOF ratio approximates 1.0 (no fade between twitches).

four), the magnitude of each twitch will increase progressively, following the order of reappearance. That is, the increase in the magnitude of twitch one will be followed by two, three and four. As a result, a progressive decay, or fade, is observed within the TOF (Figure 52.3). The presence of fade during TOF is a clear indication of residual block. This fade can be quantified by measuring the magnitude of the fourth twitch over that of the first twitch (TOF ratio T4 : T1). In the absence of block, the TOF ratio is 1.0. A TOF ratio of 0.9 or less is evidence of impaired neuromuscular transmission. While fade may be visually apparent during moderate residual block, fade during mild to shallow block (TOF ratio 0.5–0.9) is not detectable by subjective methods [10]. The fact that assessment of neuromuscular transmission is performed with values obtained within each train is an important clinical advantage; baseline values are not needed. Hence, monitoring can begin even after neuromuscular block has been established. Moreover, the patient's position may be altered, or even the site of stimulation, without affecting the ability to detect residual paralysis. This is an important feature in animals ventilated for long periods, where positioning may need to be altered periodically. Moreover, since modern devices measure triaxial AMG, no calibration is necessary. This differs from older devices (such as the TOF-Watch® series, Organon, Swords Co., Dublin, Ireland) which only measured AMG in one axis. In those devices, a baseline calibration was required, and lateral movement introduced errors during monitoring.

The use of submaximal current is yet another advantage of TOF stimulation. Other patterns of stimulation, such as the single twitch, which is used in dose-finding studies of NMBA, rely on supramaximal current for nerve stimulation. The response to nerve stimulation (i.e. the magnitude of muscular contraction) increases as the current delivered is increased, until a plateau in the response is seen. At that point, maximal current has been reached. Supramaximal stimulation is typically carried out with currents 10–20% above the maximal current. In dogs, supramaximal currents are in the order of 40–60 mA. Such currents generate discomfort when applied and the anesthetic depth needs to be adjusted to prevent a response. Because TOF examines the ratio between twitches within each train (T4 : T1), the absolute magnitude of a twitch is not important. As a result, lower, submaximal currents can be used, as long as all four twitches can be evoked with said current. Studies in humans demonstrated that both twitch magnitude as well as discomfort increased as the current was increased from 20 to 50 mA. However, the TOF ratio with the lowest current did not differ from that with the highest, suggesting that lower, less painful currents, can be used reliably to evaluate neuromuscular transmission with TOF [18].

Clinical Monitoring, Maintenance, and Reversal of Neuromuscular Block

Neuromuscular monitoring is not only necessary to avoid residual paralysis, but it is also a useful to guide administration of NMBAs and to plan reversal. Complete block, that is, the total absence of responses to nerve stimulation, is generally both unnecessary and undesirable. While only a small number of procedures benefit from such practice, most surgeries, and certainly mechanical ventilation, can be achieved with a depth of neuromuscular block characterized by the presence of one response to TOF stimulation. Maintaining a TOF count of one ensures an adequate depth of relaxation for most procedures while permitting reversal in a reasonable period with AChE inhibitors.

With this goal in mind, NMBAs administration, whether by repeated boluses or infusions, can be titrated so that a TOF count of one is present. If boluses are administered, doses are usually injected when the second response becomes apparent. In the case of infusions, the infusion rate is increased if a TOF count of two is observed and is decreased when the TOF count is zero. The practice of administering doses of NMBAs at fixed predetermined intervals, or to infuse the agent at a rate that produces complete block (no responses to TOF) carries the risk of overdosing. Once all responses are absent, assessing accumulation of the NMBA becomes a difficult task; any increase in plasma levels of the NMBA from that point onward will not be reflected by TOF stimulation. The post-tetanic count is an alternate pattern of stimulation that allows some differentiation on depth of block beyond a TOF count of zero. However, and as mentioned above, such depth of blockade is generally unnecessary.

Effective reversal with AChE inhibitors is possible when at least a TOF count of one is present. Hence, maintaining that depth of relaxation ensures that reversal agents can be administered at any point. If the TOF count is zero, it is advisable to discontinue the administration of the NMBA and delay reversal until at least one twitch returns in response TOF stimulation. The period until return of T1 will vary, depending how much agent had accumulated while the TOF count was zero. In general, the more shallow the level of block, the quicker and more consistent will AChE inhibitors restore neuromuscular transmission [5].

As mentioned above, AChE inhibitors will produce bradycardia unless they are preceded by an antimuscarinic agent, such as atropine or glycopyrrolate. If the changes in hemodynamics that can result from these agents are to be avoided, reversal can be omitted as long as neuromuscular transmission is assessed objectively so that residual block can be prevented. In such cases, NMBA administration is interrupted and recovery occurs spontaneously. The time

of spontaneous recovery will vary depending on the NMBA that has been used, the total dose administered, the dosing regimen, and other factors such as temperature, pH, electrolyte abnormalities, and use of concomitant agents such as aminoglycoside antibiotics or volatile anesthetics, which can potentiate NMBAs. A slow recovery of neuromuscular function is generally not a problem as long as the patient remains unconscious, the airway is secured, and ventilation is provided [19].

Current and Future Advancements in Neuromuscular Blockade

Several new agents have either reached the market or are in active research. Despite a wide variety of compounds, efforts seem to be targeted to an easier and more complete recovery of neuromuscular function.

Sugammadex is the first specific relaxant binding agent commercially available [20, 21]. When injected, it binds to rocuronium in an irreversible manner. The sugammadex–rocuronium complex is devoid of relaxing properties. As sugammadex binds to rocuronium, the concentration of free rocuronium in plasma drops and rocuronium moved out of the neuromuscular junction and into the systemic circulation, only to be rendered ineffective by sugammadex. As a result, neuromuscular transmission is restored almost immediately. Although sugammadex is designed to bind with rocuronium, similar effects have been demonstrated when vecuronium was used. Because sugammadex is free from adverse hemodynamic effects, this technique is not only faster than the more traditional reversal with AChE inhibitors but also more stable. The risk of incomplete reversal, while possible, is decreased, as long as the dose of sugammadex is appropriate. These virtues of sugammadex have been documented in dogs [22].

Calabadion is unrelated to sugammadex (it belongs to the cucurbit[n]uril family), but it can also bind to NMBAs, rendering them devoid of relaxing properties. Unlike sugammadex, which is selective for rocuronium, calabadion binds to agents of both the aminosteroid and isoquinoline families [23]. While still under research, calabadion is a promising alternative for restoring neuromuscular function after the injection of several NMBAs.

Unlike sugammadex or calabadion, which are new reversal agents, gantacurium and CW002 are novel NMBAs belonging to a new family, the fumarates. Gantacurium is an ultrashort-acting neuromuscular blocker, whereas CW002 is an intermediate-acting agent. Both agents have undergone tests in different species [24–27]. Regardless of the expected duration of action, their effects can be terminated almost instantly by administration of L-cysteine. These novel agents may offer the practitioner both an ultrashort-acting nondepolarizing blocker, which currently does not exist, and an intermediate-acting agent with the availability of complete, almost immediate reversal, devoid of significant hemodynamic effects.

Troubleshooting

Open Circuit

An open circuit exists when disconnections at the monitor or between the monitor and the patient occur. Most commonly, an electrode has become disconnected from the patient. This also occurs when alligator clips are placed directly on the patient rather than attached to a needle.

Inability to Calibrate and Erroneous Values

This is frequent problem with older AMG monitors that measured acceleration only over one axis. In such circumstances, lateral movement of the acceleration-sensitive crystal would produce errors. This is solved only by securing the animal's limb in a way that evoked twitches (carpal or tarsal flexion) occurs over one axis only.

Unstable Values

Unstable readings (consecutive up and down results) may occur if the limb is not secured and there is excessive movement if the patient moves voluntarily from discomfort during nerve stimulation, or if carpal or tarsal flexion become impeded (for example, if the limb is dragging over the table or drapes rather than flexing unopposed).

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Section Seven

Clinicopathologic Techniques

53

Blood Sample Collection and Handling

Courtney Waxman and Tami Lind

The information obtained from collection and analysis of blood samples often plays a critical role in the diagnostic process. It is therefore crucial that blood samples are obtained and handled properly to preserve the integrity of the specimen and to provide accurate results.

A number of issues can adversely affect the quality of blood specimens. Aggressive collection techniques including rapid aspiration of blood through a small-bore needle, prolonged or improper tourniquet use, excessive redirection of the needle (or "fishing") to access a vein, delay between the time of sample collection and time of sample analysis, and other practices are frequently associated with poor sample quality. Improper handling issues including failure to invert collection tubes adequately to mix the anticoagulant and blood, improper or delayed centrifugation, inadequate filling of blood tubes, improper storage of collected samples, and other handling errors may also have a negative impact on sample integrity and quality.

Safety Concerns

Several safety issues need to be considered during collection and handling of blood samples. Conscious patients need to be restrained in a manner that provides for the safety of the person performing the venipuncture, the assistant, and the patient. It is imperative that personnel providing restraint are properly trained in techniques that facilitate control of the patient while minimizing the risk of injury to the animal or staff. Fear-free handling of patients during venipuncture can also be beneficial to the patient as well as the staff.

Sharps safety practices must be strictly observed to minimize the risk of injury from needles or catheters. Needles should not be recapped and must be disposed of in puncture-resistant biohazard containers. Safety devices, such as syringes equipped with self-capping needles (Figure 53.1) and catheters should be used whenever possible. Additionally, needleless ports (Figure 53.2) allow for the administration of fluids and drugs, as well as collection of samples from intravenous (IV) lines and catheters without the risk of a sharps injury.

Aseptic technique should be used when collecting samples, especially in immune-compromised patients. Proper hand hygiene and the use of examination gloves significantly reduces the risk of infection to both the patient and the handler [1, 2].

Once the sample is collected, all tubes must be labeled with the patient's name, identification number, and date. Blood is considered a biohazard; proper packaging is essential for specimens submitted to commercial laboratories. Failure to label or package samples according to the laboratory's specifications may result in rejection of the sample. Serum and/or plasma must be separated from whole blood by centrifugation. All personnel using the centrifuge should be cognizant of safety and appropriate use guidelines and should read the operator manual for each piece of equipment to ensure proper use. Ideally, centrifuges should not be operated without a cover or lid in place. When in use, covered centrifuges should never be opened until the rotor has come to a complete stop. Tube caps or stoppers should be securely in place on all samples placed in the centrifuge, and any spills need to be cleaned immediately, following all appropriate safety precautions.

Venous Blood Sample Collection

Venous blood samples may be collected by direct insertion of a needle into a vein (venipuncture) or by aspiration of blood through a peripheral or central IV catheter. The steps involved in collecting a blood sample are similar for all

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Figure 53.1 Syringe with safety recapping device for needle.



Figure 53.2 Luer-lock needleless ports.

veins, with slight variations in the patient positioning/restraint and technique.

Venipuncture Equipment

Several factors play a role in the quality of a blood sample, and the person performing the venipuncture should be familiar with these variables to ensure sample quality to achieve accurate results. Needle selection is based on the size and fragility of the vein, the volume of blood required, and the frequency of sampling anticipated. Needle sizes most commonly selected for venipuncture are 20–22 gauge; smaller needles ranging from 23 to 28 gauge may be used when veins are extremely small or fragile, or when frequent sampling is required (i.e. bi-hourly glucose curves). Needles at the larger end of the spectrum are preferred when large sample volumes are required. The size of the needle used for venipuncture is a prime determinant of the rate at which a sample may be aspirated; the larger the needle, the faster the rate. Rapid aspiration of blood through a small-bore needle creates shear forces resulting in hemolysis and may adversely affect test results.

Syringe size is determined by the volume of sample required. The use of large syringes has been associated with application of excessive negative pressure during sample aspiration and may cause hemolysis and vascular collapse. To ensure sample quality, the person performing the venipuncture should apply steady, gentle traction on the syringe plunger in such a manner that the syringe fills at the same rate at which the plunger is retracted. Repetitive application and release of negative pressure on the syringe, or "pulsing," does not increase the rate or volume of sample collection; this technique causes the vessel to collapse and frequently results in hemolysis.

Besides the traditional needle and syringe setup, alternative venipuncture equipment includes butterfly catheters and Vacutainer® (Becton, Dickinson, Franklin Lakes, NJ) systems. Butterfly catheters come in a variety of gauges and can be especially useful in fractious patients or when collecting samples from small or fragile veins. Vacutainer systems (Figure 53.3) allow blood to be drawn directly into the collection tube(s), facilitating immediate contact with the anticoagulant to ensure the correct blood/anticoagulant ratio and decrease hemolysis to help preserve the integrity of the sample.

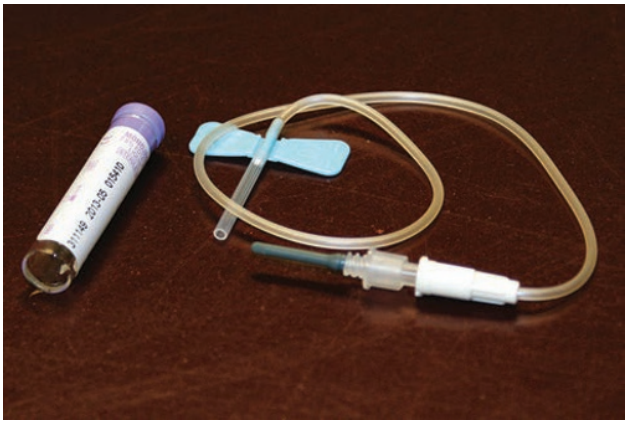


Figure 53.3 Vacutainer® system (Becton, Dickinson, Franklin Lakes, NJ).

Box 53.1 Order of Blood Tube Collection for Venous Blood Samples [11]

1) Blood culture (yellow or blood culture bottles)
2) Sodium citrate (light blue)
3) Serum or serum separator (red or red/gray)
4) Lithium/sodium heparin (green)
5) EDTA (lavender or pink)

Order of Draw

When blood is drawn for sample collection in multiple blood tubes, it is important that the samples are collected in a specific order (Box 53.1). Filling tubes in the improper order may result in unintended contamination of the sample with anticoagulants and erroneous results (i.e. false elevation of potassium levels and decreased levels of calcium due to serum or plasma contamination with potassium EDTA from the lavender-top tube).

Whenever feasible, extra blood should be collected above the minimum amount required to run diagnostic tests; this is done to allow for human error, machine error, and the possibility of needing to dilute the sample. When filling the blood tubes, it is best to remove the needle from the syringe so that the blood is directly instilled inside the tube rather than sticking the syringe needle into the rubber stopper, to minimize hemolysis. If the blood is instilled in a tube with anticoagulant, it should be gently inverted to mix the sample. If the blood is instilled in a tube without an anticoagulant, it should be stored in an upright position to allow the sample to clot; this prevents the cells from affixing to the rubber stopper or inner wall of the blood tube. The diagnostic testing ordered will determine whether the sample needs to be stored at room temperature or refrigerated.

Venipuncture Sites and Considerations

The most common sites for venipuncture include the jugular, cephalic, medial saphenous or femoral, lateral saphenous, and dorsal pedal veins. Factors influencing site selection include the sample volume required, the accessibility of the vein, the skill of the person performing the venipuncture, the condition of the skin over the site, the patient's condition and behavior, and the potential need for vascular access (i.e. IV catheter placement). The vein selected should be easily accessible; excessive probing or redirection of the needle is associated with hemolysis and poor sample quality. To minimize the risk of infection, venipuncture should not be performed at any site where there is evidence of pyoderma or loss of skin integrity.

The patient's condition may play a significant role in the site selection. Patients with heart disease, poor perfusion, or anemia may have reduced vessel filling, making visualization difficult. Patients in respiratory distress may not tolerate lateral positioning and restraint, so an alternative site, patient position, or restraint technique should be considered.

If there is any concern regarding the patient's coagulation status, samples should be collected from a peripheral vein using the smallest-bore needle that is practical for the volume of blood needed. It has been suggested that samples should not be collected from the jugular vein of coagulopathic patients. Jugular venipuncture should also be avoided in patients with head trauma or other neurologic disorders because occlusion of the jugular vein may result in a significant increase in intracranial pressure. Trauma patients may have significant injuries to various body regions (e.g. cervical spine, thoracic/pelvic limbs) that may affect the person's ability to perform a venipuncture. The person performing the venipuncture may need to select an alternate venipuncture site while keeping the patient's condition and comfort in mind. Cephalic venipuncture should be reserved for patients not requiring IV catheterization, as the cephalic vein is the ideal site for peripheral catheterization and the vessel should be kept intact.

Jugular Venipuncture

The jugular vein (Protocol 53.1) is located centrally on the neck on the lateral aspect of the trachea and esophagus in the jugular furrow and above the thoracic inlet. When a large sample volume is required, it is generally advisable to select this vessel, as it is the largest (Figure 53.4).

Cephalic Venipuncture

The cephalic vein (Protocol 53.2) is located on the cranial aspect of the thoracic limb between the elbow and the carpus. This site is commonly used in dogs and cats as it is the next largest vessel after the jugular. The cephalic vein is readily visible and easily accessible (Figure 53.5).

Lateral Saphenous Venipuncture

The lateral saphenous vein (Protocol 53.3) is located on the lateral aspect of the pelvic limb between the knee and hock. This site is most commonly used in dogs for lesser blood volume collection as this is a smaller vein (Figure 53.6). The distal lateral saphenous vein is typically readily visible and easily accessible, but the vessel tends to move or "roll" more; applying mild pressure to the proximal portion of the vein helps the distal portion be less prone to movement.

The lateral saphenous may be an alternate site to the cephalic vein if the patient is fractious or uncooperative.

Medial Saphenous (Femoral) Venipuncture

The medial saphenous (femoral) vein is located on the medial aspect of the pelvic limb between the knee and

Protocol 53.1 Jugular Venipuncture Protocol

Procedure

1) Restrain the patient in sternal, standing, sitting, or lateral recumbency, with the head elevated.
   Note: Dorsal recumbency may also be used, provided that the jugular vein is readily accessible (reverse the direction of the needle, performing the venipuncture with the needle facing the thoracic inlet rather than the chin).
2) Clip the area over the jugular vein if preferred. Prepare the area aseptically according to hospital policy.
3) Perform hand hygiene.
4) Occlude the vessel by applying pressure in the jugular furrow lateral to the thoracic inlet. Once the vessel is occluded, palpate for the vein horizontally across the furrow.
   Note: The purpose of occluding the vein is to confine its movement and allow it to fill with blood for easier palpation and sample collection.
5) Insert the needle, bevel (opening) up, through the skin and into the vein (~20-degree angle); once blood is noted in hub, decrease the angle of the syringe and advance the needle around 0.5–1.0 cm into the lumen of the vessel.
6) Apply gentle negative pressure to aspirate blood into the syringe.
7) Once the desired sample volume is obtained, release negative pressure and withdraw the needle from the vein.
8) Apply direct pressure to the jugular site for a minimum of 30 seconds or until there is no evidence of continued bleeding.



Figure 53.4 Jugular venipuncture.

Protocol 53.2 Cephalic Venipuncture Technique

Procedure

1) Restrain the patient in sternal, standing, sitting, or lateral recumbency, with the head and pelvic limbs controlled and the thoracic limb extended forward.
2) Clip the area over the distal cephalic vein if preferred. Prepare the area aseptically according to hospital policy.
3) Perform hand hygiene.
4) An assistant occludes the vessel by applying pressure with their thumb immediately distal to the elbow.
5) Once the vessel is occluded, palpate for the vein horizontally across the limb.
   Note: An alternative to having an assistant occlude the vessel is to use a tourniquet.
6) Grasp the distal portion (metacarpal) of the thoracic limb and apply traction to minimize movement. The vein may be stabilized by placing the thumb along the lateral aspect of the vein and stretching the skin downwards.
7) Insert the needle, bevel (opening) up, through the skin and into the vein (~20-degree angle); once blood is noted in hub, decrease the angle of the syringe and advance the needle around 0.5–1.0 cm into the lumen of the vessel.
8) Apply gentle negative pressure to aspirate blood into the syringe.
9) Once the desired sample volume is obtained, release negative pressure and withdraw the needle from the vein.
10) Apply direct pressure to the cephalic site for a minimum of 30 seconds or until there is no evidence of continued bleeding.

Note: A pressure wrap may be used if bleeding is excessive.



Figure 53.5 Cephalic venipuncture.

Protocol 53.3 Lateral Saphenous Venipuncture Protocol

Procedure

1) Restrain the patient in right or left lateral recumbency with the head and thoracic limbs controlled and the pelvic limb extended forward.
   Note: Large patients may require two assistants to provide safe restraint.
2) Clip the area over the distal saphenous vein if preferred. Prepare the area aseptically according to hospital policy.
3) Perform hand hygiene.
4) An assistant occludes the vessel by applying pressure with their hand wrapped around the proximal aspect of the hindlimb, slightly cranial to over the knee.
   Note: An alternative to having an assistant occlude the vessel is to use a tourniquet.
5) Once the vessel is occluded, palpate for the vein horizontally across the limb.
6) Grasp the distal portion (metatarsus) of the pelvic limb and apply traction to minimize movement. The vein may be stabilized by placing the thumb along the lateral aspect of the vein and stretching the skin downwards.
7) Insert the needle, bevel (opening) up, through the skin and into the vein (~20-degree angle); once blood is noted in hub, decrease the angle of the syringe and advance the needle around 0.5–1.0 cm into the lumen of the vessel.
8) Apply gentle negative pressure to aspirate blood into the syringe.
9) Once the desired sample volume is obtained, release negative pressure and withdraw the needle from the vein.
10) Apply direct pressure to the lateral saphenous site for a minimum of 30 seconds or until there is no evidence of continued bleeding.
    Note: A pressure wrap may be used if bleeding is excessive.



Figure 53.6 Lateral saphenous venipuncture.

Protocol 53.4 Medial Saphenous (Femoral) Venipuncture Technique

Procedure

1) Restrain the patient in semi-sternal, right, or left lateral recumbency with the head and thoracic limbs controlled and the pelvic limb extended forward.
2) Clip the area over the distal medial vein if preferred. Prepare the area aseptically according to hospital policy.
3) Perform hand hygiene.
4) Flex the non-dependent pelvic limb and hold it tucked against the caudal abdomen. The assistant uses the side of their hand to occlude the medical saphenous (femoral) vein. An alternative to having an assistant occlude the vessel is to use a tourniquet.
5) Once the vessel is occluded, palpate for the vein horizontally across the limb.
6) Grasp the distal portion (metatarsus) of the dependent pelvic limb and apply traction to minimize movement. The vein may be stabilized by placing the thumb along the lateral aspect of the vein and stretching the skin downwards.
7) Insert the needle, bevel (opening) up, through the skin and into the vein (~20-degree angle); once blood is noted in hub, decrease the angle of the syringe and advance the needle around 0.5–1.0 cm into the lumen of the vessel.
8) Apply gentle negative pressure to aspirate blood into the syringe.
9) Once the desired sample volume is obtained, release negative pressure and withdraw the needle from the vein.
10) Apply direct pressure to the medial saphenous (femoral) site for a minimum of 30 seconds or until there is no evidence of continued bleeding.

Note: A pressure wrap may be used if bleeding is excessive.

hock (Protocol 53.4). This site is most commonly used in cats for lesser blood volume collection as this is a smaller vein (Figure 53.7). The medial saphenous/femoral vein is typically readily visible and easily accessible and can vary in its depth below the skin.

The medial saphenous may be an alternate site to the cephalic vein if the patient is fractious or uncooperative.



Figure 53.7 Medial saphenous (femoral) venipuncture.



Figure 53.8 Dorsal pedal venipuncture.

Dorsal Pedal Venipuncture

The dorsal pedal vein is located on the dorsal aspect of the pelvic limb between the hock and metatarsus (Protocol 53.5). This site is less commonly used as a primary venipuncture site, often being reserved when other veins have been exhausted (Figure 53.8). The dorsal pedal vein is somewhat visible and accessible, and venipuncture of this vessel typically causes slightly more patient discomfort.

Venous Blood Sample Collection from a Catheter

Blood samples may be collected from both peripheral and central IV catheters. Hemolysis has been associated with samples collected from IV catheters and appears to be directly proportional to the size of the catheter used. In a study comparing samples collected by venipuncture using a 21-gauge Vacutainer needle and evacuated sample tubes to samples collected through a peripheral IV catheter at insertion, hemolysis was noted in 3.8% of the samples obtained through venipuncture and in 13.7% of those obtained through a catheter [3]. Further analysis

Protocol 53.5 Dorsal Pedal Venipuncture Protocol

Procedure

1) Restrain the patient in sternal or lateral recumbency with the head and thoracic limbs controlled and the pelvic limb extended.
2) Clip the area over the pedal vein if preferred. Prepare the area aseptically according to hospital policy.
3) Perform hand hygiene.
4) An assistant occludes the vessel by applying pressure with their hand wrapped around the distal aspect of the hindlimb, caudal to the hock.
   Note: An alternative to having an assistant occlude the vessel is to use a tourniquet.
5) Once the vessel is occluded, palpate for the vein horizontally across the limb.
6) Grasp the distal portion (paw) of the pelvic limb and apply traction to minimize movement. The vein may be stabilized by placing the thumb along the lateral aspect of the vein and stretching the skin downwards.
7) Insert the needle, bevel (opening) up, through the skin and into the vein (~20-degree angle); once blood is noted in hub, decrease the angle of the syringe, and advance the needle around 0.5–1.0 cm into the lumen of the vessel.
8) Apply gentle negative pressure to aspirate blood into the syringe.
9) Once the desired sample volume is obtained, release negative pressure, and withdraw the needle from the vein.
10) Apply direct pressure to the dorsal pedal site for a minimum of 30 seconds or until there is no evidence of continued bleeding.

Note: A pressure wrap may be used if bleeding is excessive.

showed that the primary variable was the diameter of the IV catheter as demonstrated by the percentage of hemolyzed samples obtained. Specifically, this study documented that there was no evidence of hemolysis in samples obtained from 16-gauge catheters, whereas all samples drawn from 24-gauge catheters were hemolyzed; a small percentage of samples drawn from catheters ranging from 18 to 22 gauge also demonstrated hemolysis [3].

Sample Collection from Peripheral Intravenous Catheters

Peripheral catheters are most commonly used for sample collection immediately following catheter placement (Figure 53.9), which minimizes the number of needlesticks for the patient and possibly expedites sample collection (Protocol 53.6).

Sample Collection from Central Intravenous Catheters

Central venous catheters include peripherally inserted central catheters and jugular central catheters; these devices are frequently used to facilitate multiple fluid type administration and serial blood sampling in hospitalized patients. The concerns associated with blood sampling through central lines include an increased risk of catheter complications including infection and occlusion, the possibility of inaccurate results due to sample dilution from IV fluids and/or medications, or hemolysis due to improper collection technique [4]. Both the discard method and the push–pull method of sample collection have been shown to yield accurate results when properly performed.

Protocol 53.6 Blood Sample Collection from a Peripheral Intravenous Catheter

Procedure

1) An assistant restrains the patient based on the venous access site chosen.
2) Clip the selected venous access site and prepare aseptically according to hospital policy.
3) Perform hand hygiene.
4) Place the peripheral IV catheter aseptically.
   Note: Catheters intended for blood sample collection should not be preflushed to avoid sample dilution.
5) Attach a syringe (size appropriate for sample volume required) to the hub of the catheter and apply gentle traction to aspirate blood.
   Note: It is imperative that the catheter be stabilized throughout the collection procedure.
6) Following sample collection, carefully disconnect the syringe and insert a luer-lock T-port or male adapter plug into the catheter hub.
7) Inject the blood sample into the appropriate collection tubes.
8) Secure the catheter and flush with 0.9% sodium chloride according to hospital policy.

Discard Method

The discard method (Protocol 53.7) involves drawing a volume of blood from the catheter and discarding it to minimize the risk of sample dilution or interfering substances. Significant concerns with this method include iatrogenic anemia, as well as an increased risk of catheter-related infections due to the handling of the catheter hub or port.

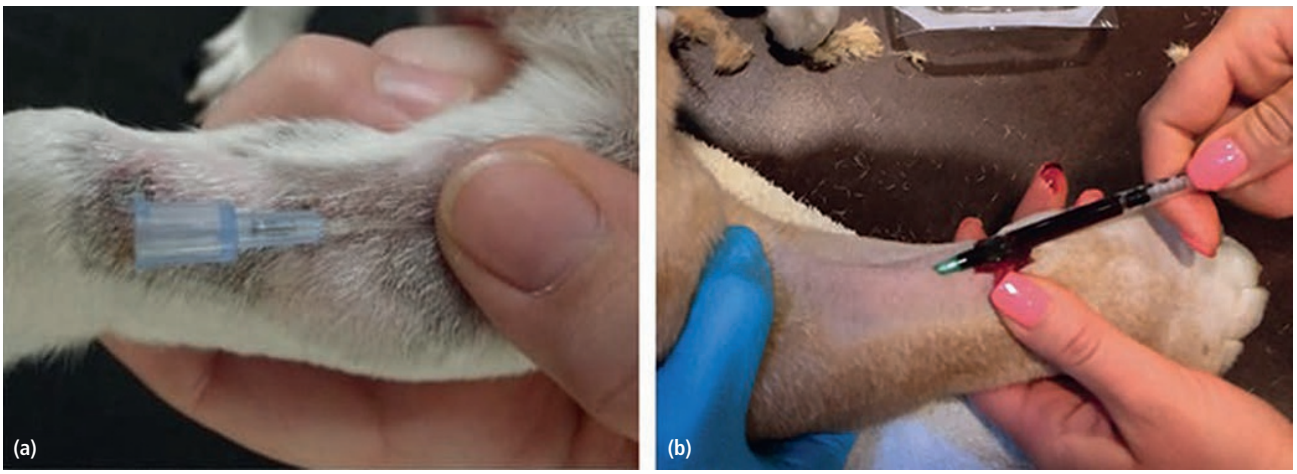


Figure 53.9 Blood collection from a peripheral catheter. (a) Apply slight digital pressure over the end of the catheter to occlude the catcher and minimize blood loss following catheter placement. (b) Attachment of a syringe to the catheter hub to collect the blood sample.

Protocol 53.7 Discard Method

Procedure

1) Discontinue administration of all fluids and medications prior to sample collection.
2) Disinfect the catheter port hub according to hospital protocol.
3) Perform hand hygiene and don exam gloves.
4) Flush the catheter with 3–5 ml 0.9% sodium chloride.
5) Attach a syringe to the catheter hub and aspirate blood.
   Minimum of three times: priming volume of the catheter for routine assays [4].
   Six to twelve times: priming volume for coagulation assays [8].
6) Remove the discard syringe and attach a fresh syringe for sample collection; apply gentle traction to aspirate blood.
7) Depending on the patient's condition, the discard sample may be reinfused. Preheparinizing the discard syringe will minimize clotting if reinfusion is planned.
8) Following sample collection, flush the catheter with 3–5 ml 0.9% sodium chloride or heparinized saline according to hospital policy.
9) Restart infusion of intravenous fluids and medications.

Push–Pull or Mixing Method

The push–pull or mixing method of sample collection (Protocol 53.8) involves aspiration and injection of blood through the catheter hub or port (repeatedly four times), followed by sample collection into a fresh syringe. The potential advantages of this method include decreased risk of iatrogenic anemia with frequent sampling and decreased risk of catheter-associated infections through minimizing extraneous handling of the catheter. As in the discard method, all fluids or medications being administered through the catheter are temporarily stopped, and the catheter hub or port is cleaned with antiseptic solution [5].

Inline Sampling

Another method that is very effective for collection of samples from central catheters is the use of an inline sampling system. These systems feature a reservoir, stop cock, and a sampling port (Figure 53.10). They are connected between the IV line and the catheter. With these devices, "discard" blood is drawn into the reservoir and then returned to the patient without the use of multiple syringes. This is a newer method being made available for our veterinary patients.

Protocol 53.8 Push–Pull Method

Procedure

1) Discontinue administration of all fluids and medications prior to sample collection.
2) Disinfect the catheter port hub according to hospital protocol.
3) Perform hand hygiene and don exam gloves.
4) Attach a syringe to the catheter hub and aspirate blood (minimum of three times the catheter priming volume).
5) With the syringe still attached to the hub of the catheter, reinfuse the blood.
6) Repeat steps 3 and 4 at least three times.
7) Discard the empty mixing syringe and attach a fresh syringe.
8) Aspirate the required sample volume and inject it into the appropriate blood collection tubes.
9) Following sample collection, flush the catheter with 3–5 ml 0.9% sodium chloride or heparinized saline according to hospital policy.
10) Restart infusion of intravenous fluids and medications.



Figure 53.10 Inline blood sampling system.

Blood Culture Samples

Blood cultures may be helpful to identify causative agents in patients with suspected bacteremia. Samples collected for blood culture (Protocol 53.9) require strict

Protocol 53.9 Blood Culture Collection

- It is recommended that a minimum of three samples be obtained from different vascular sites over a period of time [9]:
  - 10–30 minutes for critical or septic patients
  - 24 hours for noncritical patients
- The general sample volume recommendations are as follows:
  - Cats and small dogs: 1 ml
  - Medium dogs: 2–3 ml
  - Large dogs: 3–5 ml
- Volume must be sufficient for a 1:10 ratio of blood to broth [9].

Procedure

1) Disinfect the diaphragm of the culture tube/bottle with 70% isopropyl alcohol or iodine according to hospital policy and allow to air dry.
   Note: Keep the blood culture tubes in an upright position.
2) Clip the area over the venipuncture site and perform an aseptic preparation according to hospital policy.
3) Perform hand hygiene.
4) Don sterile gloves to collect the blood sample.
5) Insert the needle, bevel (opening) up, through the skin and into the vein (~20-degree angle); once blood is noted in hub, decrease the angle of the syringe and advance the needle around 0.5–1.0 cm into the lumen of the vessel.
6) Apply gentle negative pressure to aspirate blood into the syringe.
7) Once the desired sample volume is obtained, release negative pressure and withdraw the needle from the vein.
8) Apply direct pressure to the venipuncture site for a minimum of 30 seconds or until there is no evidence of continued bleeding.
   Note: A pressure wrap may be used if bleeding is excessive.
9) Following sample collection, place a new needle on the syringe and inject the blood into the culture tube/bottle, dividing the sample between aerobic and anaerobic media.
   Note: Avoid injection of air into the culture tube/bottle.
   Note: Invert each bottle two to three times to thoroughly mix the blood and broth.
10) Maintain samples at room temperature; sensitive bacteria may perish under refrigeration.

aseptic technique, and proper sample handling is essential for successful and accurate culture results (Figure 53.11). In most bacteremic patients, the number of organisms in circulation is very small; the volume of blood collected needs to be sufficient to account for the low concentration of bacteria. The current recommendations are that multiple samples be collected from different vascular sites over a period of several minutes to several hours. Venipuncture is the preferred sample collection method; indwelling catheters have been shown to have a higher percentage of false-positive results due to colonization of the catheter.

Arterial Blood Sample Collection

Arterial blood samples are useful for assessment of blood gas status, which includes oxygenation, ventilation, and acid–base balance. These samples may be collected by direct arterial puncture or through an arterial catheter.

A number of arterial blood gas (ABG) syringes prefilled with lyophilized heparin are readily available on the commercial market (Figure 53.12). These syringes are available in either vented or unvented styles and typically range from 1 to 3 ml in volume. The unvented syringe is handled like a standard syringe; the needle is inserted into the artery with the plunger positioned at the base of the barrel and then gently retracted to draw blood into the syringe. The vented syringe allows the person performing the venipuncture to position the plunger at the desired draw volume in the barrel; once the needle enters the artery, blood should fill the syringe to the level of the plunger.

Commercial ABG syringes are preferred for arterial sampling. Blood gas syringes may be created in the hospital by drawing 1000 IU/ml heparin into the syringe, allowing it to coat the interior of the barrel, and then expressing the heparin from the syringe prior to sampling. Note that samples obtained using non-commercial syringes with liquid heparin may lead to dilutional error [6]. To minimize hematoma formation, small-bore needles (25 or 27 gauge) should be used for sample collection.

It is important to note that arterial puncture is considered to be significantly more painful than peripheral venipuncture. It is crucial that the patient be properly restrained



Figure 53.11 Blood culture collection. (a) Blood sample collection following sterile technique (sterile preparation, sterile gloves). (b) Replacing the blood draw needle with a sterile needle. (c) Disinfecting of the culture tube diaphragm using a 70% alcohol wipe. (d) Instilling the blood sample into the blood culture tube.

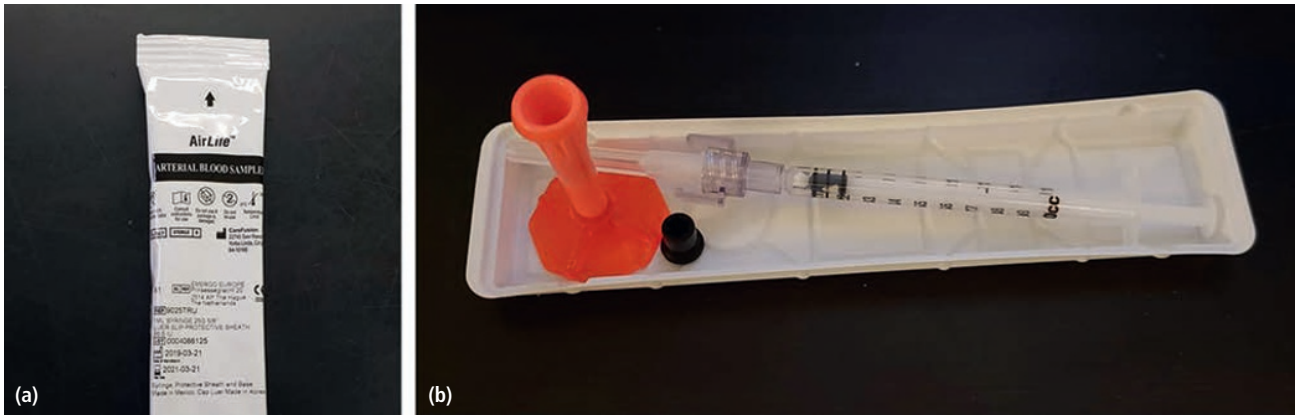


Figure 53.12 Arterial sample commercial kit. (a) The outer packaging. (b) Replacing the kit contents – 1-ml syringe with lyophilized lithium heparin anticoagulant. After arterial blood collection, the needle can be placed directly into the orange needle stopper, or the needle can be removed, and the black rubber stopper can occlude the syringe.

for the procedure; the use of a small volume of local or topical anesthetic at the site is often beneficial.

Direct Arterial Puncture

The dorsal pedal and femoral arteries are most commonly chosen sites for arterial puncture (Protocol 53.10). On rare occasions, samples may be collected from the radial artery, the artery in the pinna of the ear, or from the sublingual artery in anesthetized or unconscious patients. The dorsal pedal artery is the preferred sampling site because there appears to be a lesser incidence of mixed arterial/venous samples, and it is relatively easy to apply a pressure bandage following sample collection (Figure 53.13). The drawback to the dorsal pedal artery is that it is significantly smaller than the femoral artery and may be difficult to access in some patients. The femoral artery is typically easy to palpate and may be easier to access, but it appears more likely to yield mixed samples, and it is difficult to place a pressure bandage to control bleeding following arterial puncture. If an arterial puncture is needed and there is concern for coagulopathy, it is recommended to choose a site where a pressure wrap can be applied, such as the dorsal pedal artery.

Arterial Catheter Sampling

Arterial catheters are placed for the purposes of continuous, direct blood pressure measurements and the repeated sampling of arterial blood. Sampling from an arterial catheter (Protocol 53.11) minimizes the need to perform arterial punctures.

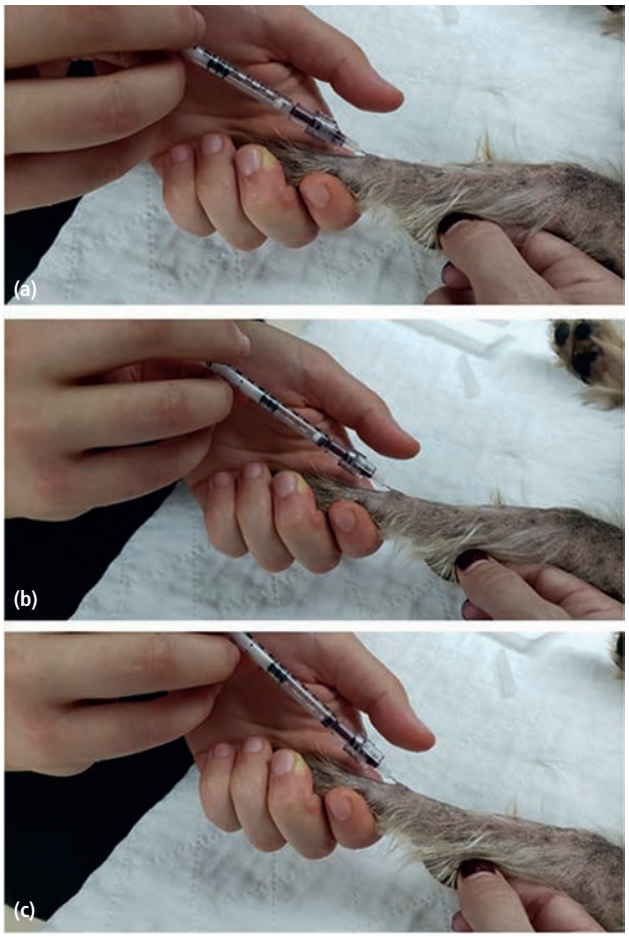


Figure 53.13 Direct arterial puncture technique. (a) Initial puncture into the dorsal pedal artery. (b) The syringe filling with each arterial pulse to mix with the lyophilized lithium heparin anticoagulant. (c) The syringe continues to fill with each arterial pulse.

Protocol 53.10 Arterial Puncture Technique

Procedure

1) Restrain the patient in right or left lateral recumbency with the head and thoracic limbs controlled and the pelvic limb extended forward. You may require two assistants to provide safe restraint.
2) Locate the artery by palpating the patient's arterial pulse.
3) Clip the area over the artery and aseptically prepare the puncture site according to hospital policy.
4) Perform hand hygiene and don exam gloves.
5) Palpate and stabilize the artery:
   - A single digit may be placed directly over the artery; with a heparinized syringe, the needle is advanced into the vessel at a 45-degree angle directly below the finger
     or
   - Place a digit on each side of the artery (~1 inch apart); have the needle between the fingers and advance into the vessel at a 90-degree angle [10].

   Note: Hold the syringe in a "pencil" grip and insert the needle bevel (opening) upward.
6) During arterial puncture, blood should pulsate into the syringe; gentle aspiration of the plunger may be necessary.
7) Once the sample volume has been allowed to fill the syringe (minimum of 0.5–1 ml), apply direct pressure for a minimum of 20 minutes and monitor the site closely to ensure that there is no continued bleeding.
8) Expel all air is from the syringe immediately after sample collection and plunge the needle into a rubber stopper to occlude exposure of the sample to room air.

Note: It is recommended that samples are analyzed immediately, but they may be placed in an ice bath for up to one hour [8].

Protocol 53.11 Arterial Catheter Sampling

Procedure

1) Perform hand hygiene and disinfect the sampling port or catheter hub according to hospital protocol.
2) Attach a non-heparinized syringe and aspirate a discard sample:
   - Minimum of three times: priming volume of the catheter for routine assays [4].
   - Six to twelve times: priming volume for coagulation assays [8].
3) Remove the discard syringe and attach a new heparinized syringe to the catheter; turn the stopcock (if present) to a 45-degree angle during syringe change.
   Note: Discard samples from an arterial catheter should be reinfused into a venous catheter.
4) Aspirate the sample, disconnect the syringe from the catheter; immediately expel all air, then occlude the syringe tip to prevent exposure of the sample to room air.
   Note: It is recommended that blood gas samples are analyzed immediately, but they may be placed in an ice bath for up to one hour.
5) Following sample collection, flush the arterial catheter with 0.9% sodium chloride or heparinized saline according to hospital protocol.

Proper Specimen Handling

The accuracy of hematology, blood chemistries, and other laboratory results is directly related to the quality of the sample submitted for analysis. Although the first step in acquiring a high-quality sample requires using appropriate collection techniques as previously noted, the manner in which the sample is handled following collection is equally important.

Handling of Venous Samples

During or immediately after collection, blood samples should be injected into the appropriate sample tubes for the tests required; all tubes must be properly labeled. Each tube needs to be inverted several times to ensure appropriate mixing of the sample with the tube's anticoagulant. Inversion should be smooth and requires a complete turn of the wrist 180 degrees and back [7]. Tubes should never be shaken or mixed aggressively. The number of inversions is dictated by the tube being filled: Serum separator, serum, and clot activator tubes require five inversions; sodium citrate tubes require three to four inversions, and all other additive tubes (EDTA, heparin, plasma separator, etc.) require eight to ten inversions [7].

Following inversion, it is recommended that serum and serum separator tubes sit at room temperature for 30 minutes to facilitate adequate clot formation [7]. Plasma separator and all other additive tubes may be submitted for analysis or centrifuged immediately following inversion. Serum and gel tubes should be centrifuged within two hours of collection.

Proper centrifugation techniques also play an important role in sample quality. All personnel using the centrifuge should be cognizant of safety and appropriate usage guidelines. Ideally, centrifuges should not be operated without a cover or lid in place. Stoppers should be securely in place on all samples placed in the centrifuge, and any spills must be cleaned immediately.

Centrifuges must be properly balanced (Figure 53.14) when in use, both to obtain quality samples and to promote safety of personnel using the instrument. This is achieved by ensuring that tubes are filled with equal volumes of fluid and placed opposite each other in the centrifuge. If tubes are filled unequally or an uneven number of tubes need to be centrifuged, balance tubes may be used by filling the same size tubes with an equivalent amount of water. Atypical noise or vibration are good indicators that the centrifuge is unbalanced and should be immediately turned off; once the samples are correctly balanced, the centrifuge may be restarted.

Gel tubes include serum separator and plasma separator tubes and should be centrifuged at room temperature at 1000–1300 RCF (relative centrifugal force) or 2000–2500 RPM (rotations/minute) for 10 minutes in a swinging-bucket centrifuge or 15 minutes in a fixed-angle centrifuge [7]. Sodium citrate tubes should be spun at 1500 RCF or about 2500–3000 RPM for 15 minutes; all other tubes (EDTA, heparin, serum, etc.) should be centrifuged at 1300 RCF or 2500 RPM for 10 minutes [7]. To ensure sample quality, it is important to make certain that all similar samples are centrifuged for the same length of time; a

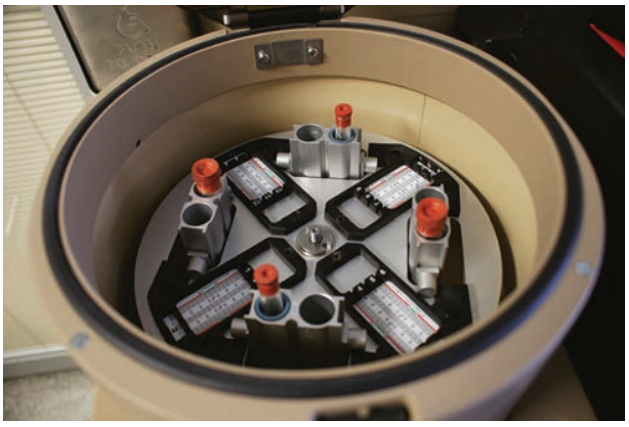


Figure 53.14 Correctly balanced centrifuge.

packed cell volume obtained from a sample centrifuged for three minutes may yield significantly different results from one centrifuged for four minutes. Following centrifugation, plasma or serum should be immediately removed from the tube and placed in an appropriate sample container labeled with the patient's identification information.

Most serum and plasma samples, once removed from the red blood cells, are stable for up to 8 hours at room temperature or up to 48 hours in the refrigerator; samples needing to be kept beyond 48 hours should be placed in plastic tubes and frozen. Special handling is required for certain specimens. Adrenocorticotropic hormone, angiotensin-converting enzyme, ammonia, acetone, lactic acid, pyruvate, and renin specimens are temperature sensitive and should be placed in an ice bath immediately following collection; these samples also require transport in a chilled container. Bilirubin and erythrocyte protoporphyrin are very light sensitive; wrapping the tubes in aluminum foil decreases exposure to light and helps maintain the stability of the sample. It is advisable to contact the reference laboratory for detailed collection, handling, and submission protocols whenever there is a concern about a specific sample or test.

Troubleshooting Technical Problems Associated with Blood Sample Collection and Handling

Hemolysis

Hemolysis can interfere with a number of laboratory test results. Potassium and lactate dehydrogenase may be falsely elevated due to release of intracellular material; tests requiring analysis by spectrophotometry may be invalid due to interference. Hemolysis may be secondary to disease processes such as immune-mediated hemolytic anemia, heavy metal toxicity (zinc), or parasitism (*Babesia*); improper collection or handling of blood samples is also commonly implicated in hemolysis (Protocol 53.12). To try to prevent hemolysis, use the largest bore needle possible.

Sample Dilution

Blood samples may be inadvertently diluted during collection, which can result in erroneous results. Sample dilution is typically attributed to inadequate sample to anticoagulant ratio, inadequate discard volume during sample collection from a central line or arterial catheter, and aspiration of tissue fluids during venipuncture (relatively rare).

A common dilemma that arises in veterinary medicine is the need to collect sodium citrate samples from very small patients for coagulation assays. Commercially available sodium citrate collection tubes require either 1.8 or 2.7 ml of blood. Coagulation testing demands a correct blood to anticoagulant ratio because dilute samples will not yield accurate results.

Protocol 53.12 Hemolysis Troubleshooting Checklist

Procedure

1) Choose an appropriate size needle and syringe or Vacutainer™ needle and tubes; avoid very small needles and large syringes.
2) Aspirate sample gently; avoid excessive negative pressure or pulsing. Ensure that tourniquet use or vessel occlusion is limited to two minutes or less; one minute is preferred.
3) Avoid excessive isopropyl alcohol use; allow alcohol to dry prior to venipuncture.
4) Invert the sample tubes following collection; allow serum samples to stand in a vertical position for 30 minutes to promote adequate clot formation prior to centrifugation.

Platelet Clumping

Platelet clumping is commonly encountered and may be due to prolonged collection times, delayed mixing of blood and anticoagulants, or other factors. It is acceptable to use blood from a sodium citrate tube for platelet counts when there is clumping in the EDTA sample; counts performed on sodium citrate samples should be multiplied by 1.1 to account for the dilutional differences between the tubes.

Delayed Sample Separation or Analysis

Delays in separating or analyzing samples may have a significant negative effect on the quality of the specimen. Delayed analysis of arterial and venous blood gas samples may result in considerable changes in the partial pressure of oxygen or carbon dioxide in arterial blood and pH. Delayed centrifugation of serum and plasma samples allows prolonged contact with red blood cells and often results in erroneous sample results, including decreased glucose results and elevations in ammonia.

Troubleshooting Patient Problems Associated with Blood Sampling

Information obtained from blood tests is often vital to the diagnostic process, and serial blood tests are frequently required to monitor a patient's condition and the efficacy of the treatment regimen. In some cases, obtaining blood samples can be problematic for the patient.

Iatrogenic Anemia from Frequent Sampling

Frequent blood sampling, especially from small or anemic patients, can be associated with significant blood loss to a degree that is detrimental to the patient's condition. This is especially concerning in small patients, neonates, geriatric animals, and patients with anemia. The volume of blood that is safe to collect from a patient is no more than 10–15 ml/kg, with a maximum collection volume of 20 ml/kg. For instance, if a patient weighs 10 kg, the volume of blood that is safe to collect is 100–150 ml (maximum 200 ml) over a two-week period. When blood sample collection exceeds 10 ml/kg, the patient's intravascular volume should be replaced with isotonic crystalloids. There are several strategies to minimize blood loss in these patients:

1) Use Microtainer™ or other "mini" collection tubes to minimize sample volume requirements.
2) Use point-of-care testing equipment that requires minimal sample volumes instead of sending blood to reference laboratories that require larger sample volumes.
3) Use the push–pull or mixing method rather than the discard method when pulling blood samples from an IV catheter.

Hematoma Formation

Hematomas (bruises) form when blood leaks from a vessel and pools under the skin. Hematomas may be minimized by holding direct pressure over a venipuncture site for at least 30 seconds and for a minimum of 20 minutes over an arterial puncture site. Pressure bandages may be placed over sampling sites on the limbs; these bandages need to be removed and the site should be carefully checked within 15–30 minutes of application. Patients requiring frequent sampling or with coagulopathies may benefit from a central catheter to allow for administration of fluids and medications as well as to facilitate serial blood sampling.

Summary

- The venipuncturist needs to be cognizant of safety concerns at all times.
- The quality of a blood sample and the accuracy of test results are significantly affected by collection and handling techniques.
- The venipuncturist should assess each case individually and choose venipuncture sites and techniques best suited to that patient.
- Blood cultures require strict aseptic technique and specialized handling.
- Arterial and venous samples for blood gas analysis require special handling and must not be contaminated with room air.
- Hemolysis is a significant issue that affects sample quality and can be minimized when proper techniques are used.
- Risks to the patient including catheter-related infections and iatrogenic anemia can be significantly decreased by using aseptic techniques and minimizing the volume of blood drawn or discarded.

Acknowledgment

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In-House Hematologic Evaluation

Karl E. Jandrey and Andrew Burton

Introduction

In-house evaluation of hematologic samples has many advantages, and the information obtained may be life saving for some critically ill patients. The ability to rapidly identify the presence and cause of anemia (such as reviewing blood smears for infectious disease or spherocytes indicative of immune-mediated hemolytic anemia, IMHA), evaluate platelet adequacy, or perform blood typing for blood transfusions can facilitate immediate treatment of emergency and critically ill patients which may improve outcomes. Some measurements may also be more accurate if performed in-house, owing to the stability of the sample or reduction in processing, which may induce errors.

Completion of these tests requires not only the correct instruments and supplies, but also personnel who are trained and knowledgeable in the use of equipment and interpretation of results. This was highlighted in one study which showed that in-house interpretation of canine and feline blood smears by emergency room personnel at a tertiary referral hospital frequently showed poor agreement with review by a clinical pathologist. Many important abnormalities such as spherocytes, toxic changes in neutrophils, nucleated red blood cells, infectious agents, and neoplastic cells frequently were not accurately identified [1]. Additionally, accuracy of in-house testing requires knowledge of quality control and diligent maintenance of supplies and tests. In-house hematologic evaluation is thus not a replacement for send-out laboratory analysis but a complementary tool that is incredibly useful in the care of emergency and critically ill patients.

This chapter reviews the components of blood that are routinely assessed in-house, as well as the supplies and steps to evaluate these parameters with accuracy, precision and highest quality.

Composition of Blood

Blood comprises cells (including erythrocytes, leukocytes and platelets) suspended in a fluid component called plasma. Plasma, in turn, consists mostly of water, as well as plasma proteins, inorganic salts, lipids, hormones, carbohydrates, and vitamins. Plasma contains clotting factors and is obtained when blood is collected in tubes with anticoagulant (e.g. EDTA or lavender-top tubes) is centrifuged. Fluid obtained from centrifugation of whole blood is called serum, and is devoid of clotting factors and fibrinogen. The total protein of serum is typically 0.2–0.5 g/dl lower than that of plasma.

Sample Collection and Handling

Safety

The safety of clinic personnel and patients is of utmost importance. Biologic samples should be treated with caution, as the potential for zoonotic infection may exist, and some diseases may be transmissible to other patients. Materials for collection and processing of blood, such as needles and glass slides, may also cause harm to personnel or patients if not handled correctly.

Clinic personnel handling blood should wear appropriate personal protective equipment (PPE), including latex, rubber, or nitrile gloves, as well as protective clothing such as a lab coat or scrubs. Protective eyewear such as safety glasses should also be worn. Sharp implements such as needles and glass slides should be disposed appropriately in biohazard containers. Such supplies should never be reused, and needles should never be recapped. In the event of broken glass or other sharp objects that may puncture

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the skin, use only mechanical means such as forceps or a brush and pan to remove hazardous material.

If blood or other biologic waste is spilled, ensure that appropriate PPE is worn. Cover the spill area with paper towel, and then pour a freshly mixed (no more than 24 hours old) 10% bleach and water solution over the area, starting from the outside edges of the spill inward. Allow the mixture to sit for up to 20 minutes (especially if there is concern for infectious disease), prior to cleaning the surface with more paper towel and bleach solution as required. Waste from the clean-up procedure (including gloves worn) must be disposed of with other biohazardous waste. Wash hands thoroughly after clean-up is complete.

Blood Collection and Handling for Hematologic Evaluation

For details on blood sampling from patients, refer to Chapter 53. EDTA is the preferred anticoagulant for complete blood counts in cats and dogs, and is present in purple- or lavender-top tubes. Other anticoagulants are available, and citrate (pale blue-top tube), for example, is the preferred anticoagulant for assessment of coagulation.

Collection of blood directly into a vacuum tube is preferred, as this can reduce clot formation and activation/clumping of platelets. This method may also reduce iatrogenic hemolysis, which can affect erythrocytes (including hematocrit and erythrocyte indices), as well as plasma protein and fibrinogen measurements. Ensure that the tubes are filled completely to the recommended levels. Underfilling of tubes may reduce hematocrit due to dilution from anticoagulants. Excessive EDTA may also cause osmotic shifts of fluid from erythrocytes and affect morphology and erythrocyte indices. Once filled, the tubes should be gently inverted multiple times to thoroughly mix the blood and anticoagulant.

Blood should be visually inspected within the tube. Samples should be checked for any gross clots, which may affect platelet concentrations or estimates, as well as hematocrit and leukocyte concentrations/differentials. The color of the blood should also be evaluated and logged. A pink discoloration may be seen with lipemia, and brown discoloration can be present in cases of methemoglobinemia. Additionally, red granules present within well-mixed blood may indicate agglutination of red blood cells.

Aggregation of platelets and leukocytes tends to increase with both storage and cooling of blood, and it is recommended that samples are processed and evaluated as quickly as possible. Blood smears should be made immediately, and it may be beneficial to make smears from fresh whole blood that has not been anticoagulated.

Microhematocrit Tube Evaluation

Evaluation of a microhematocrit tube is a common starting point for hematologic evaluation of the patient and offers a plethora of information quickly with minimal supplies. The packed cell volume (PCV) can be measured to help assess erythrocyte mass. The plasma or serum can be evaluated visually, and the total protein can also be measured. Protocol 54.1 provides more details about preparing the microhematocrit tube. Briefly, the tube is filled to around 75% capacity, and one end is plugged with clay. The tube is centrifuged at 10 000 rpm for five minutes, which delineates the blood sample into three distinct layers within the tube: a layer of red blood cells adjacent to the end of the tube sealed with clay, a layer of white blood cells and platelets (also known as the "buffy coat" above the red blood cell layer), and the acellular plasma layer above the buffy coat (Figure 54.1). From this tube, parameters including the PCV, buffy coat, plasma appearance, and total protein can be evaluated.

Packed Cell Volume

There are many methods available to assess erythrocyte mass, including hematocrit, PCV, hemoglobin concentration and red blood cell concentration. PCV is the term used when the hematocrit is determined via centrifugation, evaluating the red blood cells as a percentage of blood volume. Results of PCV can be available quickly, and when performed appropriately are accurate and reproducible, making PCV determination ideal for serial monitoring of erythrocyte mass within patients. PCV will overestimate the hematocrit slightly, due to the trapping of plasma between red blood cells. Values for PCV typically are 2–3% higher than calculated hematocrit [2]. Refer to Protocol 54.1 for further details about performing a PCV measurement.

PCV values can be determined from a microhematocrit tube using of a variety of methods, including scales on the centrifuge, handheld cards, or specialized microhematocrit readers. One popular tube reader is a plastic sheet called a Critocaps™ chart (Critocap, Oxford Labware/Sherwood Medical, St. Louis, MO). With this reader, the centrifuged microhematocrit tube is placed perpendicular to the chart lines with the clay–erythrocyte interface on the zero line. The tube is moved along the chart until the 100% line intersects the plasma–air interface in the center of the meniscus in the microhematocrit tube. The PCV is read as a percentage directly from the chart at the erythrocyte–buffy coat interface (Figure 54.2).

PCV can also be obtained by dividing the length of red blood cell layer by that of all three layers of the microhematocrit tube (red blood cells, buffy coat, and plasma) and

Protocol 54.1 Packed Cell Volume Determination

Principle

Packed cell volume is the ratio of erythrocyte relative to whole blood, expressed as a percentage. This value can be used as a measure of erythrocyte mass in a patient to determine if they have normal erythrocyte mass, low erythrocyte mass (anemia) or elevated erythrocyte mass (erythrocytosis).

Specimen

Anticoagulated blood (e.g. EDTA)

Materials and Equipment

- Plain microhematocrit tubes (blue ring)
- Microhematocrit centrifuge
- Clay or tube sealing compound
- Microcapillary reader or hematocrit card
- Kimwipes® or gauze

Notes

The end of the microhematocrit tubes with the blue ring is fire-sealed by the manufacturer to prevent breakage during centrifugation. Always place the clay/sealant into the end with the blue ring.

Procedure

1) Correct mixing of blood is imperative. Samples should be thoroughly mixed, ideally for 5 minutes on a rocker, or by inverting the samples gently a minimum of 25 times.
2) Remove the rubber stopper of the EDTA tube, pointing the tube away from the face.
3) Insert the end of the microhematocrit tube without the blue ring, and fill the tube to 75% capacity. *Do not* fill the tube to capacity.
4) Gently wipe the outside of the tube to clean off any blood.
5) Seal the end of the microhematocrit tube with the blue ring with clay or sealing compound until the entire space under the blue ring is sealed.
6) Place the microhematocrit tube into the centrifuge with the sealed end toward the rubber gasket and balance the centrifuge with another sample or empty tube in the opposite position of the centrifuge.
7) Secure both lids of the centrifuge (inner screw lid and outer latch).
8) Centrifuge samples at 10 000 RPM for 5 minutes.
9) Once the centrifuge has stopped completely, remove the microhematocrit tube and read the packed cell volume on a capillary reader or card reader per the instructions below.

Operation

Reading the Packed Cell Volume Using a Hematocrit Card

1) Place the reading card on a flat surface.
2) Place the microhematocrit tube on the left side of the card, aligning the clay sealant/red blood cell interface with the line at the bottom of the card.
3) Slide the tube along the card, keeping the clay/red blood cell interface aligned with the bottom line, until the top of the plasma–air interface is aligned with the line at the top of the card.
4) Check to ensure both portions of the tube align with their respective lines.
5) Find the line that intersects the tube at the red cell/buffy coat interface. Do not include the buffy coat as part of the erythrocyte volume.
6) Read and record the value to the nearest whole number. Read the value from directly overhead to avoid parallax error.

Reading the Packed Cell Volume Using a Capillary Tube Reader (Rectangular Type)

1) Place the microhematocrit tube into the groove of the hematocrit reader.
2) Align the clay sealant/red blood cell interface with the line at the bottom of the reader.
3) Slide the microhematocrit tube until the plasma–air interface is aligned with the top line of the reader (corresponding to 100 on the scale).
4) Slide the horizontal bar up/down so that the line intersects at the red cell/buffy coat interface. Do not include the buffy coat as part of the erythrocyte volume.
5) Read and record the value from the scale to the nearest whole number. Read the value from directly overhead to avoid parallax error.

multiplying by 100 to obtain the percentage of blood volume of erythrocytes:

$$\frac{Length\ of\ erythrocyte\ layer\ in\ microhematocrit\ tube}{Length\ of\ all\ 3\ layers\ of\ microhematocrit\ tube} \times 100 = PCV(\%)$$

Buffy Coat

The buffy coat refers to the layer between the red blood cells and the acellular plasma. The buffy coat is typically a very narrow layer, but may be enlarged in patients with elevated white blood cell concentrations or platelet

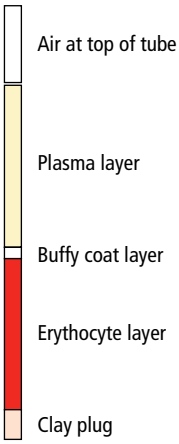


Figure 54.1 Diagram depicting the layers of the microhematocrit tube. Erythrocytes are spun to the bottom of the tube above the clay plug. A small clear layer sits above the erythrocytes known as the 'buffy coat' which contains leukocytes and platelets. Acellular plasma then sits atop the buffy coat.

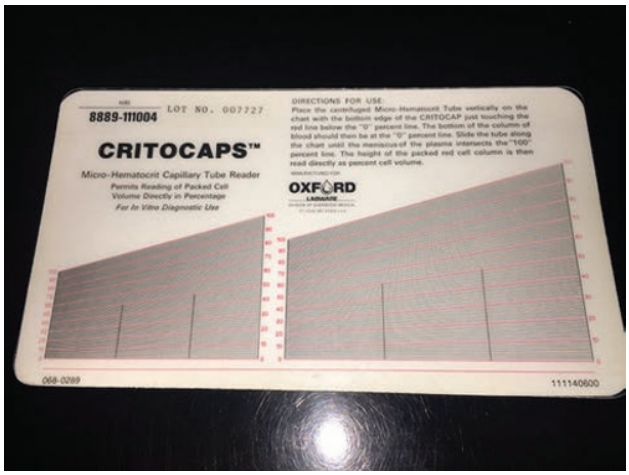


Figure 54.2 A common microhematocrit card reader.

concentrations. The buffy coat may also be difficult to see in patients with low white blood concentrations.

Plasma Appearance

The microhematocrit tube allows visualization of plasma, which may offer important clinical information. Plasma in dogs and cats is normally clear, and any discoloration of the plasma should be noted. Yellow discoloration may suggest hyperbilirubinemia, which can be seen secondary to liver disease or hemolysis. Red discoloration may also indicate intravascular hemolysis; however, this will not differentiate between pathologic intravascular hemolysis in the patient (e.g. secondary to IMHA or Heinz body anemia) and hemolysis that occurred due to traumatic venipuncture or prolonged storage of samples. White or opaque plasma is indicative of lipemia.

Total Protein

After PCV has been measured and the buffy coat and plasma have been visually inspected and reported, the microhematocrit tube is broken just above the buffy coat. A small amount of plasma is then placed on a refractometer to determine total protein. See Protocol 54.2 for details of total protein determination. Note that hemolysis or lipemia may falsely elevate total protein measurements.

Total Protein and Total Solids

The angle of refraction produced by a serum or plasma sample is due to the combined concentration of all its solutes, also called "total solids." Protein is the predominant solute; however, nonprotein substances such as urea, glucose, electrolytes and lipids contribute to the angle of refraction. Total protein and total solids are *not* interchangeable terms. In health, serum/plasma concentration of total solids is approximately 1.5–2 g/dl higher than the concentration of total protein. Almost every refractometer uses a conversion factor, such that they are measuring total protein and not total solids [3].

Maintenance of Refractometers

The zero scale can be tested using distilled water. This should be checked daily for non-temperature corrected models, and adjusted as needed. Temperature affects refractometer results, and the instruments should be kept at room temperature. The glass platform in the refractometer should be cleaned with distilled water between each measurement.

Fibrinogen

Fibrinogen can be measured in microhematocrit tubes, due to precipitation of fibrinogen exclusively when tubes are heated to 56°C (± 1°C) for three minutes [4]. The difference between the total protein of plasma from a normally processed microhematocrit tube, and that of one incubated under the above conditions gives an estimate of fibrinogen concentration. Protocol 54.3 gives details of the fibrinogen heat precipitation method. It is important to note that this technique is useful to determine elevations in fibrinogen but is not accurate in identifying low fibrinogen concentrations [5].

Blood Smear Preparation

For detailed instructions on blood smear preparation, please refer to Protocol 54.4. Some important points to keep in mind when preparing blood smears:

- Blood smears should be prepared soon after collection – ideally within one to two hours to prevent storage artifact.

Protocol 54.2 Plasma Protein Assessment with Refractometer

Principle

Proteins in solution alter the refractive index proportional to their concentration, allowing an estimation of plasma protein.

Specimen

Anticoagulated blood (e.g. EDTA)

Materials and Equipment

- Microhematocrit tube
- Microhematocrit centrifuge
- Clay or tube sealing compound
- Glass slide or etcher (for scoring)
- Refractometer calibrated for plasma protein reading
- Deionized water

Notes

Check refractometer daily with deionized water to verify reading of 1.000.

Procedure

1) Mix blood in EDTA tube thoroughly (gently invert at least 25 times).
2) Remove the rubber stopper of the EDTA tube, pointing the tube away from the face, and fill the microhematocrit tube to 75% capacity.
3) Seal the end of the tube with clay or tube sealing compound.
4) Place the microhematocrit tube in the centrifuge with the clay end toward the rubber gasket. Balance the centrifuge with another microhematocrit tube (empty or another sample). Secure both lids of the centrifuge (inner screw lid and outer latch).
5) Centrifuge samples at 10 000 RPM for 5 minutes.
6) Remove microhematocrit tube from the centrifuge and score the tube with a glass slide or etcher just above the buffy coat.
7) Using both thumbs and forefingers, place gentle, even pressure either side of the scored region away from the face to break the microhematocrit tube.
8) Using the end that was not scored (to avoid placing glass fragments on the refractometer), place 1–2 drops of plasma onto the glass measuring prism of the refractometer and close the cover plate immediately.
9) Press the cover plate gently with fingers to spread plasma evenly over the reading prism.
10) Point the instrument toward a bright light source.
11) Take the reading at the interface where the light and dark fields cross the scale.
12) Clean the prism surface with deionized water and dry with a soft cloth immediately after reading.

- Blood in EDTA tubes should be at room temperature prior to slide preparation and should be thoroughly mixed prior to removing blood by gently inverting the tubes at least 20 times.
- Blood smears should be dried immediately after being made to prevent contraction of red blood cells, which can impede evaluation of morphology (including echinocyte formation, or refractile inclusions).

A good quality blood smear will have three major components: a feathered edge, a monolayer, and a body.

1) The feathered edge should give the smear a bullet or thumbprint shape and have a fine, feathery appearance.
2) The monolayer is the area just behind the feathered edge, where 50% of red blood cells are touching their edges, while 50% of red blood cells are not touching other cells. It is imperative for a monolayer to be present for accurate cellular assessment, and it is in this area where cellular morphology and platelet estimates will be performed. If slides are too thick, red blood cell morphology is distorted, while leukocyte morphology will be affected in thin smears. Platelet estimates also critically require a monolayer to be present.
3) The body makes up the majority of the smear and should blend smoothly into the monolayer region.

Blood smears may lack the above components or suffer from other artifacts of preparation. These errors, as well as possible causes and solutions for these artifacts are summarized in Table 54.1.

Staining Samples

Romanowsky-type (Wright or Wright–Giemsa) are the most common stains used for blood films in veterinary medicine. These stains contain a mixture of eosin (staining red/pink) and oxidized methylene blue dyes (staining blue). Many rapid stains for in-house use also are available, including Diff-Quik® (Dade Behring Inc., Newark, DE) and Hema 3™ (Fisher Scientific, Pittsburgh, PA). Although methanol is used as the fixative, these are aqueous stains.

Blood films should be fixed in methanol within one to four hours (preferably within one hour) of preparation, and only when the slide is completely dry. Quality of rapid stains may

Protocol 54.3 Fibrinogen Heat Precipitation Method

Principle

Heating plasma to 56 ± 1°C will precipitate fibrinogen, but not albumin and other noninflammatory globulins.

Specimen

EDTA or citrate anticoagulated blood with no clots.

Materials and Equipment

- Plain microhematocrit tubes
- Clay or tube sealing compound
- Microhematocrit centrifuge
- Heat bath
- Thermometer
- Timer
- Glass slide or etcher
- Refractometer

Notes

- Heparinized samples cannot be used.
- Clotted samples cannot be used.
- Hemolysis or lipemia may give erroneous results.

Procedure

1) Mix blood in EDTA tube thoroughly (gently invert at least 25 times).
2) Remove the rubber stopper of the EDTA tube, pointing the tube away from the face, and fill 2 microhematocrit tubes to 75% capacity.
3) Seal the end of the tube with clay or tube sealing compound.
4) Place the microhematocrit tubes in the centrifuge with the clay end toward the rubber gasket in opposite positions to balance the centrifuge. Secure both lids of the centrifuge (inner screw lid and outer latch).
5) Centrifuge samples at 10 000 RPM for 5 minutes.
6) Remove microhematocrit tubes from the centrifuge.
7) Place one of the tubes into the heat bath and start timer for 5 minutes.
8) Immediately score the other microhematocrit tube with a glass slide or etcher just above the buffy coat and read the plasma protein using a refractometer (refer to Protocol 54.2). Record this value.
9) Repeat step 8 for the heated sample after the 5-minute incubation period.
10) Using the plasma protein readings from the unheated and heated samples, calculate the fibrinogen estimate per the calculation in the next section.

Calculation

Fibrinogen (mg/dl) = (unheated plasma protein [g/dl] – heated plasma protein [g/dl]) × 1000

be increased by allowing the blood film to remain in the fixative for one to two minutes. Slides cannot be overfixed; however, underfixation of slides may lead to cellular lysis or poor staining quality. See Protocol 54.5 for more details on using Romanowsky-type stains. It is important to note that aqueous stains may not stain mast cell and basophil granules well [6].

Stain abnormalities may include excessive pink or blue coloration of the cells, or excessive stain precipitation:

- Excessively pink: Low stain pH, inadequate staining time, degraded stains or excessive washing.
- Excessively blue: High stain pH, prolonged staining, insufficient washing [7].
- Stain precipitation: may be present if the slide was stained too long, if the slide was not washed sufficiently, or if the stains need to be filtered or replaced.

Reticulocyte stains are used to quantitate reticulocytes in circulation (i.e. they help to determine the regenerative response by the bone marrow in cases of anemia) and to highlight Heinz bodies in erythrocytes. These stains cause precipitation of ribosomal ribonucleic acid in immature red blood cells, which stains deep blue. These stains are commercially available but can also be produced by dissolving 0.5 g of new methylene blue (NMB) and 1.6 g of potassium oxalate in 100 ml of distilled water [8]. To prepare a slide with reticulocyte stains, equal volumes of blood and stain (following filtration to avoid stain precipitation artifact) are mixed together in a test tube and incubated at room temperature for 10–20 minutes. Short incubation times may result in poor staining and underestimation of reticulocyte counts and Heinz bodies. After incubation, blood slides are made and examined See Protocol 54.6 for more details on reticulocyte evaluation.

Blood Smear Evaluation

Blood smears should be evaluated in a routine fashion to ensure that all components are assessed. A good approach is to divide the blood smear into four components:

1) Background, quality and feathered edge
2) Erythrocytes
3) Leukocytes
4) Platelets.

Protocol 54.4 Blood Smear Preparation

Principle

Examination of a blood smear is an integral part of a complete blood count. Many important findings that can be seen on blood smear evaluation cannot be accurately identified by automated machines, including neoplastic cells, toxic changes within neutrophils, immature granulocyte precursors and infectious agents.

Specimen

Anticoagulated whole blood (EDTA preferred)

Materials and Equipment

- Latex or nitrile gloves
- Pencil
- Premium, precleaned glass microscope slides with a frosted edge (at least 3)
- ± Camelhair brush
- Anticoagulated blood in EDTA tube that is at room temperature and thoroughly mixed
- Plain hematocrit tube

Notes

- Latex or nitrile gloves should be worn when handling blood and making blood smears.
- Blood smears should be prepared soon after collection; ideally within 1–2 hours.
- If using blood from an EDTA tube:
  - Blood must be at room temperature. If the EDTA tube has been stored in the refrigerator, bring to room temperature prior to slide preparation.
  - Gently invert the tube around 20 times to thoroughly mix the blood immediately prior to slide preparation.

Procedure

1) Use a pencil to write the patient details on the frosted edge of the slide.
2) If not using premium, precleaned slides, brush the slide with a camelhair brush to remove any glass grit or debris.
3) Place the glass slide on a flat surface.
4) Fill a plain hematocrit tube with blood from the EDTA tube and place a finger over the end of the tube to prevent blood from dripping.
5) Place an approximately 3–5 mm diameter drop of blood at one end of the slide. Do not tap the hematocrit tube against the glass slide.
6) Use a finger on one hand to hold the slide in place. Hold a second spreader slide between the thumb and index finger of the dominant hand at a 30–45° angle,[a] in front of the drop of blood.
7) Back the spreader slide into the drop of blood, and as soon as the blood starts to spread along the edge of the spreader slide, push the spreader slide forward in a smooth, moderately fast motion. Use only enough pressure to keep the spreader slide on the glass slide.
8) Dry the slide quickly by waving it or blowing gently on the slide.

[a] This angle may be increased if blood is thin (e.g. in cases of severe anemia), or decreased if blood is thick (e.g. in cases of erythrocytosis) to create a better monolayer.

Background, Quality and Feathered Edge

It is important to assess the quality of the slide prior to evaluation of the various components. The slide should have the three major components (feathered edge, monolayer and body) and should be well stained. The background may offer important information about the patient. The background may have a deep blue or scalloped appearance in patients with high total protein concentrations, and pools of smooth blue material may be seen in patients with cryoglobulinemia, which can be seen secondary to some neoplasms such as multiple myeloma (Figure 54.3).

It is also important to evaluate the feathered edge of the slide, as many components (particularly large elements) of blood may be pushed to the edge of the smear. Infectious agents such as microfilaria from heartworm disease (*Dirofilaria immitis*) or *Hepatozoon* spp. frequently are pushed to the feathered edge (Figure 54.4). Platelet clumps as well as leukocyte clumps also are often seen at the feathered edge, and should be noted, as these may affect both automated results and estimates made on blood smears. Finally, large, atypical cells such as mast cells or neoplastic cells may also be seen more frequently at the feathered edge of the slide.

Erythrocytes

Erythrocyte Distribution

Evaluation of erythrocytes starts at lower magnification (e.g. 100× or 200× magnification) looking at distribution of the red blood cells. Patients with anemia (decreased red blood cell mass) frequently will have a long monolayer, and the body of the smear will be short. Patients with erythrocytosis (increased red blood cell mass) may have a very short,

Table 54.1 Artifacts of Blood Smear Preparation.

Problem	Potential cause	Potential fix
Too thin	Drop of blood too small	Increase size of blood drop
	Wrong spreader slide angle	Increase angle of spreader slide
Too thick	Drop of blood too big	Decrease size of blood drop
	Wrong spreader slide angle	Decrease angle of spreader slide
Blood pushed off edge of slide	Drop of blood too big	Decrease size of blood drop
Streaking (linear)	Slow spreading	Spread blood in smooth constant motion.
	Dried blood on spreader slide	Clear end of spreader slide if reusing for multiple smears
	Particulate matter on slide	Clean surface of slides used for blood smears
Streaking (horizontal)	Hesitation or irregular pressure when spreading	Push spreader slide in smooth, single motion
Uneven distribution of red blood cells	Excessive downward pressure	Apply only enough pressure to keep spreader slide on blood smear slide
Flat end of smear (no feathered edge)	Slow spreading	Spread blood in smooth, constant motion
	Abrupt end to motion of spreading	Push spreader slide to end of smear; do not stop abruptly during spreading
	Lifting spreader slide at end of motion	Do not lift up with spreader slide at the end of the smearing process
Narrow smear	Smear made before blood spread across spreader slide	Allow blood to spread along full edge of spreader smear
	Wobbling of spreader slide	Hold spreader slide stable with even pressure

Protocol 54.5 Romanowsky-Type Rapid Staining of Blood Smears

Principle

Romanowsky-type rapid stains can be used to highlight cytoplasmic and nuclear features of cells on peripheral blood smears.

Specimen

Completely dry blood smear prepared on a glass slide.

Materials and Equipment:

- Clean benchtop with absorbent pads
- Timer
- Alcohol fixative solution
- Romanowsky rapid stains (eosin and methylene blue)
- Forceps
- Distilled water
- Lint-free wipes (e.g. Kimwipe®) or gauze

Notes

- Ensure that the blood smear is completely dry prior to staining.
- Slides cannot be overfixed, but they can be underfixed. Leave slides in the fixative solution for 1–2 minutes to improve staining quality.
- The number of dips and time spent in the eosin and methylene blue stains can be tailored to the needs of the sample and observer to increase or decrease stain intensity.
- Ensure clean stain solution is used to prevent stain precipitation or contamination artifact.

Procedure

1) Hold the slide with forceps and dip it into the fixative solution multiple times to completely cover the slide.
2) Leave the slide in the fixative solution and set timer for 1–2 minutes.
3) After the timer alarms, use forceps to remove the slide from the fixative and allow excess fixative to drain by holding the slide onto an absorbent pad.
4) Use forceps to dip the slide into solution 1 until the stain completely covers the slide. Dip the slide a further 4–6 times for approximately 1 second per dip.
5) Allow excess stain to drain from the slide by using forceps to hold the slide onto an absorbent pad.
6) Repeat steps 4 and 5 for solution 2.
7) Rinse the slide thoroughly using distilled water.
8) Stand the slide vertically to dry with the feathered edge facing upward. The backside of the slide may be wiped with a Kimwipe or gauze to speed the drying process.

Protocol 54.6 New Methylene Blue Staining for Reticulocytes and Heinz Bodies

Principle

Methylene blue reagent in supravital stains precipitates aggregates of RNA. These aggregates are visible under the microscope as dark blue granules within immature red blood cells (RBC), and are classified as reticulocytes. Increased numbers of such cells in circulation indicates release of immature red blood cells by the bone marrow, and typically a regenerative response by the bone marrow. Such stains also stain aggregates of oxidized hemoglobin (Heinz bodies).

Specimen

Anticoagulated sample (EDTA preferred).

Reagents

- Commercially prepared reticulocyte stain (NMB). Reagent contains 1.6% (w/v) reagent grade potassium oxalate monohydrate, 0.5% (w/v) NMB in distilled, reagent grade water.
- Mix reagent well prior to use. Filtering the reagent is preferable – use Whatman #4, 11-inch paper.
- Periodic filtering may be required if increased precipitation is seen in stored solution.

Notes

- Optimal results obtained on fresh samples (refrigerated < 48 hours old).
- Gently mix NMB stain prior to use.

Materials and Equipment

- Microscope slides
- Disposable pipettes
- Glass or plastic test tubes
- Timer
- Microscope with 100× oil objective
- Immersion oil
- Manual hand counters (×2)

Procedure

1) Place 1 drop of reticulocyte stain into a test tube.
2) Add 1 drop of well-mixed patient blood to the test tube containing reticulocyte stain and mix thoroughly.
3) Set the timer for 10 minutes and allow the mixture to sit.
4) After 10 minutes, mix the contents of the test tube again.
5) Use a clean pipette and place 1 small drop of well-mixed sample onto a glass slide and prepare a blood smear per Protocol 54.4.
6) Allow slide to dry completely.

Counting Reticulocytes

- Mature erythrocytes have a homogeneous blue/green appearance.
- Immature erythrocytes will contain RNA that will appear as fine dots (punctate reticulocytes) or larger strings of granules (aggregate reticulocytes).
- Count both punctate AND aggregate reticulocytes in dogs. Count only aggregate reticulocytes in cats.

Procedure

1) Scan the dried, stained smear on low power (20× objective) to find a monolayer of erythrocytes.
2) Using the 100× oil objective, count the number of reticulocytes seen per 250 erythrocytes counted. Use one manual hand counter to tally the total number or red blood cells counted, and another to tally the number of reticulocytes counted. Record this number.
3) Move to a different monolayer area of the slide and repeat this count.
4) Repeat steps 2 and 3 until a total of 1000 cells has been counted.
5) Calculate the percentage of reticulocytes using the following formula:

$$\%reticulocytes = \frac{\#\,reticulocytes}{1000} \times 100$$

or use the following condensed formula:

$$\%\text{reticulocytes} = \left(\#\,\text{reticulocytes} / 1000\,\text{RBC}\right) \times 0.1$$

6) The absolute reticulocyte concentration is much more meaningful than percentage, and can be calculated as follows:

$$\text{absolute reticulocyte concentration} = \text{RBC count}\left(10^6/\mu\text{l}\right) \times \%\text{reticulocytes}$$

Counting Heinz Bodies

1) Scan the dried smear on low power (20× objective) to find a monolayer of erythrocytes.
2) Using the 100× oil objective, count the number of Heinz bodies seen per 100 erythrocytes. Use one manual hand counter to tally the number of red blood cells counted, and another to tally the number of Heinz bodies seen.
3) Move to a different monolayer area of the slide and repeat the count.
4) Repeat steps 2 and 3 until a total of 400 cells has been counted.
5) Calculate the percentage of Heinz bodies:

$$\%Heinz\ bodies = \frac{\#\,Heinz\ bodies}{400} \times 100$$

or use the following condensed formula:

$$\%\text{Heinz bodies} = \left(\#\,\text{Heinz bodies} / 400\text{RBC}\right) \times 0.25$$

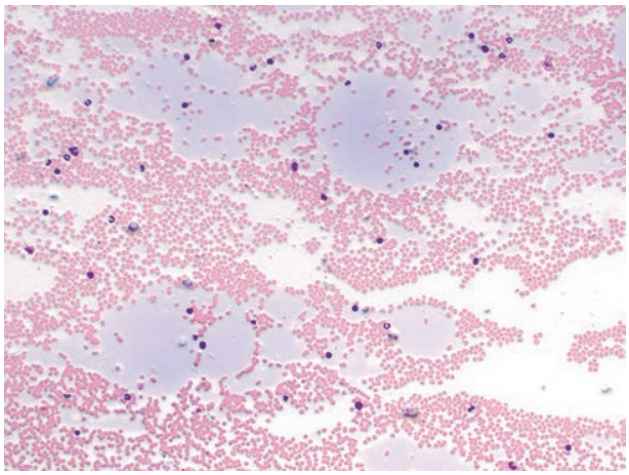


Figure 54.3 Large pools of pale blue cryoglobulin can be seen interspersed between erythrocytes and leukocytes. Cat blood, 200× magnification, Wright-Giemsa.

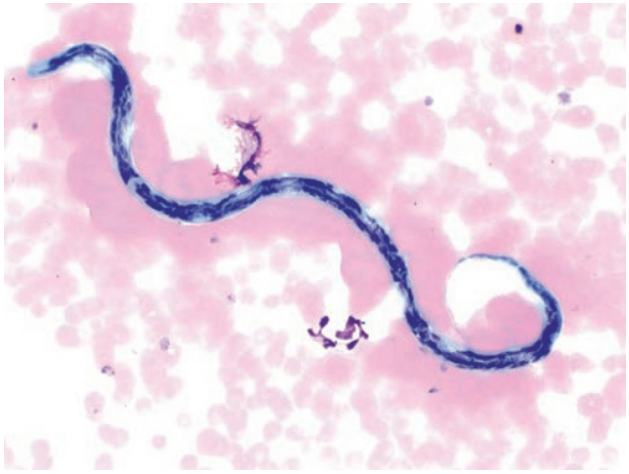


Figure 54.4 *Dirofilaria immitis* microfilaria. Dog blood 500× magnification, Wright-Giemsa.

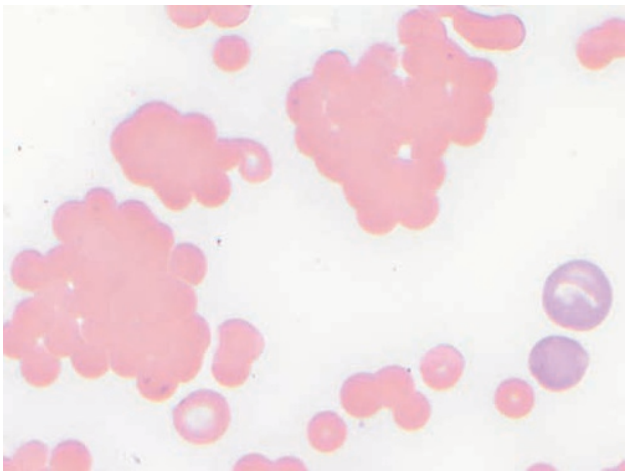


Figure 54.5 Agglutination in a dog with immune-mediated hemolytic anemia. Note the clumping of red blood cells, giving them the appearance of bunches of grapes. Dog blood, 1000× magnification, Wright-Giemsa.

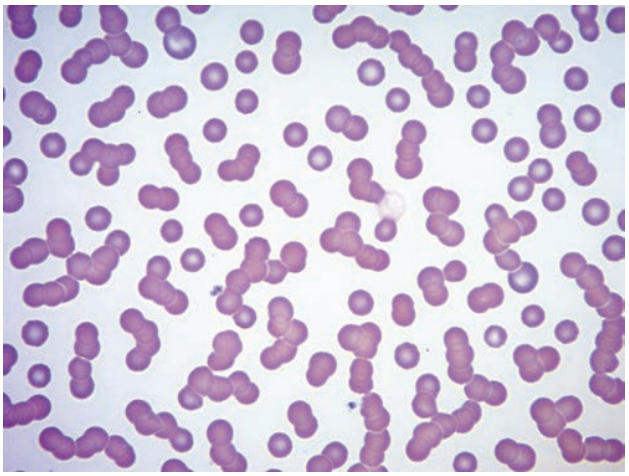


Figure 54.6 Rouleaux formation. Note the distribution of red blood cells in long chains, giving them the appearance of stacks of coins. The total protein in this patient was 10 g/dl. Dog blood, 500× magnification, Wright-Giemsa.

or non-existent monolayer. This may be helpful as a qualitative assessment of erythrocyte mass if PCV is not available. Also important is to evaluate for any agglutination or rouleaux formation. Agglutination is characterized by dense aggregation of red blood cells often with the appearance of "bunches of grapes" (Figure 54.5). Agglutination indicates the presence of surface-bound immunoglobulins to erythrocytes, usually immunoglobulin (Ig)M type due to the greater number of binding sites, and distance between these binding sites (vs. other immunoglobulins such as IgG) [9]. Rouleaux on the other hand is characterized by linear arrangements of red blood cells, like linear stacks of coins (Figure 54.6). Mild rouleaux can be normal in cats. Prominent rouleaux in cats, and any rouleaux in dogs, is most commonly associated with hyperglobulinemia.

The distinction between agglutination and rouleaux is not always easy by visual inspection, and a saline agglutination test may be performed (see Protocol 54.7 for details of this procedure). A rapid way to differentiate rouleaux from agglutination is to mix five drops of 0.9% NaCl with a drop of anticoagulated blood on a glass slide, with a coverslip placed on top. This slide is then examined as a wet mount under the microscope. Dilution with saline will disperse red blood cells individually if rouleaux is present, but agglutination will persist.

Erythrocyte Morphology

It is essential to evaluate erythrocyte morphology within the monolayer of the slide, where half the erythrocytes are touching each other and half are not touching other red blood cells. The morphology of erythrocytes can reveal a

Protocol 54.7 Saline Agglutination Test

Principle

Agglutination refers to clumping of red blood cells. Autoagglutination is caused by surface-bound immunoglobulins. Rouleaux formation refers to stacking of red blood cells (RBC), which is commonly due to increased concentrations of fibrinogen or globulins. Cats may have increased rouleaux formation normally. Agglutination and rouleaux formation can be difficult to differentiate on blood smears. Agglutination may cause erroneous automated erythrogram results including decreased RBC and increased MCV.

Specimen

Anticoagulated whole blood (EDTA preferred).

Reagent

Phosphate buffered normal saline.

Materials and Equipment

- Glass slides and coverslips
- Disposable pipettes
- Microscope
- Glass or plastic test tubes

Notes

- It may be useful to create a coverslip slide with 1 drop of undiluted blood to compare with the results of the dilution.
- If agglutination is confirmed, it may be appropriate to heat the EDTA sample to 37°C for 15 minutes prior to running (or rerunning) through automated machines.

Procedure

1) Label the test tube and glass slide with patient information.
2) Use a pipette to place 1 drop of well-mixed anticoagulated blood into the test tube.
3) Use a new pipette to place 4 drops of saline into the test tube.
4) Gently mix the blood and saline mixture.
5) Place 1 drop of the 1:5 dilution onto the glass slide, and cover with a coverslip.
6) Examine the slides using the 10× and 40× objectives on the microscope.

Interpretation

- If red blood cells disperse and are no longer clumped, rouleaux is present and the saline agglutination test is said to be negative.
- If red blood cell clumping persists, or if red blood cells separation is unclear, a 1:10 dilution may be performed. This may be required in patients with marked hyperglobulinemia.
- If red blood cell clumping does not disperse after a 1:10 dilution, the sample is positive for autoagglutination.

wide range of pathology within the critically ill patient. Some of the most important morphologic changes are discussed below.

- Normal morphology:
  Erythrocytes in both dogs and cats are anucleate, biconcave cells that appear round. In dogs, normal erythrocytes have central pallor, which should take up about one third of the cell. Normal erythrocytes of cats lack any central pallor. Canine red blood cells are approximately 6–8 μm in diameter; feline red blood cells are smaller but have more size variation normally.
- Polychromasia:
  Polychromatophils appear as blue-tinged erythrocytes when stained with routine hematologic stains, owing to the presence of increased residual ribosomes and polyribosomes, indicating immature cells. These cells are counted as reticulocytes by automated machines. Presence of these cells in circulation typically indicates a regenerative response by the bone marrow toward anemia, which is helpful as it generally indicates that the anemia is due to either destruction of erythrocytes (e.g. hemolysis) or hemorrhage. The presence of polychromasia on a blood smear is more accurate at predicting a bone marrow regenerative response than red blood cell indices from automated machines such as mean corpuscular volume or mean corpuscular hemoglobin concentration [10]. It takes approximately three to five days for a regenerative response by the bone marrow, so the absence of polychromatophils does not preclude an emerging regenerative response.

Reticulocyte Evaluation

Protocol 54.6 provides details on preparing slides for reticulocyte evaluation. To determine the reticulocyte percentage, 1000 erythrocytes are examined. The cells are examined for any blue granular inclusions ("reticulum") indicative of precipitation of ribosomal RNA present in immature red blood cells. As red blood cells mature, the amount of this material decreases from coarse aggregates (known as aggregate reticulocytes), to fine stippled inclusions (known as punctate reticulocytes). In dogs, mostly

aggregate reticulocytes are typically present, and correlate closely to the number of polychromatophils that would be seen in slides stained with Romanowsky-type stains. In cats; however, it is important to classify reticulocytes as either aggregate or punctate type, because maturation of reticulocyte to mature erythrocytes is slower in cats than dogs. The number of aggregate reticulocytes will still correlate closely with the number of polychromatophils seen on Romanowsky-type stained slides, but punctate reticulocytes will not stain as polychromatophils, despite indicating a regenerative response.

Once determined, the percentage of reticulocytes can be multiplied by the red blood cell count (RBCC) to determine a concentration of reticulocytes in circulation. Manual erythrocyte counts are not accurate enough to be useful.

Poikilocytosis

Poikilocytosis is the term used to describe any deviation in the normal shape of erythrocytes. It is important to review red blood cell morphology on 1000× magnification. There are many classic shape changes linked to underlying pathophysiology. Some are of particular importance in critically ill patients, and are described in detail below.

Spherocytes

Spherocytes are smaller than normal erythrocytes, lack central pallor, and have dense coloration (Figure 54.7). They form due to loss of cell membrane, that frequently occurs in cases of IMHA, in which spherocytosis is a hallmark finding [11]. Spherocytes are not pathognomonic for IMHA, however, and may also be seen (albeit less commonly) in cases of zinc toxicity [12], bee sting or snake envenomation [13, 14], or secondary to some hemic parasites [15]. It is essential to evaluate for spherocytes within the monolayer of the slide. Evaluation of red blood cell morphology too close to the feathered edge may result in a false positive diagnosis of spherocytes, as red blood cells are able to sphere and lose central pallor toward the feathered edge.

Schistocytes

Schistocytes are fragments of red blood cells, often appearing as elongated cells with sharp extremities (Figure 54.8). They can be seen secondary to microangiopathy associated with disseminated intravascular coagulation (DIC) in dogs, and less commonly in cats [16, 17]. Importantly, the absence of schistocytes does not preclude DIC, and other diagnostic tests such as platelet concentration, D-dimer levels, activated partial thromboplastin time (aPTT)/prothrombin time and fibrinogen concentration should be evaluated if there is suspicion for DIC [18]. Schistocytes can also be seen in other disease states, such as liver disease, iron deficiency anemia, and glomerulonephritis.

Leptocytes

Leptocytes have an increased surface membrane area to volume ratio, which often gives them a folded appearance. A classic form of leptocyte is a codocyte or "target cell". These cells have a central region of density, giving a "bulls eye" appearance. Low numbers of target cells can be normal in dogs, but are seen in increased numbers in regenerative anemias, especially as polychromatophils may appear as target cells. They are also seen in increased numbers in iron deficiency, and rarely in cases of liver disease.

Inclusions

It is important to look for any inclusions within erythrocytes, as these can have important clinical significance. It is also important to be able to distinguish true inclusions

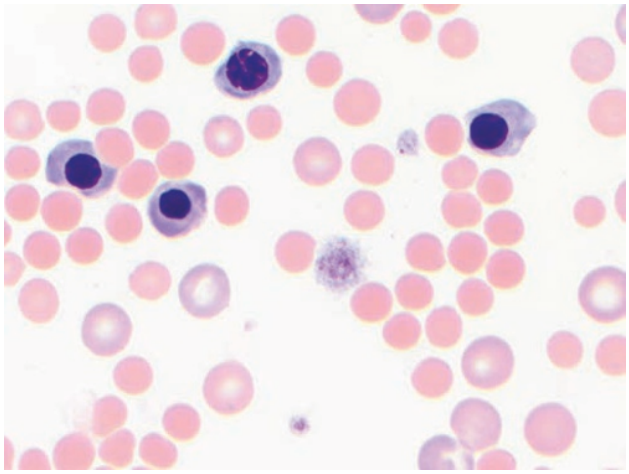


Figure 54.7 Spherocytes from a dog with immune-mediated hemolytic anemia. Note that the spherocytes that have no central pallor, and are smaller and more dense than other red blood cells. Dog blood, 1000× magnification, Wright-Giemsa.

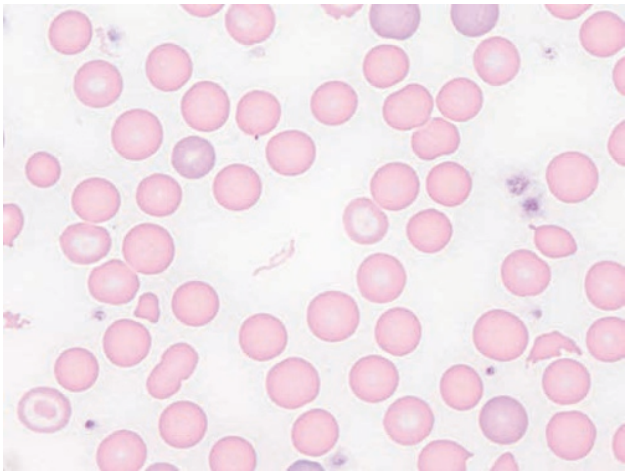


Figure 54.8 Schistocytes. Note the schistocyte in the middle of the image as a fragmented cell with sharp borders. Dog blood, 1000× magnification, Wright-Giemsa.

from artifacts, such as stain precipitation that might cover cells, or refractile inclusions that can result if blood smears are not dried quickly, or if they are still wet during staining. A good rule to keep in mind is that true inclusions will always be in the same plane of focus as the rest of the red blood cell and will never be refractile if moving the fine focus of the microscope up and down.

Heinz Bodies

Heinz bodies often appear as small round projections from the side of the cells, but may also be seen as pale, round inclusions within the cell (if viewed from above) (Figure 54.9). These inclusions represent aggregates of precipitated, oxidized hemoglobin, and hence are the same color, or slightly paler than that of a normal red blood cell when using routine Romanowsky-type stains. Heinz bodies stain deep blue using reticulocyte stains such as NMB, which can make them much easier to visualize and quantitate (Figure 54.10).

Feline hemoglobin has eight reactive sulfhydryl groups (compared with four in canine hemoglobin), predisposing this species to oxidative damage [19]. Additionally, cats have non-sinusoidal spleens that are less able to remove denatured hemoglobin [20]. Heinz bodies may therefore be present in up to 5% of erythrocytes in normal cats [21].

Oxidative damage can result from exposure to some drugs or toxins, including acetaminophen [19], zinc, propylene glycol or ingestion of garlic or onions, as well as in some disease conditions such as diabetes/diabetic ketoacidosis [21], hyperthyroidism, and secondary to lymphoma [22].

Howell–Jolly Bodies

Howell–Jolly bodies should not be confused with Heinz bodies and are easily distinguishable. They appear as small, round, densely purple inclusions even using routine Romanowsky-type stains and represent small remnants of nuclear material; hence, their other name of micronuclei (Figure 54.11). Howell–Jolly bodies are normally removed by the spleen as red blood cells pass through inter-endothelial slits of the splenic sinuses. Low numbers may be an incidental finding (especially in cats, due to a non-sinusoidal spleen), and increased numbers may be present in cases of regenerative anemia, post splenectomy, or secondary to some drugs including glucocorticoids or chemotherapeutic agents [23].

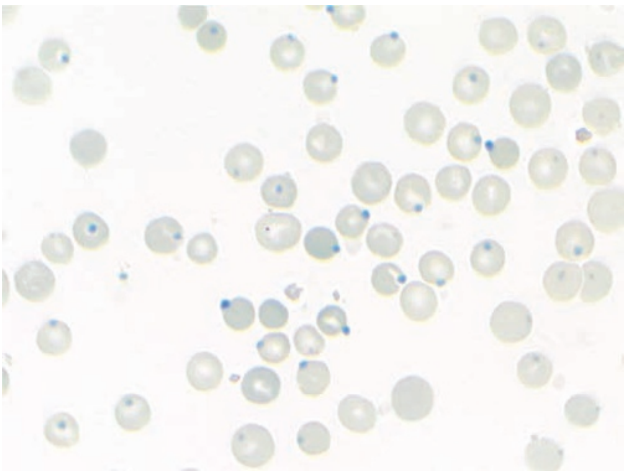


Figure 54.10 Heinz bodies, new methylene blue stain. The stain highlights the Heinz bodies seen in Figure 54.9. Cat blood, 1000× magnification.

Parasites

Many important infectious agents may affect erythrocytes of dogs and cats, and it is important to evaluate red blood cells closely at 1000× magnification, especially in cases of

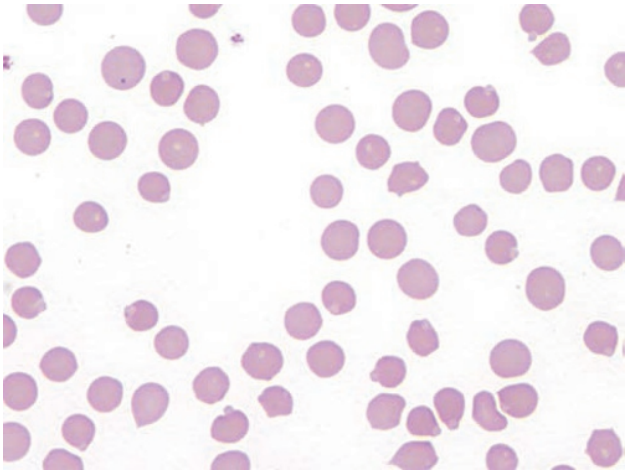


Figure 54.9 Heinz bodies. Numerous Heinz bodies are present, seen both as small round projections from the side of some cells, and as clear round inclusions overlying other red blood cells. Cat blood, 1000× magnification, Wright-Giemsa.

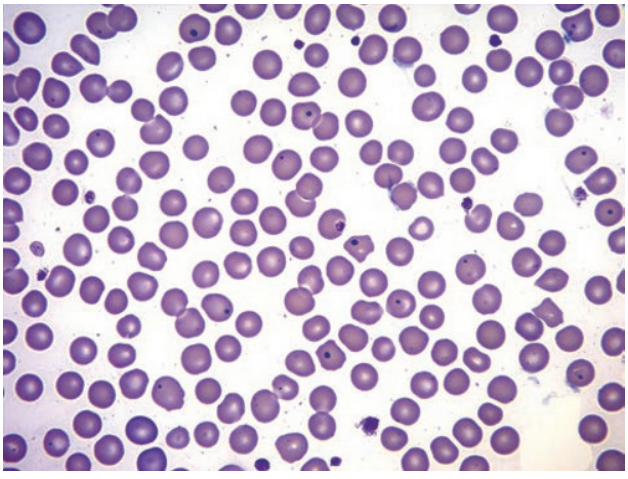


Figure 54.11 Howell–Jolly bodies. Numerous red blood cells contain small, densely blue, round inclusions knows as Howell–Jolly bodies. Dog blood, 500× magnification, Wright-Giemsa.

regenerative anemia. These may be intracellular (including protozoa such as *Babesia* spp. (Figure 54.12), *Cytauxzoon felis* (Figure 54.13) or viral inclusions (e.g. associated with Distemper in dogs), or may be epicellular (such as bacterial *Mycoplasma* spp.). As mentioned previously, it is important to differentiate infectious agents from artifacts such as stain precipitation or refractile artifacts.

Nucleated Red Blood Cells

Mature red blood cells lack nuclei, but immature red blood cells in the bone marrow have nuclei. As the cells mature, the nucleus becomes smaller and more condensed, prior to the cells losing nuclei before release from the bone marrow. Nucleated red blood cells can be difficult to differentiate from lymphocytes. Metarubricytes have condensed nuclei and a moderate rim of hemoglobinized cytoplasm, while rubricytes have round nuclei with clumped chromatin, and the cytoplasm may be deep blue (basophilic rubricytes) or the same color as polychromatophils (polychromatophilic rubricytes) (Figure 54.14). Rarely, very young, immature red blood cell precursors may be present, and the presence of rubriblasts may indicate an erythroleukemia, particularly in cases of non-regenerative anemia [24]. A mild increase in nucleated red blood cells can be seen with regenerative anemia; however, large numbers in circulation indicate a breakdown of the blood/bone marrow barrier, which can be seen secondary to hypoxia (associated with severe anemia), drug or toxin exposure (including lead poisoning and chemotherapeutic agents) [25], heatstroke [26], or primary bone marrow disease, including neoplastic and infectious etiologies. The presence of nucleated red blood cells is also associated with increased mortality in acute trauma patients [27].

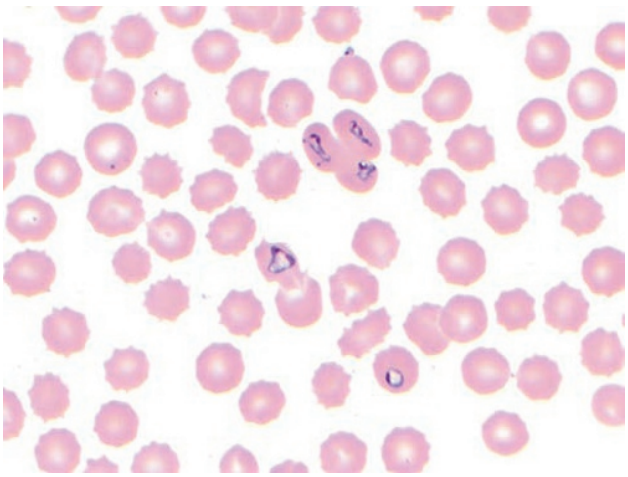


Figure 54.12 *Babesia canis*. Teardrop-shaped *B. canis* organisms are seen within some erythrocytes. Dog blood, 1000 × magnification, Wright-Giemsa.

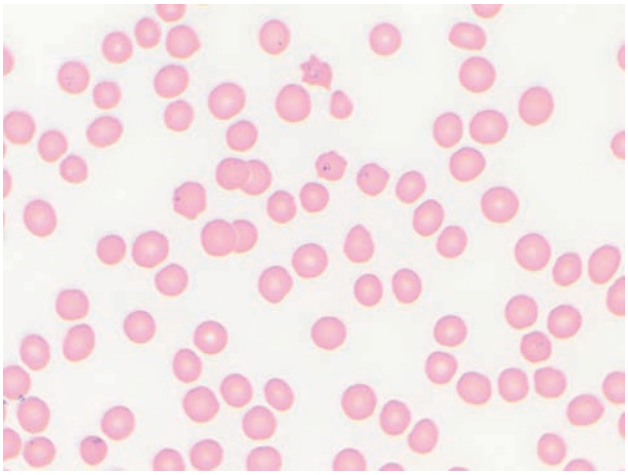


Figure 54.13 *Cytauxzoon felis*. Small, round *C. felis* organisms are seen within red blood cells. Cat blood, 1000 × magnification, Wright-Giemsa.

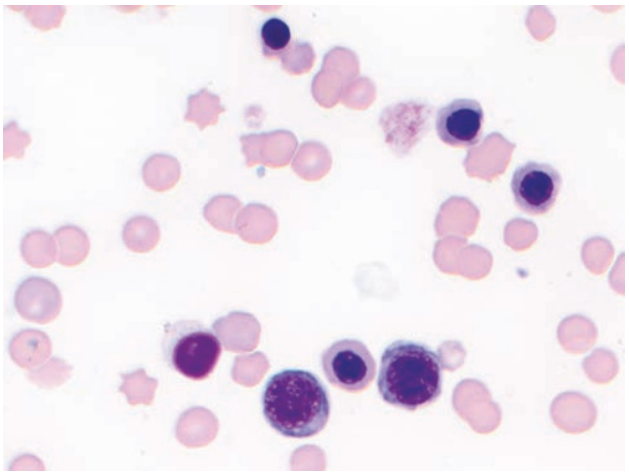


Figure 54.14 Nucleated red blood cells, including metarubricytes, polychromatophilic rubricytes and basophilic rubricytes. Dog blood, 1000 × magnification, Wright-Giemsa.

Leukocytes

Estimating the Leukocyte Concentration

Estimation of the leukocyte concentration can be performed on a blood smear. The estimation should be performed in the monolayer of the slide. The estimation can be performed on any microscope objective where the observer feels they can most accurately count the number of leukocytes present. If there are very few leukocytes present (e.g. leukopenia), a lower objective should be chosen (e.g. 20 × objective) so that the estimate is not underestimated. Conversely, in cases of leukocytosis, a higher objective (e.g. 40 × or 100 × objective) should be chosen to avoid double counting of cells and for efficiency of the procedure. Within the monolayer, count the number of leukocytes present per field. Repeat this step for a total of 10 fields to account for any variation in leukocyte distribution

across the slide, and then divide the sum of leukocytes in all 10 fields by 10 to obtain the average number of leukocytes per field. This value is then multiplied by the square of the objective on which the leukocytes were counted (Eq. 54.1):

$$\frac{Sum\ of\ leukocytes\ in\ ten\ 1000x\ magnification\ fields}{10} \times \left(objective\right)^2 = Estimate\ of\ leukocyte\ concentration\left(/l\right) \quad (54.1)$$

For example, if there is an average of 13 leukocytes counted per field on the 40× objective, the leukocyte concentration estimate would be 13 × 1600 = 20 800 cells/μl.

Manual Total Leukocyte Counts

Manual total leukocyte counts can also be performed using commercially available kits using reservoirs and pipettes to dilute samples and lyse red blood cells prior to counting cells in a hemocytometer chamber. Such counts require special equipment and training and also are labor/time intensive. In an emergency or critical care setting, such procedures may not offer any more clinically useful information than the leukocyte estimates outlined above, especially in cases of marked leukopenia or leukocytosis when animals likely are to be the most critical.

Performing a Leukocyte Differential

In addition to an appreciation of the total number of leukocytes present in circulation, the types of cells present is also critically important. If possible, a 200-cell differential (vs. a 100-cell differential) should be performed to increase the accuracy of the differential [28]. This may not be possible in cases of severe leukopenia. Additionally, manual differential counts are inherently imprecise. It is important to scan the entire slide to gain an appreciation of the types of leukocytes present, as well as any clumping of cells which may affect the differential [29].

The differential should be performed in a systematic fashion within the monolayer to avoid counting the same leukocytes multiple times. The differential is performed by identifying 200 consecutive leukocytes, and should include cells from both the center and edges of the slide to account for different distribution of cell types. After completion of the differential, the percentages of each cell type are multiplied by the total leukocyte concentration to obtain the absolute number per microliter of blood.

To perform an accurate differential, the observer must be able to accurately identify granulocytes (neutrophils, eosinophils, basophils) and mononuclear cells (lymphocytes and monocytes). In addition to normal morphology of these cells, there are important morphologic changes that frequently are seen in emergency or critically ill patients that must be identified. These include a left shift and toxic changes within the neutrophil line, infectious agents, and neoplastic cells. These will be described in more detail below.

Neutrophil Morphology

Two important morphologic changes that should be assessed in neutrophils include immature neutrophils (left shift) and toxic changes.

Left Shift

Neutrophils are produced in the bone marrow from maturation of precursor cells, starting with myeloblasts, and maturing through promyelocytes, myelocytes, metamyelocytes, band neutrophils to mature segmented neutrophils. Neutrophils are released into circulation from a storage pool of cells, that in dogs is equal to approximately a five-day supply of neutrophils based on normal rates of utilization [30]. The presence of immature neutrophils such as bands, or even earlier precursors, in circulation is referred to as a "left shift" and indicates that the stimulus for neutrophils from the bone marrow exceeds what the bone marrow can release from storage pools alone. Left shifts typically indicate underlying inflammation in the patient, and this finding should prompt investigation for a nidus of inflammation or infection.

- Degenerative left shift:
  A degenerative left shift refers to when the number of granulocytic precursors (bands, metamyelocytes, myelocytes, etc.) exceed that of mature neutrophils in circulation. The presence of a degenerative left shift suggests an exhausted supply of leukocytes and is associated with an increased risk of death or euthanasia in hospitalized dogs and cats [31, 32].
- Toxic changes:
  Toxic changes suggest dysplasia from accelerated granulopoiesis, most frequently associated with inflammation. Changes mostly affect the cytoplasm, and may include Döhle bodies, vacuolation, and increased basophilia (Figure 54.15). Giant neutrophils may also be seen. Toxic changes in neutrophils have diagnostic and prognostic significance. They can be helpful to differentiate inflammation from redistribution of neutrophils (e.g. neutrophilia associated with a corticosteroid or epinephrine response) when bands are absent. The presence of toxic changes is associated with increased hospitalization time in both dog and cats, and increased severity of toxicity confers an increased risk of mortality in hospitalized dogs [33, 34]. It is important to note that automated machines, including those at diagnostic laboratories, do not reliably evaluate neutrophils for toxic changes, and evaluation of a blood smear is essential.

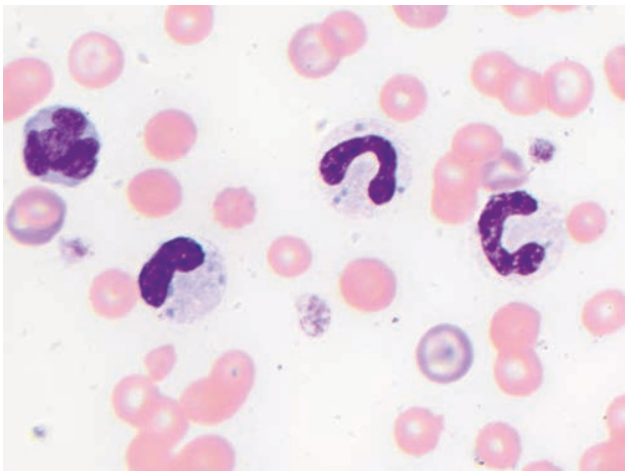


Figure 54.15 Band and metamyelocyte neutrophils with moderate toxic changes including Dohle bodies and increased cytoplasmic basophilia. Dog blood, 1000 × magnification, Wright-Giemsa.

Infectious Agents

Infectious agents may be seen within leukocytes, and careful examination of the slides at 1000 × magnification is recommended. Some infectious agents that may be seen include Rickettsial organisms, other bacteria (such as cases of bacteremia), viral inclusions (e.g. distemper), and even fungal organisms.

Neoplastic Cells

Neoplastic cells may be seen in circulation. These cells may be large, immature and blastic, such as in the case of the leukemic phase of large cell lymphoma, or cases of acute leukemia (Figure 54.16). They may also be small; however, such as in cases of chronic lymphocytic leukemia, where lymphocyte concentrations may exceed 1 000 000 cells/μl, and this disease is mostly indolent and slowly progressive [35] (Figure 54.17).

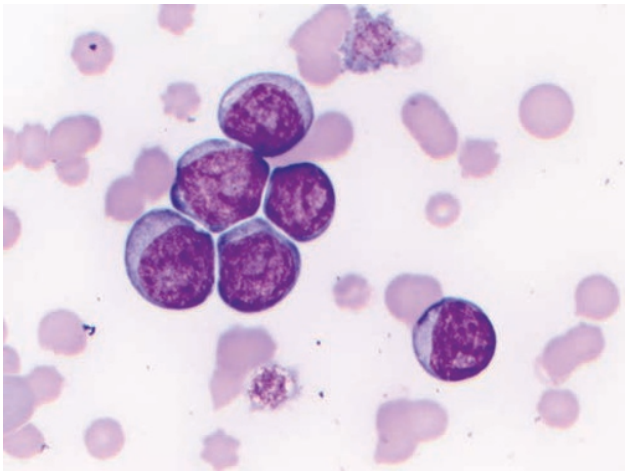


Figure 54.16 Acute lymphoid leukemia. Large, immature lymphocytes in circulation in a case of acute lymphoid leukemia. Dog blood, 1000 × magnification, Wright-Giemsa.

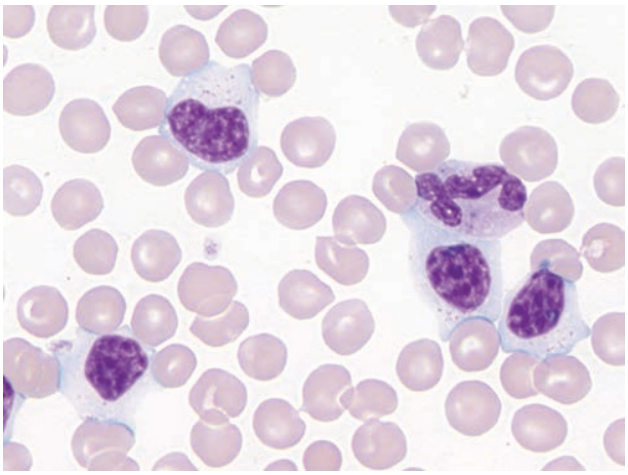


Figure 54.17 Chronic lymphocytic leukemia. A monomorphic expansion of small, mature lymphocytes is present, with a perinuclear packet of granules in a case of chronic lymphocytic leukemia. Dog blood, 1000 × magnification, Wright-Giemsa.

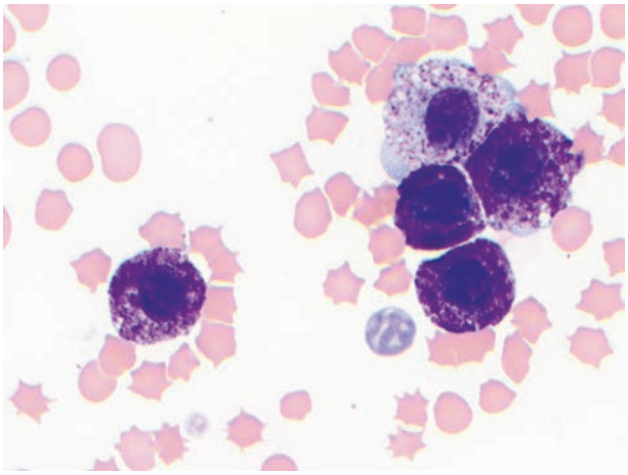


Figure 54.18 Mast cells in circulation (mastocytemia) in a cat with visceral mast cell neoplasia. Cat blood, 1000 × magnification, Wright-Giemsa.

Mast cells may be present in circulation, and evaluation of a buffy coat preparation may be useful to increase the chance of finding mast cells (Figure 54.18). For more information about preparing buffy coat smears, see Protocol 54.8. The significance of mast cells in circulation (mastocytemia) differs dramatically between cats and dogs. In cats, mastocytemia is most commonly associated with mast cell neoplasia such as visceral or cutaneous mast cell neoplasia. Rarely, mastocytemia may be associated with other neoplasms such as lymphoma, or with non-neoplastic conditions such as chronic renal disease [36]. Conversely, mastocytemia in dogs is most commonly associated with inflammatory disease, and only rarely indicate underlying mast cell neoplasia [37].

If any atypical cells are seen, it is recommended that slides and blood are submitted for review by a board-certified clinical pathologist.

Protocol 54.8 Buffy Coat Slide Preparation

Principle

Buffy coat preparations concentrate nucleated cells. This may facilitate the identification of mast cells, other atypical nucleated cells, or infectious agents such as Rickettsial bacteria.

Specimen

Anticoagulated whole blood (EDTA preferred); less than 24 hours old.

Materials and Equipment

- Microhematocrit tubes
- Clay or tube sealing compound
- Microhematocrit centrifuge
- Glass slide or etcher
- Glass slides

Procedure

1) Proper mixing of blood is imperative. Samples should be thorough mixed, ideally for five minutes on a rocker, or by inverting the samples gently a minimum of 25 times.
2) Remove the rubber stopper of the EDTA tube, with the tube pointing away from the face.
3) Fill 4 microhematocrit tubes to 75% capacity.
4) Gently wipe the outside of the tube to clean off any blood.
5) Seal the end of the microhematocrit tube with clay or sealing compound.
6) Place the microhematocrit tubes into the centrifuge with the sealed end toward the rubber gasket and balance the centrifuge with the tubes on opposite sides.
7) Secure both lids of the centrifuge (inner screw lid and outer latch).
8) Centrifuge samples at 10 000 RPM for 5 minutes.
9) Use a glass slide or etcher to score the tubes just below the white blood cell and platelet (buffy coat) layer.
10) Place gentle pressure at the score line to break the tube, breaking in the direction away from the face.
11) Tap the section of the microhematocrit tube with the white blood cell layer gently onto a clean glass slide, mixing cells and a small amount of plasma.
12) Make a pulled smear by placing a second clean glass slide on top of the slide with the white blood cell–plasma mixture and gently pull the top spreader slide over the bottom slide to create a monolayer of cells.
13) Repeat steps 9–12 for all microhematocrit tubes to make several buffy coat smears.
14) Allow the slides to completely dry before staining the slides per Protocol 54.5.

Platelets

Enumeration of platelet concentration and assessment of platelet adequacy is a critical component of evaluating primary hemostasis in critically ill patients, especially those with clinical signs such as petechiae on the skin, or bleeding from mucosal surfaces. Platelets may clump, causing spuriously low results on automated machines, and evaluation of a blood smear should be performed in every patient with a low automated platelet concentration.

Evaluation of platelet adequacy on a blood smear begins with scanning of the feathered edge on low power magnification; typically 100× or 200× magnification, looking for platelet clumps or aggregates (Figure 54.19). Clumping of platelets is common, particularly in cats, and after traumatic venipuncture. Such aggregates may affect the platelet concentration reported by CBC analyzers, which can result in a spuriously decreased platelet concentration. If platelet clumps are present, a smear made from fresh whole blood from a non-traumatic venipuncture may be useful to better assess platelet adequacy.

Estimating the Platelet Concentration

Estimation of the platelet concentration can be performed on a blood smear. This should ideally be performed on a blood smear prepared from fresh whole blood, as some anticoagulants can promote platelet aggregation and cause a spuriously low platelet estimate. It is also essential that the platelet estimate is within the monolayer of the slide, so that the platelet concentration is not overestimated (if performed in the body of the smear) or underestimated (if performed too close to the feathered edge).

Within the monolayer, count the number of platelets present in a 1000× magnification field. Repeat this step for a total of 10 fields to account for the variation in platelet distribution across the slide, and then divide by 10 to obtain

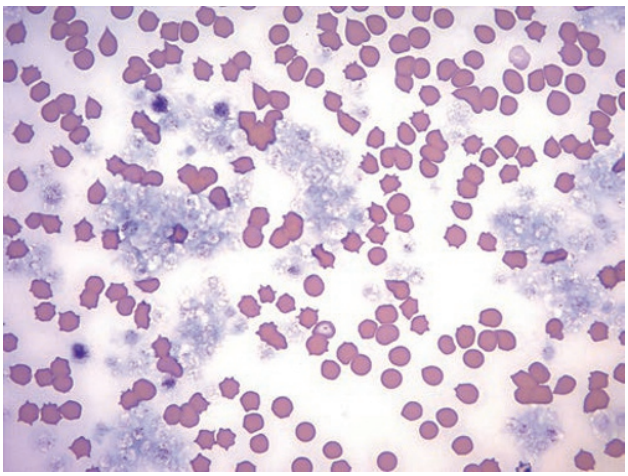


Figure 54.19 Large platelet clumps. Cat blood, 500× magnification, Wright-Giemsa.

an average number of platelets per 1000 × magnification field. Within the monolayer, 1 platelet is representative of approximately 10 000 to 20 000 platelets/μl, so multiplying the average number of platelets by 15 000 will give an estimate of the platelet concentration per μl (Eq. 54.2):

$$\frac{Sum\ of\ platelets\ in\ ten 1000 \times magnification\ fields}{10} \times 15{,}000 = Estimate\ of\ platelet\ concentration\left(/l\right) \quad (54.2)$$

Mean Platelet Volume

The mean platelet volume (MPV) is the average volume of a single platelet. This value can only be obtained from automated hematology analyzers and is available from most in-house machines. There is an inverse correlation between platelet concentration and MPV in dogs and cats [38]. MPV values are highly susceptible to preanalytical and analytical errors, particularly in cats, because of platelet clumping [39].

Evaluating Platelet Morphology

Platelets are clear to pale purple with variably prominent pink granules (typically more prominent in cats) and are approximately 2–4 and 2–6 μm in diameter in dogs and cats, respectively. Cats have greater variability in platelet size, possibly due to an altered M-loop region in β1-tubulin compared to other species, which results in a higher MPV than other domestic animals [40].

Giant platelets are approximately the size of a red blood cell, and can be seen in cases of thrombocytopenia if the bone marrow is mounting a regenerative response. Platelet mass, and not platelet concentration, provides negative feedback on thrombopoietin.

Coagulation

Major tests of coagulation available in the emergency room and intensive care unit include the activated clotting time (ACT), prothrombin time (PT), activated partial thromboplastin time (aPTT), and buccal mucosal bleeding time (BMBT).

Activated Clotting/Coagulation Time

The ACT tests the intrinsic and common pathways of coagulation. It does not test factors in the extrinsic pathway (i.e. factors III and VII). The ACT has many limitations; the first is poor sensitivity. The test may require greater than 90–95% deficiency of a single factor to be prolonged. The other major problem of the test is reproducibility, due to many factors that may affect results, including temperature and length of incubation period, as well as how the tube is inverted and inspection times if the test is performed manually and not by an automated machine. Severe thrombocytopenia (< 10 000 platelets/μl) may also prolong ACT [41].

ACT may be performed either by visual clot detection or using point-of-care devices. Visual clot detection involves a special tube that contains clot activators such as kaolin or celine–kaolin glass beads such as those in MAX-ACT™ (Helena Laboratories, Beaumont, TX). Diatomaceous earth is no longer available as a clot activator, and any reference intervals based on this activator may no longer be appropriate. Reference intervals for normal patients using the MAX-ACT tubes in one published study were 55–85 seconds for cats, and 55–80 seconds for dogs [42]. The tube should be kept at 37 degrees C and gently rotated every 5–10 seconds with visualization for clot formation. The time taken to the start of clot formation is recorded as the ACT. Refer to Protocol 54.9 for further details on manual ACT.

Automated evaluation of ACT may also be performed using point-of-care machines such as iSTAT® (Abbott Laboratories, Abbot Park, IL), which use cartridges containing clot activators such as kaolin or cellite. These machines use electrochemical sensing to measure thrombin generation (rather than fibrin formation).

Activated Partial Thromboplastin Time and Prothrombin Time

The aPTT is used to assess the intrinsic and common pathways of coagulation, while the prothrombin time is used to assess the extrinsic and common pathways. In general, the tests will be prolonged if there is a greater than 70% decrease in any given coagulation factor.

Agreement between point-of-care analyzers and laboratory methods generally is good (up to 87.5% for aPTT and 97% for prothrombin time) [43]. Interestingly, the prothrombin time test on most point-of-care instruments has high specificity, but low sensitivity, while the aPTT has very high specificity (up to 100%), but low sensitivity [43–45]. In another study using blood from healthy dogs, prothrombin time and aPTT results from in-house analyzers had moderate correlation with results from a laboratory, but results were still considered clinically reliable [46]. It is important to note that such studies are performed in controlled settings with trained personnel and excellent quality control, and may not translate to clinical practice. Point-of-care tests should be calibrated with control tests frequently and maintained per manufacturer recommendations.

It is recommended that citrated whole blood be used for in-house testing of prothrombin time and aPTT. Both tests may be falsely prolonged if the citrate tube is underfilled, or if erythrocytosis is present. Platelet concentrations and

Protocol 54.9 Activated Clotting Time

Principle

Activated clotting time assesses the intrinsic and common pathways of coagulation. Surface activators within ACT tubes activate coagulation via the intrinsic pathway, and the time to clot formation is measured as the activated clotting time.

Specimen

Fresh whole blood.

Materials and Equipment

- ACT tube (e.g. MAX-ACT (Helena Laboratories, Beaumont, TX))
- Timer
- 37°C water bath

Notes

- Do not use tubes past their expiration date.
- Use fresh whole blood from a fresh, non-traumatic venipuncture. Do not use blood from sampling lines.
- Factors that may artifactually prolong ACT include hypothermia, hemodilution, or severe thrombocytopenia.

Procedure

1) Fill ACT tube with fresh whole blood per manufacturer guidelines.
2) Begin timer as soon as tube is filled with blood.
3) Gently mix blood in tube at 37°C for 30 seconds.
4) Place tube in 37°C water bath and assess for clot formation every 5–10 seconds by tipping the tube gently on its side.
5) Record the time taken for a clot to be first seen. This time is the activated clotting time.

function will not affect results. It has been suggested that citrate tubes for coagulation testing be stored on ice; however, multiple studies report that storage of citrated whole blood from clinically ill dogs at room temperature for 24 hours does not result in statistically or clinically significant changes in prothrombin time or aPTT [47, 48]. Changes can occur, however, after 48 hours of storage at room temperature.

Buccal Mucosal Bleeding Time

BMBT is used to assess platelet function. The test is highly imprecise, with repeat bleeding times varying by as much as 87 and 120 seconds in cats and dogs, respectively [49, 50]. Factors that may affect results include how the incision is created [51], how tightly the lip is held/tied back, and any disruption of the incision. Additionally, bleeding times may be falsely prolonged in patients with anemia [52]. Protocol 54.10 provides further details of the BMBT procedure.

Protocol 54.10 Buccal Mucosal Bleeding Time

Principle

The buccal mucosal bleeding time (BMBT) is a point-of-care test used to evaluate primary hemostasis.

Materials and Equipment

- Bleeding time device (to create standard incision)
- Gauze
- Filter paper
- Timing device

Notes

- Factors that may affect BMBT results:
- Device used to create incision
- How tightly the lip is held back
- Use of sedation
- Disruption of the incision
- Anemia

Procedure

1) Restrain the patient in lateral recumbency, preferably without sedation.
2) Evert the upper lip to expose the mucosal surface; use gauze tied around the maxilla to hold the lip in place. There should be congestion of the mucosal vessels but blood flow should not be occluded by the gauze tie.
3) Place bleeding time device flush against the mucosal surface of the lip, avoiding obvious surface vessels.
4) Activate the trigger mechanism in the bleeding time device.
5) Start the timer and allow the incision to bleed freely.
6) Use filter paper to blot excess blood from the area but be careful not to disturb the incision or clot formation.
7) Stop the timer when blood no longer stains the filter paper.

Blood Typing

Dogs

Dog erythrocyte antigen (DEA) 1.1 and 1.2 are the most important surface antigens, owing to their high antigenicity. Dogs do not have natural antibodies for 1.1 and 1.2, but do have natural antibodies for DEA 3, 5 and 7, but reactions from these antigens are generally mild or delayed.

In a laboratory, typing is performed using polyclonal antibodies using a tube agglutination method. In-house typing kits are available that test for DEA 1.1. The most common tests are the DMS CARD test (CARD; DMS Laboratories, NJ) and the Quick Test DEA 1.1 (CHROM; Alvedia, France). The benefit of the CHROM testing system is that is it not affected by autoagglutination. In one study, CHROM was 93% accurate and 100% specific, which is important because there were no false positive results [53]. It is less sensitive than both CARD and the gold standard gel column test.

Cats

Major blood types in cats include blood type A, B or AB. Recently, the MiK antigen has also been identified. There is no universal blood type in cats, and cats have naturally occurring antibodies to these surface antigens. Type B cat that receive type A blood may have fatal reactions, making blood typing essential.

Two major in-house blood typing kits for cats include Rapid® VetH Feline (CARD, DMS Laboratories, NJ), and Quick Test A + B, (CHROM, Alvedia, France). In a study comparing these kits to the gold standard (tube agglutination test), CARD had 91.4% agreement, and CHROM had 94.8% agreement [54]. Presently, there is no in-house testing kit for the MiK antigen.

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In-House Evaluation of Hemostasis

April Summers, Jocey Pronko, and Julien Guillaumin

Hemostasis is defined as the ability of the body's systems to maintain both blood fluidity and blood vessel integrity to preserve normal blood function. It is a complex physiologic process that stops bleeding at the site of vascular damage. When the vasculature is damaged, there is a cooperative response in both the endothelium and the blood that ultimately results in the formation of a clot. Hemostasis involves not only primary and secondary hemostasis but also fibrinolysis and amplification of inhibitory pathways.

The process of hemostasis is usually divided into two distinct but overlapping phases: primary hemostasis and secondary hemostasis. Primary hemostasis refers to the formation of the initial platelet plug following vascular damage, whereas secondary hemostasis refers to the formation of crosslinked fibrin across that platelet plug. The traditional model of coagulation simplifies secondary hemostasis into two distinct pathways: the intrinsic pathway and the extrinsic pathway, both of which converge into the common pathway. This model is particularly useful for interpreting in vitro tests of coagulation [1]. In vivo, however, coagulation is more accurately depicted by the cell-based model of coagulation, which is separated into three distinct phases: initiation, amplification, and propagation. Initiation begins when a vessel is damaged and tissue factor is exposed on tissue factor-bearing cells. This results in the generation of small amounts of thrombin. The amplification phase is marked by platelet activation and the accumulation of large amounts of tenase and prothrombinase. Afterwards, stabilization of the clot is achieved via large-scale thrombin generation (the propagation phase) and the activation of factor XIII (fibrin stabilizing factor) [1, 2].

Fibrinolysis refers to the breakdown of the clot, which then returns the vasculature to its normal patency. It is the enzymatic dissolution of fibrin into fibrin degradation products by plasmin. This reaction is catalyzed by tissue plasminogen activator (tPA), which converts plasminogen to plasmin [2].

Hemostatic testing can be used to identify and characterize hemostatic defects in patients. It is important to note that the tests performed in vitro may not accurately reflect changes occurring in vivo.

Testing for Defects in Primary Hemostasis

Primary hemostatic problems refer to platelet disorders, either decreased numbers (i.e. thrombocytopenia) or decreased function (i.e. thrombocytopathia). Platelets (also called thrombocytes) are small anuclear cellular fragments derived from larger cells called megakaryocytes in the bone marrow. Platelets are a major component of the blood and play an integral role in primary hemostasis, the repair of damaged vasculature, the inflammatory cascade, and wound healing. They have a mean circulating lifespan of four to six days in dogs and cats.

Platelet Count

The platelet count is the number of platelets per microliter of blood and is used to assess total platelet mass. A decreased platelet count is referred to as thrombocytopenia and an increased platelet count is called a thrombocytosis. Thrombocytopenia can be caused by a number of different mechanisms including loss (e.g. from acute hemorrhage), decreased production (e.g. from primary bone marrow disease), increased consumption (e.g. in abnormal clot formation, or thrombosis), destruction (i.e. immune-mediated thrombocytopenia), sequestration in the spleen, or artifactual (i.e. clumping). Thrombocytosis may be due to a

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myeloproliferative disorder or increased bone marrow production of platelets secondary to cytokine release or erythropoietin production.

Platelet counts can be obtained via impedance analyzers, laser flow cytometers, quantitative buffy coat analyzers, and peripheral blood smears.

Platelet Evaluation via Thrombogram

Platelets can be evaluated via a thrombogram, the term used to refer to the portions of a complete blood count related to platelets. The thrombogram includes analysis of total platelet count, mean platelet volume (MPV) and size, platelet distribution width, platelet morphology, and thrombocrit (or plateletcrit) [3]. MPV is a measure of the mean platelet size; increases in this value can suggest the presence of young platelets due to increased production [4]. A decrease in MPV can be associated with immune mediated destruction. MPV, like most parameters, should be interpreted in light of the sample preparation and analyzer used to assess it. Sample storage time, temperature, and the use of anticoagulants can all affect MPV. This value may be falsely increased when lipid droplets or cell fragments are mistakenly labeled as platelets by impedance analyzers and flow cytometers [4]. Platelet distribution width reflects platelet anisocytosis as calculated from individual platelet volume and gives an estimate of total variation in platelet size [3]. Plateletcrit, the product of MPV and platelet count, and expressed in percentage, is generated by certain analyzers [3].

It is important to interpret thrombogram data in light of the analyzer used. For instance, impedance analyzers separate platelets and erythrocytes based on size. Impedance analyzers can thus be inaccurate when the sizes of platelets and erythrocytes overlap [5]. This can occur when there are large platelets or when erythrocytes are small (such as with iron deficiency anemia). Cats are known to have small erythrocytes and large platelets; thus, impedance analyzers may have inaccurate counts in this species. Impedance analyzers can also incorrectly count clumps of platelets as leukocytes. In contrast, flow cytometers provide more reliable cell counts because these machines sort cells according to their internal complexity, and therefore can more accurately identify platelets.

Blood Smear Platelet Estimate

Microscopic examination of a peripheral blood smear allows for rapid, bedside estimation of the platelet count. The number of platelets/μl can be estimated by counting the number of platelets seen per high power (100×) field in the monolayer in at least 10 high power fields, then multiplying the *average* count in those 10+ fields by 15000–20000/μl [6–8]. It is important to thoroughly assess the feathered edge of the blood smear to investigate for platelet clumps, which can falsely decrease the platelet count, causing a pseudothrombocytopenia [7]. While platelet clumping invalidates platelet counts and platelet estimates, abundant numbers of platelet clumps generally rule out a clinically significant thrombocytopenia. Clumped platelets occur commonly in cats and less commonly in dogs, though platelet clumping can be noted in either species since platelets can become activated during blood sampling and subsequently clump together [9]. Spontaneous bleeding typically does not occur when platelet counts exceed 10000–20000/μl, and surgical bleeding typically does not occur when platelet counts exceed 50000–60000/μl [10].

Buccal Mucosal Bleeding Time

Buccal mucosal bleeding time (BMBT) is the time to form the primary hemostatic plug in vivo and is therefore used to detect defects in primary hemostasis. It is usually performed to assess platelet function after thrombocytopenia has been ruled out. A platelet count should always be performed prior to assessing BMBT, since thrombocytopenia affects the results of the test. Indications for BMBT include patients that have a suspected defect in primary hemostasis but have an adequate platelet count. BMBT is used to measure the duration of hemorrhage resulting from a small, standardized incisional injury made with a spring-loaded disposable device to the mucous membrane on the inside of the lip (the buccal mucosa). The incisional injury is small enough such that a platelet plug alone is adequate to stop the bleeding regardless of secondary hemostatic (clotting factor) status. Prolonged bleeding times can be seen in patients with von Willebrand disease (vWD), other thrombocytopathia (e.g. inherited or acquired diseases, aspirin administration), thrombocytopenia, and endothelial diseases. For this reason, BMBT is used preoperatively as a screening test for patients suspected to have vWD or other thrombocytopathias.

Normal BMBT is less than 4.0 minutes in dogs and less than 1.5 minutes in cats. It may be prolonged in patients with severe anemia due to decreased blood viscosity [11]. While BMBT can be useful to detect primary hemostatic defects, there is high intra- and interobserver variability as many technical variables can alter the results [12]. For instance, one must be careful that the filter paper does not touch the cut surface and thus interfere with formation of the platelet plug. Often a gauze tie is placed around the muzzle to fix the lip in an everted position for this procedure; if the tie is overly tight, it can occlude blood flow or cause engorgement, either of which could falsely affect bleeding times. Finally, if excessive pressure is placed on the spring-loaded incisional device, the incision may be deeper than intended and trigger secondary in addition to primary hemostasis. Care must be taken when performing

Protocol 55.1 Acquiring Buccal Mucosal Bleeding Time

Items Required

- Appropriately sized spring-loaded device
- Gauze tie
- Filter paper
- Stopwatch

Procedure

1) Remove the spring-loaded device from the packaging without touching the aperture slot.
2) Remove the safety clamp.
3) Place the patient in lateral recumbency.
4) In dogs, a gauze strip can be used as a tie to expose the buccal mucosal surface, hold the lip up, and provide moderate mucosal engorgement. However, it should not be placed too tightly as to occlude blood flow. An alternative is to manually lift and hold the lip steadily for the duration of the test.
5) Choose an area on the buccal mucosa that is devoid of visible blood vessels.
6) Place the device on the mucosa.
7) Press the release button of the incision device.
8) Start the stopwatch immediately after bleeding begins.
9) Allow for undisturbed bleeding of the cut surface.
10) Absorb draining blood every 10 seconds with the filter paper without touching the cut surface.
11) Stop the stopwatch immediately after bleeding stops and read the buccal mucosal bleeding time.

the BMBT to ensure the most reliable result (Protocol 55.1). If BMBT is prolonged, vWF assessment and platelet function testing may be indicated.

Although BMBT can be easily performed in clinical practice, the gold standard for platelet function assessment is turbidimetric aggregometry, which can be done in some reference laboratories. Other point-of-care devices, such as the PFA-100® (Siemens Medical Solutions, Malvern, PA), can provide bedside platelet function analysis. As they are uncommon in practice, these devices are not covered in this chapter.

Secondary Hemostasis

Prothrombin Time and Activated Partial Thromboplastin Time

The prothrombin time (PT), also known as one-stage prothrombin time, is used to evaluate the extrinsic and common pathways of the classic model of the clotting cascade. Specifically, factors X, VII, V, II (prothrombin), and I (fibrinogen) are evaluated. The activated partial thromboplastin time (aPTT; also known as partial thromboplastin time) is used to evaluate the intrinsic and common pathways of the classic model of the clotting cascade. As such, the aPTT evaluates factors XII, XI, X, IX, VIII, V, II (prothrombin), and I (fibrinogen). Both PT and aPTT may not be prolonged until factor activity has decreased to 60–70% of normal.

Normal hemostasis depends on a balance between pro- and anticoagulant proteins as well as the fibrinolytic system. It is important to note that the division of the intrinsic and extrinsic pathways is an artifactual by-product of in vitro coagulation testing and is not how coagulation works inside the animal. In vivo, these two pathways interact and both must be activated for appropriate hemostasis.

PT and aPTT are useful in diagnosis of coagulation disorders and for monitoring anticoagulant therapy. Heparin therapy, by inhibiting the intrinsic pathway, is known to prolong aPTT [13]. Measurement of PT and aPTT is warranted in animals suspected of having coagulation disorders such as liver failure, vitamin K antagonism, and consumptive coagulopathies like disseminated intravascular coagulation. Because it affects factor VIII, patients with vWD may have a prolonged aPTT.

Vitamin K deficiency, usually secondary to anticoagulant rodenticide toxicosis, affects factors II, VII, IX, and X. PT is a particularly useful diagnostic test in patients exposed to anticoagulant rodenticide because factor VII has the shortest half-life of these vitamin K-dependent factors, at 6.2 hours in dogs. Because of the short half-life of factor VII, the PT is usually first to be prolonged, usually 24–36 hours after ingesting an anti-vitamin K rodenticide. As time progresses without treatment, and the functional vitamin K factors are depleted, aPTT becomes prolonged as well.

The PT and aPTT are generally measured using plasma from blood collected into a blue-top tube with 3.2% sodium citrate as an anticoagulant with a ratio of nine-to-one for the blood-to-citrate. Collection tubes need to be filled to the appropriate level to ensure the correct blood-to-anticoagulant ratio [14]. The samples should then be gently inverted at least six times to ensure appropriate mixing of citrate with the blood. These assays are generally performed within one hour of sample collection, although longer storage times have been reported [15]. In fact, some reports show that aPTT is not substantially affected by refrigeration for at least 24 hours following sampling [16].

Manual Measurement of Prothrombin Time

PT can be measured manually (Protocol 55.2) by adding tissue thromboplastin reagent followed by a reagent (such as calcium chloride) to recalcify the sample and

Protocol 55.2 Manual Prothrombin Time

Items Required

- Citrate tube
- Blood collection supplies
- Tissue thromboplastin – calcium chloride reagent
- 12 × 75 test tube
- Stopwatch
- Heat block 37°C

Procedure

1) Perform venipuncture and place whole blood into sodium citrate tube for a ratio of 9 : 1. Invert the tube gently at least six times to ensure adequate mixing of blood and anticoagulant.
2) Centrifuge as soon as possible and collect the plasma (i.e. the supernatant after centrifugation).
3) Incubate plasma for two to three minutes until it reaches 37°C.
4) Incubate a 12 × 75 mm tube with 0.2 ml of the thromboplastin-calcium reagent until it reaches 37°C.
5) Inject, with force, 0.1 ml of the plasma into the tube with the tissue thromboplastin–calcium chloride reagent. The thromboplastin triggers the clot formation while calcium reverses sodium citrate's anticoagulant effects. Confirm volumes with reagent manufacturer's recommendations.
6) A clot should fully form in 6–20 seconds. A clot that takes longer than 20 seconds to form is considered to demonstrate a prolonged prothrombin time.
7) If the result is achieved in less than 20 seconds, the results should be repeatable within ± 1 second. If the result is achieved in longer than 20 seconds the test should be repeatable within ± 2 seconds.

reverse citrate's effects [17]. Clot formation should be detected within 20 seconds.

Analyzer Measurement of Prothrombin Time and Activated Partial Thromboplastin Time

Point-of-care analyzers are available for measuring PT and aPTT using non-anticoagulated or citrate anticoagulated whole blood [18]. These point-of-care analyzers are beneficial in that results are obtained within minutes. To achieve adequate mixing of blood and sodium citrate, the blood should be collected five minutes prior to testing and well mixed with the citrate prior to analysis. The analyzer accepts a very small sample of blood and moves it into the testing chamber, where clot formation occurs within the cartridge. The clot is detected by light-emitting diode optical detectors and, as the clot is formed, blood flow is impeded and the blood movement slows. The PT and aPTT are recorded when the movement of blood decreases below a predetermined rate. Studies have shown that abnormalities detected by point-of-care assays are similar to clinical laboratory tests [18, 19].

PT and aPTT are generally considered to be prolonged if the patient's time is 25–30% longer than the upper end of the reference interval. Although rare, a shortened PT can be caused by an excessive plasma-to-anticoagulant ratio, increased tissue factor concentrations due to traumatic venipuncture, a hypercoagulable state, or may merely represent the normal range for the patient [20]. Inflammatory conditions may also shorten PT and aPTT times due to increased fibrinogen concentration [21]. Artifactual results can be caused by improper collection, storage, and processing. Results that do not correlate with the clinical picture should thus be interpreted with caution.

Activated Clotting Time

Activated clotting time (ACT) is used to detect defects in the intrinsic and common pathways. Abnormally increased ACT indicates that the activity of one or more of the coagulation factors is reduced by at least 85–95%. ACT is frequently used for monitoring heparin anticoagulation therapy, especially with high doses of heparin when aPTT cannot be used. It is most commonly used in settings of invasive intravascular procedures including cardiac catheterization, vascular surgery, and hemodialysis [22]. aPTT is a test that involves an in vitro clotting reaction and is not clinically useful with high-dose heparin administration, as the blood will not clot. ACT uses whole blood that is added to a tube containing an activator of the intrinsic coagulation cascade such as kaolin, celite, glass beads, phospholipid tissue extract, diatomaceous earth, or other substance, followed by measurement of the time required for clot formation. While the two tests are used for similar purposes, ACT is less sensitive than aPTT and relies on the patient's platelets, phospholipids, and calcium to support the reaction. ACT can be affected by a number of different factors including hemodilution, hypothermia, factor deficiency, and platelet dysfunction. Severe thrombocytopenia is also known to prolong ACT.

The ACT can be evaluated manually (Protocol 55.3) by collecting blood into a gray-top vacutainer that contains diatomaceous earth. In this method, the ACT is the time in seconds to the first visible clot formation while gently rocking the tube every 5–10 seconds after initially incubating the tube for 60 seconds at 37 °C. ACT can also be performed with the use of specialized point-of-care coagulation analyzers. There are assays that use mechanical methods such as a mechanical plunger (ACT Plus®, Medtronic, Minneapolis, MN) or displacement of a magnet (Hemochron®, Werfen,

Protocol 55.3 Manual Activated Clotting Time Evaluation

Items Required

- Vacutainer tube with diatomaceous earth (gray top)
- Blood sample
- Stopwatch
- 37°C incubator

Procedure

1) Collect 2 ml of blood directly into a gray-top vacutainer tube that contains diatomaceous earth.
2) Begin a timer as soon as the blood enters the tube.
3) Gently invert the tube to mix its contents and place it in a 37°C incubator.
4) Rock the tube back and forth gently every 5–10 seconds.
5) Examine the tube for clot formation at 60 seconds, and then every 5 seconds thereafter.
6) Record the time to blood clot formation. Normal time to clot is less than 120 seconds.

Bedford, MA). ACT can be tested on i-STAT® machines (Abbott Point of Care, Princeton, NJ) using ACT cartridges with either kaolin or celite as activators. The benefits of this type of equipment include increased automation and decreased sample volume compared with other methods, including manual ACT evaluation.

Viscoelastic Testing

Viscoelastic hemostatic assays (VHA) are used for the global assessment of hemostasis. In human medicine, VHA is commonly used to monitor for response to treatment, while in veterinary medicine it is commonly used for the diagnosis of different hemostatic states including: normocoagulable, hypocoagulable, hypercoagulable, excessive fibrinolysis, or decreased fibrinolysis. This method of assessing hemostasis provides a graphical tracing of clot formation and breakdown as well as numerical information reflecting the time it takes for initial fibrin formation, the kinetics of the fibrin clot to reach maximum strength, as well as the overall strength and stability of the clot. Many different analyzers are available.

Thromboelastography (TEG) and rotational thromboelastometry (ROTEM) are the most commonly studied point-of-care tests for viscoelastic testing in small animals. Both systems use whole blood and measure coagulation by assessing the shear elasticity as it clots. Whole blood is placed into a heated cuvette and a vertical pin is held in the blood sample. The difference between TEG and ROTEM is which part of the system rotates, although the information provided is quite similar. With TEG, the cup rotates clockwise and counterclockwise around the static pin as the blood clots. Fibrin strands and platelet aggregates form between the pin and the walls of the cup, which result in torque on the pin. The pin movements are monitored and converted into an electrical signal. The opposite happens with ROTEM, as the pin oscillates and the cuvette is held stationary [23]. The impedance of pin rotation as fibrin strands are formed is detected and a tracing is generated [24]. Although both tests are useful in rapidly diagnosing coagulopathies, and although the various time points and other variables provided by those devices appear similar, differences in activators and other variabilities limit direct comparison of VHA variables between the two methodologies [25, 26]. Differences in results of VHA variables have been noted even with the use of the same machine at different institutions, and it is recommended that each institution establish its own reference intervals.

TEG provides information on initiation, amplification, and propagation of coagulation in addition to fibrinolysis. Several different result parameters are reported in the machine's graphical tracing and the tracing's associated numerical values. Time is represented on the TEG tracing's X axis while the Y axis represents the clot formation kinetics. Specific values reported from the TEG tracing include the reaction time (R), which is the time to first clot detection and reflects coagulation factor activity; the kinetics of clot formation (K), which is the time from R until the tracing reaches 20 mm on the Y axis, which reflects the speed of clot formation; and the alpha angle (α) is the angle created by the tangent from R on the X axis and point K, and represents conversion of fibrinogen to fibrin. Maximum clot strength is represented by maximum amplitude, and data associated with fibrinolysis are provided by the clot lysis time and the percent lysis 30 minutes after maximum amplitude.

ROTEM provides information similar to that from TEG. The time to initiation of clotting, thrombin formation, and the start of clot polymerization are reported as the clotting time. The kinetics of clotting are reported as two closely linked parameters: clot formation time and alpha angle, similar to K and alpha angle from the TEG. The maximum clot firmness and amplitude obtained at 10 minutes, which correlate with platelet count and function, are provided. Lysis 30 minutes after clotting time and maximum lysis are also provided for information regarding the stability of the clot and fibrinolysis.

There are newer viscoelastic testing devices designed with miniaturization and automation in mind for rapid, global, point-of-care coagulation assessment (e.g. VCM Vet®, Entegrion, Durham, NC). This instrument uses fresh

whole blood and is available as an in-house diagnostic. Once the fresh whole blood is loaded into the cartridge, the cartridge can be inserted into the fully automated system. The system provides similar information about time to initiation of clot formation, rate of clotting, maximum clot strength, and fibrinolysis. Owing to its ease of use, these smaller viscoelastic testing devices are commonly found in veterinary emergency clinics, although peer-reviewed data are scarce. The TEG 6s® system (Haemonetics Corp., Braintree, MA) is a newer VHA that uses a multichannel microfluidic cartridge, which enhances portability and eliminates the need for pipetting. These cartridges mix small blood samples with reagents automatically and can run up to four assays in parallel. It offers the potential for more reliable measurements due to decreased user-related and vibrational errors, though some studies have shown that this assay is still susceptible to vibrational artifact [27, 28]. Thus far, clinical experience with the use of this assay is limited.

Protocol 55.4 Acquiring Whole Blood Clotting Time

Items Required

- Clean, dry, glass blood tube
- Blood sample drawn with any gauge needle
- Stopwatch

Procedure

1) Obtain 2 ml of venous blood via venipuncture.
2) Transfer directly into a clean, dry glass tube and hold at 21°C (room temperature).
3) Gently rock the tube back and forth every 30 seconds.
4) The time to the beginning of clot formation should be recorded.
5) Normal animals generally have clotting times of 3–13 minutes.
6) If the clot breaks down or fails to coagulate, this indicates coagulopathy.

Whole-Blood Clotting Time

Whole-blood clotting time (WBCT), also known as the Lee–White clotting time, red-top tube clotting time, or glass-tube clotting time, is a very simple, useful, point-of-care diagnostic test used in the assessment, diagnosis, and monitoring of patients with coagulopathies (Protocol 55.4). It is commonly used as an alternative bedside test when more sophisticated coagulation assessment devices are unavailable or in resource-poor countries for detecting coagulopathies in patients that have sustained snake bites [29]. The premise of this test is to take a small volume of blood and transfer it directly into a glass tube incubated at room temperature and assess formation of a clot. In normal animals a clot should form in 3–13 minutes and the time to clot formation is referred to as WBCT [30]. A variation of this test is referred to as 20-minute WBCT, in which clot formation is not assessed until 20 minutes of room temperature incubation have passed [31]. Other studies using human blood have been performed with clot formation assessed at the 30 minute mark [31]. At the end of the designated time, the clot is graded 0, 1, or 2. Grade 0 would refer to normal coagulation as a solid clot. Grade 1 refers to a partial or semisolid clot that breaks apart and grade 2 refers to uncoagulated blood that would easily pour out of the tube if inverted [31]. The clot, once formed, should retract to 50% within one to two hours of formation [30]. WBCT can be an imprecise test as it is not standardized [32]. Specifically, as platelets are needed for activation of the clotting cascade, prolonged clotting times can be seen in thrombocytopenic patients due to a phospholipid deficiency. As such, its use has decreased as the availability of automated devices have increased. However, it can be a useful test in veterinary clinics where point-of-care automated devices are unavailable.

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Electrolyte Evaluation

Louisa J. Rahilly and Katherine Vachon

Electrolytes are pivotal players in maintaining homeostasis. They have key roles in such physiologic functions as maintaining intracellular and extracellular fluid volumes and distribution, energy production and use, electrical conductivity and muscle contraction in cardiac, skeletal, and vascular smooth muscle, and clot formation. Many disease processes can cause electrolyte abnormalities including gastrointestinal disease, endocrine diseases such as diabetes, hyperadrenocorticism, hypoadrenocorticism, and thyroid disorders, renal and urinary disease, various neoplasms, sepsis, and skin disorders. Treatment interventions in critically ill animals can also precipitate electrolyte disorders or clinically significant shifts in electrolyte distribution and levels within the body.

Both abnormally high and low electrolyte changes have been demonstrated to increase mortality in dogs and cats [1, 2]. Appropriate treatment of electrolyte abnormalities may result in decreased morbidity and mortality. Many of the clinical signs of electrolyte abnormalities can be masked by or are thought to be due to the underlying disease state. The role of the emergency and critical care technician is to monitor for potential detrimental effects of electrolyte abnormalities, appropriately obtain samples and use in-house analyzers, and alert the clinician to important changes noted on laboratory monitoring over the course of hospitalization.

Electrolyte concentrations in serum, plasma, or whole blood samples are ultimately the results of the combination of intake, excretion, shifts between the intracellular and extracellular space (note that we sample the extracellular space in the clinical setting), and artifactual influences in vitro [3, 4]. Accurate measurement of electrolytes in the critical care setting is both necessary and challenging, owing to common artifacts such as dilution or binding with anticoagulants, sample exposure to air, and concurrent sample or patient abnormalities that spuriously alter electrolyte measurements. Electrolytes often need to be evaluated in the clinic using point-of-care (POC) analyzers because changes in electrolyte concentrations can alter the treatment course on an hour-by-hour basis. Understanding the methodology and potential artifacts that may be caused by the POC analyzer in use is important, as some physiologic abnormalities, such as lipemia or hyperbilirubinemia, necessitate particular methodology and submission to a reference laboratory for accurate electrolyte measurement [5]. This chapter introduces methods of electrolyte quantification and commonly used veterinary electrolyte analyzers, the physiologically significant electrolytes, disease states or treatments that may affect them, and recommendations on sample handling to ensure correct measurement and interpretation.

Methods of Electrolyte Quantification

Methods of electrolyte evaluation include flame photometry, ion-selective electrode (ISE) assays, and enzymatic spectrophotometry (ESP) [5, 6]. When serially evaluating a patient's electrolytes, values should always be measured with the same methodology and, ideally, the same analyzer because reference intervals and readings vary among different methodologies and analyzers [5, 7–9]. Flame photometry is typically only available in reference laboratories, making it impractical to use this methodology for POC analysis. It was the gold standard for electrolyte analysis but has become obsolete due to both logistics and inaccuracies in measurement with concurrent lipemia, hyperproteinemia, hemolysis, or hyperbilirubinemia [4, 6]. ESP has clinical value in enabling electrolyte measurement using in-house chemistry analyzers but it has demonstrated the most interference in situations of hemolysis, hyperproteinemia, and lipemia [6, 10, 11]. Uremic samples with

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alterations in bicarbonate and potassium may also affect samples analyzed by ESP [11]. Updated chemistry analyzers that use ESP, such as the Catalyst One® chemistry analyzer (IDEXX, Westbrook, ME) and the Vetscan® (Abaxis, Union City, CA) use filters to attempt to minimize the interference created by these abnormalities. One should interpret results in severely lipemic, icteric, hemolyzed, or hyperproteinemic samples carefully, however, and submit a paired sample to a reference laboratory that uses ISE to confirm in-house results.

ISE is now the method of choice and the reference method recommended by the Lab Medicine and Clinical and Laboratory Standards Institute for the validation of POC analyzers [6, 7, 11]. The basic principle of ISE measurement is the comparison of an unknown value against a known value to compute the sample's electrolyte concentration (VetLyte® electrolyte analyzer, IDEXX, Westbrook, ME). Essentially, an ion-sensitive membrane undergoes a reaction based on the electrical charge of the ion (electrolyte) causing a change in the specific ion-generated voltage. The analyzer then compares the change in voltage on the sample side of the membrane with a reference solution. An algorithm using the measured voltages and a calibration curve derived from the ion concentration of known solutions determines the electrolyte concentration within the sample. Commonly used in-house veterinary analyzers that use ISE include the i-STAT® (Abbott, Abbott Park, IL) Nova® (Nova Biomedical, Waltham, MA), VetLyte, iSmart-30, and EasyLyte® (Medica, Duluth, MN). Table 56.1 details each of these analyzers, which electrolytes they measure, and select operator information.

Electrolytes vary as to the best sampling method and possible artifacts associated with measurement, the specifics of which are covered in the subsequent sections. However, overall electrolyte analysis should be performed on serum samples that are collected anaerobically [3, 7, 8, 12–14]. Anaerobic conditions are most important for ionized calcium and magnesium analysis [8, 13–15]. Whole blood, anticoagulated whole blood, and plasma can also be used, but one must consider various possible artifacts and the risk of clot formation prior to the completion of analysis when using whole blood. Careful attention to the timing of sample collection and measurement is pivotal because various electrolyte concentrations can be affected by increased exposure time to platelets, exposure time to white blood cells, and alterations in protein binding associated with continuing cellular metabolism [7, 8, 15].

Individual Electrolytes

Table 56.2 details normal intervals and critical alert values for each of the physiologically significant electrolytes. It is important to remember, however, that these intervals should be used only as guidelines because each analyzer has its own manufacturer-determined reference intervals [5]. A reference interval specific to the analyzer used should be consulted when interpreting all electrolyte measurements. These ranges should be specific for sample type (i.e., plasma versus serum) and species within each analyzer.

Sodium

Sodium is the most abundant extracellular ion. It functions in determining the distribution of water throughout the body in the extracellular and intracellular compartments [4, 16–20]. Disorders in sodium concentration are nearly always the result of changes in the volume of body water [4, 16, 17, 19, 21]. The sodium concentration should therefore be viewed as a reflection of relative free water in the body, rather than necessarily the amount of sodium in the body [16, 17, 19, 21]. "Free water" is water that is free of sodium.

An increased sodium concentration is due to either the loss of body fluid containing relatively less sodium than plasma (hypotonic fluid), fluid containing no sodium (free water), or the ingestion or iatrogenic administration of relatively high-sodium substances [4, 16, 17, 20]. Disease processes that can result in hypotonic fluid loss include gastrointestinal and kidney disease; osmotic diuresis from hyperglycemia, ketonuria, or mannitol administration; or postobstructive diuresis [17, 22]. Lack of access to water and neurologic disease resulting in decreased water intake can also result in hypernatremia [17, 19]. Animals with underlying metabolic disorders such as hyperadrenocorticism, hyperthyroidism, and hepatic disease have decreased urine concentrating abilities and are at risk of developing hypernatremia in the critical care setting if the patient experiences decreased water intake due to nausea or sedation [17]. Although not significantly hypernatremic, Greyhound dogs have slightly higher sodium concentrations than non-Greyhound dogs; the reason for this is still being elucidated [23]. Processes resulting in the loss of fluid that contains virtually no sodium (i.e. pure free water loss) include diabetes insipidus and panting [17, 19].

Hypernatremia can also result from excess sodium retention or intake as opposed to excessive free water losses. Hyperaldosteronism, a rare disorder in dogs and cats, results in increased sodium retention [17, 24]. Dietary indiscretion can cause hypernatremia if the animal ingests excessive salt in the form of substances such as table salt, sea water, homemade playdough, solid ice melts, or paintballs [17]. Iatrogenic causes of hypernatremia include the administration of high-sodium-containing solutions (sodium bicarbonate, hypertonic saline, and sodium phosphate enemas), cathartics such as activated charcoal, and drugs that can affect the kidney's ability to concentrate urine

Table 56.1 Common POC electrolyte analyzers used in veterinary medicine.

Analyzer	Method	Electrolytes Measured (Reportable Range)	Sample Type Based on Manufacturer Recommendation	Sample Size	Maintenance/ Operator tips
Catalyst One Veterinary Chemistry Analyzer Idexx	ESP	Na^+(85–180mmol/L) K^+(0.8–10mmol/L) Cl^-(50–160mmol/L) iPhos(0.2–16.1mg/dL) tMg (0.5–5.2mg/dL)	WB Serum Plasma Urine	700µL	• Monthly quality control checks with internal and external cleaning • Reboot analyzer weekly • Slides should be stored in the freezer; warming is not required prior to running sample
Easy-Lyte PLUS Hemagen	Direct ISE	Na^+(20–200mmol/L) K^+(0.2–40mmol/L) iCa^{++}(0.1–6.0mmol/L) Cl^-(25–200mmol/L) Urine: Na^+(25–1000mmol/L) K^+(1.0–500mmol/L) Cl^-(25–500mmol/L)	WB Serum Plasma Urine	Blood:100µL Urine: 400µL	• Automatic or on demand calibration • Easy to use; two button operation
EPOC (Element POC) Heska	ISE[a]	Na^+(85–180mmol/L) K^+(1.5–12.0mmol/L) iCa^{++}(0.25–4.0mg/dL) Cl^-(65–140mmol/L)	WB Serum	>92µL	• Real time samples must be heparinized as calibrates and internal QC automatically prior to each sample (170sec) • Minimal maintenance • Handheld • Cartridges have a 180 Day shelf life • Sample run window 7.5min such that real time blood draws must happen in this window
Gem Premier 5000	Direct ISE	Na^+(100–200mmol/L) K^+(1.0–20.0mmol/L) iCa^{++}(0.11–5.0mmol/dL) Cl^-(40–170mmol/L)	WB	150µL	• Monthly internal cleaning with supplied wipes • Change GEM PAK montly to perform automated QC • Performs continuous analyzer checks before, during and after every sample run
i-Smart 30 VET electrolyte analyzer-Woodley[b]	Direct ISE	Na^+(20–250mmol/L) K^+(0.5–20.0mmol/L) Cl^-(20–250mmol/L)	WB Plasma	60µL	• No user maintenance required • Autocalibration • Cartridges stored in refrigerator, run at room temperature • Cartridges good for 4 weeks once warmed to room temperature • Results in 35 sec
i-STAT Alinity Abaxis Abbott	ISE*	Na^+(100–180mmol/L) K^+(2.0–9.0mmol/L) iCa^{++}(0.25–2.5mg/dL) Cl^-(65–140mmol/L)	WB with or without heparin	150µL	• Handheld, portable, and has touch screen • Rechargeable battery • Calibration performed automatically each time a cartridge requiring calibration is use • Only maintenance required is a software update every 6 months • Cartridges must be refrigerated. When removed from the fridge cartridges must be stored at room temperature and are good for up to 2 weeks

(*Continued*)

Table 56.1 (Continued)

Analyzer	Method	Electrolytes Measured (Reportable Range)	Sample Type Based on Manufacturer Recommendation	Sample Size	Maintenance/ Operator tips
Radiometer ABL90 Flex	ISE* or ESP[c]	Na^+(116–180mmol/L) K^+(2.1–10.5mmol/L) iCa^{++}(0.5–2.48mg/dL) Cl^-(86–151mmol/L)	WB	65µL	• Clot detection • Automatic QC management • Automatic sample mixing • Cassettes stored at 36-46°F, refrigerated • Can use syringe, capillary tube or test tube to enter a sample
Response910 VET Veterinary Chemistry Analyzer-Diasys	ESP	Na^+(100–180mmol/L) K^+(1.5–8.5mmol/L) tCa(1.0–16.0mg/dL) Cl^-(40–170mmol/L) tMg(0.2–5.0mg/dL) iPhos(0.2–30mg/dL	WB Plasma Serum CSF Urine	2–30µL	• Minimal maintenance • Monthly calibration • Store reagent 35.6°–77°F • Reagent life is 12 months
Rapidpoint 500 Siemens	ISE[a]	Na^+(100–200mmol/L) K^+(0.5–15.0mmol/L) iCa^{++}(0.2–5.0mg/dL) Cl^-(65–140mmol/L)	WB	Capillary tube: 100µL Syringe: 200µL	• ~60 sec to results • Automatic QC • No maintenance requirements • Cartridge life is 28 days
Stat Profile Critical Care Xpress Nova	Direct ISE	Na^+(80–200mmol/L) K^+(1.0–20.0mmol/L) iCa^{++}(0.1–2.7mg/dL) Cl^-(50–200mmol/L) iMg^{++}(0.1–2.5mmol/L)	WB, heparinized	135–250µL[d]	• Automatic QC • Automated maintenance
VetLyte Idexx	ISE[a]	Na^+(40–205mmol/L) K^+(1.5–15.0mmol/L) Cl^-(50–200mmol/L)	WB Serum Plasma	95µL	• No longer being • Weekly maintenance and cleaning • Daily conditioning • Monthly cleaning of electrode
Vetscan VS2 Analyzer Zoetis	ESP	Na^+(110–170mmol/L) K^+(1.5–8.5mmol/L) tCa(4.0–16.0mg/dL) Cl^-(80–135mmol/L) iPhos(0–20mg/dL)	WB Serum Plasma	100µL	• Minimal Maintenance: clean filter on back of analyzer • Self Calibrates • Automatic QC and calibration • Results in 12 minutes
Xpedite ISE VET Veterinary Electrolyte Analyzer-Diasys	ISE[a]	Na^+(40–200mmol/L) K^+(1.7–15.0mmol/L) iCa^{++}(0.3–5.0mmol/L) Cl^-(50–200mmol/L) Urine: Na^+(3–300mmol/L) K^+(5–120.0mmol/L) Cl^-(15–300mmol/L)	WB Serum Plasma Urine	95µL	• No daily maintenance required • Automatic QC checks

FF, flame photometry; ESP, enzymatic spectrophotometry; ISE, ion selective electrode; Na^+, sodium; K^+, potassium; Cl^-, chloride; iPhos, inorganic phosphate; iCa^{++}, ionized calcium; iMg^{++}, ionized magnesium; tMg, total magnesium; WB, whole blood; QC, Quality Control; min, minutes.

[a] Machine methodology stated as potentiometric or ion sensitive electrodes without specification of direct or indirect.

[b] i-Smart 30 PRO is a similar analzyer which measures hematocrit in addition to electrolytes

[c] Radiometer methodology for electrolytes is determined by settings for electrolytes as ISE vs. ESP

[d] Volume required for Nova dependent on which chemistry is being run

Table 56.2 Reference ranges and alert values of each electrolyte[a].

Electrolyte	Normal Range Canine	Normal Range Feline	Critical Care Alert Values[b]	Common Situations that may Cause Erroneous Results
Na^+	139–150mmol/L	139–150mmol/L	<120mmol/L >170mmol/L Change: >1.0mmol/L/hr >10mmol/L/24h	• Lipemia • Hemolysis • Hyperbilirubinemia • Hyperproteinemia • Sodium heparin
K^+	3.4-4.9mmol/L	147-162mmol/L	<2.5mmol/L >6.0mmol/L	• Thrombocytosis • Leukocytosis • Potassium EDTA • Delay in serum or plasma separation
Cl^-	106-127mmol/L	112-129mmol/L	<90mmol/L	• Lipemia • Hemolysis • Hyperbilirubinemia • Hyperproteinemia • Sodium heparin
iPhos	2.5-6.0mg/dL	3.1-7.5mg/dL	<1.5mg/dL	• Hemolysis • Citrate, Oxalate and EDTA anticoagulation • Hyperlipidemia • Hyperbilirubinemia • Delay in serum or plasma separation
iCa^{++}	1.12-1.40mmol/L	1.2-1.32mmol/L	<0.75mmoL/L >2.0mmol/L	• Aerobic sampling • Heparin dilution • Heparin binding • Delay in analysis
iMg^{++}	0.42-0.58mmol/L	0.47-0.60mmol/L		• Heparin dilution
tMg	1.5-2.5mg/dL	1.5-3.0mg/dL		• Heparin binding • Delay in analysis

[a] Catalyst DX chemistry analyzer (IDEXX Laboratories, Westbrook, ME), operator 's guide, and iSTAT Alinity (Abaxis, Union City, CA), user manual.
[b] Critical care alert values refer to values that suggest impending severe physiologic compromise. Any value outside of the reference range is considered clinically significant, however.

(gentamicin, amphotericin B, methoxyflurane, and vinca alkaloids) [17, 19, 22]. As evident by this extensive list of causes, many animals admitted to a veterinary intensive care unit are at risk of having or developing hypernatremia. Serial measurements to monitor for a rising sodium concentration are necessary to catch an animal trending toward hypernatremia before the clinical signs of hypernatremia develop, at which point irreversible neurologic damage may have occurred.

Hypernatremia results in cellular dehydration in the brain as water moves from the intracellular to the extracellular space along its osmotic gradient [19, 20]. The brain tissue shrinks, which can result in tearing of blood vessels and subarachnoid and intracerebral hemorrhage [17, 19]. Clinical signs are related more to the rate of increase of the sodium concentration rather than to the absolute concentration, and they include lethargy, nausea, depressed mentation, ataxia, and weakness [17, 20]. Neurologic signs can progress to seizures, coma, and death [17, 20].

Decreased sodium concentrations are caused by a relative gain of free water or hypotonic fluid within the extracellular space [16, 21]. This may be due to a true increase in total body water or a decrease in body fluids with sodium losses exceeding water losses [16, 20]. Measured hyponatremia may be due to an increase in osmotically active particles such as glucose or mannitol in the extracellular fluid causing a dilution of sodium molecules in the serum [16, 20, 21]. Puppies have been documented to have lower sodium concentration than adult dogs, with some puppies being below the reference interval up until approximately 11 weeks old [25]. Gastrointestinal disease, severe burns, and hemorrhage all cause fluid loss that can result in hypovolemia with normal sodium concentration [20, 21]. Sometimes, however, these clinical syndromes result in hyponatremia from an increase in antidiuretic hormone (ADH) levels in the face of hypovolemia, also called nonosmotic ADH release; ADH functions to retain free water and in this scenario prioritizes blood volume over the sodium

Table 56.3 Various fluid solutions and their sodium and potassium concentrations[a] (mEq/L).

Fluid Type	Sodium	Chloride	Potassium	Calcium	Magnesium
5% Dextrose in water	0	0	0	0	0
0.45% Saline	77	77	0	0	0
2.5% Dextrose in 0.45% Saline	77	77	0	0	0
Lactated Ringers Solution	130	109	4	3	0
Plasma-Lyte A	140	98	5		3
Normosol-R	140	98	5	0	3
0.9% NaCl	154	154	0	0	0
Vetstarch	154	154	0	0	0
7.5% Hypertonic NaCl	1232	1232	0	0	0

[a] Values reported based on each fluid type's drug insert

concentration, something that is typically kept relatively constant in the body [21]. Water retention and resultant hyponatremia can occur in cases of hypothyroidism with myxedema coma and in water intoxication [20, 26]. Loss of sodium through the kidneys can occur with hypoadrenocorticism, diuretic administration, or sodium-wasting renal disease [20]. A pathologic syndrome known as the syndrome of inappropriate antidiuretic hormone secretion (SIADH) has been described in people as well as veterinary patients [16, 20, 21, 27]. It is characterized by inappropriate release of ADH resulting in the retention of free water [16, 20, 21, 27]. This syndrome has been associated with certain neoplasms, pulmonary disease including pneumonia in a dog, central nervous system disease, perioperative status, and certain drugs [16, 20, 21, 27]. There is documentation of SIADH in human patients associated with the administration of fentanyl, a drug used commonly in critically ill or injured veterinary patients [28]. Disease processes associated with decreased effective circulating volume such as hepatic disease and primary cardiac disease can cause hyponatremia as the thirst center is activated, increasing water consumption [16, 20, 21]. Administering relative free water in the form of intravenous nutrition has also been associated with hyponatremia [29].

Similar to hypernatremia, the severity of the clinical signs associated with hyponatremia depend on the rate of drop as well as the absolute sodium concentration [20, 21]. Signs often do not occur unless the sodium concentration is less than 125 mmol/l. [20, 21] Vomiting, ataxia, depression progressing to obtundation, seizures, and coma may occur as a result of cerebral edema as water moves into neurons [21, 26].

Treatment of sodium concentration abnormalities involves addressing the underlying cause and slowly returning the sodium concentration to normal with the administration of relatively hypertonic (in the case of hyponatremia) and relatively hypotonic (in the case of hypernatremia) fluids. Table 56.3 details the sodium concentration of various electrolyte solutions that can be used to treat sodium abnormalities. Diuretic administration may be necessary to encourage sodium or water excretion [16, 17, 19–21]. The fluid of choice depends on the desired rate of sodium drop and the volume status of the patient. Cases of chronic sodium concentration abnormalities (> 48 hours) should be treated with the goal of gradually correcting the sodium concentration; acute cases can be addressed more aggressively [20, 21]. Sodium concentration should not be changed more frequently than 0.5–1.0 mEq/kg/hour, 10 mEq/l over 24 hours, or more quickly than 18 mEq/l over 48 hours [17, 20, 21]. Rapid adjustment of sodium concentration can result in neurologic sequelae.

In the case of hyponatremia, neuronal dehydration resulting in a syndrome known as osmotic demyelination syndrome (ODS, previously called central pontine myelinolysis) can occur if the sodium concentration is raised too fast during treatment [20, 21]. ODS is characterized by water moving out of brain cells during the correction of hyponatremia [20, 21]. Signs of ODS can be delayed one to six days and neuronal damage can be irreversible, making careful sodium monitoring vital because clinical signs may not immediately be apparent [20]. In the case of hypernatremia, rapid drops in sodium concentration can cause cerebral edema as extracellular water moves into neurons [20]. Unlike in ODS, the signs of cerebral edema and rising intracranial pressure due to overzealous correction of hypernatremia appear at the time of treatment and include depressed mentation, pupillary changes, thoracic limb rigidity, and seizures. The technician must monitor closely for these signs and pay close attention to serial sodium concentrations. Initially, sodium concentration should be reassessed every two to six hours during the treatment of sodium derangements, depending on the severity of

clinical signs, the degree of abnormality, and the duration of the disorder [20, 21].

Sodium Concentration Measurement

The reference standard for sodium measurement was traditionally flame photometry [16], which evaluates the entire volume of the sample, including both the aqueous and the nonaqueous phases [16]. Plasma is 93% water by volume, and sodium is present in the aqueous phase of plasma [16]. The distribution of sodium within only the aqueous portion of the sample results in an artifactually lower measured concentration of sodium than is truly present [16]. This difference is typically negligible, but in situations of hyperlipidemia and hyperproteinemia the nonaqueous portion of plasma increases [16]. When the nonaqueous portion of plasma is increased, the measured concentration of sodium will be significantly lower than the actual value, invalidating the results [16]. This artifact is known as pseudohyponatremia [16].

Direct measurement via ISE measures sodium in water only, avoiding the artifact caused by increased lipid or protein in samples [16, 20, 21]. Indirect ISE, however, involves a dilution step that is accounted for by adjusting the sodium concentration by a correction factor of 0.93 (93%), the plasma component of water in a "normal" sample [21]. Indirect ISE will therefore also reduce the "measured" sodium concentration if protein or lipid reduce the relative amount of the aqueous phase of the sample [21]. Low protein in the sample can result in falsely elevated values if measured via indirect ISE [21].

ESP can be used to measure sodium concentrations but can be inaccurate for hemolyzed, icteric, or hyperproteinemic samples and therefore should be used with caution in these circumstances [6]. Serum should be submitted to a reference laboratory that uses ISE for sodium and chloride concentration evaluation in icteric, lipidemic, or hyperproteinemic patients if the in-house electrolyte analyzer uses ESP to verify the in-house results. It also has higher normal reference intervals for sodium concentration than flame photometry or ISE, so analyzer-specific intervals must be used when interpreting results [5, 6].

Sodium heparin contains relatively high concentration of sodium (160 mEq/l) and chloride (166 mEq/l) and can falsely increase the measurement of both these electrolytes when whole blood is anticoagulated with sodium heparin at concentrations greater than 3.9% [3]. Thus, if one uses sodium heparin to anticoagulate the sample prior to measurement of sodium or chloride concentrations, preparation of an "evacuated syringe" (Protocol 56.1) or the purchase of commercially available syringes containing lyophilized 40 iu/ml heparin is necessary to avoid artifact [3]. One could also use lithium heparin for measurement of sodium or chloride concentrations [3].

Protocol 56.1 Preparation of a Heparinized Syringe or "Evacuated Syringe" for Blood Sampling

Items Required

- 3-ml syringe with a 22-gauge needle attached
- Liquid sodium heparin 1000 iu/ml concentration

Procedure

1) Aspirate 0.5 ml heparin into the syringe and set the heparin bottle aside. Leave the 22-gauge needle attached to the syringe.
2) Draw the plunger back to the 3-ml mark to coat the inner surface of the syringe
3) Expel all the air and heparin.
4) Draw 3 ml of air into the syringe and expel it forcefully.
5) Repeat step 4 a total of three times.
6) Perform venipuncture.
   - 0.5 ml sample is 7.8% Na heparin: inaccurate for all electrolyte measurements
   - 1.0 ml sample is 3.9% Na hHeparin: accurate for Na+ and K+ measurements
   - 3.0 ml sample is 1.3% Na heparin: likely acceptable for iCa++ and iMg++ measurements

iCa++, ionized calcium; iMg++, ionized magnesium.

Chloride

Chloride is an extracellular ion that functions with sodium in maintaining water balance and has an important role in acid–base balance [30]. Chloride concentration often changes with the sodium concentration as a reflection of free water, but certain situations can alter chloride concentration independent of changes in sodium. To determine whether the change in chloride concentration is due to an alteration in sodium, formulas were developed (Eq. 56.1) that function to determine the chloride concentration as high, low, or normal outside of the influences of free water [30]:

$$\text{Dog Cl corrected} = \text{Cl} \times 146 / \text{Na}^{+}$$
$$\text{Cat Dog Cl corrected} = \text{Cl} \times 156 / \text{Na}^{+} \quad (56.1)$$

Hypochloremia can be caused by vomiting, gastrointestinal obstruction, metabolic alkalosis, and therapy with diuretics that block absorption of chloride in the kidneys (e.g. furosemide) [31, 32]. Hypochloremia is more common than hyperchloremia in cats with sepsis and the systemic inflammatory response syndrome; the hypochloremia in these patients may represent a compensatory change associated with metabolic acidosis primarily due to uremia or ketosis, in which non-chloride, non-bicarbonate anions accumulate [33, 34]. Hyperchloremia is often the result of loss of bicarbonate-rich, chloride-poor fluid in diarrhea

and renal disease, and it is associated with a metabolic acidosis [32]. The administration of bicarbonate-free fluids such as 0.9% NaCl is thought to contribute to hyperchloremia associated with illness and can potentially cause iatrogenic hyperchloremia in critically ill patients [32, 35, 36].

The clinical signs of chloride derangements are typically associated with the underlying disease process or acid–base abnormality. Hyperchloremia may exacerbate cardiovascular compromise, acute kidney injury, coagulation disturbances, and gastrointestinal dysfunction [35, 36]. Treatment of chloride derangements involves addressing the underlying disorder and stopping any iatrogenic cause.

Chloride Concentration Measurement

Chloride measurement is similar to that for sodium in that it can be falsely lowered by lipemia and hyperproteinemia and falsely increased by the use of sodium heparin for anticoagulation [3, 6]. Direct ISE, particularly in the presence of low bicarbonate concentrations, is the more accurate method of measuring chloride concentration [37]. Chloride concentration is more affected by dilution with sodium heparin than sodium concentration [3]. The sensitivity of chloride concentration measurements to sodium heparin anticoagulant is likely caused by the high concentration (166 mmol/l) of chloride within sodium heparin. Because the concentration of chloride within sodium heparin is significantly higher than normal chloride concentrations, it spuriously increases the chloride measurement of the sample. Accurate chloride analysis should either be performed on serum, plasma, or whole blood that is anticoagulated with lithium heparin.

Red blood cells in venous blood carry more intracellular chloride than red blood cells in arterial blood, and the chloride concentration in venous blood is 3–4 mEq/l lower than arterial blood [30]. This variation is due to chloride shifting out of red blood cells in the presence of higher oxygen and lower carbon dioxide (generally the environment within arterial blood and room air relative to venous blood) [30]. If a sample is exposed to room air and permitted to "arterialize," the chloride concentration measurement will be falsely increased. Anaerobic sampling is therefore necessary when determining and interpreting chloride concentrations from venous blood [30, 38].

Potassium

Potassium is the most abundant cation in the body, but it is located primarily intracellularly, making measurement in whole blood, serum, or plasma an imperfect reflection of total body stores [16, 39]. The large concentration gradient of potassium across cell membranes enables potassium to be a key player in the excitability and function of nerve and muscle cells, including those in cardiac and vascular smooth muscle [39]. Potassium concentration is a reflection of intake (primarily through nutrition), loss into the gastrointestinal tract (a factor exacerbated in animals with gastrointestinal disease), kidney function, as the bulk of potassium is eliminated through the kidneys, and transcellular potassium shifts as occurs in some acid–base disorders and under the influence of insulin [32, 39].

Hyperkalemia can be caused by decreased renal excretion in acute kidney failure, end-stage kidney disease, or hypoadrenocorticism, urinary obstruction or intra-abdominal urine accumulation, and syndromes of massive cell death such as tumor lysis syndrome or rhabdomyolysis [32, 39]. Drug therapy with angiotensin-converting enzyme inhibitors (e.g. benazepril and enalapril), nonsteroidal anti-inflammatory agents, beta-blockers (e.g. atenolol), potassium itself, potassium-sparing diuretics (e.g. spironolactone), and heparin have been shown to cause hyperkalemia in people and may result in similar changes in small animal patients [16]. Patients at higher risk are those prescribed a combination of these drugs and those with decreased renal delivery of potassium, as might be seen in dogs and cats with cardiac disease and dehydration [39, 40]. A group of disease processes can cause concurrent hyperkalemia and hyponatremia with a decreased sodium-to-potassium ratio. This is the classic electrolyte picture with hypoadrenocorticism [41]. A decreased sodium-to-potassium ratio can also be seen in dogs and cats with gastrointestinal disease, kidney disease, pregnancy, body cavity effusions, and cardiac disease resulting in decreased effective circulating volume. Decreased effective circulating volume triggers free water retention and hyponatremia with concurrent decreased renal delivery of potassium for excretion, resulting in hyperkalemia [40, 42, 43].

Hyperkalemia can be life threatening as a result of the increased potassium concentration's effect on cardiac cell conduction and function [32, 39]. Clinical signs include muscular weakness with progression to paralysis [32, 39]. Cardiac toxicity is manifested by classic electrocardiogram (ECG) abnormalities, including tall, tented T waves, a prolonged PR interval, loss of the P wave, bradycardia, and widening QRS complexes (see Figure 56.1) [32, 39]. Ventricular tachycardia and fibrillation can occur [32]. In severe hyperkalemia, the ECG progresses to a sine-wave pattern as the QRS complexes merge with the T wave. This latter rhythm is an indication of impending cardiac arrest, and rapid intervention is indicated [39]. Life-threatening hyperkalemia generally occurs at potassium concentrations greater than 7.5 mEq/l [32], but concurrent abnormalities such as hypovolemia, acidemia, hypocalcemia, and hyponatremia may exacerbate the condition and trigger rapid cardiac collapse at lower potassium concentrations [39]. Treatment of hyperkalemia involves diuresis with intravenous fluid therapy, relieving urinary obstruction if present, cardiac protection with calcium gluconate or calcium chloride administration, and initiating intracellular movement

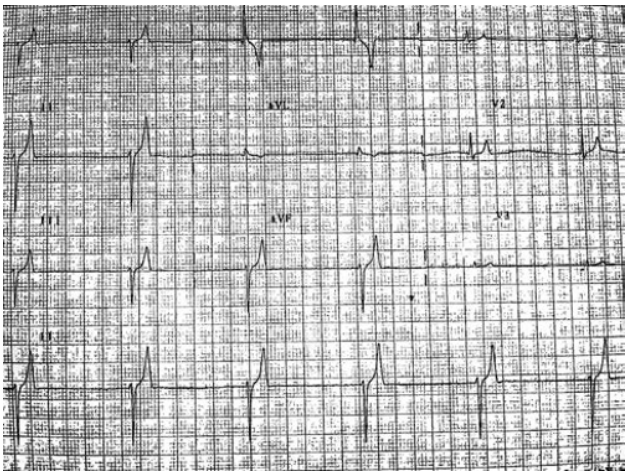


Figure 56.1 A 6-lead ECG showing some classic changes associated with hyperkalemia: tall, tented T waves; loss of the P wave; and widening QRS complexes.

of potassium with insulin, dextrose, beta agonists (e.g. terbutaline or albuterol) or bicarbonate therapy [32, 39, 44]. In the ICU, management of hyperkalemia also includes reducing potassium supplementation in fluids and drugs, and using only potassium-restricted diets for nutritional support. In refractory kidney failure, hemodialysis or peritoneal dialysis is required to treat hyperkalemia [32, 39].

Low potassium concentration, or hypokalemia, is typically the result of excessive potassium loss through the gastrointestinal tract or through the kidneys due to renal disease or osmotic diuresis (e.g. hyperglycemia, ketonuria, postobstructive diuresis); hypokalemia can also result from intracellular potassium shifts in the face of metabolic alkalosis [32, 39]. Hypokalemia is often confounded by decreased potassium ingestion due to nausea or anorexia [32, 39]. Iatrogenic causes of hypokalemia include diuretics, steroids, beta agonists (e.g. terbutaline, albuterol), or insulin and dextrose therapy [32]. Hypomagnesemia can cause refractory hypokalemia in which the potassium concentration does not return to normal despite aggressive supplementation [32]. Re-feeding of cats and dogs that have suffered prolonged starvation can result in hypokalemia due to insulin release in the face of depleted electrolyte stores [45, 46]. Hyperaldosteronism causes increased renal excretion of potassium and is a rare cause of severe hypokalemia in dogs and cats [24].

Clinical signs of hypokalemia generally occur with serum potassium concentrations less than 2.5 mEq/l and include generalized weakness characterized classically by cervical ventroflexion that can progress to paralysis and respiratory failure, inability to concentrate urine, anorexia, vomiting, and decreased bowel motility [32, 39]. Cardiac conduction abnormalities can occur and manifest on ECG by a depressed ST segment, decreased amplitude or inversion of the T wave, increased P wave amplitude, and prolonged PR and QRS intervals [32]. Multiple arrhythmias may also occur [32, 39].

Treatment of hypokalemia involves potassium supplementation orally or parenterally, depending on the severity of hypokalemia and whether or not the patient is tolerant of enteral treatments [32, 39]. Concurrent magnesium administration may be necessary to treat hypokalemia effectively [32]. Charts exist with recommended potassium supplementation based on the degree of hypokalemia, but these should be viewed as a starting point. Rate of potassium administration depends on the degree of total body depletion, renal and gastrointestinal losses of potassium, and rate of diuresis. Potassium concentration should be monitored at least once a day in patients on potassium supplementation and more frequently in animals with documented hypokalemia or pathophysiologic processes that may cause hypokalemia. Parenteral potassium supplementation should rarely exceed 0.5 mEq/kg/hour and should never exceed 1.0 mEq/kg/hour because rapid potassium administration can cause cardiac arrest [32]. The rate of potassium supplementation should be checked frequently, and fluids with potassium additives should not be administered rapidly as a bolus. Box 56.1 demonstrates how to determine the rate of potassium supplementation based on the concentration of potassium in the solution. When administering potassium in particular, but true of all hyperosmolar electrolyte additives, the infusate should be diluted to less than 550 mOsm/l to avoid phlebitis in peripheral, small, lower-flow veins [47]. Potassium additives in fluids have been found to be inaccurate in terms of actual potassium concentration of the fluid delivered compared with intended concentration [48]. However, adequate mixing of the fluid bag characterized by inversion of the bag four times results in more accurate potassium delivery [48].

Potassium Concentration Measurement

Artifactually increased potassium concentrations can be caused by anticoagulation with potassium EDTA as well as prolonged exposure to potassium-containing cells such as platelets and white blood cells [16, 43]. Thrombocytosis and leukocytosis have both been shown to cause pseudohyperkalemia [12, 43]. Artifactual hyperkalemia can be seen in blood samples from Japanese dog breeds (e.g. Akita, Shiba Inu) because of leakage of potassium from these breeds' potassium-rich red blood cells [12]. Potassium concentrations are generally higher in serum than in plasma due to release of potassium from platelets during clotting [12]. Canine serum potassium concentrations are generally 0.35 mmol/l higher than in canine plasma, and feline serum potassium was found to be 0.47 mmol/l higher than in feline plasma [12]. This expected variation between serum and plasma samples necessitates consideration of the sample type used to create the reference interval on the

Box 56.1 Back-Calculation to Ensure Safety of Potassium Supplementation in mEq/kg/hour

Method

1) Determine the amount of potassium in the solution by adding the amount of supplemented potassium to the amount already present in the stocked fluid (Table 56.3).
2) Divide the total amount of potassium by the total volume of the fluid to determine the concentration of potassium in the solution.
3) Determine the total amount of potassium being administered/hour by multiplying the amount of potassium/milliliter by the fluid rate of the patient in ml/hour.
4) Determine the rate of administration in mEq/kg/hour by dividing the amount of potassium/hour by the weight of the patient in kilograms.

Case example

A 10-kg Boston terrier being treated with lactated ringer solution with potassium 40 mEq/l added at a rate of 40 ml/hour.

1) Total amount of potassium in 1L of solution:
$$40\text{mEq}(\text{supplemented}) + 4\text{mEq}(\text{already present in fluid}) = 44\text{mEq}$$
2) Concentration of potassium in solution:
$$44\,\text{mEq}(\text{total amount potassium}) \div 1000\,\text{ml}(\text{total volume}) = 0.044\,\text{mEq/ml}$$
3) Amount of potassium administered/hour:
$$0.044\text{mEq/ml} \times 40\text{ml/hr} = 1.76\text{mEq/h}$$
4) Rate of administration:
$$1.76\text{mEq/h} \div 10\text{kg} = 0.18\text{mEq/kg/hour}$$

analyzer being used [12]. Serial samples on the same patient should always be run using the same sample type (i.e. serum or plasma) on the same machine. Although the changes caused by anticoagulation with potassium EDTA or potassium leakage from cells are typically minimal, they may result in pseudohyperkalemia or pseudonormokalemia if the patient's actual value is at the high end of normal or just below the reference interval, respectively [12]. Ideally, potassium concentration should be determined from whole blood, heparin anticoagulated whole blood, or plasma rapidly separated from the cell pellet to minimize length of exposure to potassium-containing cells [12].

Hemolysis and elevated total protein concentration appear to have less of an effect on potassium measurement than on sodium and chloride measurements, although analysis with ESP showed more variation in potassium measured in hemolyzed samples than either flame photometry or ISE [6]. Like sodium and chloride, however, values obtained via ESP are generally higher than those obtained via flame photometry or ISE [6]. Because ESP potassium concentration measurement can show more variation than other methods, samples for plasma potassium measurement should be submitted for ISE analysis to a reference laboratory in patients with severe hemolysis or hyperbilirubinemia if the available POC analyzer uses ESP.

Samples anticoagulated with heparin can cause dilution and artifactual lowering of potassium concentrations [3]. Syringes should be prepared for blood sampling using the "evacuated syringe" technique (Protocol 56.1) [3]. Use of a commercially available syringe coated with lyophilized 40 iu/ml heparin is also an option to avoid dilutional artifact [3].

Phosphorus

Phosphate is a vital intracellular ion in many physiologic processes including maintenance of cell membrane integrity, enzyme activation and deactivation, acid–base balance, clot formation, and energy production and use [49–51]. Most of the phosphorus in the body is within the mineralized bone matrix with the remainder mostly intracellular [49, 50]. Only a small portion (< 1%) is present in the extracellular fluid [49, 50]. Only one third of total plasma phosphorus, in the form of inorganic phosphate, is measured by chemistry analyzers [50]. Similar to potassium, changes in total body stores of phosphorus are not necessarily reflected by blood phosphate concentration [49, 50]. An increased blood phosphorus concentration may occur in situations of normal or low total body phosphorus levels. Transcellular shifts contribute to many of the alterations seen in phosphate concentrations measured in critically ill patients [49, 50].

Hypophosphatemia can be caused by a number of disease processes, as well as arising secondary to treatment of various disease states. It can be seen in patients with

decreased intestinal absorption, increased renal losses, hypomagnesemia, rapid tissue regeneration (i.e. liver regeneration after an episode of acute hepatic failure), head trauma, sepsis, hypothermia, hyperparathyroidism, vitamin D deficiency, and hypercalcemia associated with certain cancers. Hypophosphatemia can also be caused by the shifting of phosphorus into cells with respiratory or metabolic alkalosis, insulin, or dextrose administration (particularly in diabetic ketoacidosis), or re-feeding after prolonged starvation [45, 46, 49–51]. Respiratory alkalosis with associated hypophosphatemia can be seen in critically ill patients who are hyperventilating as a result of pain, anxiety, fever, or seizures [49]. Treatment of patients with phosphate-binding antacids (e.g. aluminum hydroxide) or aggressive diuresis in the face of decreased intake can result in hypophosphatemia [50].

Clinical signs generally do not occur unless hypophosphatemia is severe [49–51]. The most significant clinical sequelae of hypophosphatemia are decreased delivery of oxygen to the tissues due to an increased affinity of hemoglobin for oxygen, impaired white blood cell function, and the development of hemolytic anemia a result of altered integrity of the red blood cell membrane [49, 50]. Decreased oxygen delivery to tissues may cause subtle clinical signs, but severe cases may demonstrate neuromuscular depression first noted with diaphragmatic weakness, muscle fasciculations, and progressive neurologic signs such as ataxia and seizures [49, 51]. Other signs of hypophosphatemia such as ileus, anorexia, and vomiting are often thought to be part of the underlying disease [49]. Hemolysis in dogs has not been documented until phosphorus concentrations are less than 0.5 mg/dl, but cats appear to be more sensitive and have been shown to hemolyze at 1 mg/dl [49]. A decreased hematocrit may take 24–48 hours to become apparent following hemolysis triggered by hypophosphatemia [49, 50]. In addition to hemolysis, severe hypophosphatemia can cause myocyte damage with neuromuscular pain and pigmenturia [51]. Treatment of hypophosphatemia involves managing the underlying condition, addressing any acid–base abnormalities contributing to transcellular shifts, and administering phosphorus supplementation either enterally or parenterally in more severely affected or nauseated animals [49]. It is recommended to treat phosphorus concentrations under 3 mg/dl [49]. Parenterally administered potassium phosphate should be diluted before use because it is hypertonic and can cause tissue irritation [49]. It should be administered in calcium-free fluids (0.9% saline or 5% dextrose in water) to prevent precipitation of calcium salts [49]. Similar to other electrolytes, the dose depends on the rate of phosphorus loss and the underlying cause of hypophosphatemia. Recommended doses of parenteral phosphorus range from 0.01–0.06 mmol/kg/hour [49, 50]. Phosphorus concentrations should be monitored every three to six hours to ensure appropriate supplementation [49]. Toldimfos sodium is a derivative of phosphoric acid that has been used to treat severe hypophosphatemia [51]. Sodium, potassium, and ionized calcium concentrations should also be closely monitored while patients receive phosphorus therapy because it can cause increases in these electrolytes, and calcium concentration can drop due to binding of calcium to phosphorus [49].

Hyperphosphatemia is generally caused by decreased urinary excretion of phosphorus due to kidney disease or urinary obstruction; increased phosphorus release during cell death (e.g. tumor lysis syndrome), hypoparathyroidism, or iatrogenically through administration of phosphate-containing enemas [50]. Phosphate concentration is normally higher in growing animals up to one year of age due to rapid bone turnover; therefore, adult reference intervals usually underestimate the normal growing animal's phosphorus concentration [50]. Intoxications with vitamin D-containing rodenticides, anti-psoriasis creams (e.g. calcipotriol and calcipotriene), or zinc phosphide (a rodenticide used in gopher bait) can cause serious and potentially fatal hyperphosphatemia [50].

Clinical signs of hyperphosphatemia include weakness, tetany, seizures, tachycardia, and torsades des pointes, a potentially fatal cardiac arrhythmia [50]. Initial treatment should directly address severe sequelae of hyperphosphatemia such as seizures and torsades des pointes. Following stabilization, therapy to lower phosphorus concentration includes diuresis with crystalloid therapy and potentially dextrose administration to drive phosphorus into cells [50].

Phosphate Concentration Measurement

Phosphate should be measured on serum or heparinized plasma using ESP or flame photometry [49, 52]. Citrate, oxalate, and EDTA can interfere with the enzymatic assays and should therefore not be used [49]. Hemolysis of the sample causes artifactual hyperphosphatemia as inorganic phosphate is released from red blood cells and quantified along with the serum fraction. Similar to potassium, serum or plasma should be separated from cells within one hour of sampling to prevent leakage of cellular phosphorus into the sample [49]. Hyperlipidemia and hyperbilirubinemia may falsely decrease the phosphate concentration due to interference with colorimetric determination [49].

Calcium

Like phosphorus, most of the body's calcium is stored in bone with less than 1% readily available in plasma. Calcium exists in three forms in plasma or serum: ionized or "free" calcium (iCa^{++}), complexed or chelated calcium bound to other substances such as phosphate and sulfate, and protein-bound calcium [13]. Ionized calcium is the biologically

active form of calcium and has a role in many physiologic functions including intra- and extracellular messaging, cardiac conduction, and muscle contraction [53].

The most common cause of hypercalcemia in veterinary patients is neoplasia, specifically lymphoma [18, 53]. Other neoplasms can also cause hypercalcemia [18, 53]. Other causes of hypercalcemia include kidney failure, hyperparathyroidism, hypoadrenocorticism, and systemic pyogranulomatous disease [18, 53]. Intoxications that can result in hypercalcemia include vitamin D rodenticides, antipsoriasis creams (e.g. calcipotriol, calcipotriene), and day-blooming jasmine ingestion [18].

Hypercalcemia results in nausea, anorexia, abdominal pain, constipation, polyuria and polydipsia, and soft tissue mineralization [18]. It is treated with promotion of calciuresis by intravenous fluid therapy, furosemide, and glucocorticoids; treatment of the underlying cause; and medication to inhibit bone resorption (e.g. calcitonin and bisphosphonates) [18].

Hypocalcemia is common in critically ill humans and has been documented in hospitalized cats and dogs [54, 55]. It can occur in many disease states such as kidney failure, diabetic ketoacidosis, pancreatitis, eclampsia, malabsorptive syndromes, feline lower urinary tract obstruction, ethylene glycol toxicosis, tumor lysis syndrome, hypomagnesemia, and sepsis [56]. The systemic inflammatory response syndrome in a number of disease states including neoplasia, trauma, and gastrointestinal disease seems to be linked to hypocalcemia [55]. Iatrogenic causes of hypocalcemia include parathyroidectomy, phosphate enema, sodium bicarbonate, and furosemide. Multiple blood transfusions can cause hypocalcemia because the anticoagulant in the transfused blood products chelates calcium [56].

Clinical sequelae of hypocalcemia include restlessness or excitation, facial rubbing, muscle tremors, tetany, seizures, tachycardia, hyperthermia, and cardiopulmonary arrest [56]. Hypotension has been documented in human patients [56]. ECG changes include a prolonged QT interval [56]. Symptomatic hypocalcemia is life threatening and should be treated. Emergent treatment involves intravenous administration of 10% calcium gluconate at a dose of 1.0–1.5 ml/kg over 20–30 minutes with continuous ECG monitoring to watch for bradycardia [18]. If bradycardia occurs, the infusion should be discontinued until a normal heart rate returns [18]. Severe cases may require a constant rate infusion of 10% calcium gluconate (5–10 ml/kg over 24 hours) or repeated doses every six to eight hours [18]. Calcium solutions should not be added to bicarbonate-containing solutions because calcium carbonate can precipitate [18]. Careful monitoring of ionized calcium concentration to watch for over-correction and repeated patient assessment for the clinical signs of hypercalcemia or relapsing hypocalcemia are necessary [18].

Calcium Concentration Measurement

Calcium can be measured as either total calcium (a reflection of ionized and bound forms in plasma) or ionized calcium [16]. Total calcium can be affected by protein concentration, as a portion of total calcium is protein bound. Historically, formulas were created to correct total calcium for the influence of hypoalbuminemia [13, 54]. However, these formulas have since been shown to cause inaccurate interpretation of calcium concentration in the face of hypoproteinemia [13, 54]. Total calcium measurements correlate poorly with ionized calcium in situations of renal insufficiency and acid–base imbalance [8, 15]. Total calcium measurement can also be spuriously affected by hemolysis, hyperbilirubinemia, and lipemia [13]. Ionized calcium assessed by ISE, which minimizes interference by other ions, protein, hemolysis, and lipemia, is considered the most accurate and clinically appropriate way to measure calcium [13, 54].

Measurement of ionized calcium must be carefully planned and performed due to potential artifact associated with exposure to air, the anticoagulant used, hemodilution with the anticoagulant, and alterations in ionized calcium with prolonged handling time. Calcium can be measured in whole blood, plasma, or serum, but reference intervals for each sample type may vary slightly depending on the analyzer.

Ionized calcium concentration is affected by pH because hydrogen ions compete with ionized calcium for binding sites on albumin and displace ionized calcium from the protein [13, 14]. Acidemia, therefore, is associated with an increase in ionized calcium concentration. Exposure of the sample to air decreases a sample's carbon dioxide tension, raising its pH and falsely lowering the ionized calcium concentration [7, 8, 13, 15]. The change in ionized calcium with exposure to air appears to be about 7.2% and thus will only yield results in a different category (high vs normal vs low) if a patient has a mildly increased or low end of normal calcium concentration [14]. It has been found that there is acceptable correlation between ionized calcium concentration measurements sampled anaerobically and those measured aerobically with correction to a pH of 7.4 [13]. Automatic correction using formulas based on the predictable linear change in ionized calcium with changes in pH are available with some POC analyzers [3, 8, 13]. One must be very careful in veterinary medicine, however, because most POC analyzers are using an algorithm to correct the ionized calcium to a pH of 7.4 based on human data that are not validated in dogs and cats [3, 8, 13]. If an analyzer has an algorithm for ionized calcium correction to a pH of 7.4 that has been validated in dogs and cats, corrected aerobic measurements can be considered accurate. In most cases, however, this is not the case, and these authors recommend strictly anaerobic sampling for measurement of ionized calcium concentrations.

Anaerobic blood sampling is best performed using Vacutainer® tubes (Becton Dickinson, Franklin Lakes, NJ). Silicone serum separator tubes should be avoided because the silicone releases calcium and falsely increases the ionized calcium value [13]. Once the blood is clotted, the sample is centrifuged for serum separation. Serum can be removed from the tube using a spinal or other long needle attached to a non-air-containing syringe through the red-top stopper without exposing the sample to air [13]. Anaerobic sampling can also be achieved through the use of a sampling line (a central venous catheter or arterial catheter). Anaerobic sampling from a sampling line and gentle negative pressure avoiding air bubbles or air spaces from a venous sampling line. A rubber stopper can be placed on the needle if the sample cannot be placed directly into the cartridge or analyzer.

Calcium concentration also changes with time after a sample has been collected as cells undergo metabolism and produce lactic acid, which drops the pH in the sample [7]. Various studies have demonstrated slightly different results regarding the length of time an ionized calcium sample is stable at varying temperatures, but conservative collective interpretation of these studies indicates that if a sample is not analyzed within eight hours of collection, it should refrigerated (4°C) for up to 48 hours or frozen at −20°C for up to one week [7, 8, 15].

Sodium and lithium heparin bind or chelate calcium, which causes ionized calcium in the sample to drop [3, 7, 13]. Zinc heparin displaces calcium from binding sites and causes ionized concentrations to increase [13]. Oxalate, citrate and EDTA also bind calcium. Therefore, none of these anticoagulants should be used when collecting a sample for measurement of ionized calcium concentration [13]. It has been shown that 40 iu/ml of dry coated lithium heparin syringes or 3-ml syringes specially self-prepped with liquid sodium heparin do not cause significant dilution or binding of ionized calcium or magnesium [3].

Ionized calcium concentrations are lower in plasma than in whole blood or serum [8, 15]. This may be due to increased exposure time to heparin, causing increased binding to heparin [8, 15]. We recommend using either non-anticoagulated whole blood or low concentration (≤ 30 iu/ml) heparin anticoagulated whole blood for measurement of ionized calcium concentrations. Samples must be run relatively quickly after they are obtained to avoid premature clotting or prolonged exposure to heparin. Whole blood can be used to measure calcium concentration with the Vetscan i-STAT 1 to avoid error caused by anticoagulant, but efficient sampling and running are necessary to avoid the sample clotting. Most other POC analyzers that measure ionized calcium require anticoagulation (Table 56.1), and low concentration (≤ 30 iu/ml) heparin should be used. Animals with hypercoagulable states and cats are more likely to clot prematurely. Samples obtained from animals with a known or suspected hypercoagulable state should be anticoagulated with a low (≤ 30 iu/ml) concentration of heparin.

Magnesium

Magnesium functions mainly in the production and use of energy, maintenance of the sodium-potassium gradient across cell membranes, regulation of intracellular calcium, and muscle and cardiac contraction [57]. Magnesium is a predominantly intracellular ion distributed mostly in bone and muscle. Similar to calcium and phosphorus, approximately 1% exists in plasma and interstitial body fluid [57]. The plasma component has three portions: ionized, protein bound, and complexed to molecules such as phosphate and bicarbonate [57]. Ionized magnesium is the physiologically active form [57].

Hypomagnesemia is a common condition in critically ill people, dogs, and cats [57–59]. It is linked to a number of other electrolyte disorders including hypokalemia, hypocalcemia, hypophosphatemia, and hyponatremia [57, 60]. Causes of hypomagnesemia include gastrointestinal losses, decreased intake or malabsorption, acute pancreatitis, kidney disease, diabetes mellitus, hyperthyroidism, hyperparathyroidism, sepsis, hypothermia, re-feeding after prolonged starvation, and massive blood transfusion [57, 60]. Iatrogenic causes of hypomagnesemia include insulin administration causing intracellular shifts, increased renal losses of magnesium due to the administration of furosemide, and osmotic agents such as mannitol, as well as medications such as cyclosporine, pamidronate, beta agonists, and angiotensin-converting-enzyme inhibitors (e.g. enalapril, benazepril) [32, 57].

Clinical sequelae of hypomagnesemia include cardiac arrhythmias, anemia, neuromuscular weakness that can manifest as dysphagia or dyspnea if the esophagus or respiratory muscles are affected, muscle twitching, ataxia, seizures, coma, hypertension, refractory hypokalemia, and hypocalcemia [32, 57, 60]. ECG changes include prolonged PR interval, widened QRS complex, depressed ST segment, and a tented T wave [60]. Multiple arrhythmias can occur, including torsades des pointes [57]. Additionally, hypomagnesemia can trigger systemic inflammatory changes [57]. Treatment of hypomagnesemia involves the parenteral administration of magnesium sulfate or magnesium chloride at a rate determined by the degree of depletion, level of kidney function and the estimated rate of ongoing losses [32, 57]. Parenteral magnesium should be diluted to below a 20% solution as some of the formulations are hyperosmolar [57]. Some balanced electrolyte fluids including Plasmalyte-A® (Abbot Laboratories, Abbott Park, IL) and Normosol-R® (Hospira, Lake Forest, IL) contain

magnesium, and magnesium concentrations in these fluids should be taken into account in calculating magnesium dosages [57]. Magnesium infusions are being used therapeutically as treatment beyond supplementation in human critical care for an array of critical syndromes including systemic inflammatory response syndrome, severe tetanus, and reperfusion injury [57]. The use of this electrolyte as adjunctive therapy requires critical care staff awareness of treatment complications and necessary monitoring during infusions. Magnesium supplementation can cause burning at the catheter site, cutaneous vasodilation, and hypotension [57]. Iatrogenic overdose of magnesium can cause vomiting, hypotension, bradycardia, flaccid paralysis, and severe mental obtundation [61]. Intravenous calcium gluconate counteracts the physiologic effects of magnesium and can be used along with 0.9% NaCl diuresis to reverse these complications [61]. Blood magnesium concentrations should be monitored during magnesium supplementation as well as concentrations of potassium, sodium, calcium, and chloride [57].

Hypermagnesemia appears to be less common than hypomagnesemia [57–59]. It is most commonly caused by decreased renal clearance due to kidney disease but can also be seen with some endocrinopathies such as hyperparathyroidism, hypoadrenocorticism, and thyroid disorders [32, 57]. Hypermagnesemia can be caused iatrogenically through overdosing parenteral magnesium formulations or through the administration of multiple doses of magnesium-containing cathartics or antacids, particularly in patients with renal insufficiency [32, 57, 61].

As noted in the description of magnesium overdose, hypermagnesemia can cause obtundation, weakness, and hypotension [57, 61]. ECG changes include a prolonged PR interval and widened QRS complexes [57, 60]. Severe hypermagnesemia can cause respiratory depression to the point of apnea, coma, and cardiac arrest [57, 60]. Therapy for hypermagnesemia includes diuresis with fluid therapy and potentially furosemide [32, 57]. Calcium gluconate therapy should be used in severe cases if severe obtundation, respiratory depression, hemodynamic instability, or arrhythmias are present [17, 57]. Dialysis may be necessary if kidney function is severely decreased [57].

Magnesium Concentration Measurement

Because magnesium is primarily intracellular, it is still unclear which clinical magnesium sample best reflects total body stores [57]. Clinicopathologic options for magnesium concentration quantification include ionized magnesium, total serum or plasma magnesium, intracellular quantification of magnesium via nuclear magnetic resonance spectroscopy and fluorescent dye labeled magnesium, or magnesium loading tests to determine total body depletion [57]. A study evaluating intracellular versus total serum magnesium in dogs with gastric dilatation and volvulus demonstrated no correlation between the two methods [62]. Because ionized magnesium is the biologically active form, and total magnesium concentration is influenced by factors similar to those that affect total calcium concentration (protein concentration, kidney function, acid–base balance), ionized magnesium concentration is theoretically the POC analyte of choice [7, 8, 57, 63]. ISE assay is the method of choice for determining magnesium concentration because there is less interference caused by icterus, lipemia, and hemolysis [54].

Sample handling and causes of artifact are similar for ionized magnesium as they are for ionized calcium. Aerobic sampling causes artifactual decreases in ionized magnesium due to an increased pH and increased protein binding [8]. Both lithium and zinc heparin formulations can interfere with the magnesium electrode and cause falsely increased ionized magnesium concentrations [15, 64]. Serum values of ionized magnesium may be higher than for whole blood or plasma due to possible release of magnesium from platelets during clotting. Despite this, anaerobically obtained serum samples are considered the most accurate for determination of magnesium and calcium concentrations due to lack of dilution, binding, or interference from anticoagulant. Sample storage for measurement of magnesium is similar to that for calcium; ionized magnesium concentration should be analyzed within eight hours if kept at room temperature (22°C), within 24 hours at 4°C, and within one week if stored at −22°C [8]. Ionized magnesium concentration drops with time if samples are held too long or stored inappropriately. Feline samples are stable for 24 hours at 2°C, 72 hours at 4°C, and 4 weeks at −20°C [63].

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57

Acid–Base Evaluation

Kate Hopper

Acid-base evaluation is recommended for all patients with significant illness or injury. In these patients, the magnitude of acid–base derangement is difficult to ascertain by clinical signs alone, and the measurement of acid–base status is an important component of the initial assessment and ongoing patient monitoring.

Sampling and Storage of Blood for Acid–Base Measurement

For acid–base measurements, venous or arterial blood can be used. In health, there is a small but clinically unimportant difference between venous and arterial acid–base values (Table 57.1) [1]. In clinical scenarios of poor perfusion such as circulatory shock, there can be a significant variation between arterial, central venous, and peripheral venous values.

The technique for collecting a blood sample for pH and blood gas analysis is outlined in Protocol 57.1. Dilution of the blood sample by anticoagulant should be minimized. Use of commercially available blood gas syringes or filling anticoagulated hematocrit tubes or cartridges designed for blood gas evaluation is ideal.

Blood should be collected anaerobically. Exposure to small air bubbles allows equilibration of gases between the blood and the air. Air mixing will lower the partial pressure of carbon dioxide, which will change the pH. The sample should be analyzed as soon as possible after collection to minimize in vitro changes due to metabolism and diffusion of gases into and through the plastic wall of the syringe. For acid–base evaluation (not partial pressure of oxygen), samples can be kept in a syringe at room temperature for up to two hours before analysis [2]. It is not recommended to store samples in vacuum tubes or screw top tubes for acid–base evaluation.

Overview of Acid–Base Interpretation

Acid–base balance is the evaluation of hydrogen ion concentration [H^+] in the body. Hydrogen ions are important to normal physiology and severe abnormalities in [H^+] can directly impact cell and organ function. In clinical medicine, evaluation of acid–base balance is even more important as a diagnostic and monitoring tool. Blood gas machines need very small volumes of blood and provide immediate results, making them ideal for use in the emergency room and intensive care setting. Clinically [H^+] is measured as pH, a unitless value that has an *inverse* relationship with [H^+]. Increased [H^+] causes a decrease in pH, an acidemia. A decreased [H^+] causes an increase in pH, an alkalemia.

The contributions to acid–base balance are divided into respiratory (controlled by the lungs) and metabolic (all other mechanisms) components. The respiratory contribution to the acid–base disturbance is defined by the partial pressure of carbon dioxide (PCO_2), while the metabolic contribution is defined by the bicarbonate concentration or base deficit. We will focus on bicarbonate concentration [HCO_3^-] at this time. The pH is determined by the ratio of [HCO_3^-] to PCO_2:

$$pH \sim \frac{[HCO_3^-]}{PCO_2}$$

From this relationship, it can be appreciated that an acidemia (low pH, high [H^+]) may be due to decreased [HCO_3^-], increased PCO_2, or both these changes, while an alkalemia (high pH, low [H^+]) may be due to increased [HCO_3^-], decreased PCO_2 or both these changes (Figure 57.1).

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Companion website: www.wiley.com/go/burkitt/monitoring

Table 57.1 Arterial versus venous blood gas values for normal dogs (mean ± standard deviation) [1].

Measure	Arterial	Mixed venous	Jugular venous	Cephalic vein
pH	7.40 ± 0.03	7.36 ± 0.02	7.35 ± 0.02	7.36 ± 0.02
PCO_2 (mmHg)	37 ± 3	43 ± 4	42 ± 5	43 ± 3
Base deficit (mmol/l)	−2 ± 2	−1 ± 1	−2 ± 2	−1 ± 1
Bicarbonate (mmol/l)	21 ± 2	23 ± 2	22 ± 2	23 ± 1
Total CO_2 (mmol/l)	22 ± 2	24 ± 2	23 ± 2	24 ± 2
PO_2 (mmHg)	102 ± 7	53 ± 10	55 ± 10	58 ± 9

Source: Adapted from Ilkiw 1991.

Protocol 57.1 Protocol for Collecting a Blood Sample for pH and Blood Gas Analysis

Items Required

- Use either a commercial blood gas syringe with dry anticoagulant or prepare a sampling syringe by drawing an aliquot of liquid heparin solution into it and then blowing as much of the liquid heparin out of the dead space of the syringe as possible. If acid–base analysis is to be performed in anticoagulated hematocrit tubes or cartridges, a small volume of whole blood in a plain syringe is collected and immediately transferred.
- If blood is to be taken from a catheter:
  - Use a separate syringe with about 0.5 ml of heparinized saline to scavenge 3–5 ml fluid and blood from the catheter prior to sample collection.
  - Use a separate syringe with about 3 ml of heparinized saline to flush the catheter after the fluid-scavenged blood mixture has been returned to the patient (see Chapter 53).

Procedure

Blood taken via direct vessel puncture:

1) Gather supplies.
2) Perform hand hygiene ± wear gloves.
3) Clip the fur and aseptically prepare the skin over the intended puncture site.
4) Aseptically puncture the vessel and anaerobically obtain an appropriate volume of blood.

Blood taken from a catheter:

1) Scrub the injection port with antiseptic solution. The fluid infusion must be stopped.
2) Remove (scavenge) at least 3 ml blood from the catheter into syringe containing 0.5 ml heparinized saline.
3) Switch to a new, empty blood collection syringe and collect the desired volume of blood.
4) Return the scavenged blood-heparinized saline mixture to the patient.
5) Flush the catheter with heparinized saline.

Storage and analysis:

1) Analyze the samples immediately.
2) If the sample cannot be analyzed within 30 minutes, store anaerobically at room temperature. If analysis will be delayed for greater than 30 minutes, samples should be collected in a glass syringe and stored in ice water.
3) Mix the sample prior to analysis by vigorously rolling the syringe between your hands.
4) Insert the blood anaerobically into the analyzer as per the manufacturer's guidelines.
5) Input the patient's identification and temperature.

Other Measures of the Metabolic Contribution

The metabolic component is commonly evaluated from the $[HCO_3^-]$ but other parameters can be used instead. Base deficit/excess is a value calculated by many blood gas machines. The terms *base deficit* and *base excess* have become interchangeable; the measured value defines if there is a deficit or gain in base. For the purpose of this chapter, the term base deficit will be used. The normal

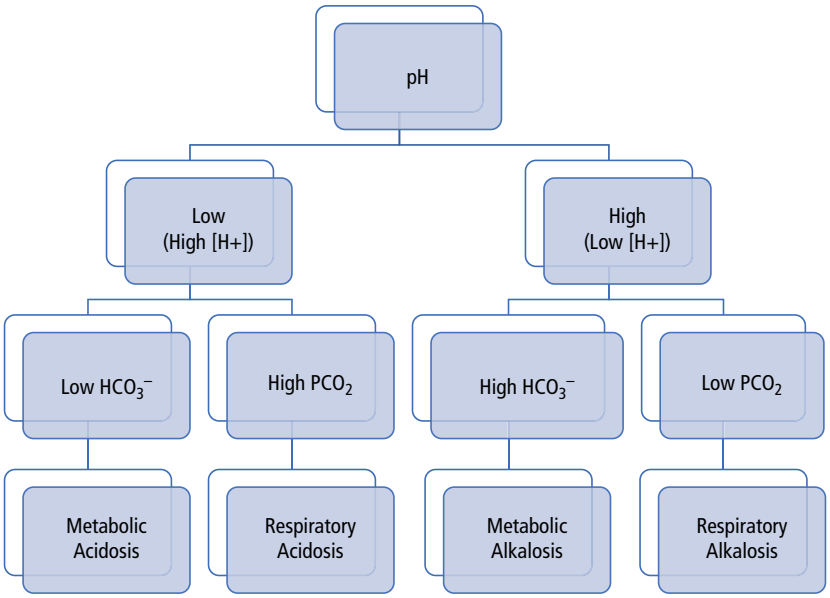


Figure 57.1 Algorithm for acid–base evaluation.

range for base deficit in dogs and cats is slightly negative as a result of their carnivorous diet. If the measured patient value for base deficit is more negative than the reference interval for the species, it represents a metabolic acidosis while values more positive than the reference interval are a metabolic alkalosis.

Total carbon dioxide (TCO_2) is another measure of the metabolic contribution to acid–base balance. It is often a parameter reported on biochemistry panels and is a value similar to [HCO_3^-]. Given the confusing name, it is important not to confuse the metabolic parameter TCO_2 with the respiratory parameter PCO_2.

Compensation

In response to a primary disturbance of the respiratory or metabolic system, the body will attempt to correct the abnormality in pH with changes in the opposing system. For example, a metabolic acidosis will normally be accompanied by a compensatory respiratory alkalosis. As a result, the abnormality in pH is minimized (the ratio of HCO_3/PCO_2 is less abnormal). Compensation returns the pH toward normal but does not return the pH completely to normal. Respiratory compensation to a primary metabolic abnormality is very rapid and expected to be evident at the time of blood gas evaluation [3]. The absence of respiratory compensation is always considered abnormal. Primary respiratory acid–base disorders will have a small degree of "acute metabolic compensation," but full metabolic compensation takes hours to begin and days to complete [3]. For this reason, the absence of any substantial metabolic compensation to a primary respiratory disorder may simply reflect insufficient time for compensation.

Acid–Base Analysis

When making an acid–base diagnosis, the patient values of pH, PCO_2 and [HCO_3^-] are compared to a species-specific reference interval as shown in Table 57.2.

The pH should be evaluated to determine if it is normal, low (acidemia), or high (alkalemia). The PCO_2 is evaluated as either normal, low (respiratory alkalosis), or high (respiratory acidosis). The [HCO_3^-] is either normal, low (metabolic acidosis), or high (metabolic alkalosis). Once these evaluations have been made, the next step is to determine if a primary process (respiratory or metabolic) can be identified. The primary process is the one responsible for the abnormality in pH. When a primary process is identified, the opposing system should then be evaluated for appropriate compensation (Examples 57.1 and 57.2).

In Example 57.1, the only process that can be considered responsible for the acidemic pH is the metabolic system; the primary disturbance is therefore a metabolic acidosis. Respiratory compensation is fast and should always be

Table 57.2 Arterial acid–base values for normal individuals; mid-normal (reference interval).

Measure	Human [4]	Dog [1, 5, 6]	Cat [6–8]
pH	7.40 (7.35–7.45)	7.39 (7.35–7.43)	7.39 (7.33–7.45)
$PaCO_2$ (mmHg)	40 (35–45)	37 (31–43)	32 (26–38)
Bicarbonate (mmol/l)	24 (22–26)	22 (19–25)	18 (15–21)
Base deficit (mmol/l)	0 (−2 to +2)	−2 (+1 to −5)	−6 (−3 to −9)

Example 57.1 Acid–Base Disorder 1

Parameter	Patient	Reference interval	Interpretation
pH	7.26	7.35–7.45	Acidemia
PCO_2 (mmHg)	30	32–43	Respiratory alkalosis
Bicarbonate (mmol/l)	12	18–24	Metabolic acidosis
Base deficit (mmol/l)	–10	–4 to –2	Metabolic acidosis

Example 57.2 Acid–Base Disorder 2

Parameter	Patient	Reference interval	Interpretation
pH	7.50	7.35–7.45	Alkalemia
PCO_2 (mmHg)	15	32–43	Respiratory alkalosis
Bicarbonate (mmol/l)	18	18–24	Normal
Base deficit (mmol/l)	−2	−4 to −2	Normal

Example 57.3 Acid–Base Disorder 1

Parameter	Patient	Reference interval	Interpretation
pH	7.036	7.35–7.45	Acidemia
PCO_2 (mmHg)	62	32–43	Respiratory acidosis
Bicarbonate (mmol/l)	14	18–24	Metabolic acidosis
Base deficit (mmol/l)	−8	−4 to −2	Metabolic acidosis

Example 57.4 Acid–Base Disorder 2

Parameter	Patient	Reference interval	Interpretation
pH	7.396	7.35–7.45	Normal
PCO_2 (mmHg)	59	32 – 43	Respiratory acidosis
Bicarbonate (mmol/l)	30	18 – 24	Metabolic alkalosis
Base deficit (mmol/l)	+10	−4 to −2	Metabolic alkalosis

present to a primary metabolic abnormality. A respiratory alkalosis would be the appropriate compensation for a primary metabolic acidosis so in this example the acid–base analysis is a primary metabolic acidosis with respiratory compensation. In example 57.2, the respiratory alkalosis is clearly responsible for the alkalemia. There is no evidence of metabolic compensation, but as metabolic compensation is slow to occur, its absence suggests an acute respiratory acid–base disorder. The acid–base analysis is an acute respiratory alkalosis.

Simple Versus Mixed Disturbances

Acid–base abnormalities can be simple disorders of only one system (respiratory or metabolic), and any change in the other system is purely compensatory (such as Examples 57.1 and 57.2). When there are abnormalities in both the respiratory and metabolic systems, neither of which can be considered compensatory, it is classified as a mixed disorder. In this situation, there is no primary disturbance since both disturbances are "primary" (independent of one another). For example, when both the respiratory and metabolic systems have the same abnormality (both have an alkalosis or both have an acidosis), it is a mixed disorder. A normal pH in the face of abnormal PCO_2 and HCO_3^- also indicates a mixed disorder since compensation alone will not return pH to normal. In example 57.3, both the respiratory system and the metabolic system contribute to the acidemia. This animal has two problems (not one disorder with compensation). As such, this is a mixed disorder of both respiratory and metabolic acidosis.

In example 57.4, there is a normal pH, but abnormalities are present in both the PCO_2 and bicarbonate. By definition, this is a mixed disorder. There are two acid–base disorders present: a respiratory acidosis and a concurrent metabolic alkalosis. These abnormalities are of similar magnitude so their effect on pH effectively cancels each other out, ultimately leaving the patient with a normal pH. It is important to recognize that this cannot be a primary disorder with "excellent" compensation (i.e. primary metabolic alkalosis with "excellent" respiratory compensation) as even perfect compensation does not return the pH back to the normal range.

The Respiratory Component of the Acid–Base Balance

The respiratory contribution to the acid–base balance is defined by the PCO_2. The arterial PCO_2 is regulated by the respiratory center of the brain by adjusting how the animal

Box 57.1 Common Causes of Respiratory Acid–Base Disorders

Respiratory Acidosis (Hypercapnia)

- Hypoventilation – diseases causing reduced respiratory rate and/or tidal volume
  - Brain disease
  - Cervical spinal cord disease
  - Peripheral nerve or neuromuscular junction disease
  - Muscle fatigue or myopathy
  - Airway obstruction
  - Anterior displacement of the diaphragm by abdominal space filling disorders
- Carbohydrate-rich parenteral nutrition solutions in debilitated patients
- Bicarbonate therapy in patients with respiratory compromise
- Rebreathing of exhaled alveolar gases due to mechanical dead space
- Malignant hyperthermia
- Venous blood gas only - poor tissue perfusion

Respiratory Alkalosis (Hypocapnia)

- Hyperventilation – diseases causing increased respiratory rate and/or tidal volume
  - Hypotension
  - Fever and heat-induced illness
  - Systemic inflammatory response
  - Pulmonary thromboembolism
  - Pulmonary parenchymal disease
  - Excitement and exercise
  - Pain

breathes. Breathing faster and/or deeper (hyperventilation) will decrease PCO_2 in arterial blood ($PaCO_2$), while breathing slower and/or shallower (hypoventilation) will increase $PaCO_2$. In most situations, there is only a small difference between arterial and venous PCO_2 ($PvCO_2$). But in states of poor tissue perfusion (e.g. circulatory shock), $PvCO_2$ may not accurately reflect $PaCO_2$.

Respiratory Acidosis

The causes of respiratory acidosis (elevated PCO_2) are cited in Box 57.1. An elevated PCO_2 level can also be described as hypoventilation or hypercapnia. Significant elevations in $PaCO_2$ (> 60 mmHg) may warrant treatment. Without supplemental oxygen therapy, this magnitude of hypoventilation is likely to be associated with hypoxemia. Hypercapnia causes cerebral vasodilation, which increases cerebral blood flow that may be harmful in patients with intracranial disease. With proper support and time for compensation, and in patients without intracranial disease, considerably higher PCO_2 values may be permissible without apparent harm to the patient.

The first treatment for primary respiratory acidosis is effective treatment for the underlying disease process. The symptomatic therapy for hypoventilation is support of the respiratory system and may require positive pressure ventilation if severe.

Respiratory Alkalosis

The causes of respiratory alkalosis are listed in Box 57.1. A decreased PCO_2 level can also be described as hyperventilation or hypocapnia. Treatment for respiratory alkalosis is largely focused on resolution of the underlying disease that is causing the hyperventilation.

The Metabolic Component of Acid–Base Balance

Unlike the respiratory component of acid–base balance, there are many different contributors to the overall metabolic acid–base balance. Evaluation of the primary disease processes evident in the patient and additional parameters such as the anion gap and lactate concentration are all considered when determining the cause of metabolic acid–base disorders.

Metabolic Acidosis

The causes of metabolic acidosis are listed in Box 57.2. Metabolic acidosis can occur by one of two mechanisms: the gain of acid or the loss of bicarbonate. In the clinical setting, acid gain is usually associated with an increase in anion gap, while diseases associated with the loss of

Box 57.2 Common Causes of Metabolic Acid–Base Disorders

Metabolic acidosis with normal anion gap

- Gastrointestinal losses of bicarbonate
- Renal loss of bicarbonate/acid retention (renal tubular acidosis; hypoadrenocorticism)
- Large-volume 0.9% saline administration

Metabolic acidosis with increased anion gap[a]

- D = diabetic ketoacidosis
- U = uremia
- E = ethylene glycol intoxication
- L = lactic acidosis
- Methanol intoxication
- Salicylate poisoning

Metabolic alkalosis

- Gastric losses of acid (vomiting due to a pyloric obstruction, gastric suctioning)
- Furosemide administration
- Hyperaldosteronism
- Organic anion (lactate, acetate, citrate) administration
- Bicarbonate administration

[a] Anion gap may not always be elevated as predicted.

bicarbonate are associated with a normal anion gap. See below for more explanation of the anion gap. Common causes of a high anion gap metabolic acidosis can be recalled with the acronym DUEL: D, diabetic ketoacidosis; U, uremia; E, ethylene glycol intoxication; L, lactic acidosis.

The treatment of metabolic acidosis should be primarily aimed at correction of the underlying disease process, and should be the only therapy necessary if the metabolic acidosis and the pH disturbance is mild to moderate and the underlying disease is readily treatable. The kidney plays an important role in regulation of the metabolic acid–base balance. As such, kidney disease commonly causes metabolic acidosis in dogs and cats. If there is no immediate treatment to improve kidney function, bicarbonate administration to improve the acid–base abnormality maybe considered. Bicarbonate therapy may also be considered in severe metabolic acidosis of other causes in some circumstances. Guidelines for the calculation of bicarbonate dosage are detailed in Protocol 57.2. Sodium bicarbonate administration may be associated with a number of adverse effects; these problems and their avoidance are detailed in Box 57.3.

Metabolic Alkalosis

The causes of metabolic alkalosis are listed in Box 57.2. The treatment of metabolic alkalosis relies on effective treatment of the underlying disease process. Coexistent electrolyte abnormalities such as hypochloremia and hypokalemia potentiate metabolic alkalosis and should also be treated.

Anion Gap

The anion gap is a calculated value that can be useful in determining the cause of a metabolic acidosis. It is calculated using the equation:

$$AG = \left(Na + K\right) - \left(HCO3 + Cl\right) \quad (57.1)$$

where Ag is the anion gap; Cl, chloride; HCO3, bicarbonate; K, potassium; Na, sodium.

There is no anion gap in reality (the number of cations always equals the number of anions). In this calculation, $Na + K$ normally exceeds $Cl + HCO_3$ by 15–20 mmol/l (varies between laboratories). Normally, the negative charges on albumin comprise most of this apparent gap. Phosphate and lactate make up a small portion of the gap in the normal animal, but this can increase in disease states [9].

Metabolic acidosis due to a gain of acid is usually associated with an increased anion gap. Common causes include lactic acid, ketoacids, elevated phosphate levels, or acid intoxicants such as glycolic acid from ethylene glycol and salicylic acid from salicylate (aspirin).

Metabolic acidosis can also be caused by renal or gastrointestinal bicarbonate losses without an increase in anion gap. As a result, evaluation of the anion gap can provide

Protocol 57.2 How to Calculate and Administer a Dose of Sodium Bicarbonate

Procedure

1) Use the base deficit value determined by the blood gas machine or estimate the base deficit from the equation:

$$\text{Patient}\left[HCO_3^-\right] - \text{normal}\left[HCO_3^-\right]$$

2) Estimate the total bicarbonate deficit from the equation:

$$\text{Bicarbonate}(\text{mmol}) = \text{base deficit} \times 0.3 \times \text{body weight}(\text{kg})$$

3) Administer 50–75% of this calculated dose to avoid creating an iatrogenic metabolic alkalosis.
4) Administer dose slowly over a minimum of about 30 minutes (more commonly over several hours).
   a) Undiluted sodium bicarbonate (1 mmol/ml) has an osmolality of ~2000 mOsm/l, which can cause phlebitis with extended infusions and a sodium concentration of 1000 mmol/l, which can cause hypernatremia with large infusions.
   b) Recommend diluting sodium bicarbonate with a sodium-free fluid such as dextrose 5% in water or sterile water. See Table 57.3 for some possible dilutions.
   c) Sodium bicarbonate can be administered with other fluids but is not compatible with many drugs. Compatibility should be verified before co-administering drugs.

Table 57.3 Suggested dilutions for sodium bicarbonate 8.4% (1 mmol/ml).

Sodium bicarbonate (1 mmol/ml)	D5W or sterile water	Total number of parts	Approximate Final osmolality
1 part	1 part	2 parts	100 mOsm/l
1 part	2 parts	3 parts	667 mOsm/l
1 part	3 parts	4 parts	500 mOsm/l
1 part	4 parts	5 parts	400 mOsm/l
1 part	5 parts	6 parts	333 mOsm/l
1 part	6 parts	7 parts	286 mOsm/l

Box 57.3 Adverse Effects of Sodium Bicarbonate Administration

- Excessive alkalinization of the patient: calculate dosages carefully; monitor acid–base balance.
- Hypokalemia: administer carefully and with concurrent potassium supplementation in hypokalemic patients; monitor potassium concentration.
- Low ionized calcium concentration: administer carefully, potentially with concurrent calcium supplementation in hypocalcemic patients; monitor ionized calcium.
- Hypercapnia: administer carefully with carbon dioxide monitoring in patients with mild hypoventilation. Sodium bicarbonate administration is contraindicated in patients with moderate or severe hypoventilation.
- Phlebitis with continuous infusions through a peripheral vessel: ideally dilute fluid for infusion to an osmolality < 600 mOsm/l unless administering through a central catheter.
- Hypernatremia with large infusions of undiluted solution: monitor sodium with repeated dosages of sodium bicarbonate; where possible, dilute for administration.
- Volume overload: if not diluted sodium bicarbonate is a hypertonic saline solution that can impact vascular volume. When diluted it becomes a significant volume for administration and should be used with caution in at-risk patients.

diagnostic insight regarding the potential cause of a metabolic acidosis. Unfortunately, the anion gap may not be elevated despite the presence of added acids, and should be considered supportive information for diagnosis, but not definitive. In the presence of hypoalbuminemia, the anion gap loses sensitivity and can be normal despite the presence of gained acid [10].

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58

Osmolality and Colloid Osmotic Pressure

Elke Rudloff and Angel Rivera

Water is the most essential nutrient of the body. Within the vessel, water is the transport medium that delivers oxygen, solutes, and hormones to the interstitium while removing waste products for breakdown and excretion. Within the interstitial space, water provides the environment for these substances to move between the capillary and the cell. Within the cell, water provides a medium for organelles and for cell membrane expansion. Water also provides a means to dissipate heat through evaporation.

Identifying water imbalance in the critically ill animal can be one of the most important challenges of patient management. Qualitative information of a patient's water needs is obtained through evaluation of physical parameters of perfusion and hydration [1]. Quantitative information can be obtained with laboratory evaluation of osmolality as well as colloid osmotic pressure (COP). This chapter focuses on the basic concepts behind the role of osmolality and COP in the movement of water in the body, as well as understanding, measuring, and interpreting osmolality and COP.

Physiology of Water Movement: Osmolality

An *osmole* is a unit term used to describe a particle that contributes to the osmotic pressure of a solution; 1 osmole is equivalent to 1 mole (6.02×10^{23} particles) of any non-dissociable substance, regardless of the substance's composition, charge, size, or weight [2]. Osmoles are often expressed in terms of *milliosmoles* (1 osmole = 1000 milliosmoles). *Osmolarity* describes the total concentration of all solutes dissolved in a volume of water and is expressed as milliosmoles/liter (mOsm/l). *Osmolality* describes the total concentration of all solutes dissolved in a mass of solution and is expressed as milliosmoles/kilogram (mOsm/kg). Because 1 l of water weighs approximately 1 kg and because water is the solvent in biological systems, the terms *osmolarity* and *osmolality* are often used interchangeably when discussing the composition of body water, except when significant hyperlipidemia exists.

Water moves freely across nearly all membranes separating the intravascular, interstitial, and intracellular compartments in the body. The two important factors that affect the movement of water across the membranes are: (i) the difference in the concentration of water molecules (which relates to the number of solutes or *osmoles*) on one side of the membrane compared with the other, and (ii) the difference in the hydrostatic pressure on one side of the membrane compared with the other.

Water is an uncharged molecule, and its movement is governed passively by its chemical gradient, also called a concentration gradient, across a membrane: Water molecules will move from areas of higher water concentration to areas of lower water concentration. The pressure that generates this passive water movement (*diffusion*) along a concentration gradient is called *osmotic pressure*. Areas of high osmotic pressure have relatively low water concentration in relation to solute, whereas areas of low osmotic pressure have relatively high water concentration in relation to solute. Osmotic pressure is therefore a chemical pressure generated by the particles (solutes) dissolved in water that tends to hold water on the particles' side of a membrane permeable only to water.

Hydraulic pressure in the vessels is the physical pressure generated by the heart and conveys a hydrostatic pressure of blood at the level of the capillary network. Hydrostatic pressure in the interstitium is created by the interaction of collagen fibrils, fibroblasts, and the lymphatics, which are dynamic. By expanding, contracting, and pumping, they influence interstitial hydrostatic pressure, which is kept more negative than the intravascular space in health. Water is in equilibrium across a membrane when the net driving

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force for water movement is zero. In other words, at equilibrium, water does not move across a membrane because the osmotic pressure gradient ("holding water in") is equal to the hydrostatic pressure gradient ("pushing water out") across that membrane.

The primary solutes that produce osmotic pressure in the extracellular (intravascular and interstitial spaces combined) compartment of dogs, cats, and people are the most plentiful dissolved substances: sodium and its accompanying anions, glucose, and urea [2, 3]. The primary solutes that produce intracellular osmotic pressure are potassium and magnesium because they are the most plentiful dissolved intracellular molecules [2, 3]. Effective osmolality (also called *tonicity*) is generated by solutes that are unable to pass between the intracellular and extracellular compartments. Because they do not freely cross the cellular membrane, they can affect water movement (they are "effective"). When there is a difference in tonicity between the intracellular and extracellular compartments (i.e. an osmotic gradient), water will freely move from the compartment with fewer solutes to the compartment with more solutes until the osmolality (concentration of water in relation to solutes) is equal between compartments. This process is called *osmosis*, a term that specifically denotes the diffusion of water molecules (Figure 58.1).

Like water, urea passes across most mammalian membranes without energy and is therefore considered an *ineffective osmole*, except under rare conditions. Urea can act as an effective osmole when changes in urea concentration occur more rapidly than its equilibrium occurs across the cell membrane, such as urea extraction during dialysis or intravenous infusions of urea.

The osmolality in the plasma and interstitial (taken together, the "extracellular") compartments is equal because the solutes that contribute the most to osmolality (i.e. sodium and its accompanying anions, glucose, and urea) pass freely across the *endothelial* membrane, as does water. However, changes in plasma osmolality affect water movement between the intracellular and extracellular compartments since those solutes do not cross the *cell* membrane freely. Rapid increases in plasma (and thus extracellular compartment) osmolality cause water to move from the cells into the extracellular fluid space, dehydrating the cells. This is the principle by which hypertonic saline helps treat cerebral edema.

Isotonic solutions that contain added dextrose and parenteral nutrition are hyperosmolar compared with normal plasma. Their infusion in peripheral veins can create a local osmolar gradient causing water to move out of the endothelial cell and into the plasma, leading to endothelial cell dehydration and damage at the site of injection with localized pain and tissue swelling (phlebitis). In contrast, when large volumes of 5% dextrose in water are rapidly infused, the dextrose is quickly metabolized and the remaining hypotonic water will decrease plasma osmolality, resulting in water moving from the extracellular space into the cells, causing cell swelling.

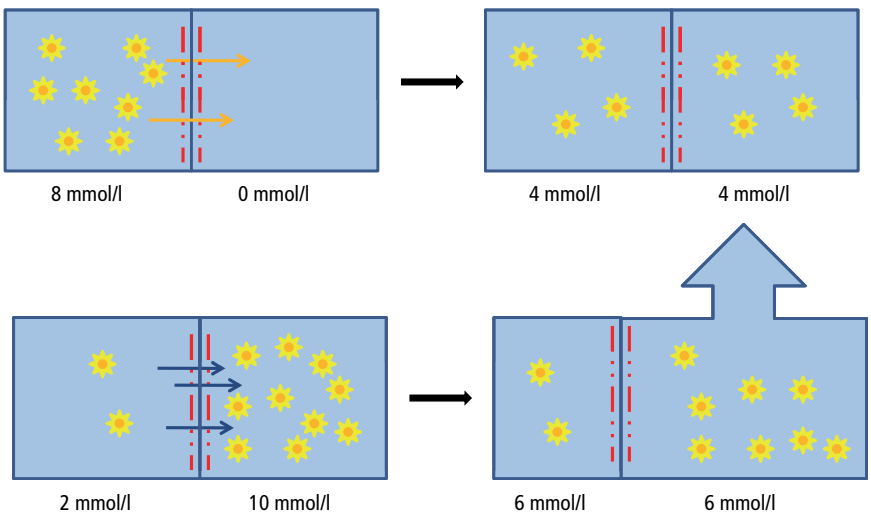


Figure 58.1 Osmotic forces across a semipermeable membrane. The top figure demonstrates that when more solutes (star-shaped dots) exist on one side of a semipermeable membrane (dashed lines) and the membrane is permeable to those solutes, solute particles will diffuse across the membrane until they are in equal concentration on either side of the membrane. Permeable solutes are thus considered ineffective osmoles because their presence on one side of the membrane (or the other) is ineffective at causing *water* to move across the membrane; the solutes move instead. The bottom figure demonstrates that when more solutes exist on one side of a semipermeable membrane and the membrane is **not** permeable to those solutes, water moves across the membrane until the solutes are in equal concentration; this movement is caused by *osmotic pressure*. Note that the water movement in the bottom figure has caused the right-hand compartment to expand in size, denoted by the large blue arrow in the lower right image. When the impermeable solutes that generate the osmotic pressure are colloidal particles (by convention in medicine, particles that are too large to pass freely from the intravascular into the interstitial space), then the pressure generated is called *colloid osmotic pressure*.

The brain is most susceptible to rapid changes in plasma osmolality (changes in excess of 30–35 mOsm/kg), and damage caused by rapid swelling or shrinking of neurons can result in altered mentation, seizures, and intracranial bleeding [4]. The brain is encased in the skull, such that any type of increase in brain volume from neuronal swelling will compress and damage functional neurons and brain vessels. However, when plasma osmolality increases slowly, brain cells accumulate organic osmolytes (e.g. amino acids, glutamine, inositol) that prevent the development of a transcellular osmotic gradient and maintain intracellular water content [5].

When a patient is hyperosmolar (e.g. a diabetic cat with hyperglycemia and ketosis), special attention should be given to the type and rate of fluids infused. It could be highly detrimental to infuse large volumes of hypotonic fluids (e.g. lactated Ringer's solution or "half-strength" hypotonic solutions) because a large osmotic gradient could result in a rapid shift of water into brain cells. To prevent rapid osmotic shifts during fluid administration, isotonic fluids with an osmolality closer to the patient (e.g. Normosol®-R, Abbott Laboratories, North Chicago, IL) are used, or fluids can be modified by mixing with concentrated NaCl solutions to make the infusion fluid osmolality similar to the patient's osmolality, so that they produce less of an osmotic gradient.

Physiology of Water Movement: Colloid Osmotic Pressure

The sum of all forces that affect water movement across the endothelium have traditionally been summarized in the Starling principle, which holds that the capillary endothelium is the working barrier between the plasma and the interstitium [6]. Across that barrier, the plasma COP is the greatest force that opposes hydrostatic pressure and prevents movement of water out of the vessel and into the interstitium [3, 7]. COP is the osmotic pressure generated by the large molecules (primarily proteins) that do not readily move across the capillary membrane, which creates an osmotic effect (Figure 58.2) As it pertains to proteins, synonyms of COP include *protein osmotic pressure* and *oncotic pressure*. Albumin is the most abundant

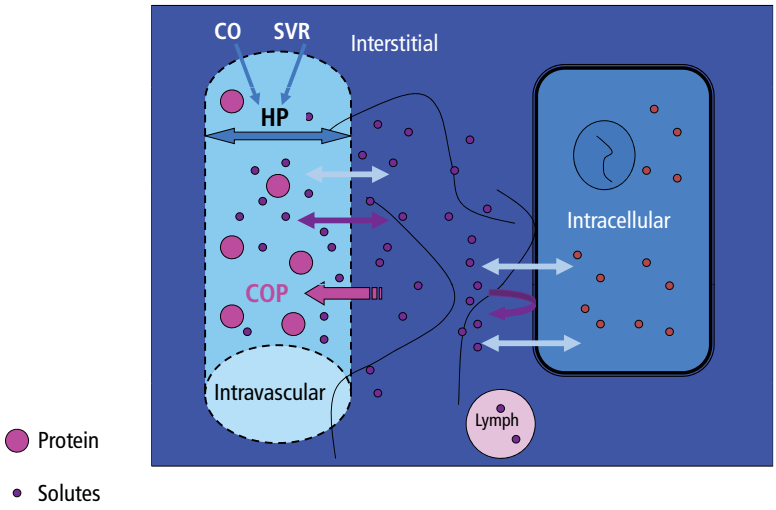


Figure 58.2 Relationship of forces governing water movement between the fluid compartments. Total body water is distributed between two major body compartments: the intracellular space and the extracellular space. The extracellular compartment is further divided into the interstitial space and intravascular space. The cell membrane is freely permeable to solute-free water (white arrows), which moves in and out of the cell as a result of osmotic gradients established between the intracellular and interstitial fluid compartments. The concentration gradient of solutes on either side of a membrane dictates how water will move by osmosis across the cell membrane. Changes in the intracellular solute concentration affect the osmolar gradient and depend on the various molecules' abilities to move through the cell membrane by active membrane pumps and restricted channels. The capillary membrane is permeable to water and small solutes, and is relatively impermeable to blood cells and large molecules such as proteins. The capillary endothelium is coated by a complex matrix called the glycocalyx, and the space between endothelial cell and glycocalyx is called the "subglyceal space." The most abundant small solutes in the extracellular fluid are sodium (and its major paired anions chloride and bicarbonate), glucose, and urea, which pass freely across the vascular membrane and thus at equilibrium are at equal concentrations in the vascular and interstitial spaces. Modified Starling's forces dictate fluid movement and retention in the intravascular space. The capillary hydrostatic pressure (HP) is produced by the forces generated by cardiac output (CO) and systemic vascular resistance (SVR). The intravascular colloid osmotic pressure (COP) opposes the HP, retaining fluid in the vessel. The endothelial surface layer creates a barrier that under normal conditions prevents the movement of large molecules, primarily proteins, out of the vessels. Under normal conditions, the concentration of proteins is greater in the blood vessel than in the subglyceal space, promoting water retention within the capillary. Lymphatic vessels (lymph) are within the interstitial matrix and carry excess fluid, proteins, and solutes from the interstitium back to the circulation.

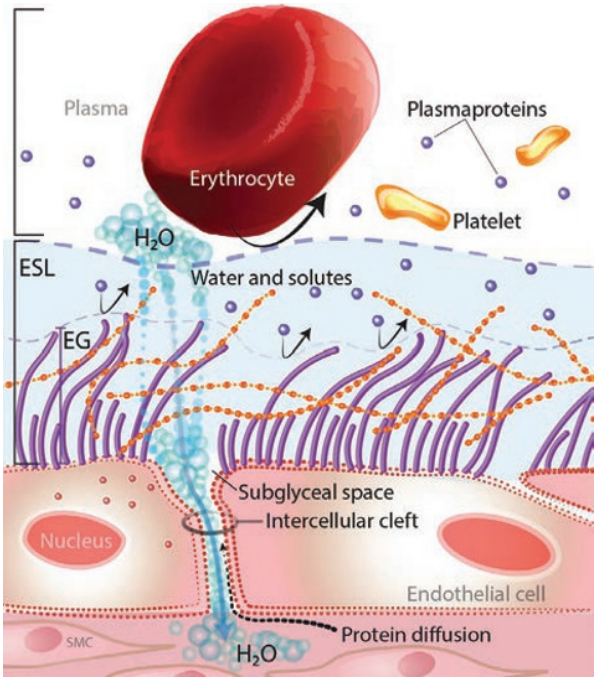


Figure 58.3 Functional compartments involved in transvascular fluid flux. Plasma proteins are largely reflected off the surface of the endothelial glycocalyx (EG), while water and small solutes pass according to the transendothelial hydrostatic pressure gradient. This leads to the generation of a space immediately underlying the endothelial glycocalyx (i.e. subglyceal space) that contains fluid of very low colloid osmotic pressure. Protein diffusion from the interstitium into the subglyceal space is mitigated by the filtrate convection in the opposite direction. ESL, endothelial surface layer; SMC, smooth muscle cell. *Source:* © Veterinary Emergency and Critical Care Society, 2020; reproduced with permission [8].

protein dissolved in the plasma, and at normal concentrations, it produces approximately 75% of the total intravascular COP. Other proteins such as fibrinogen and globulins contribute the remaining 25%. The albumin molecule expresses a negative charge that attracts positively charged sodium ions, increasing the water attraction of albumin. This additional water-holding effect is called the *Gibbs–Donnan effect* and increases the COP by approximately 20%.

Recently, the traditional Starling principle has been modified to take into consideration the osmotic asymmetry to the continuous endothelium (Figure 58.3). The new exchange pathway model (the modified Starling principle) recognizes the presence of the endothelial glycocalyx, a fur-like structure that is composed of a polysaccharide mix of proteoglycans, glycoproteins, and glycosaminoglycans that lies along the surface of and is in contact with the endothelium, forming an endothelial surface layer (ESL) [8]. The ESL functions as the actual oncotic barrier between the vessel lumen and the interstitium. A subglyceal space between the endothelial glycocalyx and the endothelial cells is completely separated from the plasma by the ESL. In health, this space remains protein-free, and the COP of the sub-endothelial glycocalyx space has replaced the interstitial COP as the oppositional force to the capillary hydrostatic pressure. Solute-free fluid and small solutes pass through inter-endothelial pathways of the ESL into the interstitium.

When there is inflammation or injury to the ESL and/or endothelium, protein molecules can pass from the intravascular compartment into the interstitial compartment causing a decrease in plasma COP and an increase in subglyceal and interstitial COP. When plasma COP is significantly reduced, intravascular water retention is reduced. This can result in hypovolemia, interstitial edema, and fluid losses into body cavities (e.g. pleural space, abdominal cavity, gastrointestinal tract; Figure 58.4). Hypovolemia reduces oxygen delivery to the tissues. Significant interstitial edema inhibits transport of metabolic substances to and from the cell. Significant leakage of fluid into the lung interstitium, pleural space, and abdominal cavity may negatively impact oxygenation, ventilation, and work of breathing.

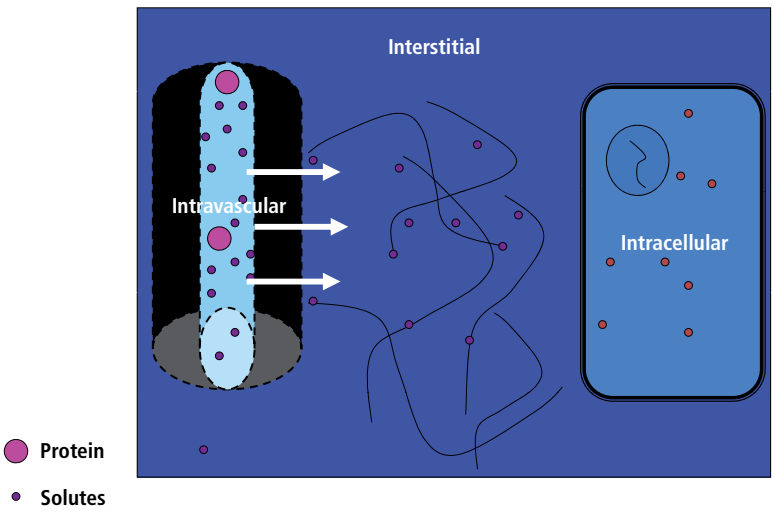


Figure 58.4 Decreased intravascular colloid osmotic pressure. Decreased intravascular colloid osmotic pressure results in movement of intravascular water into the interstitium due to consistent hydrostatic forces in that direction, which can lead to reduced blood flow through the capillary, interstitial edema, and decreased tissue oxygen delivery.

Synthetic colloid fluids (e.g. hydroxyethyl starches) and hemoglobin-based oxygen carriers contain large molecules that generate COP. When the molecules are larger than the inter-endothelial gaps, they can support intravascular COP during states of hypoproteinemia and increased capillary permeability. Synthetic colloid fluids are commonly used to resuscitate and maintain intravascular volume in critically ill patients with hypovolemic shock and with diseases causing a systemic inflammatory response (e.g. pancreatitis, severe gastroenteritis, pneumonia) associated with increased endothelial permeability. Albumin-containing transfusions and lyophilized albumin are also used to support intravascular COP and may play a role in maintaining the endothelial glycocalyx.

Frequent monitoring of both the patient as well as laboratory monitoring of osmolality and COP can provide the veterinary team with information on a patient's needs and response to specific therapy affecting fluid balance.

Calculating Plasma Osmolality

Osmolality can be *calculated* by knowing a patient's blood urea nitrogen (BUN), glucose, and sodium (Na^+) concentrations. Four equations are reported to be used in the calculation of osmolality [7]. The two that correlate most accurately to measured plasma osmolality are as follows: [7]

$$\text{Osmolality, mOsm/kg} = \left[2[\text{Na} + \text{K}], \text{mEq/l}\right] + \left[\text{glucose, mg/dl}/18\right] + \left[\text{BUN, mg/dl}/2.8\right]$$

$$\text{Osmolality}\left(\text{mOsm/kg}\right) = \left[1.86\left(\text{Na, mEq/l} + \text{K, mEq/l}\right) + \left(\text{glucose, mg/dl}/18\right) + \left(\text{BUN, mg/dl}/2.8\right)\right]/0.93$$

Both equations assume that 1 l = 1 kg.

The constants 18 and 2.8 convert the mg/dl of glucose and BUN, respectively, to mOsm/l [2]. In the second equation, the 1.86 accounts for the incomplete dissociation of the salts, and the 0.93 accounts for the body percentage of water when measuring whole blood [9, 10]. Because they are the only elements in these equations, an increase or decrease in *calculated* osmolality is always caused by an increase or decrease in sodium, potassium, glucose, or BUN. Other substances will increase osmolality, but the only way to detect them is by *measuring* osmolality using an osmometer. In contrast to *calculating* osmolality, an osmometer *measures* osmolality and thus not only accounts for the normal solutes (BUN, glucose, Na^+) but also for other small solutes, including toxins. There is normally a 10 mOsm/kg difference between calculated and measured osmolality (*osmolar gap*) due to normal solutes that are not included in the osmolality calculation (e.g. albumin, cholesterol) [7]. If the osmolar gap is significantly increased, toxin exposure should be considered, as well as certain conditions such as diabetic ketoacidosis (Box 58.1).

Box 58.1 Substances that Increase Osmolar Gap

- Acetone
- Ethylene glycol
- Ethanol
- Ether
- Glycerol
- Inositol
- Isopropyl alcohol
- Ketones
- Lactate
- Mannitol
- Methanol
- Paraldehyde
- Sorbitol

Measuring Plasma Osmolality

A variety of osmometers are available for clinical laboratory monitoring. Some measure osmolality using a freezing-point depression method (Figure 58.5), and others use room-temperature controls and vapor-point depression (Figure 58.6). The freezing-point depression determination of osmolality compares the freezing point of solute-free water and the freezing point of the sample. Water has a freezing point of 0°C, and a solution with saline concentration of 1 mOsm/kg has a freezing point of −1.858°C. Osmotically active substances decrease the freezing point, and the freezing-point depression difference is translated by a thermistor into measured mOsm/kg.

A vapor-point depression osmometer analyzes the vapor pressure of an osmotically active solution: the lower the vapor pressure, the higher the osmolality of a solution. A sample of solution is pipetted onto a small solute-free paper disk that is inserted into the sample chamber that contains a thermocouple hygrometer. The temperature of the sample and the temperature of the chamber equilibrate. An electrical current is passed through the thermocouple, cooling it to a temperature below dew point. Water condenses from air in the chamber to form microscopic droplets on the surface of the thermocouple. When the temperature of the thermocouple reaches the dew point, condensation ceases, causing the thermocouple temperature to stabilize.

In contrast to freezing-point depression osmometers, vapor-point depression osmometers are not affected by



Figure 58.5 Fiske 210 freezing-point depression osmometer (Cardinal Health). Osmotically active substances decrease the freezing point, and the freezing-point depression difference is translated by a thermistor into measured milliosmoles. This type of osmometer has been the most common one encountered in veterinary medicine because it can detect consumed alcohol-based toxins such as ethylene glycol. Single-sample freezing-point depression osmometers for clinical use have largely been discontinued; however, many are still available refurbished from medical suppliers or on resale websites.



Figure 58.6 VAPRO® 5520 vapor pressure osmometer (Wescor, Logan, UT). Sample volumes of 10 µl are typically used, but the osmometer allows for testing of sample volumes as small as 2 µl. Results are available in 20 seconds and expressed as mmol/kg. The osmometer stores up to 32 sample results and can be easily connected to a printer or computer to download data. The instrument needs to be calibrated and has control solutions for high, normal, and low osmolality.

artifacts caused by increased viscosity of a solution, suspended particles, or other conditions. However, because vapor-point depression osmometers require larger sample volumes and do not detect alcohols such as ethylene glycol, they are used less frequently in clinical medicine. Freezing-point depression osmometers can detect not only nonvolatile particles but also commonly encountered toxic volatile alcohols that can increase the osmolar gap. The Advanced Micro Osmometer Model 3300 (Advanced Instruments, Inc., Norwood, MA), a freezing-point depression osmometer, has been validated for measuring plasma and whole blood samples in normal dogs [7].

Osmometers can test a variety of bodily fluid and tissue samples. Plasma, serum, whole blood, and urine are the most commonly tested samples in veterinary medicine. Serum can be separated using a serum separator. Plasma and whole blood samples can be collected into lithium heparin tubes, while other anticoagulants could affect results. Technical support and equipment manuals should be consulted on sample handling in case there are nuances related to the specific osmometer being used. Serum osmolality can be compared with urine osmolality to evaluate for water imbalances. As the serum osmolality rises, the urine osmolality should also rise. The normal kidney will reabsorb water from the renal tubules in the hyperosmotic patient, which concentrates the urine. When there is an excess of water in the body, normal kidney function dilutes the urine, eliminating extra body water to return serum osmolality to normal. Normal serum osmolality ranges from 290 to 310 mOsm/kg in the dog and from 290 to 330 mOsm/kg in the cat [11]. Whole blood osmolality is a little higher than plasma osmolality [7]. Normal urine osmolality can range between 161 and 2830 mOsm/kg in dogs [12]. The urine osmolality will increase when water intake is withheld as long as kidney function is normal. Each individual laboratory should establish the normal reference interval in each species for its osmometer.

Measuring Colloid Osmotic Pressure

COP cannot be accurately predicted and must be directly measured using a colloid osmometer (Figures 58.7 and 58.8). The colloid osmometer uses a semipermeable membrane to simulate the role of the natural vascular membrane in the determination of COP responsible for water flow between interstitial fluid and blood. The colloid osmotic effects of both natural and synthetic colloid molecules are measured by colloid osmometers. The normal values reported for plasma COP are 16.7–28.9 mmHg in the healthy dog and 18.3–30.8 mmHg in the healthy cat [12–15]. Whole blood COP is reported to be 17.9–27.1 mmHg in the healthy dog and 18.9–30.4 mmHg in the healthy cat [16, 17] When whole blood is being measured, the sample should be collected with lyophilized heparin. Each individual laboratory will establish the normal reference interval in each species for its colloid osmometer.

Severe hemolysis (with release of hemoglobin into plasma), as well as severe hyperglobulinemia (caused by



Figure 58.7 Model 4420 Colloid osmometer (Wescor, Logan, UT). Heparinized whole blood, plasma, and serum can be analyzed. Sample volume requirements are normally 350 µl; however, special procedures allow for measurements of sample volume as low as 125 µl. The osmometer requires frequent calibration with high (25 mmHg), low (15 mmHg), and normal (20 mmHg) reference solutions. The membrane requires periodic changing, and saline solution is regularly infused to prevent the membrane from drying out.

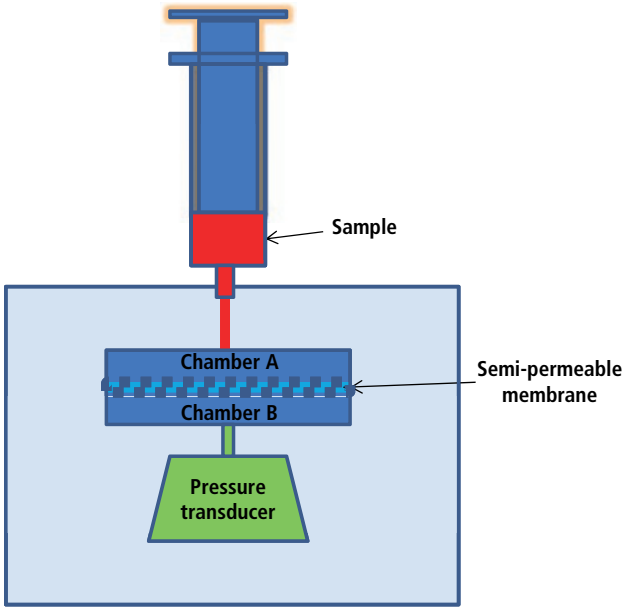


Figure 58.8 Principles of the colloid osmometer. The sample is injected into chamber A and allowed to equilibrate with the reference chamber B, which contains 0.9% NaCl. The artificial membrane does not allow molecules greater than 30 000 Da in size to pass. The colloid osmotic pressure (COP) of the sample causes water and small solutes to move from chamber B to chamber A, which causes a reduction in pressure in chamber B. The negative pressure produced is measured by the pressure transducer and equals the COP of the sample in chamber A. The results are displayed in mm Hg, cm H_2O, or kilopascals (kPa).

Box 58.2 Causes of Hypoalbuminemia

Decreased production (liver failure):

- Portosystemic shunt
- Chronic active hepatitis
- Acute hepatotoxicity

Increased loss:

- Protein-losing glomerulonephropathy
- Protein-losing enteritis
- Systemic inflammatory response syndrome
- Acute allergic reaction

multiple myeloma or feline infectious peritonitis) and hyperalbuminemia can increase COP. A decrease in COP can indicate dilution of the blood or a deficiency in intravascular albumin molecules (Box 58.2).

In summary, osmolality and COP play an important role in water homeostasis of our patients. Veterinary technicians who understand the physiology and monitoring of osmolality and COP, and how it relates to abnormal water balance, will have a greater ability to anticipate and prevent morbidity in their patients.

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Body Fluid Collection and Handling

Adesola Odunayo and Eric Hilton

Laboratory tests are used in diagnosis and monitoring of responses to treatment in healthy and sick veterinary patients. It is estimated that about two thirds of important clinical decisions about patients are based on laboratory test results [1]. Many of these tests usually require acquisition and analysis of bodily fluid samples (i.e. blood, urine, effusion samples).

Errors in laboratory tests are common. These errors may occur prior to the testing (preanalytical), while the test is being run (analytical) or after the test is completed (postanalytical) [1]. Body fluid collection method, method of storage, length of storage, and sample processing techniques (all sources of preanalytical error) can affect the results and interpretation of specific laboratory tests. Thus, it is important that veterinary professionals follow standardized recommendations to minimize preanalytic errors.

This chapter focuses on handling techniques for body fluid sample to maximize the accuracy of the diagnostic test and provides an overview of sample collection methods (see Chapter 53 for blood sample collection and handling).

Body fluid samples should be collected in a clean and sterile way (if appropriate) with minimal stress on the patient. The samples should be transferred to the appropriate storage tubes immediately. Storage tubes should be clean, sterile (if appropriate) and should not be expired. Table 59.1 provides an outline of the commonly available storage tubes used for storage and transport of body fluid samples to a reference laboratory.

To ensure positive identification and optimal patient outcome, all patient samples should be labeled as soon as they are collected. While specific details should align with the preferences of the reference laboratory performing the test, the patient name, client's last name, patient's unique identifying number, and type of specimen should always be included on the label. Multiple samples from the same patient on the same day should be labeled with the time of collection, as well as the site of collection, if appropriate. All samples designated to be transported to an external laboratory should be packaged appropriately to prevent spillage or breakage. Cool packs should be used for temperature-sensitive specimens and the sample should be transported as quickly as possible to minimize transport artifacts.

Urine

Urine is a major diagnostic specimen in veterinary patients. Common tests performed on urine samples include urinalysis, urine culture and sensitivity, urine protein/creatinine ratio, urine electrolytes, fungal antigen tests, and urine cortisol concentrations [2].

Collection

There are multiple ways of obtaining urine samples in veterinary patients. Collection technique may depend on operator preference, patient compliance, and the diagnostic rationale obtaining the urine sample; for example, a cystocentesis may be preferred for urine sample collected for urine culture and sensitivity, while a free-catch collection is perfectly acceptable to evaluate urine protein : creatinine ratios [2, 3].

Asepsis should be strictly maintained during urine collection. Urine samples should be collected into a clean container with minimal contact of the urine sample with the animal's body [4]. Ideally, new (plastic or glass) clean containers with tight-fitting lids should be used [4].

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Table 59.1 Storage tubes commonly used for transport of body fluid samples.

Tube type	Tube contents/preservative	Common indications
EDTA (lavender/ purple top)	Dipotassium EDTA (ethylenediaminetetraacetic acid)	Tube of choice for cytology samples. Prevents clotting. Allows preservation of white and red blood cells, platelets, and infectious agents. Used to determine the total nucleated cell count, white blood cell count and total protein. (Note that underfilling an EDTA tube can result in falsely increased refractometric total protein). The EDTA tube can also be used for tests such as PCR and virus isolation. EDTA is bacteriostatic and is not appropriate for culture.
Heparinized (green top)	Sodium heparin	Rarely used for body fluid analysis. May occasionally be useful for fluid samples that require biochemical analysis of their contents (sodium, potassium, glucose, creatinine). Note that potassium cannot be measured from an EDTA tube sample because the EDTA additive is potassium EDTA.
Slides/freshly made smears	None	Direct or concentrated/centrifuged smears should be provided with EDTA samples for cytology.
Glass non-anticoagulant (red top)	None	Good for urinalysis, as well as for samples that require culture and sensitivity. Can also be used for CSF, PCR, virus isolation, or biochemical assay evaluation.
Plastic non-anticoagulant (red top)	Contains a clot activator but no anticoagulants	Best used for PCR, virus isolation tests, biochemical assay evaluation. Crystalline clot activator in the plastic red-top tube may inhibit bacterial growth or introduce artifact in urinalysis or cytology.
Plastic containers	None	Some plastic containers may be sterile and can be used for cultures and sensitivities. May also be used for urinalysis, CSF samples, biochemical assays, PCR testing, or virus isolation.
Anaerobic transport medium	Soft agar and reducing agents	Designed to maintain viability of anaerobic organisms. Ensure that the cap of the tube is replaced and tightened before transport.
Blood culture medium	Growth medium to enhance the culture of bacteria	Helps in isolation of bacteria from synovial fluid and blood.

CSF, cerebrospinal fluid; PCR, polymerase chain reaction.

Cystocentesis

Cystocentesis involves the introduction of a sterile needle directly into the urinary bladder to obtain a sterile urine sample. It is the preferred method of obtaining urine for a culture and sensitivity, although the urine collected this way can be used for any test requiring a urine sample. In brief, the patient is placed on its back (dorsal recumbency) with an assistant helping to restrain it (cystocentesis may also be performed standing or in lateral recumbency). The caudoventral abdomen is shaved and aseptically prepared. A sterile needle is introduced through the skin and directly into the urinary bladder and urine is collected into a syringe. This method is best performed with ultrasound guidance. Complications associated with cystocentesis are few but may include hemorrhage, vasovagal collapse, septic peritonitis, and seeding of urinary tract neoplasia [5, 6].

Catheterization

Urinary catheterization (see Chapter 35) is the most time consuming and labor/resource intensive method of urine collection. While sedation is generally not necessary for male dogs, it is usually required for cats and female dogs. The sample collected may be contaminated with cells and bacteria from the lower urinary tract so culture and sensitivity results should be interpreted cautiously.

Free-Catch Collection

Free-catch is a non-invasive urine collection method and the least stressful for the patient. The urine sample is collected while the animal is voiding. A midstream sample is recommended to minimize contamination with cells and flora of the lower urinary tract. This can be challenging in a cat and is sometimes challenging in female dogs. Employing a long-handled ladle or shallow tin may maximize success in dogs. Voided samples may be considered for urine culture and sensitivity following previously defined bacterial cut-off values of 100,000 colony-forming units (CFU)/ml or greater, as long as the specimens are refrigerated and plated on the day of collection [7].

Surface Collection

Surface collection is the least invasive way of collecting urine. The urine sample is retrieved from a surface on which the animal has voided, including but not limited to the cage, litter box, hospital floor, or outdoor surface. This sample is usually contaminated with debris and bacteria from the surface where it was collected. This sample is usually unsuitable for determination of bactiuria or bacterial culture. It may be used for other tests, such as fungal antigen testing, urine cortisol concentration, or urine protein : creatinine ratio.

Manual Expression

Manual expression of the urinary bladder is usually not recommended because of the risk of urinary tract rupture and the vesicoureteral reflex.

Sample Handling and Preservation

Once the urine sample is collected, it should be grossly evaluated and the urine color, as well as the transparency, should be noted [4]. Urine samples should be analyzed as quickly as possible after collection, ideally within 30 minutes, as there is no substitute for a fresh urine sample when urine is collected for urinalysis and urine culture and sensitivity [4]. If this is not possible, the sample should be refrigerated immediately and stored for no longer than 6–12 hours after collection, although urine may be stable for slightly longer or shorter periods [4, 8, 9]. The refrigerated sample should be brought to room temperature before analysis.

It is important to note that when a urine sample is obtained for culture and sensitivity, it should ideally be plated immediately. Storage of urine samples at room temperature for extended periods (longer than two hours) leads to false positive and false negative culture results [8, 10]. In one study, while CFU count was not affected, refrigeration appeared to affect sensitivity results, which may skew patient treatment, although a recent study concluded that there is minimal clinical impact if refrigerated urine samples are plated within 24 hours [11, 12]. Prolonged refrigeration ($\geq$ 24 hours) may lead to false negative culture results. Refrigeration of urine samples may also cause changes in urine pH (falsely elevated), glucose (falsely low) in the presence of bacteria [9]. Crystals may form in samples kept at room temperature or in the fridge. If there is clinical concern about crystalluria, the urine sample should be evaluated immediately [9]. Presence of crystals observed in stored samples should be validated by reevaluation of a fresh urine sample [13]. Freezing of urine samples is discouraged as this leads to disintegration of cells and casts, as well as inducing changes in the urine pH [4].

While not commonly recommended, urine sample integrity can be maintained by chemical preservatives like formalin or sodium fluoride (Table 59.2). Chemical preservatives are usually used when refrigeration is not an option. The appropriate preservative should not alter urine constituents or interfere with analytical tests [14].

Refrigerated urine samples are generally adequate for non-urinalysis or urine culture tests (urine cortisol, urine protein : creatinine ratio, or urine fungal antigen tests).

Urine samples can be stored and submitted in the syringe used for urine collection, additive-free red-top tubes (tubes with additives may inhibit bacterial growth), glass tubes, or clean additive-free plastic containers. Urine samples submitted to an external laboratory for culture and sensitivity can be submitted in an additive-free plastic or glass tube, on swabs, or in boric acid tubes (Figure 59.1) [15]. It is important to fill boric acid tubes to the line indicated

Table 59.2 Chemical preservatives for urine samples.

Chemical preservative	Advantage	Disadvantage
Sodium fluoride	Inhibits glycolysis	Interferes with dipstick test: glucose, blood, leukocyte esterase
Formalin	Preserves sediment	Interferes with dipstick tests: glucose, blood, leukocyte esterase

Source: Modified from Sink and Weinstein (2012) [14].



Figure 59.1 Storage containers that can be used for culture and sensitivity of body fluid samples; (l to r): micro culture swab, culture swab, sterile plastic container, red-top tube, red-top tube, sterile syringe, petri dish.

because high concentrations of boric acid may – kill bacteria [15]. Urine should not be stored in any tubes with additives (e.g. heparinized or EDTA tubes).

Transtracheal and Endotracheal Tube Washes and Bronchoalveolar Lavage

Transtracheal and endotracheal tube washes and bronchoalveolar lavage (BAL) are used to obtain lower airway samples. The airway sample can be employed to make a diagnosis of bacterial or fungal pneumonia, neoplasia, or inflammatory airway disease, using cytological evaluation. Culture and sensitivity can also be performed on the airway wash sample to identify predominant microorganisms and to determine the best therapeutic agent. In addition, special diagnostics, such as polymerase chain reaction (PCR), virus isolation, or specific antigen assays can be performed on the sample obtained [16].

A transtracheal wash is usually done with the patient awake or lightly sedated. This technique is easier in medium to large-sized dogs. Briefly, the area of the trachea is surgically clipped and prepped and about 5–20 ml of sterile saline in a syringe is injected into the lower airway using an over-the-needle catheter or polypropylene catheter fed through a needle [16, 17]. The patient is then vigorously coupaged to obtain about 10% of the infused volume of saline back into the syringe [17]. This technique has the benefit of being performed awake but has been shown to be inferior compared with samples obtained by bronchoalveolar lavage [17, 18].

Endotracheal tube wash is performed in cats and small dogs under general anesthesia. Briefly, the patient is induced and a sterile endotracheal tube is placed in the trachea, with care taken to avoid contaminating the tube with oropharyngeal secretions. A red rubber (or similar) catheter is passed through the endotracheal tube and saline is infused through the red rubber catheter into the lower airway and then aspirated back. Bronchoalveolar lavage is performed under general anesthesia using specialized equipment (bronchoscope). Veterinarians with advanced training most commonly perform BAL and the technique is not discussed in this chapter.

Sample Handling and Preservation

Samples should be collected using sterile techniques and immediately labeled with the patient information. The sample should be grossly evaluated and the fluid color and consistency should be noted. Cytological samples may be submitted as fluid or prepared slides, depending on the reference laboratory and the nature of material obtained [16]. In general, it is advisable to submit both. Fluid submission confers the advantage of enabling the laboratory to use cytocentrifugation technique to concentrate samples with a low cell count; however, submitting a freshly prepared smear with the fluid sample allows for interpretation of changes that occur secondary to storage [16, 19]. Slides should not be refrigerated but should be stored and transported at room temperature.

For best results, processing of transtracheal and endotracheal tube washes and BAL samples should be performed within one hour of collection as cell quality changes may happen quickly. Samples should be transported on ice within 60 minutes to maximize cell preservation and minimize bacterial growth [17, 19, 20]. A delay of about 48 hours is acceptable if the sample is refrigerated and the fluid sample sent on ice, if shipped to a reference laboratory overnight [19].

EDTA tubes are recommended for samples submitted for cytology to preserve cellular morphology (Figure 59.2) [16]. EDTA tubes can also be used for PCR, virus isolation, or specific antigen identification, depending on the laboratory and the specific diagnostic test run. Samples for bacterial cultures can be submitted in a sterile tube free from additives (red-top tubes, glass tubes) although samples may also be submitted in tightly sealed syringes or plastic tubes. Samples for bacterial cultures should ideally be plated within one hour of collection but refrigerated samples are also acceptable.



Figure 59.2 EDTA tube used for storage of body fluids that require cytological evaluation.

Pericardial Effusion

Pericardial effusion can present as a life-threating emergency in veterinary patients. Pericardiocentesis, the act of removing fluid from the pericardial sac, acts as a therapeutic and diagnostic intervention in those patients. The fluid removed may help to identify the cause of the effusion (lymphoma, bacterial, or fungal pericarditis), although in most cases, cytological evaluation of pericardial fluid is often nondiagnostic. In brief, pericardiocentesis (see Chapter 18) is performed awake in most patients, although light sedation may be required. After aseptic preparation, a needle is inserted between the third and sixth rib spaces on the right side, while the patient is in lateral or sternal recumbency. The patient should be closely monitored during the fluid collection for cardiac arrhythmias. In most cases, the pericardial fluid is hemorrhagic, although it may be serosanguinous in cats with heart failure.

Sample Handling and Preservation

Some of the pericardial effusion should be stored in an EDTA tube to obtain a packed cell volume (PCV) and total protein, as well as for fluid cytology, which may be helpful to identify lymphoma [21] or infectious pericarditis. Another sample should be stored in a red-top tube to help differentiate hemorrhagic pericardial effusion (unlikely to clot) from iatrogenic hemorrhage from cardiac puncture (more likely to clot) [22]. The red-top tube sample can also be used for culture and sensitivity of the pericardial effusion, if indicated. Culture and sensitivity is rarely performed on pericardial effusion but any sterile additive-free tube can be used for that purpose.

Abdominal Effusion

Abdominocentesis, the process of removing free intraabdominal fluid, is a common diagnostic and therapeutic skill. There are various methods to remove fluid from the abdominal cavity, including, blind abdominocentesis, ultrasound guided abdominocentesis, four-quadrant centesis, as well as a diagnostic peritoneal lavage (see Chapter 38).

Sample Handling and Preservation

The abdominal fluid sample should be grossly evaluated and the color noted. If the fluid is clear initially, then turns red, iatrogenic hemorrhage is likely [23].

Abdominocentesis fluid samples should be cytologically evaluated as soon as possible. A sample should be saved in an EDTA tube and some smears (ideally direct and centrifuged sample with slides labeled appropriately) should be made concurrently. The EDTA tube should be refrigerated if immediate evaluation is not possible but should be evaluated within 24 hours of collection. A PCV/total protein should be obtained from the EDTA sample to determine whether it is a hemorrhagic or serosanguinous effusion. With hemorrhagic samples, the PCV of the fluid should be at least 10–25% of the peripheral blood [22].

In patients where there is concern for peritonitis, an abdominal fluid glucose and lactate is compared with the peripheral glucose and lactate as a diagnostic test. The abdominal fluid glucose and lactate can be analyzed as soon as the fluid is removed from the abdominal cavity without transferring the sample to a storage tube. However, a heparinized tube can be used to store the sample for delayed analysis. It is important to remember that prolonged storage will lead to lower glucose and higher lactate readings.

A red-top tube (additive free), sterile plastic tube or culture swab should be used for bacterial culture and sensitivity (Figure 59.1). A red-top tube can also be used for biochemical analysis (creatinine, potassium), as indicated, such as when one is ruling out a uroabdomen.

Samples should be prioritized based on the volume of fluid available for analysis and the suspected underlying disease [24].

Pleural Effusion

Pleural effusion is removed via thoracocentesis. Many animals present with evidence of respiratory distress and a thoracocentesis provides almost immediate relief if the pleural effusion is the main cause of their respiratory difficulty. Common causes of pleural effusion includes heart failure, pyothorax, chylothorax, and hemothroax. Like the abdominocentesis procedure, thoracocentesis can be used both as a diagnostic and therapeutic tool.

Briefly, when possible, an intravenous catheter should be placed before thoracocentesis. Sedation may be required in agitated or anxious patients. After aseptic preparation of the thoracic body wall, a small-gauge needle or catheter (18–22 gauge) is introduced into the pleural space between the sixth and ninth intercostal space. The needle should be placed along the cranial curvature of the rib or in between ribs, with care taken to avoid the caudal margin of the ribs, where the nerves and blood vessels run. More specific details about thoracocentesis are outlined in Chapter 34.

Sample Handling and Preservation

The thoracic fluid sample should be grossly evaluated and the color noted. If the fluid is clear initially, then turns red, iatrogenic hemorrhage is likely [23]. A PCV/total protein should be performed to identify true hemorrhage or

serosanguinous effusion. With hemorrhagic samples, the PCV of the fluid should be at least 10–25% of the peripheral blood [22]. Pyothorax effusions may have a marked unpleasant odor, increasing the index of suspicion for an infectious etiology. Chylothorax effusions are white or milky in color and the color does not clear after centrifugation [25].

As with abdominal effusions, pleural fluid should be saved in EDTA tubes for cytological evaluation, as well as to obtain PCV/total solids and total nucleated cell count. Smears should also be made at the same time and should ideally be transported to the reference laboratory with the EDTA samples. Cytology samples should be evaluated immediately but refrigerated samples are acceptable when immediate evaluation is not feasible. An additive free red-top tube or sterile plastic or glass container can be used for culture and sensitivity samples, as well as biochemical evaluation of the fluid if needed (e.g. cholesterol and triglyceride concentrations to rule out a chylothorax) (Figure 59.1). Culture swabs can also be used for culture (aerobic, anaerobic, and *Mycoplasma* cultures). Samples for culture and sensitivity should be plated immediately but refrigerated samples are acceptable when immediate culture is not feasible.

Cerebrospinal Fluid

A cerebrospinal fluid (CSF) evaluation is indicated in patients with neurologic disease affecting the central nervous system. A CSF evaluation is useful in diagnosing infectious and inflammatory diseases in dogs and cats and may also be useful to diagnose neoplastic diseases (such as lymphoma). CSF collection may carry significant risks (hemorrhage, brain herniation, infection) and is usually performed by clinicians with specialized skills [26].

The two most common sites for CSF collection are the cisterna magna (atlantooccipital) and the caudal lumbar subarachnoid space. If focal central nervous system disease is suspected, the site of collection should be caudal to the suspected lesion [27]. If diffuse disease is suspected, CSF should be obtained from both sites if possible. A spinal needle is inserted in to the subarachnoid space while the patient is under general anesthesia. Once CSF is obtained, the stylet is removed from the spinal needle, and the CSF is allowed to flow freely in to a red-top tube. Aspiration of the fluid with a syringe is not recommended [26]. Approximately 1 ml of CSF can be safely removed per 5 kg of body weight, however, most routine sampling only requires a total volume of 1 ml for any patient [26].

Sample Handling and Preservation

The cells in CSF begin to degenerate within 30 minutes of collection, so it is important to have a plan for sample analysis before collection. This includes a plan for other diagnostic tests indicated for that patient, which will dictate what type of sample collection tube is to be used [26].

The sample should be inspected grossly once collected. Normal CSF should be colorless and clear [28]. Iatrogenic blood contamination from sampling can be determined by centrifuging the sample. A red cellular pellet after centrifugation (and the supernatant is clear) indicates peripheral blood contamination or recent hemorrhage [28]. If the supernatant is xanthochromic, it may indicate previous hemorrhage [28]. CSF samples should be collected into sterile plain tubes. EDTA tubes are not recommended for routine sampling as the additive can falsely elevate total protein concentration [27, 29].

Cytological analysis of CSF should be performed within 30–60 minutes of collection due to distortion of cell structures and reduction of total nucleated cell count from cell lysis [30]. However, some studies have suggested that the addition of fetal calf serum, hetastarch, 10% formalin, and/or fresh frozen autologous clear plasma or serum may stabilize CSF samples for 4–48 hours, depending on the medium used [30–32]. However, in a 2020 study, vetstarch did not reduce the time-dependent cellular degeneration compared with serum or saline [33].

If cytological analysis requires a delay, two aliquots of CSF can be collected and placed in sterile plain tubes. A preservative should be used in one of the tubes for cytology and cell count. The other tube can be used for protein quantification and antibody titer analysis [27]. The analysis of protein and other analytes in CSF have no time-dependent requirements and can be handled just like any other fluid [28]. Sterile additive-free tubes can be used for CSF culture.

Synovial Fluid

Arthrocentesis is the aspiration of synovial fluid from a joint space for diagnostic evaluation [34]. Synovial fluid samples are used in the diagnosis of joint disease (immune mediated, neoplastic, or infectious) but is also used to evaluate the response of such diseases to treatment [34]. Arthrocentesis is performed under heavy sedation in most patients, although general anesthesia may be required in some cases [34]. In general, joints that contain excessive fluid should be sampled; however, if none exists, at least two to three joints should be sampled [35]. If possible, the carpal and tarsal joints should be included in the joints sampled [35].

Sample Handling and Preservation

Arthrocentesis of normal joint yields only a small amount of synovial fluid (less than 0.5 ml) [34].

A sample of more than 1 ml from any joint is abnormal [34]. The sample should be visually inspected once obtained. Normal synovial fluid sample is colorless or light yellow and clear in appearance [34]. The viscosity can be assessed by placing a drop of the sample between two fingers and slowly pulling them apart. Normal synovial fluid should form a strand, which usually extends to about 2–5 cm before it breaks. Reduced viscosity is indicative of joint disease [34].

The top test priority is the microscopic examination of the fluid. A smear of the sample should be made for cytology and the rest of the fluid (if applicable) should be divided between a pediatric EDTA tube (owing to the small volume collected) and blood culture media tubes or bottles [35]. The EDTA tube is used for cytological evaluation and cell count. Aerobic bacteria is cultured most reliably from the joint by using blood culture medium, since synovial fluid culture generally has a low yield (up to 50–70% false negative results) [36, 37]. The liquid blood culture medium prevents coagulation of the fluid, dilutes inhibitors, inactivates aminoglycoside antibiotics, and limits in vitro phagocytosis of bacteria [35]. Thus, blood culture medium significantly enhances the recovery of organisms from septic joints [35]. However, aerobic culture transport media or plain sterile tubes can also be used if blood culture medium is not available (Figure 59.3) [35, 36]. Isolation of anaerobic bacteria can be accomplished using an anaerobic transport media. The reference laboratory should be alerted if *Mycoplasma* infection is suspected, as these organisms grow better on a specialized medium [35].



Figure 59.3 Storage containers that may be used for arthrocentesis samples; (l to r): pediatric blood culture tube, blood culture tube, micro culture swab, culture swab, red-top tube, and red-top tube.

Fluid samples for cell counts and cytological assessment can be stored in the refrigerator for 24 hours but it is best to make smears immediately the sample is collected [35]. If only a few drops of fluid are obtained, preparation of stained smears for cytology will provide the most diagnostic information for that patient [35].

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60

Urinalysis in Acutely and Critically Ill Dogs and Cats

Lucy Kopecny and Sean Naylor

Urinalysis is a key component of the evaluation of acutely and critically ill dogs and cats. It provides information on not just the urinary system but also systemic diseases such as diabetes mellitus and liver disease. Urinalysis is relatively simple and inexpensive and can be combined with more advanced techniques.

What Is Urinalysis?

Urinalysis includes evaluation of the physical and chemical properties of urine, urine specific gravity (USG) measurement and urine sediment evaluation. In-house testing is preferred due to faster turnaround times and improved accuracy of results, as delays in analysis can alter some urine characteristics. Urinalysis requires only basic laboratory supplies and is readily performed by trained staff.

A summary of recommended equipment and disposable supplies for urinalysis is provided in Box 60.1. When performing urinalysis, a standardized urinalysis report should be completed. An example of a urinalysis report form is provided in Table 60.1.

Urine Sample Collection and Handling

Urine collection techniques are reviewed in Chapter 59. Cystocentesis is preferred for urine collection, although these samples can have blood contamination if collection is traumatic. Catheterized (see Chapter 35) samples or samples collected by midstream free catch can have contamination from both the lower urinary and genital tracts; this can influence urine sediment examination results. It is therefore important that urine collection method is reported (Table 60.1).

Selection of urine collection technique is determined by the type of urine testing planned and the presence of certain systemic disorders and drug administration. For example, samples for measurement of the urine protein to creatinine ratio can be collected by free catch [1]. Cystocentesis should be avoided in dogs and cats with coagulation disorders or that are receiving drugs that affect coagulation, owing to the risk of hemorrhage. Common drugs that affect coagulation are those that inhibit platelet function (e.g. clopidogrel or aspirin) and drugs that affect clotting factors (e.g. unfractionated heparin, low molecular weight heparin, or rivaroxaban).

If a dog or cat is sufficiently stable, urine should be collected before any therapeutic interventions such as administration of fluid therapy or antimicrobials as these can alter some urine testing results.

Urine samples should be stored in a sterile, airtight container and analyzed within 30–60 minutes. If testing is delayed, refrigeration of the urine sample is recommended [2, 3]. Both refrigeration and increased storage time can result in crystal formation and bacterial overgrowth. Storage time and temperature do not significantly affect urine pH or USG measurement [3]. It is recommended that urine be stored for a maximum of 24 hours and that refrigerated samples be returned to room temperature before analysis [2].

For samples collected for urine culture, if immediate processing is not possible, urine should be placed in a sterile container and refrigerated during storage and shipping to the laboratory. However, if samples will be exposed to room temperature during storage and shipping, urine transport tubes are preferred [4]. Urine transport tubes contain a preservative.

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Box 60.1 Summary of Recommended Equipment and Disposable Supplies for Urinalysis

Equipment

- Centrifuge – low rotation/minute capability
- Refractometer (feline and canine specific scales preferable)
- Microscope
- Timer
- Automated dipstick reader (optional)
- Pipettor – fixed or variable volume (optional)
- Refrigerator – if unable to analyze sample in appropriate timeframe
- Automated urine electrolyte analyzer (optional)

Disposable supplies

- Specimen container – sterile, clear and tight lid
- Multiple reagent dip sticks
- Conical centrifuge tubes
- Disposable pipettes
- Disposable pipette tips (optional)
- Glass slides and coverslips
- Sediment stain (optional)
- Urinalysis report form

Urinalysis

Physical Properties

Physical properties of urine include its color and clarity. Normal urine is yellow or amber in color and clear. Changes to urine color can be caused by several endogenous and exogenous pigments. The most common color changes are red, black, or brown caused by hematuria, hemoglobinuria, myoglobinuria, and bilirubinuria. Where urine is red, hematuria can be differentiated from hemoglobinuria and myoglobinuria by centrifugation of urine. If hematuria is present, with centrifugation, red blood cells should settle at the bottom of the sample, leaving the remainder of the sample clear. In contrast, urine color change will persist if it is caused by hemoglobinuria or myoglobinuria. Plasma color can aid in differentiation between hemoglobinuria and myoglobinuria: dogs and cats with hemoglobinuria have pink- to red-tinged plasma whereas plasma is typically clear when myoglobinuria is present. Urine color cannot be accurately used to assess urine concentration.

Urine clarity is affected by urine concentration as well as the presence of red blood cells, white blood cells, epithelial cells, bacteria, crystals, and mucus in the urine. Aggregates of white blood cells, epithelial cells, and crystals can cause urine to appear flocculent [5].

Urine Specific Gravity

USG is defined as the ratio of the weight of urine to the weight of an equal volume of distilled water and depends on the number of solutes and their weight. It is a measure of urine concentration. USG varies in healthy animals, although dogs and cats with normal renal function are considered to have a USG greater than1.030 and greater than 1.035, respectively.

Collection of first morning urine samples is recommended for determination of urinary concentrating ability as this is thought to be the most concentrated urine sample of the day [6]. However, the USG of first morning urine samples can still be variable enough to alter clinical decision making between days in dogs [7].

Glucosuria causes minimal changes to USG in dogs and cats [8].

Methods

USG is measured by refractometers. Temperature-compensated refractometers are preferred as they provide accurate readings between 60 and 100 degrees F. Refractometers with scales calibrated for human urine result in falsely increased USG measurements in cats, especially in concentrated samples. Refractometers with separate calibration scales for feline urine samples should therefore be used. Quality control can be performed using distilled water, which has a specific gravity of 1.000 [9].

When USG exceeds the scale on the refractometer, the urine should be mixed with an equal volume of distilled water and USG remeasured. The numbers to the right of the decimal point are multiplied by 2 to calculate the USG [5].

Dipstick methods for measurement of USG are unreliable in dogs and cats.

Chemical Properties

Evaluation of the chemical properties of urine is most commonly performed semiquantitatively with reagent strips (dip sticks). These have color-coded pads with specific reagents. Tests include urine pH, glucose, ketones, bilirubin, occult blood, protein, urobilinogen, nitrites, and leukocyte esterase. Both nitrite and leukocyte esterase pads are unreliable in dogs and cats.

Methods

Chemical analysis of urine using reagent strips is performed using well-mixed, uncentrifuged urine. Supernatant from a centrifuged urine sample can be used if the urine is discolored due to blood. Urine chemistry is performed by

Table 60.1 Example of a urinalysis report form.

Collection Date: Time:	**Analysis Date: Time:**		**Patient name: Client name:**	
Urine collection type	**Cysto**	**U-Cath**	**Free catch: Early mid late surface**	**Comment:**
Report	**Result**	**Reference interval**	**Units**	**Comments**
Color			*Visual*	
Clarity			*Visual*	
Specific gravity			*Refractometer*	
pH, urine			*pH U*	
Protein, urine		*Neg*	*mg/dl*	
Glucose urine		*Neg*	*mg/dl*	
Ketones, urine		*Neg*	*mg/dl*	
Bilirubin, urine		*Neg*	*mg/dl*	
Hemoprotein, urine		*Neg*	*Ery/ul*	
Sediment volume		*<0.1*	*ml*	
Volume, urine		*4.5–5.0*	*ml*	
Lipid layer			*Visual*	
WBC, urine		*0–3*	*/HPF*	
RBC, urine		*0–2*	*/HPF*	
Crystals, urine		*None*	*/HPF*	
Epithelial cells, transitional		*None*	*/HPF*	
Epithelial cells, squamous		*None*	*/HPF*	
Epithelial cells, renal		*None*	*/HPF*	
Epithelial cells, caudate		*None*	*/HPF*	
Casts, hyaline		*None*	*/LPF*	
Cast, granular		*None*	*/LPF*	
Cast, waxy		*None*	*/LPF*	
Cast, cellular		*None*	*/LPF*	
Bacteria		*None*	*/HPF*	
Lipid droplets			*/HPF*	
Sperm		*None*	*/HPF*	
Other				
Other comment:				

dipping the reagent strip in urine, then removing excess urine by tapping the strip against the side of the container. The reagent strip should be placed on a horizontal surface to prevent contamination between different test pads, with timing of the reaction according to the manufacturer's instructions. Color changes on test pads are evaluated by direct visual inspection using the manufacturer provided key [10]. An automated analyzer can also be used.

The reagent pads have a limited shelf life and should be protected from sunlight, moisture, and heat.

Urine pH

Measurement of urine pH by urine reagent strips is less accurate than pH meters as urine reagent strips tend to overestimate pH values. As a result, where accurate measurement is necessary, urine pH should be measured by pH meter [11, 12].

Normal urine pH in dogs and cats is 5.0–7.5. Urine pH varies due to factors including diet and acid–base status. It can also be impacted by the presence of urinary tract

infections with urease-producing bacteria, such as *Staphylococcus* spp. [11] Urine pH cannot be used alone for evaluation of acid–base status in dogs.

Falsely low urine pH can occur if acid buffer from the protein test pad contaminates the pH test pad.

Urine Occult Blood

Heme is detected on the test pad for occult blood and positive results for occult blood can occur with hemoglobin, myoglobin, and intact red blood cells in urine. Thus, urine sediment must be examined to determine whether red blood cells are present. Centrifugation of urine also facilitates differentiation of hematuria from hemoglobinuria and myoglobinuria, with urine persistently red, brown, or black following centrifugation when hemoglobinuria or myoglobinuria is present. Myoglobinuria can be distinguished from hemoglobinuria based on plasma color: myoglobin is excreted in urine before reaching plasma concentrations that would cause discoloration of the plasma, whereas plasma is pink to red with hemoglobinuria as free hemoglobin is only excreted when plasma haptoglobin is saturated.

Hematuria can affect other urine dipstick results in dogs and cats, including measurement of urine pH, bilirubin, and ketones [13].

Hematuria can occur with diseases of the lower urinary or genital tracts and less commonly, the upper urinary tract. Systemic disorders, particularly those that affect coagulation such as severe thrombocytopenia, can also lead to hematuria. Hemoglobinuria occurs due to hemolysis, such as with immune-mediated hemolytic anemia and certain toxins and infectious agents. Myoglobinuria indicates muscle damage.

Urine Glucose

The glucose pad on urine reagent strips is specific for glucose, using a glucose oxidase reaction.

Glucosuria should be interpreted in combination with blood glucose to allow differentiation of glucosuria associated with hyperglycemia from that due to a renal tubular defect (renal glucosuria). Hyperglycemia causes glucosuria when the renal threshold for reabsorption of glucose is exceeded: this occurs when blood glucose is approximately 180 mg/dl in dogs and 280 mg/dl in cats. Cats can have transient hyperglycemia due to stress that results in glucosuria; in some cases, hyperglycemia might resolve by the time of blood sampling. In renal glucosuria, blood glucose is normal as glucosuria occurs due to decreased reabsorption of glucose in the renal tubules.

Urine Ketones

Ketones include acetoacetic acid, acetone, and β-hydroxybutyrate. The ketone test pad contains nitroprusside, which reacts with acetoacetic acid and acetone to cause a color change. The test pads do not detect β-hydroxybutyrate and are more sensitive to acetoacetic acid than acetone. If there is concern for false negative results for ketones on urine reagent strips due to the presence of β-hydroxybutyrate only, addition of a few drops of hydrogen peroxide to urine results in β-hydroxybutyrate being converted to acetoacetate [10].

Ketones are not normally present in the urine of dogs and cats. Ketonuria most commonly occurs in dogs and cats with diabetic ketoacidosis, and as a result, glucosuria is also detected. Starvation or prolonged fasting in puppies or kittens can result in ketonuria.

Urine Bilirubin

Bilirubin results from degradation of hemoglobin and is conjugated in the liver, where it is mostly excreted through bile. Some conjugated bilirubin is also excreted by the kidney. In dogs, the kidneys can form some bilirubin by metabolism of hemoglobin. As a result, small amounts of bilirubin (trace or 1+) can be identified in concentrated urine in normal dogs, particularly male dogs. Bilirubinuria is not normal in cats and therefore any bilirubinuria is significant. Clinically significant bilirubinuria indicates that hyperbilirubinemia is present.

False negative results for urine bilirubin can occur if there are testing delays where the sample is exposed to air at room temperature. Bilirubin can be oxidized to biliverdin or hydrolyzed to unconjugated bilirubin, both of which are not detected by the test pad [5].

Urobilinogen

Urobilinogen forms when conjugated bilirubin excreted into bile in the intestinal tract is degraded by bacteria. A small amount is reabsorbed and excreted in urine. Urobilinogen is unstable in urine. Positive results indicate that the bile duct is patent, but negative results can occur because of urobilinogen instability in urine, diurnal variation in excretion or due to bile duct obstruction. It is therefore considered to be of limited use clinically.

Urine Protein

Urine reagent strips provide a semiquantitative estimate of proteinuria. The test pad is more sensitive for detection of albumin than globulins; most urine protein is albumin. A

small amount of protein is present in urine normally, detected as trace to 1+ proteinuria in concentrated urine. If proteinuria is identified on urine reagent strips, the urine protein to creatinine ratio should be measured to quantify proteinuria and therefore determine if it is clinically significant.

False positive results can occur with very alkaline or concentrated urine. Blood in the urine does not affect urine dipstick protein measurement until the sample is grossly hematuric [13].

Urine Sediment Examination

Microscopic examination of urine sediment is an important part of urinalysis as normal chemical analysis of the urine does not indicate that sediment examination is also normal [14]. Urine sediment examination should be performed as soon as possible after urine collection to prevent degradation of components of the sample such as casts and cells. Crystals can also form with prolonged storage times.

Methods

Urine sediment examination is performed after evaluation of the physical and chemical properties of urine and measurement of USG. The procedure for urine sediment examination is summarized in Protocol 60.1. Standardization of urine sediment examination is important for accurate results.

Approximately 5 ml of urine is centrifuged for five minutes. Low centrifuge speeds (1500 rpm) decrease disruption of cellular elements and casts. As the quantity of sediment is related to the volume and concentration of urine being centrifuged, it is important that the volume centrifuged for urine sediment examination is standardized. After centrifugation, the supernatant is evaluated visually, then removed. The supernatant can be used for measurement of USG or urine chemistry if the sample was grossly discolored. The urine sediment is resuspended in the remaining supernatant (approximately 0.5 ml). Using a pipette, a drop of resuspended urine sediment is placed on a glass slide with a coverslip for microscopic evaluation.

The urine sediment is examined under low power (10×) for casts, crystals, and cells. Bacteria and specific cell types are evaluated at higher power (40×). At least 5–10 fields should be examined at each power. The number of red and white blood cells in each high power field (HPF) is counted and the average used to report the number per HPF. Contrast of unstained urine sediment can be improved by closing the iris diaphragm and lowering the condenser of the microscope. Dimming the microscope light can also improve contrast.

Although evaluation of unstained urine sediment is preferred, stained preparations of urine sediment may assist

Protocol 60.1 Summary of Procedure for Urine Sediment Examination

Handling

1) Transfer the urine to a clear and sterile conical test tube.
2) Store the urine in tube with a tightly sealed lid to avoid evaporation and/or contamination.
3) Take the sample (5 ml, volume is standard in our facility).
4) Analysis timing should be < 60 minutes after collection

Storage

- Refrigerate at 39.2°F (4°C).
- Refrigerated samples are brought to room temperature prior to being analyzed.

Centrifuge

- Speed 1500 rpm for 5 minutes

Procedure

1) Visually inspect the supernatant, then decant or aspirate via a pipette into a separate test tube.
2) Record the results of your visual inspection.
3) Save the supernatant; check and record the urine specific gravity; sediment volume of 0.5 ml remains (volume standardized in facility).
4) Prepare a glass slide, adding 1 drop with a coverslip.
5) Resuspend the sediment using either a "finger-flicking" technique or gentle mixing with a pipette.
6) View under a microscope: 10× (for crystals, casts, and cells) and 40× (for cells and bacteria) magnification.
7) Scan the entire field (average of 10 fields reported).
8) The sediment is best unstained. Abnormal cell identification can be facilitated by fixing a drop of resuspended sediment on a slide (same as blood smear) and staining with Wright–Giemsa stain.

urine sediment examination for those who are less experienced. Urine sediment stains include supravital stains and the Wright–Giemsa stain. Automated urine sediment analyzers have also been developed, such as the SediVue Dx™ (IDEXX, Westbrook, ME).

Red Blood Cells

Red blood cells are round to biconcave (Figure 60.1). Normal urine has less than two red blood cells per HPF. Increased red blood cells in the urine (hematuria) can arise from the kidney, ureter, bladder, urethra, prostate gland, and, depending on urine collection method, the genital tract. Traumatic urine collection by cystocentesis or catheterization can also cause hematuria.

In urine where the concentration is decreased (USG <1.006), red blood cells can lyze or become ballooned ghost cells.

White Blood Cells

White blood cells, also called leukocytes, are round and granular on unstained urine sediment examination (Figure 60.2). They are larger than red blood cells but smaller than epithelial cells. Some white blood cells (less than three white blood cells/HPF) can be present normally in urine. Increased numbers of white blood cells in the urine is called pyuria. This indicates inflammation or infection in the urinary tract; the most common cause is urinary tract infection. Mildly increased numbers of white blood cells can also be detected with hematuria. White blood cells can degrade in urine stored for prolonged periods. Hypotonic or alkaline urine can lead to lysis.

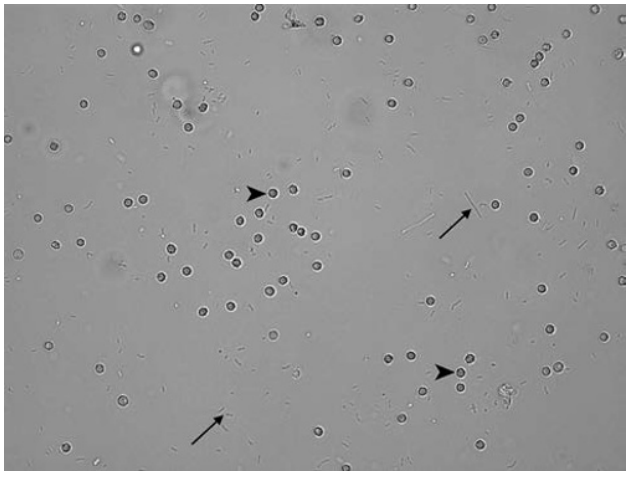


Figure 60.1 Urine sediment examination (unstained) showing red blood cells (arrowhead) and bacterial rods (arrows). *Source:* Courtesy of Dr. Demitria Vasilatis, Veterinary Medical Teaching Hospital Clinical Diagnostic Laboratory, University of California, Davis.

Epithelial Cells

Epithelial cells on urine sediment examination can arise from the kidneys, ureters, bladder, urethra, or genital tract, depending on urine collection method. Their appearance varies. Squamous epithelial cells are large, polygonal cells with small nuclei (Figure 60.3). They arise from the urethra, vagina, or prepuce, and as a result can be seen in voided or catheterized urine samples. Transitional epithelial cells vary in size; they are found in the kidney, ureter, bladder, and proximal urethra; small numbers can be seen normally in urine samples. Increased numbers of

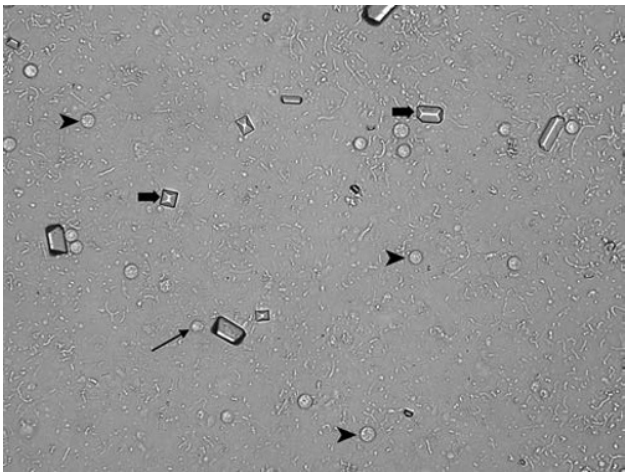


Figure 60.2 Urine sediment examination (unstained) showing pyuria (arrowhead), bacteriuria (thin arrow), and struvite crystals (thick arrow). *Source:* Courtesy of Dr. Demitria Vasilatis, Veterinary Medical Teaching Hospital Clinical Diagnostic Laboratory, University of California, Davis.

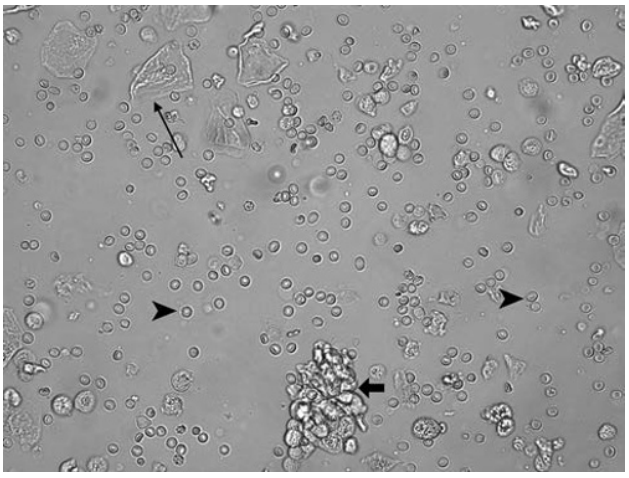


Figure 60.3 Clump of transitional epithelial cells (thick arrow), squamous epithelial cells (thin arrow) and hematuria (arrowhead) in a dog with transitional cell carcinoma on urine sediment examination (unstained). *Source:* Courtesy of Dr. Demitria Vasilatis, Veterinary Medical Teaching Hospital Clinical Diagnostic Laboratory, University of California, Davis.

transitional epithelial cells can be observed with inflammation, infection, and neoplasia of the urinary tract (e.g. transitional cell carcinoma) (Figure 60.3). Clumps of epithelial cells are called rafts. Renal epithelial cells originate from the renal pelvis or tubules and are small and round. It is abnormal to find renal epithelial cells on urine sediment examination.

Crystals

Crystals in the urine are called crystalluria. Crystals are commonly identified in dogs and cats on urine sediment examination. Urine pH, temperature, concentration of the crystals and the presence of any crystal inhibitors or promoters influence the solubility of crystals in urine. Crystals can form during storage and refrigeration of urine samples and as a result, fresh urine samples should be used for evaluation of crystalluria [3].

Struvite, amorphous phosphate, and calcium oxalate dihydrate crystals can be detected in the urine of normal dogs and cats. Struvite (Figure 60.2) and calcium oxalate dihydrate can also be identified in dogs and cats with urolithiasis. Other common crystals include urate and cystine. Both urate and cystine crystalluria are pathologic. Urate crystalluria (Figures 60.4, 60.5) can be observed in certain breeds such as the Dalmatian and English Bulldogs, as well as dogs and cats with portosystemic shunts. Cystine crystals (Figure 60.6) are associated with decreased renal tubular reabsorption of cystine as an inherited defect, with affected breeds including Scottish Deerhounds, Newfoundlands, English Bulldog, Pitbull Terrier, and Mastiff breeds. Calcium oxalate monohydrate crystals can be detected in dogs and cats with ethylene glycol poisoning (Figure 60.7).

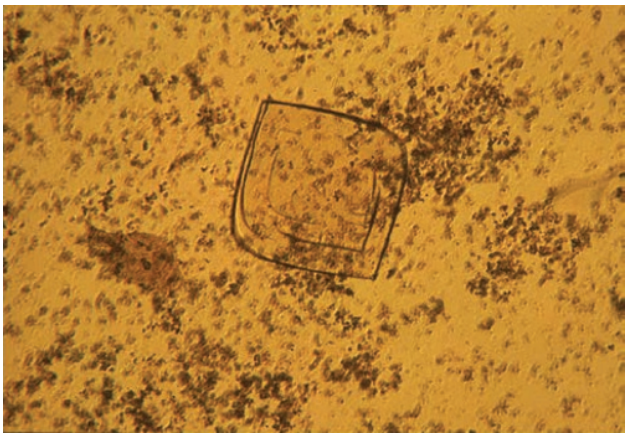


Figure 60.5 Urine sediment examination (unstained) showing uric acid and amorphous urate crystals (×25).

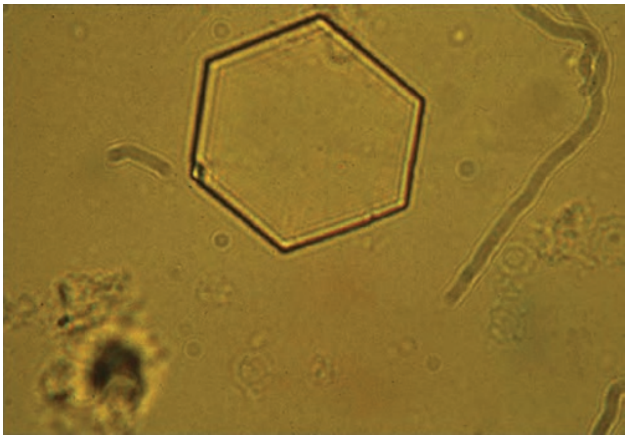


Figure 60.6 Urine sediment examination (unstained) showing cystine crystals (×250). *Source:* Taken from previous edition figure 32.3f page 416.

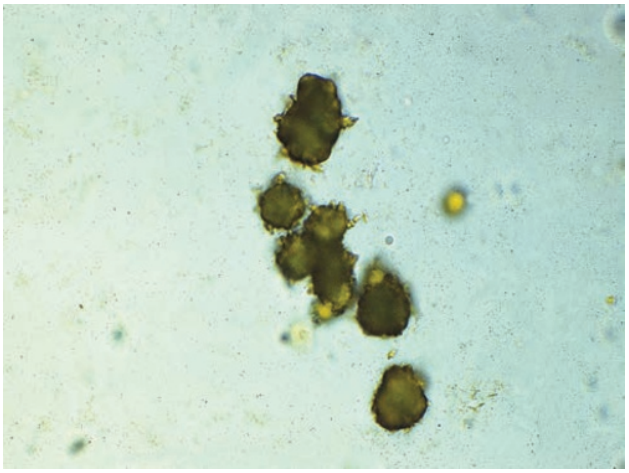


Figure 60.4 Urine sediment examination (unstained) showing "thorn-apple" ammonium urate crystals (×40).

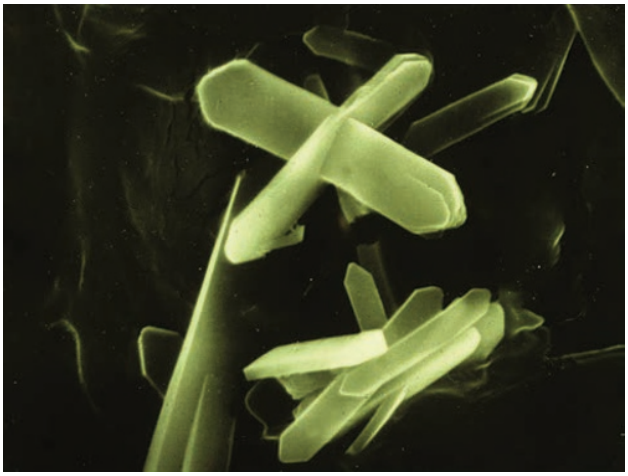


Figure 60.7 Urine sediment examination (unstained) showing calcium oxalate monohydrate crystal (×40).

Casts

Casts are cylindrical structures that are composed of a matrix of Tamm–Horsfall mucoprotein. They form in the distal tubules and structures such as cells in the tubule at the time that the cast forms can become embedded. Some types of casts can be detected in the urine of healthy dogs and cats. Increased numbers of casts in the urine is called cylindruria.

Hyaline casts are clear and homogenous. They can dissolve in dilute or alkaline urine and are generally of minimal clinical significance. Granular casts are detected more commonly and indicate the presence of tubular degeneration. They can be composed of mucoproteins, plasma proteins, degenerated cells, and tubular debris.

Cellular casts can contain white blood cells, red blood cells, and epithelial cells and indicate that renal degeneration, inflammation, or hemorrhage is present. They are not normally present in urine. They disintegrate rapidly and as a result, are only identified on fresh urine sediment examinations.

Waxy casts are homogenous and translucent with a high refractive index. Their ends may be sharply broken or square and their edges notched as they are brittle. They form as a result of degeneration of granular and cellular casts. They suggest that tubular disease is chronic and are associated with a poor prognosis.

Fatty casts are comprised of fat globules from degenerating tubular epithelial cells. They are most commonly observed in cats.

Bacteriuria

Urine is normally sterile from the kidneys to the mid-urethra. Bacteria may be seen in samples collected by catheterization or free catch due to contamination from the distal urethra or genital tract. Examples of bacteria in unstained urine sediment examinations are shown in Figures 60.1 and 60.2.

Rod-shaped bacteria are detected when their concentration is greater than 10^4 bacteria/ml urine and cocci-shaped bacteria when their concentration is greater than 10^5 bacteria/ml urine. Use of the modified Wright stain for urine sediment examination improves the sensitivity and specificity for identification of bacteriuria compared with unstained samples [15]; however, stain precipitates can resemble bacteriuria to cause false positive results for bacteria.

Others

In intact male dogs and cats, sperm can be seen in the urine. Strands of mucus or fibrin can be present in dogs and cats with inflammation of their lower urinary or genital tracts. Lipid droplets are normal in cats.

Other Techniques

Urine Protein to Creatinine Ratio Measurement

When protein is identified in the urine on chemical analysis, the urine protein to creatinine ratio (UPC) is used for quantification. This ratio estimates the daily excretion of protein in urine. To calculate UPC, urine protein concentration (mg/dl) is divided by urine creatinine concentration (mg/dl); the UPC does not have any units. As results are expressed as a ratio to creatinine, USG does not need to be considered when interpreting results [16, 17].

It is recommended that UPC measurement be considered when there is 2+ protein on the urine reagent strip, regardless of USG, and where there is 1+ protein if USG is 1.012 or less, as these values can be associated with an increased UPC [18]. Measurement of UPC on pooled urine samples, where an equal volume from each urine sample is mixed, can be used rather than measurement of UPC on multiple separate samples [19]. Collection of multiple samples for measurement (either pooled or separately) is particularly important when UPC is greater than 4, as these dogs have greater day-to-day variation in UPC [20].

In dogs and cats, UPC less than 0.2 is nonproteinuric (normal). Proteinuria is defined as UPC greater than 0.5 in dogs and greater than 0.4 in cats. Results between the nonproteinuric and proteinuric ranges are considered borderline [21]. Renal proteinuria can occur due to both tubular or glomerular disease, with glomerular disease more likely when UPC greater than 2 [22].

Proteinuria can also arise from pre- and post-renal diseases. Prerenal proteinuria occurs due to small molecular weight proteins passing through a normal glomerulus (e.g. hemoglobin or myoglobin). Postrenal proteinuria is due to protein entering the urine from the ureter, bladder, prostate, or urethra, most commonly due to infection or inflammation, neoplasia or hemorrhage.

Urine Osmolality

Urine osmolality is the number of particles of solute per kilogram of water, with urine solutes including urea, sodium, potassium, ammonium, chloride, and other anions. Urine osmolality is measured by an osmometer [23]. Urine osmolality is used to assess the concentrating ability of the kidney. It differs from USG by not being affected by the size of solutes, although there is a linear correlation between urine osmolality and USG [23].

Normal urine osmolality is approximately 360–2500 mOsm/kg in the dog and 600 and 3000 mOsm/kg in the cat [6, 24, 25]. Normal plasma osmolality is 290–310 mOsm/kg in dogs and 290–330 mOsm/kg in cats [26].

Table 60.2 Urinary biomarkers of tubular damage or dysfunction in dogs and cats [28].

Urinary biomarker	Information provided	Mechanism of change	Species
Clusterin	Tubular damage	Increased production with tubular damage	Dogs
Cystatin B	Tubular damage	Increased release from rupture and death of tubular cells	Dogs, cats
Gamma glutamyl-transpeptidase	Tubular damage	Release from tubular brush border	Dogs, cats
Kidney injury molecule 1	Tubular damage or dysfunction	Increased production in renal tubules	Dogs, cats
Neutrophil gelatinase-associated lipocalin	Tubular damage	Decreased re-absorption and increased production	Dogs
N-acetyl-β-D-glucosaminidase	Tubular damage	Cellular leakage	Dogs, cats

Source: Adapted from Hokamp and Nabity, 2016.

Urine Electrolytes

Measurement of urine electrolytes can be used to evaluate renal function and, specifically, renal handling of electrolytes and minerals such as sodium, potassium, chloride, phosphate, calcium, and magnesium. Fractional excretion is defined as the proportion of a solute that is excreted in urine compared to the proportion filtered through the glomerulus [27]. In normal dogs and cats, fractional excretion values of various solutes vary due to factors such as age and dietary intake as well as any nonurinary excretion [27]. It is performed on urine prior to centrifugation. Fractional excretion is calculated as follows:

$$\text{Fractional excretion}_{\text{electrolyte}} = \frac{U_{\text{electrolyte}} \times P_{\text{creatinine}}}{P_{\text{electrolyte}} \times U_{\text{creatinine}}}$$

Where:

$U_{electrolyte}$ = urine concentration of electrolyte
$U_{creatinine}$ = urine concentration of creatinine
$P_{electrolyte}$ = plasma concentration of electrolyte
$P_{creatinine}$ = plasma concentration of creatinine

Fractional excretion is most commonly calculated for sodium and potassium. Normal fractional excretion of sodium is less than 1% in dogs and cats. Normal fractional excretion of potassium is less than 20% in dogs and less than 24% in cats.

Measuring the fractional excretion of sodium can help to differentiate prerenal azotemia (e.g. due to dehydration or hypovolemia) from acute kidney injury, with fractional excretion expected to be normal in dogs with prerenal azotemia as the kidneys conserve sodium.

Urinary Biomarkers

Urinary biomarkers of kidney injury and dysfunction are emerging in veterinary medicine. Their goal is to identify kidney injury or dysfunction at an earlier stage than traditional biomarkers such as creatinine when interventions are more likely to be effective (Table 60.2) [28]. As some of these techniques become more readily available, their use in acutely and critically ill dogs and cats is likely to increase.

Toxin Screening

Urine can be used for screening for some toxins and drugs. This can be important in determining the cause of certain clinical signs in dogs in an emergency and critical care setting. A human rapid on-site urine multidrug test has been evaluated in dogs and was able to identify barbiturates, opiates, benzodiazepines, and amphetamines/methamphetamines, but not marijuana or methadone [29]. Urine can be used for testing for other drugs, some metals, ethylene glycol, paraquat, and alkaloids [30].

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61

Cytology

Rebecca J. Greer

Cytology is the study of individual cells at the microscopic level specifically to aid in diagnosis of abnormalities and malignancies. Microscopic examination of tissue aspirates and fluid is a valuable tool for the emergency clinician and technician as it can provide a quick diagnosis, help guide treatment, and give information about prognosis. Most samples for cytology are obtained by fine-needle aspiration (FNA) or impression smears. The FNA technique is fairly benign in most cases, which makes cytology a useful tool in critically ill patients that may not tolerate more invasive procedures. A high-quality sample is necessary for accurate interpretation. This chapter discusses some of the common situations encountered by the emergency care providers where cytology is useful. It also details the equipment required and preparation necessary to obtain the most information from cytology samples.

Indications for Cytologic Evaluation

There are many indications for evaluating cytology in emergency practice (Box 61.1). Below are the descriptions of common indications.

Effusion Analysis

Fluid accumulation in body cavities is a common presenting complaint in the emergency setting. Healthy dogs and cats have very little fluid in the pleural, peritoneal, and pericardial spaces. Accumulation of fluid in these potential spaces is pathologic and can be caused by a variety of disease processes. Collection of this fluid can be both therapeutic and diagnostic (see Chapters 18, 34, and 38 for more information regarding sample collection from these spaces). Samples should be evaluated for the presence of cells (red and white blood cells, neoplastic cells, cells from the tissue of origin) and infectious organisms (bacteria, protozoa, and fungi).

Effusions are separated into three major classifications (Table 61.1) based on protein and cellular content [1], with some overlap between the classes. Pure transudates have a low protein concentration (< 2.5 g/dl) and cellular content (< 1000/μl) [1]. Transudates are colorless and clear, and they typically occur due to decreased plasma colloidal osmotic pressure. Common causes of transudate formation are those that cause hypoalbuminemia or panhypoproteinemia, like protein-losing nephropathy or protein-losing enteropathy, respectively [1].

The second class of effusions is exudates. Exudates are characterized by high protein concentration (> 3.0 g/dl) and high cellularity (> 7000/μl) [1]. Exudates are then subclassified as septic or nonseptic. Septic exudates result from infectious causes (bacterial, viral, protozoal, fungal); nonseptic exudates are a result of sterile inflammation (as is often seen in pancreatitis) or neoplasia. Fluid that accumulates in the body cavity due to feline infectious peritonitis (FIP) is a unique example of a septic exudate. In FIP, the cell count may be less than a typical exudate (< 7000/μl), but the protein concentration in the fluid is generally very high (> 4.5 g/dl). The high protein concentration is what classifies the FIP fluid as an exudate. On cytological examination of FIP fluid there is usually granular pink material in the slide background due to precipitated protein [1].

The third class of effusions is modified transudates. Modified transudates have total protein and cell concentrations between pure transudates and exudates. The protein concentration is typically at least 2.5 g/dl and can be as high as 7.5 g/dl [1]. The cell count is usually 1000–7000/μl [1]. Modified transudates result from leakage of fluid from the lymphatics or capillaries due to increased vascular hydrostatic

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Box 61.1 Indications for Cytology

Presence of effusions:

- Pleural
- Pericardial
- Peritoneal
- Joint

Lymphadenopathy/lymphadenomegaly

Masses
Dermatologic problems
Skin lesions
Ear canals

Table 61.1 Evaluation of Effusions.

Effusion	Cell count	Total protein	Causes
Transudate	< 1000 cells/μl	< 2.5 g/dl	Hypoalbuminemia, panhypoproteinemia
Modified transudate	1000–7000 cells/μl	2.5–7.5 g/dl	Most common: heart failure, neoplasia, Inflammation, chylous, hemorrhagic
Exudate	> 7000 cells/μl	> 3 g/dl	Bacterial, viral, protozoal, fungal
Septic exudate	Visualize infectious organism(s)		
Nonseptic exudate	No infectious organism(s) seen		Inflammation, pancreatitis, peritonitis, neoplasia

pressure or increased vessel permeability, such as the ascites seen with right-sided congestive heart failure, pericardial effusion, or portal hypertension. Modified transudates are the most common effusions in dogs and cats. Hemorrhagic and chylous effusions fall into this category.

Pericardial Effusions

The etiology of pericardial effusions can be difficult to determine cytologically. Pericardial effusions can be pure transudates, modified transudates, or exudates; however, most are modified transudates (hemorrhagic). Some pericardial effusions are idiopathic and no underlying etiology can be identified. Neoplasia is the most commonly identified etiology, and premortem identification generally requires imaging such as echocardiography [2]. The common neoplasms that cause pericardial effusion in dogs are hemangiosarcoma and chemodectoma, neither of which tends to exfoliate cells; thus, cytology rarely identifies the etiology. Mesothelioma cells do exfoliate, but when they do it can be difficult to distinguish the mesothelioma cells from reactive, non-neoplastic cells. The preceding discussion explains why it can be challenging to make a diagnosis of neoplasia using cytologic evaluation of pericardial effusion, even when neoplasia is the underlying etiology. Cytology can be useful in diagnosing infectious etiologies of pericardial effusion, especially if organisms can be identified [2].

Joint Effusions

In animals with joint effusion, cytology often can identify the cause and therefore guide treatment. Cytology is the most important evaluation to perform on joint effusions, so if only a small sample can be obtained, cytology should be prioritized over total protein concentration or cell counts. Normal synovial fluid is grossly viscous and has a low protein concentration (< 2.5 g/dl) and cell count (< 3000/μl) [1]. There is very little fluid present in healthy joints. Pathologic joint effusions can be septic or nonseptic. Septic arthritis is usually caused by a bacterial or fungal infection and is usually localized to one joint, though multiple joints can be affected. Infection can be caused by hematogenous spread or by direct inoculation. The cell count in septic arthritis is typically high (5000–10 000/μl), with neutrophils as the predominant cell [1]. Often in septic arthritis the neutrophils are degenerate. Neutrophil degeneration may not always be apparent, and organisms may not be identified in septic arthritis. If enough fluid can be obtained, a culture should also be performed. Joint fluid in nonseptic arthritis has an increased cell count (1000–10 000/μl) but not usually as high as seen with septic arthritis [1]. The neutrophils are nondegenerate and no infectious organisms are present. Causes of nonseptic arthritis include ehrlichiosis, Lyme disease, drug mediated, immune mediated, and systemic lupus erythematosus. Nonseptic arthritis can also be secondary to neoplasia or infection in distant areas of the body. Neoplastic cells may be present in cases of synovial cell sarcoma, but histopathology is generally required to differentiate between reactive synovial cells and neoplasia. Trauma or coagulopathies may also cause joint effusion. In this case the fluid resembles peripheral blood [1].

Lymph Node Evaluation

Enlarged lymph nodes may be noted by owners or may be found on physical examination. Lymph nodes may be enlarged due to a primary problem within the nodes themselves or in response to disease elsewhere in the body. The prescapular ("superficial cervical") and popliteal nodes are the sites of choice to aspirate when generalized lymphadenomegaly is present. The mandibular lymph nodes are not

the best diagnostic nodes because they often have a strong inflammatory component secondary to draining the mouth that can mask other diseases. The size of the nodes should also be considered. Very large lymph nodes tend to have necrotic centers, which makes diagnosis difficult. It is advisable to sample slightly or moderately enlarged nodes if possible [1]. Common conditions that cause lymphadenomegaly include lymphoma, metastatic neoplasia, infection (bacterial, fungal, rickettsial), inflammation, and immune mediated disease.

Mass Evaluation



Masses are commonly found on and in small animals. Intra-abdominal and intrathoracic masses are generally aspirated with ultrasound guidance (Video 61.1). The sample is then evaluated to help determine whether the mass is neoplastic, inflammatory, or benign. Dermal or subcutaneous masses are generally only evaluated in the emergency setting if there is concern that they are related to significant disease pertinent to the current visit. Examples of skin or subcutaneous masses related to systemic disease include mast cell tumors, cutaneous lymphoma, and blastomycosis.

Skin and Ears

Dermatologic diagnoses are not typically intensely pursued in the emergency setting; however, there are certain urgent care situations when cytology of the skin and external ear canals can be beneficial. It is not uncommon for an animal to present to the emergency clinic with severe skin lesions, itchy skin, or otitis. Cytology is beneficial in making a correct diagnosis and therefore selecting appropriate initial treatment. Excessive amounts of bacteria may require systemic antibiotics. Severely pruritic animals require skin scraping with cytologic evaluation to look for evidence of mites. Animals with signs of ear disease require ear cytology to determine whether topical treatment alone will be adequate or if systemic treatment is necessary. Furthermore, cytologic examination of skin lesions may give a diagnosis in debilitated patients not stable enough for more invasive diagnostic tests or general anesthesia. For example, patients with systemic blastomycosis may have skin lesions that allow for rapid noninvasive cytologic diagnosis of the systemic problem.

Equipment Required

A compound binocular microscope with a set of high-quality objectives is required for diagnostic cytologic examination. The microscope should have a light source with a substage condenser to provide optimal contrast and eliminate artifact. Most microscopes have four standard objectives: 4×, 10×, 40×, and 100× oil immersion. A good quality microscope is essential.

The microscope must be adjusted before each use to provide the best image. First the oculars should be adjusted for the width of the examiner's eyes. Each ocular can be individually focused to account for differences in the focusing abilities of the viewer's eyes. Once an image is visible, a combination of adjustments will be made to achieve the best focus. Protocol 61.1 lists the general sequence of events.

For the best images, the Kohler method of illumination (Protocol 61.2) should be used and readjusted every time an objective is changed. This method optimizes the path that light takes from the light source to the viewer's eye and will help eliminate scatter. The sample must first be

Protocol 61.1 Adjusting the Microscope

Items Required

- Microscope
- Specimen slides
- Immersion oil, if 100× viewing is anticipated

Procedure

1) Place the slide on the stage; secure slide in place with the stage clips.
2) Move the 4× or 10× objective into use. Adjust the coarse focus knob to move the slide up as close as possible to the objective, taking care not to bump the slide into the lens. Using the 4× or 10× it should not be possible to touch the slide to the objective.
3) Illuminate a stained area of the slide.
4) While looking through the ocular lenses, slowly adjust the coarse focus knob (moving the specimen away from the objective) until the specimen is visible.
5) Change to the fine focus knob and bring the image into sharper view.
6) Adjust the individual oculars for the best focus.
7) Adjust the condenser lens and/or light source so the illumination is optimal.
8) Readjust the fine focus knob as needed to improve the image.
9) Move to higher objectives without changing the focus from the previous objective. Use the fine focus knob to bring the image into sharp focus.
10) Prior to viewing at 100×, apply a small drop of immersion oil to the slide where the objective will contact the slide to provide optimal image.

Protocol 61.2 Kohler Illumination

Items Required

- Microscope
- Specimen slides

Procedure

1) Bring image into focus (Protocol 61.1).
2) Completely close diaphragm.
3) Raise or lower condenser until circle of light is in sharp focus.
4) Center light in the field of view.
5) Open diaphragm until only the field of view is present (i.e., circle of light just outside field of view).
6) Open or close condenser aperture to allow best contrast.

in focus, following the steps listed in Protocol 61.1 for focusing. The diaphragm is then closed all the way, resulting in a blurry area of light in the field. Using the condenser knob, raise or lower the condenser until the circle of light is in sharp focus then use the condenser placement screws to center the light circle in the field of view. Open the diaphragm until only the field of view is illuminated, so the circle of light is just outside the field of view. Open or close the condenser aperture to allow the best contrast [3, 4].

If the image is difficult to get into focus, the lenses may need to be cleaned or the light source adjusted. Opening or closing the aperture and/or adjusting the strength of the light source may help.

Maintenance of the microscope (Box 61.2) includes weekly cleaning of the objective and ocular lenses with a lens cleansing solution. Use the recommended solution for the particular microscope but not alcohol or water because standard immersion oil is not dissolved by these substances [5]. The lens is then wiped with lens paper that has the appropriate solution applied to the paper and wiped in a circular motion. Lens paper is the only material that should be used to actually wipe the lenses because other paper or materials may scratch the lens surface. The ocular pieces can be removed from the eyepiece tubes and wiped down, but do not attempt to take the actual lenses apart. The objectives should be wiped with plain lens paper at least once a day and the high-power oil objective (100×) should have the oil removed with plain lens paper every time it is used. If the microscope is not properly maintained, the lenses will become damaged and a clear, focused image cannot be achieved. The stage should be wiped as needed. This area of the microscope can be cleaned with ordinary cleansing materials; oil spills and water will cause the slides to stick to the stage, preventing appropriate movement. The owner's manual of each microscope lists specific solutions and cleaning recommendations for that microscope. The microscope should be serviced regularly as recommended by the manufacturer. The supplies needed to acquire diagnostic samples are inexpensive and readily available (Box 61.3).

Box 61.2 Microscope Maintenance

- Wipe the 100× objective with lens paper after every use.
- Wipe objectives, oculars, and stage once daily.
- Clean lenses with cleansing solution weekly. Use the recommended solution for the particular microscope.
- Follow manufacturer recommendations regarding regular service.

Box 61.3 Equipment for Acquiring Samples

- Glass slides, preferably frosted on one end
- Coverslips: glass or plastic
- Needles: 21–25-gauge
- Syringes: 3–20-ml
- Serum tubes
- EDTA tubes
- Centrifuge for separating fluid samples
- Stains: Romanowsky type
- Pencil

Slide Preparation

Slide preparation varies by the type of sample to be analyzed (i.e. tissue vs. fluid).

Fluid

Examination can be performed on fluid directly, or samples can be centrifuged to concentrate cells. Slides from fluid samples are prepared in the same way as a blood smear. A small drop is placed near the frosted end of the slide. A second slide is backed into the sample with the acute angle toward the operator (frosted end of the slide). The second slide is then drawn away from the operator. The speed at which the slide is moved depends on the viscosity of the fluid [1]. Thinner samples should be spread faster than more viscous samples to ensure even distribution over the slide. All fluid applied to the slide originally should remain on the slide. Care should be taken not to allow excess fluid

to be drawn off the end of the slide. Neoplastic cells tend to clump and stick to the spreader slide. If excess fluid is removed with the spreader slide and discarded, valuable diagnostic material may be lost. If excess fluid is a problem, the spreader slide can be stopped before it reaches the end of the specimen slide. Excess fluid on the spreader slide can then be transferred to another slide. An alternative method is to allow the excess fluid to run back on itself for a short distance. With this procedure the thin part of the specimen can be used to estimate cell numbers while the area of excess fluid can be evaluated for abnormal cells or infectious organisms.

Samples can be centrifuged to concentrate fluids of low cellularity. After centrifuging for five minutes, the pellet is resuspended in one or two drops of supernatant. This solution is then prepared as previously described. Microhematocrit tubes can be used if there is a small volume of fluid. The sample is drawn into the microhematocrit tube and is then spun just like a packed cell volume. The buffy coat or cellular layer is then applied to a slide [1]. The slide is prepared using the squash technique described later.

Tissue

Slides of tissue samples can be obtained in several different ways. FNA is a common method to obtain samples [1]. Once the tissue sample has been obtained in the needle, the needle is removed from the syringe. A syringe filled with 1–3 ml of air is attached to the needle. The sample is then blown onto the slide using the air in the syringe. The air blowing process may be repeated a few times to ensure complete transfer of sample material to a slide (Figure 61.1a,b). Once the sample material is on a slide, a compression (squash) preparation can be made (Videos 61.2 and 61.3).

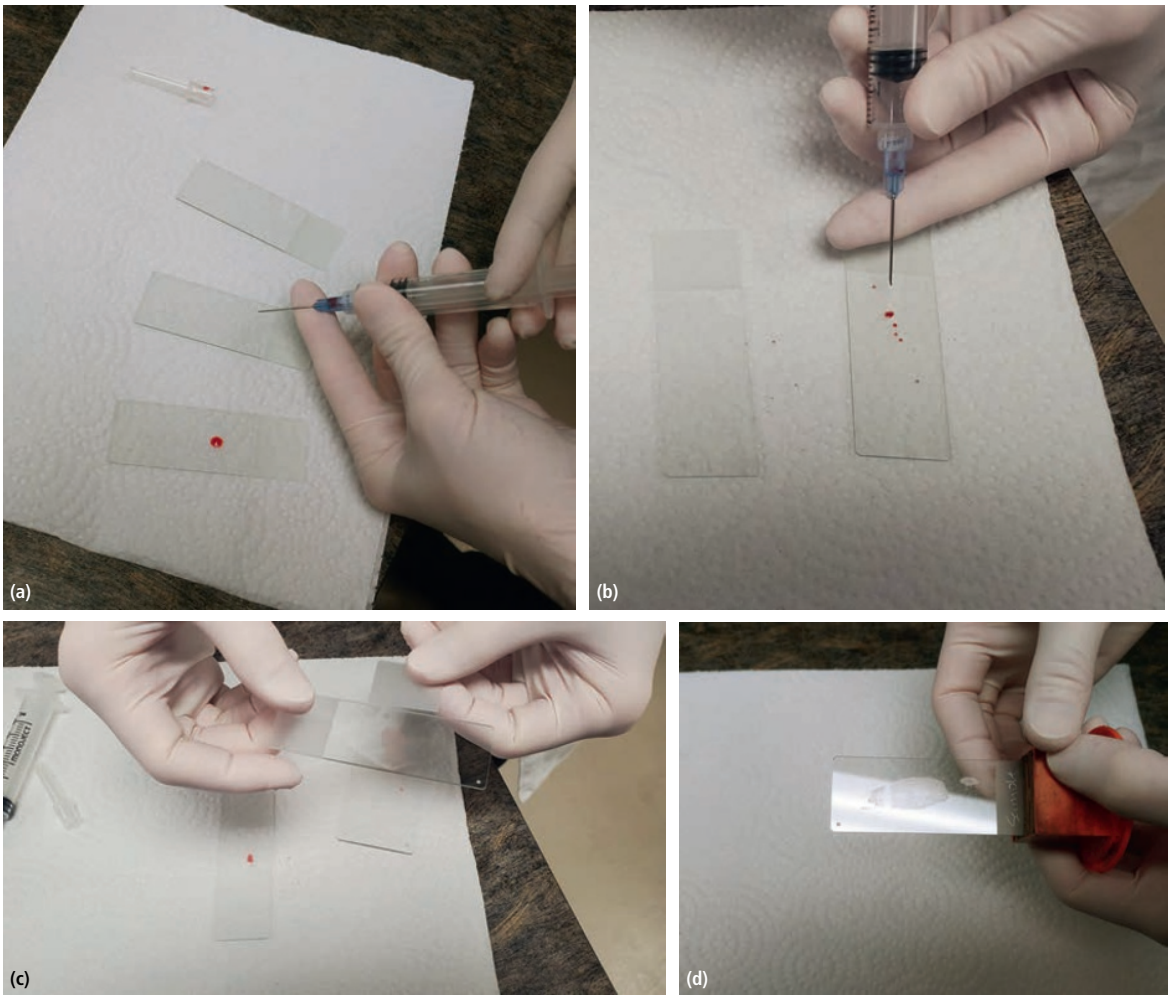


Figure 61.1 Preparing squash sample from an aspirate. (a) Aligning the needle above the slide; needle aspiration has been performed and sample is inside the needle's lumen. (b) The sample was blown out of the needle using the air in the syringe. This results in a small amount of sample on the slide. (c) A second, clean slide is aligned over the slide with the sample, the weight of the slide is used to squash the sample, and then the top (second) slide is drawn off the end of the sample slide. (d) Finished squash sample, unstained.

Squash Preparation

A squash preparation is accomplished by placing a sample on the slide and then placing a second glass slide over the sample at a right angle to the specimen slide. The sample is gently but firmly compressed. Generally, the only pressure for compression is the weight of the second slide. The second slide is then drawn away from the frosted end of the specimen slide. This process should be performed with a smooth and continuous motion [1]. Care should be taken especially with lymph node aspirates because lymphoma cells rupture easily [6]. The resulting specimen should be oblong in shape and have a cellular monolayer at the end (Figure 61.1c,d and Video 61.3 and 61.4).

Impression Smear

Slides can also be prepared by making an impression smear. This technique can be used for skin or excised tissue. With excised tissue, the cut surface is blotted with a paper towel to remove blood or other fluids. The tissue is then gently pressed or touched against a slide at several different places. The tissue should lightly stick to the slide. A clean slide can also be pressed against skin to obtain skin cytology. A variation of the slide-to-skin technique uses a clear adhesive tape preparation. A piece of transparent acetate tape is used. The adhesive side of the tape is pressed against the area of interest, being sure to leave adhesive that has not been compromised on each side for affixing to the slide. The sample area is placed on the middle of a glass slide and the tape edges used to affix the tape to the slide. The slide with adhered tape can then be stained. If using the Diff-Quik® (Andwin Scientific, Tryon, NC) solution, the fixative step is skipped because it will dissolve the adhesive, and the slide is dipped into the stains and rinsed in standard fashion. An alternative staining method is to place a single drop of stain on the slide and then affix the tape with the sample section in the stain. In this method, the stain is not rinsed off but the tape, drop of stain, slide combination is directly viewed with the microscope [7]. The tape method of obtaining samples is especially useful for the paws and around the eyes and nose [1].

After the sample is obtained and placed on a slide, the slide should be labeled with patient identification and type of sample (Figure 61.2). A pencil will write easily on the frosted end and remain visible even after staining. Marks made by pens and permanent markers will rinse off during the staining process.

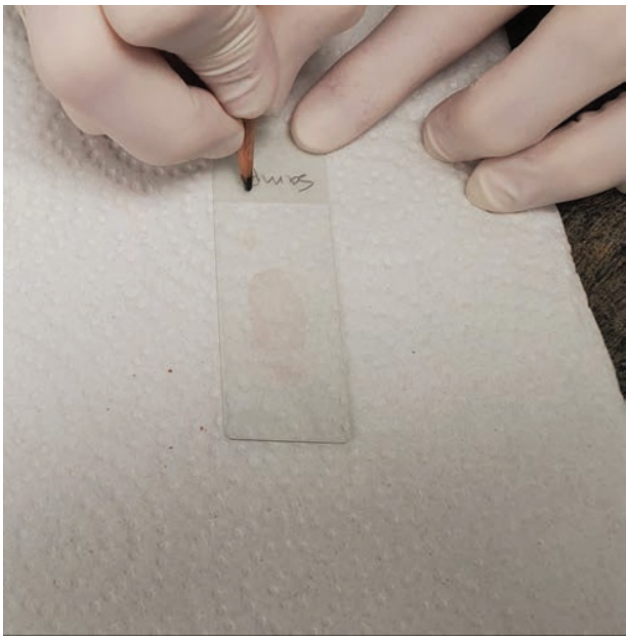


Figure 61.2 Sample labeled and ready to be stained.

Staining

Many different stains are available for staining cytology samples. The most commonly used in veterinary medicine are the Romanowsky-type stains [8], including Wright, Giemsa, and Jenner stains. Romanowsky-type stains are characterized by the use of a combination of eosin and methylene blue. Characteristically, these stains color a cell's nucleus purple and its cytoplasm blue or pink [9]. A proprietary example is the Diff-Quik stain. The Diff-Quik brand (Figure 61.3a) is one of the most commonly used Romanowsky-type stains due to the quick results and ease of use. Staining times with Diff-Quik vary depending on the thickness of the sample. Thick samples may require as much as 60–120 seconds in each solution. A general guide for staining with Diff-Quik solutions is as follows (Protocol 61.3): 60–120 seconds in fixative, 30–60 seconds in solution 1, and 5–60 seconds in solution 2. The slide should then be rinsed with cold water for 15–20 seconds to remove any stain precipitate and then allowed to air dry in a nearly vertical position [1]. If time is of the essence, the samples could be dried by blotting with bibulous paper or a hair dryer on low. Common errors in staining are discussed later.

New methylene blue is another valuable stain to have in the emergency practice because it stains nuclei, bacteria, fungi, platelets, and mast cell granules [8]. Diff-Quik solutions may not always stain mast cell granules adequately; therefore, a new methylene blue stain may help distinguish the cells in low-granule mast cell tumors. New methylene blue can also be used in cases with anemia to look for reticulocytes, which contain blue precipitating granules when stained this way. New methylene blue is more labor intensive than Diff-Quik because it requires preparing the solution and proper disposal of formalin. To prepare the solution, new methylene blue is mixed with 0.9% saline and a small volume of formalin. The solution is passed through filter paper before using to remove precipitates. The stain should be replaced and filtered weekly [1].



Figure 61.3 (a) Quick stain in glass vials for easy "dipping." (b) In fixative. (c) Solution 1. (d) Solution 2. (e) Stained sample drying.

A drop of the prepared solution is then placed on a slide, and a coverslip is placed on top. The slide should be examined immediately because the stain evaporates quickly.

Slide Scanning and Evaluation

Slides should be examined first with the 4× or 10× objective to evaluate overall sample quality and staining. Slides that are too lightly stained can be re-stained to improve quality. The slide is then scanned at 10× to find the area of the slide that will be of highest diagnostic yield. The slide should be scanned in a consistent manner to be sure the entire slide is evaluated. Starting at one corner of the slide, a back-and-forth pattern is used until the entire slide has been evaluated. Large objects such as parasites and some fungal elements may be seen at this low power. Once the initial evaluation is performed, a more detailed study is made. The best area to examine is the area where the cells are in a monolayer. The 40× objective is used to obtain an overview of the cell population. At this magnification it is possible to compare cell sizes and determine the proportions of

Protocol 61.3 General Guidelines for Diff-Quik Stain

Items Required

- Diff-Quick stains
- Specimen slides

Procedure

1) Place the specimen slide in fixative for 60–120 seconds.
2) Place the specimen slide in solution 1 for 30–60 seconds.
3) Place the specimen slide in solution 2 for 5–60 seconds.
4) Perform a 15- to 30-second slide rinse in cold water.
5) Air dry or blot gently with bibulous paper.
6) Time for staining will vary depending on thickness of sample (longer for thicker samples).

different cell types. This power should provide enough detail to make a presumptive diagnosis if there is not enough time to thoroughly examine the slide in the emergency setting. To further improve resolution, a drop of immersion oil is applied directly to the stained surface (or on top of the coverslip if one is in place), and the slide is reexamined with the 100× oil immersion objective. Take care not to touch immersion oil to *any* objective other than the oil immersion (generally 100×) because this is the only objective that is sealed and designed for oil use [10]. Oil can ruin the other unsealed objectives. This objective shows greater detail of individual cells (Protocol 61.4). Nuclear structures and cytoplasmic granules can be seen at this magnification (Figure 61.4). Cellular inclusions may also be seen at this magnification [1].

Protocol 61.4 Slide Scanning and Evaluation

Items Required

- Microscope
- Immersion oil
- Specimen slides

Procedure

1) Systematic scan at low power (4× or 10×).
   a) Use back-and-forth technique.
   b) Evaluate staining technique.
   c) Identify monolayer.
   d) Look for large organisms (fungus or parasites).
2) Increase magnification (40×).
   a) Evaluate cellular structure.
3) Increase magnification (100×).
   a) Place drop of immersion oil on slide before rotating objective fully into place.
   b) Evaluate for more detailed cellular structure.

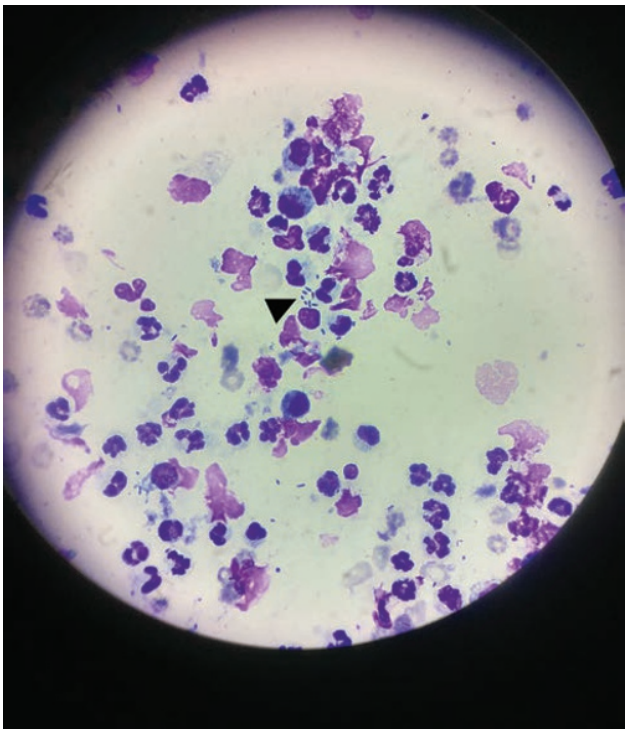


Figure 61.4 Image of a septic abdominal exudate on 100× oil immersion, intracellular bacteria at black arrowhead.

Troubleshooting

Making a diagnosis from cytology can be very rewarding. However, there are some pitfalls that can make examining slides frustrating. Here is a list of commonly encountered problems and their solutions.

Nondiagnostic Samples

Obtaining nondiagnostic samples can be frustrating, especially in an emergency situation. One of the most common causes for nondiagnostic samples is hemodilution. This phenomenon occurs commonly when aspirating vascular organs such as the spleen. If a large amount of blood is aspirated, it can be smeared like a blood film to look for diagnostic cells. The best way to avoid blood contamination in vascular organs is to use a fine-needle biopsy approach without aspiration, also called a fenestration approach [6]. A needle (alone or attached to a syringe) is directed into the area of interest. Multiple fenestrations are made in the tissue without applying negative pressure to the syringe. The needle is then attached to a syringe with air, or if already attached to a syringe, the air is used to blow the sample out and it is prepared as a squash (Videos 61.3 and 61.4). If using the aspiration technique in a tissue where peripheral blood contamination is expected to be a problem, release the negative pressure on the syringe before removing the

needle from the tissue. This precaution will prevent blood from being drawn into the syringe as the needle is removed. It will also prevent the sample from being sucked into the barrel of the syringe when the pressure in the syringe equilibrates with room air [6].

Nondiagnostic samples are also obtained if the material on the slide is too thick or if there is poor separation of cells. A common mistake is to place too much material on a slide. It is then impossible to obtain a monolayer of cells with a squash preparation. This problem can be avoided by making sure only a small amount of sample is applied to a slide [1]. Multiple slides can be made from a single FNA. It takes practice to know the right amount of material to apply to a slide.

Fractured cells and naked nuclei also make diagnosis difficult. Certain neoplastic cells are fragile and break when preparing the sample. One step to help avoid this problem is to not apply excessive vacuum when aspirating the sample. In general, 0.5–1-ml of vacuum is all that is needed. Overzealous compression of the sample during squash preparation can also fracture cells [1]. Care should be taken when preparing slides, especially of enlarged lymph nodes because lymphoma cells rupture easily.

Staining Problems

Most errors that occur with staining are due to inadequate time in the solutions or use of old solutions. Slides may appear dull or washed out. In most cases the problem can be remedied by replacing the slide into one or all of the staining solutions. Staining times vary depending on the kind of material being stained. In general, thicker cellular material requires more contact time than thinner, less cellular material. Stain precipitates may be present on the slide if it has not been rinsed adequately [8]. The same problems can be seen if the staining solutions are not maintained properly. Manufacturer recommendations should be followed regarding maintenance and changing or replacement of the solutions and their containers.

Prolonged contact time in the stain may also result in problems. When using the Romanowsky-type stains, slides may appear too pink or too blue. It is important to use the same brand of stain consistently to obtain a feel for the appropriate length of time to stain different material [8].

If technique is followed properly and the slide is still not stained appropriately, there may be a problem with the stain itself. This problem usually occurs as the stain deteriorates with age [8]. The stain should be replaced with fresh solution when this occurs (Protocol 61.5). Aggregates of stain precipitate can form and give the false appearance of cocci bacteria or inclusion bodies. False results can have life-threatening consequences in an emergency situation. For example, if these cocci-appearing aggregates are seen in a sample of abdominal fluid from a critically ill patient, that patient may be taken for an abdominal exploration resulting in an unstable patient undergoing a risky, unnecessary, and costly procedure. This problem can be avoided by changing the stain regularly. The time frame varies from clinic to clinic depending on the number of slides stained and the type of stain used. Manufacturer recommendations should be followed regarding how often to change the solutions. It is good practice to have separate stains for clean (blood, aspirates, fluid) and dirty (skin, ear canals, fecal, abscess) samples. Bacteria from dirty samples can contaminate the stain, resulting in bacteria on the slide of a sample that did not truly contain bacteria. If only one staining station is available, the stain should be changed immediately if has come in contact with infectious organisms or debris.

Protocol 61.5 Troubleshooting

Nondiagnostic Sample

1) Retake sample.
2) Decrease force of aspiration.
3) Use needle biopsy technique (fenestration approach).

Staining (too lightly stained)

1) Re-stain.
2) Replace old or used stain solution.

Stain Granules

1) Rinse slide thoroughly.
2) Re-stain new sample in fresh stain.

Conclusion

Cytology is an important tool for the emergency clinician. A diagnosis made by in-house cytology can prevent a delay in definitive treatment. A quick diagnosis benefits the patient and the owner. If a diagnosis cannot be made in the clinic, samples should be submitted to a diagnostic laboratory for analysis. Good sample collection technique and sample preparation will ensure that the pathologist has the best chance of making a diagnosis. There are some instances in which cytology will not provide an answer. In these instances, biopsies should be taken after the patient has been stabilized.

Video 61.1 Fenestrating mass with ultrasound. This video shows ultrasound guidance with the fenestration technique to obtain a cytology sample. Note in the ultrasound image as the operator performs repeated advances and withdrawals of the needle (fenestrations) to acquire tissue in the needle, rather than aspirating the

syringe plunger. This video does not include audio commentary.

Video 61.2 Squash preparation. This video reveals how to make a "squash" preparation. This video does not include audio commentary.

Video 61.3 Fenestrating mass and making slides. This video shows the fenestration technique with a needle only, then "blowing" the sample out onto a slide and creating a "squash" preparation for staining. Note that the operator performs repeated advances and withdrawals of the needle (fenestrations) to acquire tissue in the needle. After blowing the first part of the sample onto the slide, the operator removes the syringe from the needle, pulls more air into the syringe, then attaches the air-filled syringe to the needle a second time to blow any remaining sample onto the slide. This video does not include audio commentary.

Video 61.4 How to make a "squash" preparation. This video demonstrates the procedure in greater detail than in Video 61.3. This video does not include audio commentary.

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Section Eight

Infection Control

62

Minimizing Healthcare-Associated Infections

Jane E. Sykes

Nosocomial infections are infections that develop more than 48 hours after a patient has been hospitalized or that occur in a patient that has been hospitalized in the two weeks prior to the current admission [1]. The term healthcare- (or hospital-) associated infection (HAI) describes infections associated with healthcare delivery in any setting. This reflects the difficulty in determining with certainty where a pathogen was acquired in many patients. As nosocomial infections are, by definition, acquired in a healthcare setting, the veterinary team, and especially veterinary technicians who spend most time interacting with the patient, have a very important role to play in minimizing their occurrence.

At the time of writing, 2022 figures from the Centers for Disease Control and Prevention (CDC) suggest HAIs occur in 1 of every 31 hospitalized patients a year in the United States, with an annual cost of at least US$28.4 billion. The COVID-19 pandemic has adversely impacted the incidence of HAIs, with rises in reported rates and clusters of infections [2]. Both in the United States and Europe, HAIs occur with the greatest frequency in patients in intensive care units (ICU). Nosocomial infections in small-animal hospitals, including outbreaks, have been well documented in the veterinary literature, but the overall rate of HAIs is less well understood. One study estimated that the endemic rate of HAIs in critical care units at small-animal hospitals was 16.3% of dogs and 12% of cats [3]. HAIs increase morbidity and mortality, prolong hospitalization, and increase the overall cost of care, so it is important to prevent them. They also have the potential to be zoonotic, with associated public health implications.

Factors associated with an increased risk of acquiring an HAI in human hospitals are also likely to be associated with increased risk in veterinary patients; in particular, the more intensive treatment of critically ill animals with increasing use of invasive devices such as urinary and intravenous (IV) catheters, increased duration of hospitalization, increase in intensive care techniques such as mechanical ventilation, and the wider use of antimicrobial and potent immunosuppressive drugs [4, 5]. Importantly, many of the bacteria associated with nosocomial infection are normal commensal organisms that may be found on the skin/mucosa or in the gastrointestinal tract of healthy dogs and cats. Although the relationship between colonization and subsequent infection is complex, patients entering an ICU with prior colonization may be at higher risk of subsequent infection and may act as reservoirs for other noncolonized patients [6].

HAIs may complicate the course of both medical and surgical diseases. They may range from mild superficial skin infections to bacteremia with sepsis and septic shock. In human medicine, common HAI sites include bloodstream infections, urinary tract infections, surgical site infections, infectious diarrhea, and pneumonia.

Many bacterial species can contribute to HAIs. An increasing proportion appear to be resistant to multiple antimicrobial drug classes. These bacteria are of particular concern as they often resist first-line treatments and are expensive to treat. Moreover, they may represent a zoonotic risk to veterinary staff attending infected patients as well as being a risk to other patients in the ICU. Viruses such as feline calicivirus are also capable of causing HAIs, and the fungal organism *Candida* may cause opportunistic infections following overtreatment with antibacterial drugs.

Bacteria Associated with Healthcare-Associated Infections

HAIs may be caused by a large number of different bacterial species. Much of the published information focuses on infection with specific multidrug-resistant bacteria (as described below). Although not specifically covered

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here, bacteria such as *Pseudomonas aeruginosa*, *Acinetobacter baumanii*, *Serratia marcescens*, and *Bordetella bronchiseptica* are also important nosocomial pathogens.

Methicillin-Resistant Staphylococci

Methicillin-resistant *Staphylococcus aureus* (MRSA) is one of the most significant bacterial species associated with HAIs in human medicine. Staphylococci are Gram-positive cocci that are commensals of mucosa and skin. They can cause a wide range of infections following opportunistic tissue invasion, including postoperative wound infections, implant infections, bacteremia, infective endocarditis, and catheter-associated infections. The majority of MRSA isolates are also resistant to most other commonly used antibiotics, making infections hard to treat once they have occurred. Humans are generally considered to be the source of MRSA infections in dogs. Veterinary staff (including veterinary technicians) may be at increased risk of colonization with MRSA than the general human population, with colonization rates of approximately 10% being reported in several studies [7, 8]. Methicillin-resistant *Staphylococcus pseudintermedius* (MRSP) is a greater concern for HAIs in veterinary patients as it is the primary staphylococcal species that colonizes healthy dogs. MRSP infections have been well documented following surgical procedures in dogs that involve implants, especially tibial plateau leveling osteotomies. Such procedures require meticulous attention to infection control in order to prevent devastating consequences of resistant surgical site infections such as amputation or euthanasia.

Extended Spectrum Beta-Lactamase-Producing *Escherichia coli*

Escherichia coli is a Gram-negative commensal of the gastrointestinal tract. Multidrug-resistant *E. coli,* including extended spectrum beta-lactamase (ESBL)-producing *E. coli* infections, are emerging nosocomial threats in veterinary patients and are well established causes of HAIs in humans. Cultures of fecal specimens from healthy dogs and pet therapy dogs have identified colonization by ESBL *E. coli* [9, 10]. *Escherichia coli* isolates, including drug-resistant strains, are commonly implicated in HAIs, especially urinary tract infections. Prolonged ICU stay has also been shown to be associated with increasing proportions of resistant *E. coli* isolates from rectal swabs in veterinary patients [11]. ESBL-producing *E. coli* are of particular concern as they are resistant to many different antibiotics including the third-generation cephalosporins, limiting the choice of therapy for these pathogens to carbapenems (e.g. meropenem) and occasionally aminoglycosides, which can only be administered parenterally when used to treat systemic infections.

Vancomycin-Resistant Enterococci

Enterococci belong to normal human and animal gut flora. They are Gram-positive cocci with *Enterococcus faecium* and *Enterococcus faecalis* being the most frequent isolates. *Enterococcus faecium* is generally more resistant than *E. faecalis*. Vancomycin-resistant *Enterococcus* spp. (VRE) are multidrug-resistant bacterial species associated with HAIs that were first identified in human hospitals in the United States in the 1980s, and have been increasingly identified in small-animal patients. A small number of cases of clinical infection have been reported in companion animals [12, 13], and VRE have been found to colonize healthy dogs in both Europe and the United States [14]. If not susceptible to beta-lactams, treatment is challenging due to pan drug resistance. In some cases (e.g. bacteriuria), the host may clear the infection spontaneously if the underlying cause/predisposing factors for the infection can be resolved.

Transmission of Infection and Colonization

The majority of bacteria that cause HAIs are commensal organisms that invade opportunistically. The hands of healthcare workers represent the main mode of transmission of these bacteria among patients, with the main reservoir being other infected or colonized patients or colonized healthcare workers [15]. Contamination of the environment may also play a role in infection, with veterinary staff transferring bacteria that contaminate environmental surfaces to patients. Some pathogens, such as enterococci, can survive in conditions similar to those found in the hospital environment for long periods (days) and thorough cleaning of the environment is necessary to prevent outbreaks of infection.

Control Strategies for Nosocomial Infection

Infection control measures and prevention strategies are critical considering the potential for HAIs to cause increased morbidity and mortality, zoonotic infections, and hospital financial losses in association with litigation or closure. All hospital personnel play a vitally important role in prevention of HAIs. When designing a clinic strategy to minimize nosocomial infection, the following should all be considered:

- Hand hygiene
- Barrier environmental nursing hygiene technique and isolation, including identification of at-risk patients
- Antimicrobial stewardship.

Many veterinary teaching hospitals have formalized infection control programs to reduce the incidence of HAIs. The National Association of State and Public Health Veterinarians has developed a model infection control program for veterinary practices, [16] and the Ontario Animal Health Network also provides recommendations for the development of a comprehensive infection control program [17]. The reader is also referred to other resources on healthcare-associated pathogens and infection prevention and control [18]. When designing prevention and control programs, all routes of transmission should be considered, including contact (direct or indirect), airborne (aerosol or droplet), and vector borne. It also should be recognized that not all infectious agents are transmissible from one patient to another.

Hand Hygiene

Diligent hand hygiene is arguably the single most important measure used in the control of nosocomial infection [15]. Although the need and technique for surgical scrubbing is well recognized, techniques for hand hygiene outside the surgical suite are often overlooked, and experience suggests that hand hygiene is often a neglected priority in day-to-day veterinary practice. Hand hygiene can be performed in a number of ways, including:

- Handwashing with a biocide-containing hand soap (e.g. triclosan, chlorhexidine) and water
- Using alcohol-based hand sanitizers
- Surgical hand hygiene/antisepsis.

The aim of all hand hygiene procedures is to reduce the number of potentially pathogenic bacteria on the hands. Handwashing will mechanically remove organic material and transient microbes on the skin, and, when combined with an antimicrobial soap, can inactivate microflora. Furthermore, the multiplication of resident flora is also temporarily reduced if the antiseptic has persistent or residual activity. Surgical hand hygiene describes the conventional presurgical scrub procedure. This takes longer to perform than a simple antiseptic handwash but leads to a greater reduction in resident flora. When nursing patients in critical care, handwashing with a biocide-containing soap or use of an alcohol hand rub represents the most appropriate form of hand hygiene in the majority of situations.

How to Perform a Hygienic Handwash

Although it sounds simple, numerous studies have shown that handwashing is rarely performed correctly. To be effective, all surfaces of the hand and wrist must be washed. If using an antiseptic, it must also be in contact with the skin for a suitable period according to the manufacturer's instructions, which for a chlorhexidine-based solution, for example, is typically one minute. The most effective way to ensure that all parts of the hands are cleaned is to follow a staged handwash protocol (Protocol 62.1), illustrated in Figure 62.1. The CDC also provides useful information on hand hygiene [19].

When a protocol is not followed routinely, handwashing is often inadequately performed, with some areas of the hands not cleaned properly. The efficacy of a handwashing protocol can be tested using hand creams that fluoresce under ultraviolet light if not properly washed off (e.g. GlitterBug®, Brevis Coirp., Salt Lake City, UT).

Protocol 62.1 How to Perform an Antiseptic Handwash

Use free-flowing water at a temperature suitable for thorough wetting and rinsing of hands.

Procedure

1) Wet hands thoroughly and apply hand soap.
2) Rub hands together palm to palm.
3) Rub right palm over back of left hand and vice versa.
4) Rub hands together palm to palm with fingers interlaced.
5) Bend and interlock fingers (as if holding hands with yourself).
6) Clean thumbs by grasping right thumb in left palm and vice versa.
7) Use fingers of right hand to rub left palm focusing on base of left fingers and vice versa.
8) Use left hand to rub right wrist and vice versa (optional).

Notes

- Faucets should be turned off using a no-touch technique.
- Hands should be dried using paper towel.
- Paper towel should be disposed of in a foot-operated pedal bin.
- The whole process should take around 1 minute.

Hand Rubs

Complete and diligent handwashing takes time, and in a busy critical care environment it can be difficult to achieve on all the occasions in which it is required. Alcohol-based hand rubs are an alternative to antiseptic

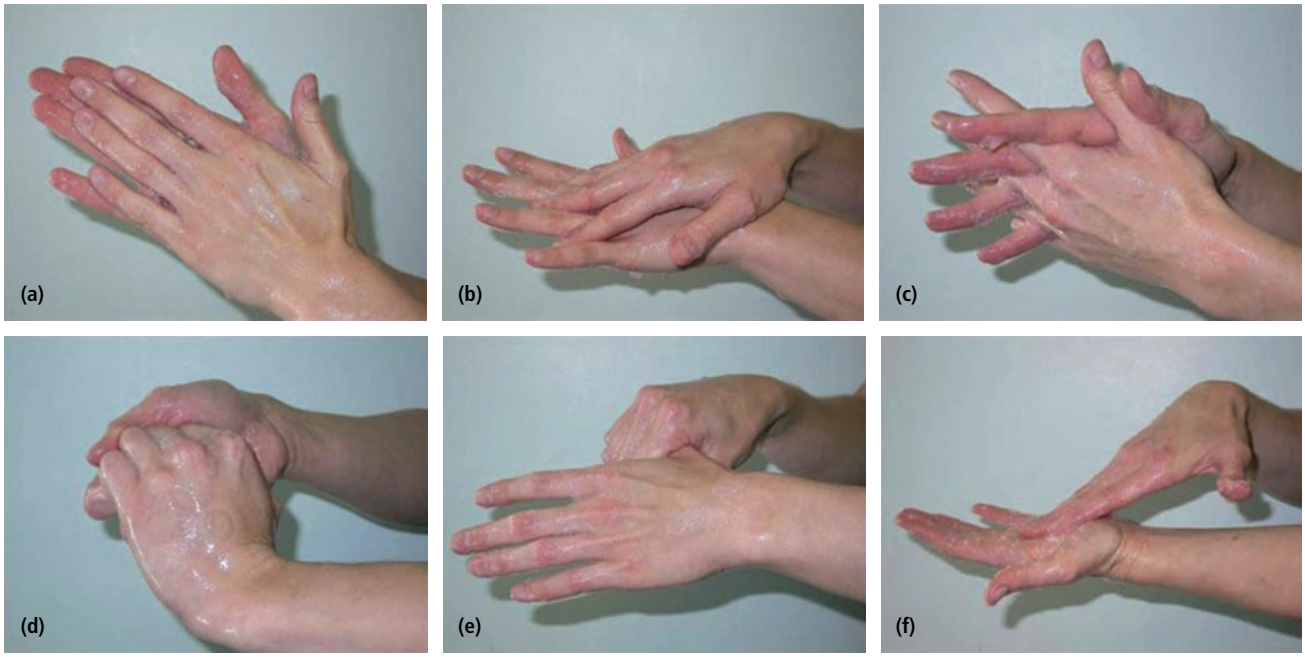


Figure 62.1 The six steps of hand hygiene. (a) Rub hands together palm-to-palm. (b) Rub right palm over back of left hand and vice versa. (c) Rub hands together palm-to-palm with fingers interlaced. (d) Bend and interlock fingers. (e) Clean thumbs by grasping right thumb in left palm and vice versa. (f) Use fingers of right hand to rub left palm focusing on base of left fingers and vice versa. *Source:* Courtesy of Professor S. Gregory MRCVS, Royal Veterinary College, UK.

handwashing that can be used provided the hands are not grossly dirty; that is, hands must be free of visible dirt, blood, or other proteinaceous material or body fluids. Use of an alcohol-based hand rub is the preferred means for hand hygiene in most clinical situations. Alcohol hand rubs should contain 60–95% ethyl or isopropyl alcohol, and they typically also contain an emollient. Both liquid and gel formulations are available. They can be bought as wall dispensers or small bottles. A total of 2–3 ml typically obtains 90% hand coverage, regardless of hand size, and should be applied to all parts of the hands (using a protocol similar to that used for antiseptic hand washing) for 15–20 seconds, allowing the hands to air dry. Hand rub use may be encouraged by wearing the small bottles on clinical clothing and/or by placing them strategically on kennel doors.

The advantages of alcohol-based hand rubs compared with antiseptic handwashing include a reduction in the time taken to complete effective hand hygiene, more rapid action, and less skin irritation. In addition, alcohol-based hand rubs do not require the presence of a sink or hand drying facilities. All these factors have been demonstrated to improve hand hygiene compliance in human medicine.

Regardless of the technique chosen, it is important that fingernails are kept short and clean and any cuts or abrasions are covered with waterproof dressings. Artificial nails should not be used [20]. All jewelry should be removed, and sleeves should be short.

Timing of Hand Hygiene and Use of Gloves

There are many occasions when hand hygiene is indicated (Box 62.1). For basic care procedures such as physical examination, correct use of an alcohol-based hand rub is sufficient. Handwashing should be used if hands are visibly soiled and after the healthcare worker performs any personal hygiene procedures, such as visiting the bathroom. Use of gloves can reduce bacterial contamination of hands as well as acting as barrier protection during contact with body fluids. Gloves do not, however, completely protect against hand contamination; contamination may occur via small defects in the glove or during glove application and removal. Gloves should be worn whenever it can be reasonably anticipated that there will be contact with potentially infectious material or nonintact skin. Gloves should be removed after each procedure and when moving from a contaminated body site to another site on the same patient. Hands should always be washed after removing gloves, and glove use should *not* reduce the frequency of handwashing.

Studies in human hospitals reveal that there are many factors associated with poor hand hygiene compliance,

Box 62.1 Hand Hygiene Guidelines

Hand hygiene should be carried out:

- Before and after touching or examining the patient.[1]
- Before and after touching any invasive devices such as intravenous catheters and urinary catheters.[1]
- After contact with any body fluids or excretions, mucosal surfaces, nonintact skin, or wound dressings.[1]
- If moving from a contaminated/infected body site to another body site of the same patient.[1]
- After contact with inanimate objects in the immediate vicinity of the patient.
- Before handling medication or preparing food.
- When arriving and leaving work.
- Before and after performing any personal hygiene procedures such as visiting the bathroom or blowing the nose.
- Before and after eating.
- Before and after removing gloves.

[1] Glove use should be considered if there is a high risk that the hands may come into contact with potentially infectious material or nonintact skin.

including being a doctor or nursing assistant (as opposed to a nurse), being male, working in a critical care environment, a high intensity of patient care, wearing gloves and gowns, undertaking activities with a high risk of cross-transmission, being too busy and failing to think about it, lack of easy availability of facilities such as sinks, and skin irritation with frequent hand hygiene [21]. A lack of knowledge is also often highlighted with some reports of skepticism about the effectiveness of hand hygiene and lack of knowledge of hospital protocols. Additional perceived barriers include lack of role models [22] and low institutional priority, with lack of administrative sanctions for noncompliers [23]. Barriers reported for veterinary practitioners are forgetfulness, being too busy, skin damage from frequent handwashing, and lack of available supplies.

Methods to improve hand hygiene compliance include education and teaching with constant reinforcement within the workplace, getting senior staff to set a good example, reminder signage, and making sure the staff-to-patient ratio is favorable. The introduction of conveniently placed alcohol hand rubs, which removes the need to handwash after every patient contact and allows opportunities for hand hygiene remote from washing facilities, also facilitates compliance. Every member of the healthcare team has a large role to play both in ensuring they themselves comply and in reminding the rest of the veterinary team as to its importance. It is also important that dispensers be refilled or replaced regularly so that they are never found empty. Posting of signage may also be of benefit.

Environmental Cleaning

Although specific responsibility for environmental cleaning may reside with one group of staff, all personnel should take some degree of responsibility for maintaining a clean clinical environment, and the policy of "clean as you go" should be adopted. This also applies to senior staff and veterinarians, who should model good practice. Positive feedback is also extremely important, and the value of performing a cleaning task well should never be overlooked. All clinics should have a fixed cleaning schedule with checks to ensure regular cleaning is being performed.

Floors and all surfaces should be cleaned at least once daily. Floors should be constructed of nonporous, nonslip materials, and ideally the junction between floor and wall should be curved to facilitate cleaning. Gross debris should be swept up and the floors cleaned with a disinfectant that is ideally virucidal, bactericidal, mycocidal, nonirritant, noncorrosive, and nonstaining (see Chapter 64 for more information). Disinfectants should be used according to the manufacturer's instructions. Mops should be kept clean and replaced on a regular basis. Mop buckets should be emptied and rinsed after each use and should also undergo regular full bucket disinfection. Floors should be deep cleaned on a regular basis with the frequency dependent on use and soiling.

Examination and operating tables should be disinfected after each use, using a suitable disinfectant with properties similar to those used on the floor. As most disinfectants are not effective in the presence of organic matter, any gross contamination should be removed using soap and water first.

Work surfaces in clinical areas and kennels should be constructed of material that can be thoroughly cleaned and is impervious to disinfectant. Seams should be sealed to prevent accumulation of contaminated material, and junctions should be rounded to facilitate cleaning. They should be regularly disinfected at least twice daily but more regularly if they become soiled. Movement of patients between kennels should be minimized. Sinks and showers must be kept clean, and in hard water areas limescale should be controlled and soap scum removed daily with a scouring preparation. Areas not immediately visible, such as the tops of kennels, should not be forgotten, and the cleaning schedule should include a frequent (e.g. weekly) general cleaning and inspection of these areas.

Garbage bins used for clinical waste should be covered and the cover should be foot operated and not allowed to overflow. Animal bedding should be cleaned of gross soiling and then washed using a hot cycle (140°F, 60°C) with a biological washing powder. Drying in a hot tumble dryer is recommended. Food and water bowls should also be made

of a material that can be disinfected. Use of disposable bedding and feeding bowls is recommended when contamination with transmissible pathogens is possible.

"High touch surfaces" such as infusion pumps, kennel doors, monitoring equipment, door handles, computer keyboards, and telephones, should be disinfected more regularly than other sites. Medical equipment used for physical examination (e.g. stethoscopes, thermometers) may also be considered in the same way. Disinfectant wipes (preferably hydrogen peroxide based) and hand rubs should be readily available near these sites.

Barrier Nursing and Isolation

All practices should have facilities and policies for isolation and barrier nursing. These facilities may be used for animals with a known infectious disease but also on occasion for patients with a known risk of contracting an infectious disease (e.g. unvaccinated or immunosuppressed animals). Barrier nursing may also be used for patients in which there is a high index of suspicion for infection with a pathogen that could be associated with HAIs pending receipt of microbiological culture results. An example might be a patient with a chronic nonhealing wounds that has received multiple courses of antibiotics.

True isolation facilities are completely self-contained areas that do not share an air space with other animal accommodation areas. As the facility must be completely self-contained, it must have its own equipment for feeding, nursing, and cleaning, including hot and cold water, medical supplies, and an examination table. Not all practices have an area that fulfills all these requirements.

For animals where strict isolation is not required or available, barrier nursing may be more appropriate. This may include patients where an increased level of vigilance is appropriate to protect the animal from infection or where the animal has a known infection but is considered a low contagious risk. Barrier nursing may be provided within a separate ward or a partitioned area of an existing ward. It should be remembered that animals that are isolated are inevitably barrier nursed; however, not all animals that are barrier nursed are effectively isolated.

Regardless, each animal being isolated or barrier nursed should have dedicated equipment. Any piece of equipment can act as a fomite and can facilitate transmission to other patients [24]. The isolation or barrier nursing area should have dedicated leads or leashes, thermometers, and stethoscopes. As far as possible, equipment including bedding and feeding bowls used in this area should be disposable, and robust cleaning protocols should be in place for any equipment that will be used subsequently with other patients. If bedding is to be reused, it should be clearly identifiable as belonging to the isolation area; for example, it may be a color different from that used in the rest of the clinic. The location of the medical record should also be considered; if paper records are used, they should not then be carried to other sites in the clinic. Dedicated pens or computer terminals should be used.

Isolated patients should not have contact with other patients. Procedures such as radiography or ultrasound that must be carried out in other areas of the clinic should be scheduled for the end of the day (if not urgent), and all staff involved should be alerted as to the nature of the patient's disease. These areas should be thoroughly cleaned and disinfected after use.

Staffing of isolation areas will depend on the size of the clinic. Ideally, one veterinary technician should be allocated to this area and this veterinary technician should have minimal responsibilities in other animal areas, especially with any high-risk patients. Protective attire (gowns, which should be water impermeable if splashes/sprays are possible; footwear protection; disposable gloves, face shields, and caps) should be worn when entering the area and must be removed on exit. For true isolation areas, a foot bath or foot mat containing an appropriate disinfectant that is properly replenished should be available on entry and exit of the area. These measures can also act to deter traffic. Personal and environmental hygiene must be strict and all waste must be disposed of safely. Staff and owners should be apprised of any possible zoonotic risks, and owners should be allowed to enter only in exceptional circumstances. If owners are allowed to visit, they should observe the same hygiene and clothing precautions as staff members. Notices should be placed at the entrance of the isolation area or around the barrier nursing area clearly stating what measures must be instituted on entry (Protocol 62.2).

The cost of isolating or barrier nursing patients is high in terms of both consumables and staffing, and therefore additional fees should be set and owners apprised of the rates. Owners should also be warned that intensive monitoring and treatment may not be possible to deliver in the isolation unit.

In some cases, it may be preferable or necessary to treat a patient with an HAI as an outpatient. Many patients with HAIs are clinically well and with appropriate owner information and consent may be managed at home. It is vital that the owners are educated as to zoonotic risks and that if there are any concerns that humans in the animal's environment are at risk, advice should be sought from medical professionals as to whether management at home is appropriate from the human health perspective. If patients are managed on an outpatient basis, it is essential that they are clearly identifiable on revisits. Owners should be educated to wait outside the clinic until their appointment time to reduce the risk of transmission to other patients in the waiting room. As far as possible, revisits should be

Protocol 62.2 Barrier Nursing an Isolated Patient

Note

Only designated personnel should enter the isolation unit; visitors are permitted only with the express permission of the attending clinician and must be accompanied at all times.

Procedure for Isolation Area

1) Wear protective clothing when entering the area, including:
   - Disposable, water impermeable apron or gown
   - Shoe covers
   - Disposable gloves
   - Caps
   - Depending on the infectious agent, face shields or N95 masks.
2) Remove and dispose of all protective clothing on exiting the isolation area.
3) Using an antiseptic hand wash, clean hands both before entering and on leaving the isolation area.
4) Provide all patients in the isolation area with a lead/leash, stethoscope, feeding utensils, litter tray (if appropriate), and thermometer for their sole use. Keep together in a plastic box and sterilize/disinfect between patients.
5) Do not remove medications from the isolation area.
6) Dispose of all clinical waste in the garbage bin within the isolation area; seal the bag and double-bag before removal from the area.
7) Use disposable bedding.
8) Thoroughly clean the isolation area once the patient has been discharged.

scheduled such that thorough cleaning of the examination room can be performed following the visit.

Antimicrobial Stewardship

Antimicrobials are commonly prescribed drugs and are often used on an empirical basis, especially while awaiting bacterial culture results. In human medicine, the CDC estimates that 50% of outpatient prescriptions are inappropriate, and in 28% of prescriptions, no antibiotic was indicated at all [25]. Moreover, inappropriate antibiotic use is associated with higher mortality, which is not necessarily reversed if the antibiotic is changed once culture results are known [26, 27]. A 2022 survey of 2410 US veterinary practitioners suggested that widespread inappropriate prescribing of antimicrobials also exists in companion animal medicine [28]. The use of most antimicrobials is uncommonly associated with observable adverse effects for that individual patient; most of the commonly prescribed drugs have a high therapeutic index and it is easy to feel that by prescribing an antibiotic we "may be doing some good and are unlikely to be doing harm" to the individual patient. However, inappropriate antimicrobial therapy can contribute to gut dysbiosis and adds unnecessary costs and inconvenience to the animal owner. Importantly, any commensal or colonizing microbes that display mechanisms of resistance will enjoy a selective advantage.

The clustering of multiple resistance genes on plasmids and other genetic elements makes the problem especially challenging, as exposure to one antimicrobial may coselect for bacteria that are resistant to several unrelated agents. When infections occur in animals with a history of antimicrobial therapy, they are more likely to be resistant than when they occur in animals that lack a history of antimicrobial therapy. For most important HAIs (e.g. those caused by multidrug-resistant staphylococci, enterococci and *E. coli*), prior colonization of the skin, mucosa, or gastrointestinal tract results in a much-increased risk of nosocomial infection with the multidrug-resistant strain [29, 30]. Thus, antimicrobial therapy should be used judiciously even when considering the individual patient. One study documented changes in fecal flora in dogs hospitalized in a veterinary ICU and showed that the proportion of dogs colonized with resistant bacteria increased with duration of hospitalization regardless of antimicrobial use. Dogs treated with enrofloxacin were 25.6 times more likely to be colonized by a resistant strain [11].

Guidelines are available from the International Society of Companion Animal Infectious Diseases to assist practitioners regarding appropriate selection and dosing of appropriate antimicrobials for different clinical situations [31–34]. Familiarity with the antimicrobial use guidelines was shown to be associated with reduced antimicrobial prescribing for conditions when antimicrobial use is typically not indicated, specifically feline lower urinary tract disease, feline upper respiratory tract disease, and canine acute diarrhea [28].

Role of Screening for Pathogenic Bacteria

Screening involves taking microbiological samples from the environment, patients, or staff to look for the presence of multidrug-resistant bacteria. Culture and molecular methods such as nucleic acid amplification tests have been used for this purpose. Routine environmental screening is costly and the results of routine environmental screening

generally do not correlate with rates of HAIs, and so has not been recommended by the CDC [35]. The CDC has outlined four indications for environmental screening:

1) To support an investigation of an outbreak of disease.
2) For research purposes.
3) To monitor a potentially hazardous environmental condition, confirm the presence of a hazardous chemical or biological agent, and validate the successful abatement of the hazard.
4) To evaluate the effects of a change in infection control practice or to ensure that equipment or systems perform according to specifications and expected outcomes.

Although advocated in the past, screening of patients and staff for colonization by MRSA is generally not recommended, because decolonization has had questionable efficacy [36].

Summary

HAIs can be associated with significant morbidity and mortality and can lead to increased expense and stress for owners, the possibility of clinic closures, and costs associated with litigation. With advancements in treatment for critically ill, nosocomial infections will become more common. Moreover, many of these infections may be with multidrug-resistant organisms, and the age of relying on ever more powerful antibiotics seems to be drawing to a close. To minimize the risk of nosocomial infection, it is vitally important that we use multiple infection control strategies based on an understanding of the epidemiology and transmission of these microbes. Diligent handwashing, good environmental cleaning, and appropriate barrier nursing and isolation are all key parts of an infection control strategy where veterinary technicians have a very large role to play.

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63

Care of Indwelling Device Insertion Sites

Helen Philp

Indwelling medical devices play an essential role in the emergency room and intensive care unit. They assist in diagnosis and patient monitoring, enable administration of intravenous fluids, blood products, nutrition and medication, and aid support of organ function [1]. Despite their widespread application, a number of complications may be encountered.

Complications

Infection is a major concern with indwelling medical devices, mainly due to their circumvention of innate defenses. For example, any device traversing the skin bypasses the protective epithelial barrier including its antimicrobial secretions, cutaneous microflora, and resident phagocytic cells [2]. Devices placed via natural orifices may similarly compromise host defenses; for example urethral catheters interfere with protective mechanisms such as the unidirectional flow of urine, periodic bladder emptying and the antimicrobial properties of urine. Potential sequelae to intravenous (IV) and/or urinary catheter-associated infections include pyelonephritis, endocarditis, and septic arthritis [3].

The consequences of device-associated infections range from localized signs to bloodstream infection, sepsis, and death. Significant association between the presence of indwelling medical devices and increased risk of sepsis has been documented in human medicine [4–7]. Nosocomial infections increase morbidity and mortality, lengthen hospital stays, and add cost to patient care (for further information, see Chapter 62). In veterinary medicine, increased morbidity or cost can lead to euthanasia, as most owners have a finite amount of money to spend. Even localized infections without systemic effects increase patient morbidity; for instance, the device may need to be removed or may cease to function. Alternatively, it may become displaced due to disruption of local tissue or loss of securement.

There are four recognized routes for contamination of intravenous catheters [8]. The most common in short-term catheters is migration of skin organisms from the insertion site. Direct contamination of the catheter by contact with hands or contaminated fluids or devices is another potential route. Finally, catheters may become hematogenously seeded from another focus of infection or infusate may be contaminated; these final two routes are less common than the first two. Contamination and bacterial colonization do not automatically lead to infection. Factors that can compound the likelihood of infection associated with a device include the material of which the device is made, host factors such as thrombus and fibrin sheath formation and the intrinsic virulence of an infecting organism, including its propensity for biofilm formation [8, 9]. Biofilm itself may become a continuing source of bloodstream infection by intermittent release of cell clusters or individual planktonic cells [10]. Additional indwelling devices and longer dwell time both appear to increase the risk of a hospitalized patient developing a nosocomial infection [1, 9].

Potential noninfectious device complications include dislodgement, lumen occlusion, patient injury (e.g. infiltration, extravasation, vascular occlusion, thrombosis, or phlebitis in the case of vascular catheters), or loss of function for another reason.

The consequences of losing function of an indwelling device for any reason could be serious. For example, a displaced thoracostomy tube may rapidly compromise a patient's ability to breathe while the loss of even a simple venous catheter can make effective patient treatment challenging and delay procedures or drug administration.

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The Value of Protocols

Adherence to protocols for the placement, handling, and maintenance of indwelling devices significantly decreases the incidence of complications [8, 11–13]. Hand hygiene is one of the most important aspects of these protocols as healthcare workers' hands represent the principal route of pathogen transmission [3, 14–17]. Transient pathogenic microorganisms are readily removed with hand hygiene and strict adherence to hand hygiene protocols has been reported to reduce nosocomial infections by as much as 40% [3, 18]. Hand hygiene also prevents colonization and infection in the healthcare worker as well as contamination of the environment [3]. An alcohol-based hand rub is generally preferred, unless hands are visibly soiled, because these rubs reduce skin irritation and improve healthcare worker compliance compared with hand washing [19]. The Centers for Disease Control and Prevention (CDC) has developed extensive recommendations for how and when to perform hand hygiene measures [14], which are detailed in Chapter 62. A hygienic handwashing protocol is available in Protocol 62.1.



Figure 63.1 Patient with esophagostomy tube and hemodialysis catheter in place (patient in right lateral recumbency with its head to the left). Care must be taken to avoid cross-contamination between these sites during inspection, cleaning, and rewrapping.

General Care of Insertion Sites

Insertion site care varies somewhat by patient and device, but some principles generally hold true across the board.

Bandaging

Insertion sites that penetrate the skin (excepting tracheostomy sites) should be kept covered with a sterile, nonadherent pad or a sterile, self-adherent bandage, and wrapped with gauze. Cast padding may be placed over the gauze to help secure it, and an outer, water-resistant wrap may be applied as a final barrier. This general bandage will work in many situations but is not appropriate for all devices. More specific wraps are described later in the chapter.

Handling

As stated above, good hand hygiene is paramount in the basic care of indwelling devices. After proper hand hygiene is performed, clean examination gloves should be worn before handling any device or insertion site. Sterile gloves may be indicated in some instances, such as when handling the inner cannula of a tracheostomy tube (Chapter 29). Care must be taken to avoid cross-contamination between devices. For example, the presence of both an esophagostomy tube and hemodialysis catheter or central line (Figure 63.1) can present a challenge in avoiding contamination of the vascular catheter insertion site. In this scenario, it is recommended to clean the catheter insertion site first and to change gloves after handling the esophagostomy tube site.

Maintenance

Most sites should be unwrapped, evaluated, and rewrapped at least once daily. The insertion site should appear clean with no redness, swelling, oozing fluid, or other signs of inflammation. The area should not feel excessively warm and should not be unduly painful. If the device is sutured in place, the sutures should be assessed for functionality and suture sites evaluated for inflammation. The device should be securely in place with no signs of slippage or migration. When a device is inserted, a note should be made on the treatment sheet as to the device's size, functionality (e.g. for an IV catheter: Does it flush? Does it aspirate?), and placement depth. Some devices have depth markers on them that can be used as a reference when performing daily evaluation. If there are no markers, the length of the device extending from the body can be measured and a note made in the treatment sheet. If the site shows signs of inflammation or infection, it should be scrubbed with a 2% tincture of chlorhexidine preparation, the chlorhexidine should be left

on the site, and the site should be allowed to air dry before rewrapping [8]. A clinician should be notified of the inflammatory change and the patient should be assessed for signs of systemic infection. Similarly, any migration from the original position or altered functionality should be reported to the clinician. Device removal is at the clinician's discretion. Insertion sites should be kept clean and dry.

Care of Associated Lines and Connections

The ideal replacement interval for infusion sets has not been established. The purpose of routine replacement is to reduce infection via colonization. However, there is some argument that replacing administration sets risks contamination during handling. Recommendations in human medicine range from intervals of four to seven days [19–25]. For veterinary patients, it would seem reasonable that disposable lines or connections used with an indwelling device can remain in place for 72–96 hours. Tubing used to administer blood, blood products, or fat emulsions (including propofol infusions) should be replaced no less often than every 24 hours [8]. Immediate infusion sets should be replaced in the event of contamination. For arterial lines, disposable, closed flush transducer assemblies are preferred and should be replaced at 96-hour intervals, together with the tubing, continuous-flush device, and flush solution [8, 25].

Injection ports should be swabbed with 70% isopropyl alcohol or an iodophor (e.g. povidone-iodine) prior to being punctured with a sterile needle [8]. Stopcocks should be aseptically capped when not in use and access via injection ports or needle-free connectors rather than direct attachment of syringes is recommended where possible [16]. Each additional access point to a device increases the risk of contamination, so port number should be kept to a minimum. Integrated devices and Luer lock design are recommended where possible [26, 27].

General Management of Device Insertion Site Infection

If a device site infection occurs, the clinician must decide whether to remove the device or leave it in place. Risks and benefits must be considered, and close monitoring of rectal temperature, blood pressure, complete blood count, and blood glucose concentration may aid in decision making. If the infection appears to be localized, it is sometimes best to leave the device in. For instance, in the author's experience, localized feeding tube insertion site infections are often treated successfully with diligent, daily site cleaning with a 2% chlorhexidine scrub; thorough site drying before bandaging; and sometimes systemic antimicrobial therapy (Figure 63.2). Minor infections related to central venous catheters in human pediatric patients are sometimes managed with diligent site care and systemic antimicrobials for up to three days, after which time the device is usually removed if the infection persists [1]; however, the CDC recommends removal of vascular catheters if there is evidence of phlebitis or purulence [8].



Figure 63.2 Sequential pictures taken over a 48 hour period show localized esophagostomy tube site infection managed with topical treatment (patient's head is to left). The tube had been removed several days prior due to owner request. The patient remained systemically well throughout.

Insertion Site Preparation for Peripheral Venous and Arterial Catheters

Venous access is required in most acutely or critically ill patients for parenteral administration of fluids and medications [28]. Many critically ill patients require multiple simultaneous constant rate infusions, which necessitates multiple venous catheter ports. Arterial catheters are indicated for direct blood pressure monitoring or frequent sampling of arterial blood. Because vascular access plays a dominant role in quality veterinary care, the proper care of venous and arterial catheters is a top priority.

Many catheter-related infections begin with catheter placement. Pathogen migration from the insertion site along the cutaneous catheter tract is the main cause of infection in peripheral intravenous catheters (PIVC) while contamination of the catheter hub is often the source of central venous catheter infections [8]. Systemic antimicrobials administered at the time of insertion appear to decrease catheter colonization, but the overall risk of catheter infection is not reduced and antimicrobials should not be administered solely for this purpose [28, 29].

Catheter Selection

Choosing the appropriate catheter is important in the prevention of catheter-associated infections. According to the CDC, catheters made of Teflon® or polyurethane are preferred over those made of polyvinyl chloride or polyethylene, to help reduce the incidence of infection [8]. Also, catheter diameter plays a role in the formation of thrombi and the smallest catheter diameter required to meet patient needs should be utilized [30–32].

Site Selection and Preparation

Site selection for placement of peripheral catheters will depend on the patient's vasculature, reason for hospitalization, mobility, and purpose for placement [32]. Distal extremity sites are ideally used first, saving more proximal sites for subsequent cannulation [33]. However, a PIVC placed over a joint or area of flexion (for example the accessory cephalic branch over the carpus in a dog), will be at higher risk of movement, occlusion, or dislodgement [25, 33]. Although saphenous catheters in dogs and cats may be expected to be at greater risk of soiling with urine and feces, this site does not appear to be associated with increased bacterial colonization [34, 35].

It is recommended that once a PIVC site has been selected, no more than two placement attempts should be made by a single person and no more than four attempts total [25]. This may not be practical in all cases but techniques such as transillumination and ultrasound (Chapter 9) could be useful in patients that are difficult to catheterize [31]. For example, shining a bright light through areas such as a dog's pinna may help to locate the artery while ultrasound guidance can be invaluable for difficult CVC placement if it is used by an operator with some experience in the technique.

The proposed insertion site should be completely clean and free of local trauma and infection. The region is clipped of fur as close to the skin as possible with a clean blade. Clipper blades must be well maintained to avoid iatrogenic trauma and bacterial contamination [36]. When possible, a margin of 1.5–2 inches (5 cm) of fur should be removed on all sides of the proposed puncture site [37].

After performing hand hygiene using either soap and water or an alcohol-based hand rub (see Chapter 62 Protocol 62.1), new gloves should be donned and the proposed insertion site scrubbed [8, 25].

Chlorhexidine, 70% isopropyl alcohol, or tincture of iodine or iodophor may be used to prepare the catheter insertion site [8]. The effectiveness of skin antisepsis is directly related to the length of time the antiseptic is allowed to act, and insufficient contact time has been cited as a reason for increased contamination rates in the fast-paced emergency department of human hospitals [38]. A 30-second scrub with 2% chlorhexidine may be the most effective at preventing catheter-related infections; a 10% povidone-iodine scrub for two minutes is also acceptable [8, 16, 26]. Potential benefits of chlorhexidine over iodine include prolonged efficacy against most nosocomial pathogens, less risk of neutralization by protein-rich biomaterials on the skin (including blood), and reduced risk of adverse effects following repeated exposure [27, 39].

While 70% isopropyl alcohol is an effective antimicrobial for the skin, it should not be used on sensitive or delicate skin, as it is drying and therefore can be damaging; sterile water can be used instead as a damp wipe/rinse in alternation with the scrub solution [15]. Additionally, alcohol inactivates povidone-iodine, so alternating these two antiseptics for site preparation is not recommended (Chapter 64). See Protocol 63.1 for instructions on vascular device insertion site preparation. Care should be taken to avoid contamination of multiple use products (for example multi-use containers of antiseptic solution or presoaked gauze sponges) [19].

Scrubbing technique has also been the focus of some human studies. The rationale for use of the classic outward circular motion is in removing bacteria from the top layer of skin and avoiding contamination [33], although some human sources consider a back-and-forth scrubbing technique to be more effective [25, 33, 39]. Whichever technique is used, the scrub should not be too vigorous, as damage to the skin increases the risk of infection.

Once the site has been cleaned, it should not be palpated again before catheter insertion [8, 25]. Gloves worn during

Protocol 63.1 Site Preparation for Peripheral Intravenous and Arterial Catheters

Items Required

- Clippers with clean blade
- Examination gloves
- Skin scrub
  - 2% chlorhexidine scrub (preferred)
  - 10% povidone-iodine scrub (acceptable)
- Skin rinse
  - 70% isopropyl alcohol
  - Sterile water (for sensitive or delicate skin, or for alternating with 10% povidone-iodine)

Procedure

1) Collect necessary supplies.
2) Clip fur with a radius of around 2 inches (5 cm) from proposed insertion site. Clip fur as close to the skin as possible.
3) Perform hand hygiene and don clean examination gloves.
4) Perform a gentle 30-second scrub with 2% chlorhexidine, or a gentle 1-minute scrub if using 10% povidone-iodine. Do not scrub so vigorously that skin is damaged.
5) Rinse the skin with 70% isopropyl alcohol if using chlorhexidine and the skin is not overly delicate or damaged. Rinse the skin with sterile water if using povidone-iodine or the skin is delicate or damaged.
6) Remove gloves, perform hand hygiene, and don clean gloves appropriate to the task prior to catheter insertion.

site preparation should be removed and hand hygiene again performed. For PIVC placement, clean gloves should be worn while the CDC recommends more strict aseptic technique be used with a minimum of a cap, mask, sterile gloves, and a small sterile fenestrated drape for peripheral arterial catheter placement [8]. Instruction on catheter placement can be found in Chapters 7 and 8.

Dressing

The role of dressings and securements in reducing catheter-associated complications should not be underestimated. In human medicine, suboptimal dressing integrity has been implicated in over 20% of catheter-associated complications while 21–71% of PIVC dressings have been found to be soiled, moist, loose, or inadequately secured at any single timepoint [40–42]. Creation of a physical barrier between the insertion site and the environment reduces microbial colonization while adequate stabilization reduces the risk of catheter dislodgement or kinking [8, 40]. Stabilization of the catheter within the vessel is also important as pressure and micro-motion of the catheter tip irritate the vessel wall, predisposing to infiltration, occlusion, and/or phlebitis [27]. The catheter insertion site may be covered with sterile gauze or a sterile, transparent, adhesive dressing [8]. In a human study on optimal catheter dressing, securement with sterile gauze and overlying tape was associated with fewer site complications and better dressing integrity [41]. However, direct application of nonsterile tape to the catheter insertion site led to increased phlebitis and infection rates. Partially used tape rolls quickly become colonized with bacteria by being exposed to multiple individuals and manipulation with ungloved hands. If nonsterile tape must be used directly at a catheter insertion site, discarding the outer layer may help to reduce the risk of infection. The site may then be dressed with clean gauze or cotton cast padding, as listed above in general recommendations.

The catheter must be protected so that the animal does not chew or lick the area. Many commercial products are available to limit animal access to vascular catheters, including Elizabethan collars, no-bite collars, catheter guards, various wrapping materials, and noxious-tasting, anti-lick sprays.

Care and Maintenance of Existing Peripheral Vascular Catheter Insertion Sites

Human guidelines recommend that PIVCs be assessed no less often than every four hours; every one to two hours for patients who are critically ill/sedated; hourly for neonatal/pediatric patients; and more often for patients receiving infusions of potentially irritating medications [25]. The dressing should be snug, clean, and dry. Loose or soiled dressings should be changed immediately, and if the dressing is soiled or wet to the level of the catheter insertion site, the clinician should be notified. During the dressing change, the site should be cleaned with 2% chlorhexidine scrub; allowed to air dry or dried with sterile gauze; and rewrapped with new, clean materials.

Topical antibiotic ointment or cream should not be applied to the catheter insertion site due to its tendency to promote fungal growth and encourage bacterial resistance [8].

The vessel above the site should not be hard or "ropey" on palpation, the skin above the site should not be excessively warm, there should be no edema proximal or distal to the catheter, and there should be no bandage strike-through or oozing at the site. If any of these abnormalities are noted, the clinician should be notified, and a dressing or catheter change considered.

There are no clear guidelines as to how often a vascular insertion site should be directly evaluated [8]. Standard veterinary practice is to unwrap and evaluate, and dress vascular catheter insertion sites with clean, new materials once daily, or more often if dressings are visibly soiled. Proper hand hygiene (washing or alcohol-based hand rub) should be performed prior to any contact with a catheter or its insertion site, and clean gloves should be worn every time the catheter is accessed (see Chapter 62 Protocol 62.1). Assessment of the catheter insertion site should be performed by palpation through the dressing to discern swelling or tenderness and by inspection if a transparent dressing is in use [8, 32]. CDC guidelines recommend that gauze and opaque dressings should not be removed if the patient has no clinical signs of infection or discomfort [8].

The catheter should be handled gently. Irritation of the vessel caused by catheter handling increases the risk for phlebitis. When accessing the catheter, be aware of the anatomy of the vessel and try to avoid bending the catheter against the direction of the vessel. Take care not to dislodge the catheter when accessing it. The catheter can be gently grasped by the hub with one hand (properly cleaned and gloved) while accessing it with the other hand so as not to dislodge the catheter or its connections during manipulations.

There should be no redness or swelling at the site. The site should be clean and dry with no oozing blood or fluid. Abnormalities should be brought to the clinician's attention.

It is recommended that a catheter be flushed after each use and at least once every six hours [32]. Flushing helps to reduce contact between incompatible drugs or fluids, limits the risk of thrombosis and phlebitis, and reduces fibrin accumulation in the internal lumen of the catheter, thereby reducing the risk of thrombosis and bacterial colonization [27].

It may be presumed that the turbulent flow created by pulsatile flushing would be more effective than the laminar flow created by continuous flushing for maintaining catheter patency. In vitro studies have shown that short boluses of flush solution interrupted by brief pauses may be more effective at removing solid deposits (e.g. fibrin, drug precipitate, intraluminal bacteria) compared with continuous low-flow techniques [19]. However, one human study found no difference in PIVC catheter patency time between the two techniques [43]. Flushing catheters with heparinized saline does not seem to be more effective at preventing catheter failure than using 0.9% saline in people [44, 45] or in dogs with short-term PIVCs [46]. Similarly, the addition of heparin to the flush solution has not been shown to increase arterial catheter patency or integrity of arterial waveforms compared with 0.9% saline [47].

Any vascular catheter that is no longer essential should be removed; however, the need for routine replacement of PIVCs is less clear. Earlier CDC guidelines recommended replacement of PIVCs in people every 24–48 hours because of higher complication rates associated with longer dwell times. Current data seem to refute this necessity and the latest CDC guidelines have relaxed the recommendation, advising that there is "no need to replace peripheral catheters more frequently than every 72–96 hours." [8] A Cochrane meta-analysis found "no evidence to support changing catheters every 72–96 hours" [48]. Patel et al. cite some disadvantages to routine catheter replacement, including patient discomfort and unnecessary cost, which is also relevant to veterinary practice [49]. An exception to this rule is if a catheter is placed during an emergency situation in which proper protocol may have been compromised. Such catheters should be replaced as soon as possible (within 48 hours). PIVC failure from infiltration and extravasation was almost 13% higher in PIVCs inserted in the emergency department compared with other departments in a systematic review [50].

Replacement of arterial catheters is only recommended when there is a clinical indication [8]. This recommendation has been challenged by data showing bacterial colonization of arterial catheters over time, and thus replacement after seven days has been suggested in people [51]. However, routine arterial catheter replacement is often limited by the number of arterial access sites and the risk of mechanical complications.

Complications of Indwelling Vascular Catheters

Despite all attempts at best practice, complications can arise and a PIVC complication rate of 21.4% was reported in a population of cats in intensive care in one study [52]. Localized infection at the site, bloodstream infection or sepsis, phlebitis, thrombosis, and edema are all potential consequences of intravascular catheters. The severity of these complications ranges from mild to life threatening. See Table 63.1 for some common complications of vascular catheters.

Table 63.1 Signs of and appropriate actions for phlebitis and thrombosis of vascular catheter insertion sites.

Condition	Signs	Action
Phlebitis	Redness around site	Inform clinician; consider catheter removal. If clinician elects to leave catheter in place, clean site with 2% chlorhexidine, allow to dry, and redress with new, clean materials
	Heat	
	Swelling	
	Pain on palpation of site or upon catheter flushing	
Thrombosis	Pain on palpation of site or upon catheter flushing	
	Vessel feels firm or "ropey"	
	Vessel appears distended without being occluded	
	Edema above or below site	
	Catheter becomes difficult to flush or aspirate	

Catheter, Line, or Dressing Damage or Dislodgement

In the event of catheter, line, or dressing complication, the catheter should be unwrapped carefully. If the catheter or insertion site is wet or dirty, the site should be cleaned with 2% chlorhexidine scrub and allowed to air dry before being rewrapped [8]. If air drying is impossible, the insertion site can be dried with sterile gauze prior to rewrapping. A wet site should not be rewrapped, as this encourages pathogen growth. Before rewrapping, make sure the catheter is at the proper angle in relation to the vessel.

Flush and attempt to aspirate blood back from the catheter while it is unwrapped to ascertain that the catheter is still in the vessel. If no blood appears after aspiration, place clean, gloved fingers just proximal to the point where the catheter tip should lie and inject saline into the catheter; if the catheter is still in the vessel one can sometimes feel a "jet" or "stream" of fluid as it flows up the vessel (particularly in a vein). If no intravascular fluid stream is palpable, watch this area closely while flushing. If the catheter tip is in the subcutaneous space, fluid will accumulate subcutaneously. Whenever flushing a catheter (wherever its tip may be), consider the size of the animal, the prescribed fluid rate, and the animal's ability to tolerate volume (and heparin, if flushing with heparinized saline). This is especially important when working with very small animals such as toy breeds, neonates, and small mammals such as ferrets.

If unable to ascertain whether an IV catheter is in the vessel, but the clinician is reluctant to remove the catheter, it should only be used to infuse balanced, isotonic fluids. Infuse slowly while observing closely for leaking or the subcutaneous accumulation of fluids. If fluids accumulate subcutaneously, the catheter must be removed. Catheters at highest risk of infiltration/extravasation are those in areas of flexion, inadequately secured and/or with vessel thrombosis or stenosis proximal to the tip, limiting blood flow [25]. To reduce the risk of infiltration/extravasation, it is recommended that two thirds of the catheter length should be within the vessel lumen.

The insertion site of an intact vascular catheter should be re-dressed with new materials, as previously mentioned, and protected from self-induced damage by using an Elizabethan collar or by wrapping more extensively and applying a commercial anti-lick product. Commercial plastic catheter guards are also available. Secure the fluid line in such a way as to minimize pulling on the catheter itself, as trauma to the vessel leads to phlebitis and thrombosis.

Edema Distal to the Catheter Insertion Site

Swelling distal to the catheter wrap usually indicates that the wrap is too tight. Swelling for this reason should be "cool" and should pit with pressure from a finger. In such cases, remove the bandage, rewrap the site more loosely with new bandage materials, and monitor. Evaluate the patient's other limbs and its skin turgor for evidence of generalized edema, which could indicate a compromise in the patient's vascular retention status due to low colloid osmotic pressure (see Chapter 58 for more details). If distal swelling persists despite a loosened bandage, inform the clinician, who may elect to replace the catheter.

If the swelling distal to the catheter is warm, the skin is reddened, the limb seems painful, or the swelling does not pit with pressure from a finger, it may be due to phlebitis or infection. The clinician should always be notified in such cases, and the catheter will likely need to be removed.

Swelling Extending Proximal to the Insertion Site

Swelling above the insertion site often indicates phlebitis or thrombosis of the vessel. The site should be unwrapped and evaluated. If no signs other than edema are present, consider the patient's overall hydration status as above.

However, if redness, heat, or pain is noted, phlebitis or thrombosis may be present. Signs are described in Table 63.1.

Phlebitis describes inflammation of a vessel wall and may be mechanical, chemical, or bacterial in origin [50, 53]. It may manifest as pain, erythema, swelling, purulent discharge, induration, pyrexia, and/or a palpable venous cord beyond the catheter tip [50]. It may remain localized to the insertion site or travel along the vessel [54]. If phlebitis is present, the catheter should ideally be removed. If vascular access is limited and the catheter is still needed, the catheter should be flushed while unwrapped to make sure there is no leakage at the site. If the site leaks when flushed the catheter must be removed. If there is no leakage at the site, the doctor should evaluate the patient and the site. If the phlebitis is minor and no signs of systemic inflammation are present, the clinician may elect to leave the catheter in place. At minimum, the site should be cleaned with 2% chlorhexidine scrub, air dried or dried with sterile gauze, and rewrapped. If phlebitis has been found, the wrapped site should be checked every 2 hours, unwrapped, and evaluated every 6–12 hours, and the patient must be monitored closely for signs of systemic inflammation. If infectious phlebitis is suspected, systemic antimicrobials are sometimes used. If there is no improvement in the phlebitis within one to three days, or if systemic inflammatory signs have evolved and are believed secondary to the catheter, the catheter must be removed. Blood cultures may be taken aseptically, one from the infected catheter, and at least one from a different site, to help confirm and diagnose a catheter-related infection (see Chapter 53 Protocol 53.9).

Vessel thrombosis occurs with thrombus formation at the tip of or along the outer length of the catheter. A thrombosed vessel can be painful and increases the chance of pulmonary thromboembolism. Thrombosis varies in severity and common sense must be used when deciding when to leave or remove a catheter. The location and degree of edema should be considered. For instance, swelling only near the insertion site might warrant less drastic action than swelling that involves a large portion of a limb, and more severe edema may warrant catheter removal more often than milder edema. The degree of hardness or "ropiness" of the vessel should also be considered. Is it mild or severe, and how much of the length of the vessel is affected? Once the vessel begins to harden, a catheter may be left in for a short period of time (usually 12–24 hours) before the condition worsens and the catheter needs to be pulled or ceases to function (leaking at site, no longer patent). If leaving a catheter in place, one may consider massaging and wrapping the limb to inhibit fluid retention.

Redness, Heat, Swelling, Pain, or Purulence at the Catheter Insertion Site

Taken together, redness, heat, swelling, and pain are signs of inflammation or infection although differentiating these etiologies can be difficult. Fever and/or local signs including phlebitis do not confirm the presence of infection while catheter-related infection can occur in their absence [35, 55]. Only 15–25% of catheters removed due to local signs proved infected upon quantitative catheter-tip culture in one human study [55]. Nonetheless, the CDC recommends removal of PIVCs if the patient develops signs of phlebitis, infection, or a malfunctioning catheter [8]. If the doctor decides to leave the catheter in place, the site and the patient should be monitored closely and cared for in the same manner as for phlebitis, detailed above. In dogs and cats, positive catheter-tip culture rates of 15.4–39.6% have been reported with isolates including *Enterobacter*, *Escherichia coli*, *Staphylococcus*, *Acinetobacter*, *Klebsiella*, and *Pseudomonas* species [35, 56]. Dextrose infusion and/or corticosteroid administration have been variably associated with increased risk of catheter-related infection [34, 56].

Oozing at the Catheter Insertion Site

Ascertain whether the tissue itself is oozing or whether fluids being placed into the catheter are leaking out at the site. This is done by unwrapping the catheter and flushing it with sterile saline. Watch the insertion site closely for leakage while flushing the catheter. Tissue oozing may indicate infection and should be treated as such (see above). If fluids administered are leaking, the catheter should be removed.

Complications Specific to Arterial Lines

The overall complication rate for arterial catheters in dogs and cats has been reported as 17.2–23.7% [57, 58]. The most commonly reported complications include catheter occlusion and inability to flush or aspirate blood, which could result from vasospasm or thrombosis [57–59]. Ischemic complications appear to be uncommon in dogs but may result from arterial occlusion in the absence of sufficient collateral circulation [59]. Particular care should be taken in the thoracic limb of the dog because the distal part of the median artery is the principal source of blood supply to the forepaw. Cats have poor collateral circulation in general. It has been recommended to avoid indwelling times of greater than 12 hours for dorsal metatarsal arterial catheters and 24 hours for coccygeal arterial catheters in cats to minimize the risk of arterial occlusion, as subsequent ischemic damage may necessitate tail or limb amputation.

Central Venous Catheters and Peripherally Inserted Central Catheters

Indications

Central lines may be used for the administration of intravenous fluids, medications, blood products, parenteral nutrition, vasoactive medications, hemodialysis, and hemodynamic monitoring [60]. They can generally be maintained for longer than PIVCs, allow repeated blood sampling, and are less prone to phlebitis [31].

Site Preparation and Insertion

Central venous catheters and peripherally inserted central catheter (PICC) lines travel to the vena cava, and because they can dwell much longer than peripheral catheters, the CDC recommends they be placed with a more stringent aseptic technique than is necessary for PIVCs [8]. During central venous catheter or PICC insertion, the operator should don a sterile gown, sterile gloves, cap, and mask, and a large sterile drape should be used to create a sterile field. Skin insertion site preparation is the same as for peripheral venous and arterial catheters, although a generous margin of fur should be clipped. Increased infection rates have been variably associated with the number of central catheter lumens [61, 62]. Guidelines recommend using a catheter with the minimum number of ports or lumens essential for patient management [8, 61].

Ultrasound guidance is frequently used in people to avoid numerous insertion attempts and can be very useful in small animal patients where catheter insertion is proving difficult.

Central venous catheters are sutured to the surrounding skin to prevent accidental dislodgement. While suture is a secure way to keep catheters in place, it does increase the potential for localized skin infections and can be uncomfortable [61]. Attention should be paid to ensuring sutures are not too tight and do not cause kinking or occlusion of the catheter.

Dressing

The central venous catheter insertion site should be covered with either sterile gauze or a sterile, transparent dressing (semipermeable to avoid moisture trapping) [8, 60]. The use of antibacterial preparations on the site after insertion is not routinely recommended because of their potential to promote fungal infections and antimicrobial resistance [8, 60, 63]. Hemodialysis catheters may be an exception to this rule.

Care and Maintenance

A central catheter should be maintained in much the same manner as a peripheral catheter. Since these catheters are accessed frequently and are expected to dwell for extended periods, good hand hygiene and donning of clean gloves when handling these catheters are imperative. The catheter should be unwrapped and the site evaluated at least every 48 hours if wrapped in gauze, or at least every seven days if covered with a transparent, adhesive dressing [8].

Generally, sites are cleaned every time they are directly evaluated with a 2% chlorhexidine scrub and allowed to air dry or are dried with sterile gauze, then dressed again with new materials. Direct site evaluation, cleansing, and redressing should also be done any time the area is soiled or the site becomes exposed.

The access ports of the central venous catheter are a potential source of contamination and should be scrubbed with an antiseptic solution, such as 2% chlorhexidine in isopropyl alcohol or 70% isopropyl alcohol prior to use [61]. Use of 10% povidone-iodine is effective but slow to dry so may not be practical [64]. All access ports should be kept covered with caps [8, 61, 65]. No advantage of heparinized saline over normal saline was demonstrated in maintaining catheter patency in a population of healthy dogs with central venous catheters [66].

Catheters should be removed as soon as they are no longer necessary but there are no standard recommendations regarding timelines [32, 60, 61]. Longer dwell time and administration of irritant medications have been associated with increased risk of complications in dogs and cats [67]. Conversely, a study of hemodialysis catheters in dogs found no difference in the prevalence of infection according to age, sex, reason for hemodialysis, catheter complications, duration of catheterization, or outcome [68]. Routine replacement of catheters in people has actually been shown to increase the incidence of infection at insertion sites and is not recommended [60].

Catheter insertion sites should be assessed for signs of infection or migration daily [8]. The site should be checked for any redness, swelling, leakage of administered fluids, or oozing. The area around the insertion site should be gently palpated for any signs of pain or hardening of the vessel. The area should also be checked for excessive warmth. If any of these signs are present, the clinician should be notified and the situation addressed.

Complications

Some risks related to central venous catheter insertion include infection, accidental arterial puncture, vein laceration, thrombosis, pneumomediastinum, and catheter misplacement [32]. The most common complications reported in dogs and cats include mechanical obstruction, skin irritation, malposition, and inflammation [67]. The risk of colonization increases with catheter duration [61]. However, it is recommended that a catheter should not be

removed based solely on the presence of fever [60]. Other noninfectious causes of fever should be considered, and other evidence of infection should be sought before replacing an existing catheter [8].

If a malfunctioning catheter must be replaced, then exchange over a guidewire is an option [61]. However, in a systematic review of catheter replacements in people, catheters exchanged over a guidewire were found to have a higher rate of colonization, exit-site infection, and catheter-related bacteremia than those placed at a new site [69]. Therefore, in the setting of infection, replacement at a new site is preferred. There are small, nonrandomized studies suggesting that, in difficult circumstances where obtaining access is difficult or may lead to further long-term complications (such as in hemodialysis patients), exchange over a guidewire may be considered [61].

As with PIVCs, having protocols in place for decision making regarding management of central venous catheters has been shown to significantly decrease the rate of infection [70].

Intraosseous Catheter Insertion Sites

The intraosseous (IO) catheter is a life-saving device, providing access to a noncollapsible venous complex in emergency situations where peripheral access cannot be achieved [71]. It is particularly useful for small, hypovolemic, and neonatal patients where PIVC placement may be very difficult and may be more efficient than a venous cutdown [72]. IO catheters can be used for infusion of drugs, fluids, and blood products [73]. IO access for drug administration during cardiopulmonary resusciation when IV access cannot be attained is recommended over tracheal administration of epinephrine in people [74, 75]. IO catheters can also be used for blood sampling although certain parameters such as potassium and glucose may not be accurate and samples must be drawn prior to drug or fluid administration [76, 77].

Insertion

IO catheter insertion site preparation is the same as for peripheral venous and arterial catheters, though sterile gloves should be worn. The femur and humerus are readily accessible and allowed the highest IO flow rates in dogs in one study [78]. Once placed, the catheter should be secured by wrapping a tape butterfly around the hub and suturing the butterfly to the skin or periosteum. Some IO catheters come with their own permanent butterflies. If possible, the secured catheter should be protected by an outer bandage. For more detail on placing and securing an IO catheter, see Chapter 7.

Maintenance

IO catheters usually dwell for only a short period of time (three to four hours), until direct venous access is achieved. If left in for an extended period, they should be maintained in much the same manner as an IV catheter. Hand hygiene should be performed, and clean examination gloves worn every time the catheter is handled. Standard practice is to flush the IO catheter with a small volume of heparinized or plain saline every six hours. Rewrapping an IO catheter is not always feasible. There may be cases in which the patient is too small, too young, or too critical to make routine rewrapping possible. Once again, common sense must be used to decide what is best for the patient. An IO catheter should be left in place for no longer than 48 hours [79].

Complications of Intraosseous Catheters

As with all indwelling devices, IO catheters carry some risks including extravasation, air embolism, skin abscessation, and osteomyelitis. However, infection is rare with adherence to aseptic placement and care of the site and catheter [71, 79].

Fluid Extravasation

Extravasation of fluid may be the most common complication of IO catheters. This usually occurs when the needle is misplaced upon insertion, either because the catheter does not penetrate fully through the cortex and into the medullary cavity or because it has passed out of the bone's medulla and through the far cortex, into the muscle. Fluid extravasation can also occur when the patient moves excessively after proper placement of the IO catheter. When hypertonic fluids or caustic medications extravasate, muscle necrosis can occur.

There are a few ways to confirm an IO catheter's position. When the IO catheter is properly positioned, it should flush easily with little resistance. Radiographic imaging shows the catheter's placement as well. If administered drugs or fluids leak at the site, one should suspect the catheter is not properly seated, and it should be removed. Replacing the IO catheter in the same site is not recommended, as fluid may escape from the defect left by the first catheter. For the same reason, it is not recommended to make more than one attempt at the same site when placing the IO catheter.

Compartment Syndrome

Compartment syndrome occurs when fluids extravasate from an IO site for an extended period of time. When a large volume of fluid leaks into the muscle, a pocket is formed within the muscle, which can cause muscle necrosis. It is recommended that there be no repeated attempts in the same bone if the first IO catheterization fails. Another site should be chosen, as fluid will sometimes leak

from the hole made on the first attempt. The best way to prevent compartment syndrome is to monitor the catheter site and associated limb closely, and if any swelling appears, remove the catheter immediately.

Infection and Osteomyelitis

Localized site infection or osteomyelitis occurs rarely, particularly if the IO catheter is removed after only a few hours [72]. Infection most often occurs when proper aseptic technique was not followed during insertion. The IO catheter should be removed if signs of infection are present; systemic antimicrobials are usually indicated.

Thoracostomy Tube Insertion Site Maintenance

Great care must be taken to keep the insertion site of a thoracostomy tube healthy. Having to remove a tube due to infection, or having the tube migrate out of the pleural space due to breakdown of the tissue at the site due to infection, can greatly compromise the patient. Care must also be taken not to introduce bacteria into the pleural space.

Prior to contacting the thoracostomy tube or insertion site, hands should be cleaned using good hand hygiene practice as described in Chapter 62. Clean examination gloves should be worn any time the site or tube system is handled. The insertion site should be kept clean and dressed. Although specific, evidence-based recommendations are unavailable, the site should probably be unwrapped, evaluated, and redressed at least as often as a vascular catheter insertion site, so at least every 24 hours if no obvious complications are present. The site should be covered with a sterile, nonadherent pad before bandaging. Self-adherent, transparent bandages can be used in place of the nonadherent pad and are preferable in smaller animals where a bulky wrap would be uncomfortable. A body stockinette can be placed to further cover the site, and the tube itself can then be fastened to the stockinette with tape and suture, or plastic clamps.

It can be very challenging to keep a thoracostomy tube site properly bandaged. Large, deep-chested dogs are especially difficult because of their body conformation. It may help to crisscross the bandage across the dog's chest. Howver, when a patient is mobile, all attempts at keeping the site covered sometimes fail. One solution is to cover the site with a sterile, self-adherent bandage. A clean T-shirt is then put on the dog and tied to a close on top of the dog's back. A hole is cut in the shirt and the thoracostomy tube is passed through the hole and clamped to the T-shirt. This method works well to keep the site covered and helps to stabilize the tube from excessive movement. This would not be suitable for a dog attached to a continuous suction device, in which case a more stabilizing wrap is needed.

Any animal with a thoracostomy tube requires constant monitoring and an Elizabethan collar. No matter how closely watched, some animals still manage to open or remove their tubes, and sometimes tubes can migrate out of the thorax and into the subcutaneous space just through the movement of the animal's body. Thus, respiratory rate and effort should be watched closely and the chest auscultated frequently (every two to four hours). Care should be taken to ensure that the tube is not accidentally pulled upon, as this can cause inflammation of the pleura and the insertion site.

Nephrostomy Tube Insertion Site Maintenance

The collecting tip of a nephrostomy tube is placed within the renal pelvis and the tube travels through the body wall with its egress end outside the skin. The draining end of the tube is outside of the animal, attached to a sterile, closed urinary collection system. Keeping the insertion site clean helps to decrease pathogen introduction into the retroperitoneal space and the kidney. The bandaged tube should be evaluated every two hours, checking for any wetness, and making sure that the bandage and tube are still wrapped in the proper position. At the author's institution, the site is unwrapped and evaluated every 8–12 hours.

Whereas most indwelling devices are situated in a fairly large space such as the pleural space, or a lumen as with a central line or a feeding tube, the nephrostomy tube is placed inside a tiny space within a delicate organ. Therefore, it is vital that there be no tugging or pulling of the tube by handlers or the animal. The tubes are usually long enough to allow them to be curled several times before being wrapped closely to the body. A "butterfly" can be made of tape wrapped around the tube and then sutured to a stockinette, which is worn by the patient, or, if the surgeon chooses, sutured directly to the patient. Most cats and small dogs seem to prefer the stockinette to a bandage wrap. A sterile, transparent, self-adherent bandage works well over the site. The stockinette provides a clean covering and takes the brunt of any pulling that may occur. Enough slack must be left in the tube so that when the stockinette is pulled, it does not affect the site. If a bandage wrap is used, the excess tubing can be curled and incorporated in the wrap so any tugging will not disturb the tube site. An Elizabethan collar should be used, and the animal watched closely.

In addition to monitoring the site for signs of infection, the nephrostomy tube site should be watched for leakage. While this could imply infection, it could also indicate retroperitoneal urine leakage. If effusion is noted from the insertion site, a doctor should be alerted immediately so that uroretroperitoneum can be ruled out.

Another complication of the nephrostomy tube is the tube becoming clogged with mineral material or debris. Once approved by the clinician, gentle aspiration and retropulsion can be attempted. If this is not successful, imaging may be necessary to determine the cause of the occlusion, and tube removal may be necessary.

Cystostomy Insertion Site Maintenance

A cystostomy tube allows drainage of urine in dogs and cats with urinary outflow obstruction or dysfunction. An example of a cystostomy tube candidate would be a cat with neurologic damage secondary to sacrococcygeal ("tail-pull") injury that may regain bladder function within days to weeks [80]. The tip of a Foley catheter, mushroom-tipped catheter, or low-profile gastrostomy tube is placed within the urinary bladder and the tube exits the abdominal wall via a stab incision. Low-profile systems usually have an exterior button or flange that sits flush with the skin. When a long tube is used, it is secured to the skin with a finger-trap suture and may be connected to a sterile, closed-collection system for continuous urine drainage. The tube can be capped and urine emptied intermittently (three to four times daily), which also allows observation for normal urination. Animals may be discharged with the tube in place and owners taught how to use it at home. Cystostomy tubes may even be maintained for several years in some cases. Beck et al. reported an overall complication rate of 49% [81]. Major complications included inadvertent removal of the tube or displacement from the bladder (which could lead to uroperitoneum), patient chewing of the tube, breakage of the tip of the catheter during removal, and fistula formation following tube removal. Other complications included irritation or inflammation around the tube exit site, urine leakage around the tube, bandage complications, and breakage of the suture securing the tube to the skin. Urinary tract infections are also common and biofilm formation may complicate this. However, prophylactic antimicrobials should not be administered.

It has been recommended that cystostomy tubes should be left in place for a minimum of 14 days to allow a stoma to form between the urinary bladder and the body wall [82]. The risk of dislodgement can be reduced by keeping the tube properly secured and careful handling to avoid unnecessary traction. Low-profile tubes have an advantage here, as they do not protrude from the body.

The stoma site should be examined and cleaned daily and kept covered with a sterile dressing. Clean gloves should be worn when handling the tube. Long tubing can be secured using a light bandage, stockinette, or T-shirt.

Ascending infection could lead to pyelonephritis or peritonitis and animals should be monitored closely for fever, abdominal pain, changes in urine character and inflammation, swelling, or discharge from the stoma site. Complete blood count, serum biochemistry, abdominal ultrasound, and urine culture will help inform management of such complications. Following removal of the tube, the stoma is expected to granulate closed in three to five days.

Surgical Drain Insertion Site Maintenance

Closed-Suction Drain Insertion Sites

Jackson-Pratt and other closed-suction drains are typically placed in the abdomen to drain peritoneal effusion postoperatively. They can also be used to drain effusion from the subcutaneous space postoperatively in cases where excessive fluid production is expected. These systems consist of a surgical drainage tube placed inside the body, which attaches to thin rubber tubing that passes through the body wall to the outside and is then attached to a rubber squeeze bulb (in the case of Jackson-Pratt drains) or other suction device. When squeezed and then closed, the Jackson-Pratt bulb creates suction, which draws fluid into the bulb where it is collected (Chapter 41).

Closed-suction drain insertion sites should be kept clean, dry, and covered. The site should be covered with a nonadherent pad or a sterile, self-adherent bandage and then wrapped. The bandaged site should be checked every two hours, and the bandage should be dry. At the author's institution, the site is unwrapped and evaluated every 8–12 hours.

Insertion sites into the cranial abdomen are fairly easy to keep covered. Insertion sites into the inguinal area are more difficult. The variations in the size and shape of the many breeds of dog make it necessary to be creative with the bandage material. It is often necessary to rewrap these sites often, as bandages seem to slide off of them easily. The bandage may sometimes become soiled with urine or feces, which can wick up toward the insertion site. If urine or feces would not come into contact with the insertion site without the wrap acting as a wick, it may be better to forgo the wrap and use only a sterile, self-adherent bandage.

The tubing of the Jackson-Pratt drain is long and can be curled and then wrapped into the bandage material, with slack enough between the site and the wrapped portion of tubing that the weight of the bulb will not pull on the site. The bulb itself can also be taped or otherwise affixed to the bandage. In cases where the site is only covered with a sterile, self-adherent bandage, a clean T-shirt can be worn by the patient, brought to the top of the back, and tied. The drain is passed through a hole cut in the shirt and attached to the shirt with a plastic clamp. A stockinette can be used in place of the T-shirt in smaller animals.

The drain insertion site should be monitored for infection, and treated accordingly, as stated earlier in this chapter for other sites. When a site is inflamed, it may allow effusion to leak out at the site. If the drain is to be left in,

some sterile gauze sponges should be added to the wrap to absorb the fluid. In cases where it is not physically possible to keep a bandage on, care should be taken to keep the site and area around the site as clean and dry as possible. Fluid should be gently and aseptically wiped from the site, taking care to wipe in motions away from the site and not toward it. A 2% chlorhexidine scrub should be used to clean the area. The area should be allowed to air dry if possible or be dried with sterile gauze. If the animal is recumbent, the limbs can be propped apart with clean towels to aid in keeping the area dry. Also, if the animal is recumbent, sterile gauze can be placed around the site without an outer wrap. Good hand hygiene as described in Chapter 62 should be employed any time the site or the drain itself is handled, and clean examination gloves worn.

Care should be taken to ensure that the tubing does not become kinked, preventing suction. The bulb should be kept empty of air and fluid to ensure adequate suction. Fluid should be emptied from the drain as often as needed, and fluid volumes recorded in the patient treatment sheet.

Penrose Drain Insertion Sites

The Penrose drain is a passive drain. It consists of a simple rubber tube placed in a wound or incision to drain fluid from the site out of the animal. This helps to prevent infection, and also aids in patient comfort.

The Penrose drain site is a messy site, with fluid draining out freely and being caught only by any bandage material. If present, the external bandage should be checked every two hours for strikethrough. When strikethrough is present the bandage should be removed and the site examined, cleaned with 2% chlorhexidine, dried with sterile gauze, and redressed. At the author's institution, if there is no strikethrough present, the site is examined, cleaned, and rebandaged every eight hours.

Care should be taken to prevent the animal licking or chewing the drain or site. This is best achieved with an Elizabethan collar.

Postoperative Wound Infusion Catheter Maintenance

A wound infusion catheter (also known as a "soaker" catheter) delivers local analgesia to a surgical site via intermittent or continuous infusion of local anesthetic agents. Commercial wound infusion catheters are available or can be fashioned from red rubber or polyurethane catheters. They are pliable and have multiple fenestrations to allow diffusion of the infused solution along the entire wound bed [83]. They are usually capped for intermittent infusion or can be attached to an intravenous administration set for continuous infusion. They are most commonly used in the authors' institution to supplement analgesia following limb amputation, large mass removal, and thoracotomy. The catheter tip is usually located in the deepest part of the incision and care must be taken to ensure all fenestrations are below the skin surface.

Commercial catheters may have a butterfly attachment that can be sutured to the skin, or the tube may be secured using a finger-trap suture. Potential complications include lidocaine or bupivacaine toxicosis, infection, hematoma formation, and nerve damage secondary to intraneural infusion [84]. Accidental intravascular injection is also possible and could lead to serious adverse consequences. Clear labeling of catheters and medications is strongly recommended to avoid this. Catheters should be kept covered with a sterile dressing and handled using clean gloves. Wound infusion catheters are generally removed after one to three days, or sooner if complications are encountered.

Feeding Tube Insertion Sites

Nasoesophageal and Nasogastric Tube Insertion Sites

Nasoesophageal (NE) tubes enter the nares and terminate in the esophagus; nasogastric (NG) tubes terminate in the stomach. The tube is generally secured with a sutured or stapled tape "butterfly" or finger-trap suture just caudal to the nostril and then again into the cheek of the animal. The nostril housing the tube should be monitored for signs of inflammation or infection such as pain or exudate, and treated accordingly.

There is risk of the tube leaving the esophagus and entering the trachea; while such migration is rare, this complication has dire consequences. At placement, the tube should be measured, and a note made in the record as to how much tubing is external. Every time the tube is used, external placement should be checked to ensure the tube has not migrated. The sutures should all be in place and the tube seated securely with no slippage visible. The tube should be aspirated and negative pressure achieved if the tube is in the esophagus; negative pressure or gastric fluid if the tube is in the stomach. If gas is aspirated, a radiograph should be taken to check tube placement before anything is instilled into the tube. Animals with NE or NG tubes should always wear an Elizabethan collar.

Esophagostomy, Percutaneous, Endoscopically-Placed Gastric and Jejunostomy Tube Insertion Sites

The insertion sites of these devices should be evaluated at least once daily. The site should be covered with a sterile, non-adherent pad or a sterile, self-adherent bandage, and wrapped with clean, soft bandage material. The animal should always wear an Elizabethan collar and the wrap

should be substantial enough to protect the site and tube in the event that the animal escapes or evades the collar. Esophagostomy tubes are especially vulnerable to clawing.

In addition to monitoring for infection, it is imperative that the device's proper placement is evaluated, as much as can be determined from the insertion site. Feeding tubes can migrate due to breakdown of the site, or tugging or pulling by the animal. If the tube is displaced, food may end up in the airways, the peritoneal cavity, or the subcutaneous space. It is impossible to ascertain whether the device is placed properly just from evaluation of the insertion site, but a healthy insertion site supports that assumption. All feeding tubes should be checked for negative pressure or reflux to ensure proper placement prior to feeding through the tube. The tube should be marked in some way so that tube migration can be assessed. The site should also be checked for leakage of food or other fluid. If any food or fluid leaks from the site, the feeding should be stopped immediately and a clinician notified.

These types of feeding tubes generally remain functional for extended periods of time (up to months) and can be maintained and used by the owner at home if they are properly educated in the care of the tube. Because of the length of time that the tube remains in the patient, a moist dermatitis sometimes develops. If the tube and sutures are all securely in place it may help to leave the site unwrapped for 15–20 minutes after each cleaning and drying. The animal should be held during this time to ensure that it does not remove the tube.

In the author's experience, minor infections of enteral feeding tube insertion sites can often be successfully treated by frequent (two to three times a day) cleaning and rewrapping. Systemic antimicrobial therapy may be considered. These patients should be closely monitored by a doctor.

An infected, soiled, or exposed site should be cleaned with a 2% chlorhexidine solution, allowed to air dry or dried with sterile gauze, and rewrapped with new, clean material. If the tissue at the site is oozing, sterile gauze can be placed over the site to absorb the fluid. It is important to keep the site as dry as possible. If the site is red and inflamed, but not oozing fluid or overly moist, povidone iodine may be helpful in eliminating the infection. In general, it is not recommended to use iodine or triple antibiotic ointment on these sites, as the excess moisture promotes the growth of certain pathogens.

Epidural Catheter Insertion Sites

Epidural catheters are often placed in cases in which pain management is expected to be difficult (Chapter 49). These catheters must be placed aseptically and maintained with stringent aseptic technique. After placement, povidone-iodine can be applied to the area around the site with sterile iodine-soaked swabs. Sterile gauze sponges can be placed under the injection cap, both to aid in keeping the catheter laying evenly along the animal's back, and to absorb any leaking fluid. The site should be covered with a clear, sterile, self-adherent bandage. It is not recommended to unwrap an epidural catheter, so the clear bandage allows for visual site evaluation every few hours. It is not unusual to note clear fluid leaking from the site. This is one reason to place sterile gauze sponges beneath the epidural catheter. If any colored fluid, swelling, or other signs of inflammation are seen, a doctor should evaluate the site immediately. If any sign of infection is seen, the catheter should be removed.

To avoid introducing bacteria into the epidural space, the injection cap to the catheter should be scrubbed with a 2% solution of chlorhexidine and a gauze sponge soaked in 2% chlorhexidine placed over it for a full three minutes prior to being punctured with a sterile needle. Proper hand hygiene should be followed, as stated above, and sterile gloves must be worn when using this device.

Extreme care should be taken to not displace the catheter. Although rare, epidural hematomas, epidural abscesses, and arachnoiditis are possible complications. There is a limit to the volume that can be injected into an epidural catheter. The doctor's orders should be specific as to that volume.

Summary

The importance of properly caring for the insertion sites of indwelling devices cannot be overstated. Most insertion sites will remain healthy and the device functional for as long as needed if well maintained. Paying close attention to the site can greatly reduce the incidence of infection and other complications.

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64

Antiseptics, Disinfectants, and Sterilization

Samantha Jones, Krystle Reagan, and Nicole Saunders

Proper hygiene within a veterinary clinic is critical to decreasing the risk of hospital-acquired infections (HAIs). Understanding the principles of proper cleaning, antisepsis, disinfection, and sterilization is essential for the development of hygiene protocols that are the pillar of infection control programs in veterinary hospitals. In a five-year period, over 80% of veterinary teaching hospitals reported the outbreak of an HAI. This finding underscores the need for infection control programs and impeccable hygiene to improve patient health.

Hygiene is a concept that encompasses several different terms. Cleaning refers to the mechanical process of removing organic material with soap and water and is an indispensable first step prior to antisepsis, disinfection, or sterilization. When these techniques are used properly, they can decrease the risk of HAI; however, when performed incorrectly or inconsistently, microorganisms can persist on surfaces or in the environment and can pose a threat to patient safety. In this chapter, we discuss the most common methods of achieving antisepsis, disinfection, and sterilization within a veterinary hospital.

Antiseptics and disinfectants are antimicrobial agents that act to decrease the microbial burden, while sterilization completely eradicates microorganisms. Antiseptics are applied to the surface of living tissue to decrease the microbial burden, while disinfectants are applied to inanimate objects to perform the same action. Antiseptics and disinfectants typically have a broad range of biologic activity depending on the chemical structure, and some will have activity against bacterial spores. There is a wide variety of antiseptics and disinfectants available, and it is critical that instructions regarding dilution and contact time are adhered to for maximal effect.

Sterilization refers to the process by which there is the complete eradication of microorganisms, and typically refers to inanimate objects such as surgical instruments, surgical implants, or other invasive devices. Several methods of sterilization are used in the veterinary setting, including gas, pressure, and chemical sterilization.

This chapter focuses on the properties of individual antiseptics, disinfectants, and sterilization procedures. Proper selection and use of these chemicals and methods are crucial to aid in the prevention of HAIs, whether the patient is undergoing a surgical procedure, has an indwelling device, is being mechanically ventilated, or is simply hospitalized. See Chapter 62 for an expanded discussion of other precautions against HAI.

Antiseptics

An antiseptic is a chemical agent or substance that kills or prevents the growth of microorganisms on living tissue through topical application. A variety of different antiseptics are available for use in veterinary medicine. Each has advantages and disadvantages, and none is effective against all organisms (Table 64.1). Commonly used antiseptics include alcohol, iodophors, and chlorhexidine.

Many antiseptics are available as aqueous solutions; some are also available as scrubs, which are detergents or soaps. Detergents and soaps are irritating and cytotoxic, and thus should only be used on intact skin, and should never contact mucous membranes or subcutaneous tissues. Product labels must always be consulted for proper mixing and diluting instructions. Over- or underdiluting the product can result in poor efficacy or exacerbation of skin irritation and toxicity, respectively.

Antiseptics are often used to clean the hands of personnel or to decrease the microbial burden on a region of a patient. It is important to ensure antiseptic techniques are practiced during the placement and management of intravascular devices, urinary catheters, and feeding tubes, and

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Table 64.1 Commonly used antiseptics.

Antiseptic	Spectrum of activity	Ineffective activity	Residual activity	Decreased effectiveness
Alcohol	Bacteria, nonenveloped viruses, some fungi, mycobacteria	Variable enveloped viruses, no spores	None	Organic material
Chlorhexidine (biguanide)	Gram-positive bacteria, some Gram-negative bacteria, nonenveloped viruses, fungi, mold, yeast	Variable Gram-negative bacteria, variable enveloped viruses, no mycobacteria, no spores	4–6 hours, occasionally up to 2 days	Organic material
Povidone-iodine (iodophores)	Bacteria, enveloped viruses, fungi, yeast, mycobacteria	Variable enveloped viruses, no spores	Up to 4–6 hours	Organic material, alcohol

during any sterile procedure. Contamination from sources within the hospital including surfaces, equipment, personnel, and the patient itself can lead to an HAI. Proper use of antiseptics to personnel and the patient can help to reduce these risks.

Alcohol

Alcohol is a commonly used product in the veterinary setting as both an antiseptic and a disinfectant. The most effective forms are ethyl and 70% isopropyl alcohol. Within two minutes 90% kill is expected, although effective kill of many bacteria occurs within 10 seconds of sustained exposure. Its main mechanism is denaturing proteins and disrupting metabolic function. This causes precipitation of cell proteins and cell lysis. Alcohol may also help to enhance the effectiveness of other antiseptics by contributing to fat solubilization. It has variable effectiveness against bacterial spores, and it is not effective in inactivating rabies virus. However, it is effective against most Gram-positive and -negative bacteria, some fungi, and some enveloped viruses. Alcohol rapidly evaporates, so it leaves no residual effects and extended exposure is needed for maximum efficacy. A quick swipe of alcohol on an animal's skin prior to venipuncture is minimally effective in antisepsis. It is important to note the flammable nature of alcohol and to exercise extreme caution when using certain equipment like cautery and lasers.

Chlorhexidine

When attempting antiseptic skin preparation for sterile procedures, such as catheter and feeding tube placements, a 2% chlorhexidine solution is the product of choice over 70% alcohol or povidone-iodine. Chlorhexidine is the most commonly used bisguanide antiseptic in veterinary medicine. Bisguanides work by disrupting the cell wall by binding with proteins within the wall. This, in turn, causes leakage and precipitation of the cell contents. There are both scrub and aqueous solution forms of this product. Bisguanides have a lower incidence of causing allergic reactions and skin irritation, and are less cytotoxic than iodine-based solutions; however, prolonged skin contact with concentrations of greater than 0.5% may be harmful and may impair fibroblast activity.

Chlorhexidine should never come in contact with eye tissue as it is toxic to eyes and should only be used in ears when the tympanic membrane is intact. Chlorhexidine is considered effective against most Gram-positive bacteria, some Gram-negative bacteria, including *Escherichia coli* and *Pseudomonas aeruginosa*, mold, yeast, and many viruses. It has variable activity against many Gram-negative bacteria and enveloped viruses and is ineffective against rabies virus. Chlorhexidine is ineffective against bacterial spores and mycobacteria. When using chlorhexidine as part of aseptic preparation, it is suggested that there is an approximately 90% kill within 30 seconds of contact time, and although a preparation time of five to seven minutes is recommended, it is possible that two 30 second preparations are sufficient. Owing to the keratin-binding properties of chlorhexidine, contact time may be a lesser issue than with other antiseptics. It has immediate antimicrobial effects and lasting residual effects when used as a wound lavage, and is generally not negatively impacted by the presence of alcohol, soaps, or lavage fluids. The effectiveness of chlorhexidine may be interfered with by a moderate amount of organic material present. As chlorhexidine's activity is pH-dependent, it is more soluble and more effective at a lower pH.

Iodophors

Povidone-iodine is the most commonly used iodophor in veterinary medicine and is usually used as a scrub or solution commonly known as Betadine® (Purdue Frederick Company, Stamford, CT), which is a 10% povidone-iodine formulation. Cadexomer iodine is available as an ointment or dressing. Iodophors contain polymerized iodine so the free iodine is slowly released, with the goal of minimizing tissue irritation and enhancing the delivery of iodine to the

tissues. The iodine penetrates the cell wall where it causes oxidation of the intracellular contents and replaces microbial contents with iodine. It has a broad spectrum of activity against Gram-positive and -negative bacteria, fungi, enveloped viruses, yeast, and mycobacteria. It is considered ineffective against bacterial spores and nonenveloped viruses. Approximately 90% kill is expected within 30 seconds of contact time although a minimum of two minutes of contact time is advised and a five- to seven-minute scrub time is recommended. Povidone-iodine has a very rapid onset of activity and up to four to six hours of residual activity. It is inactivated by the presence of blood, plasma, and organic material, so the presence of any of these substances rapidly diminishes activity. Alternating povidone-iodine with alcohol is not advised and can decrease the effectiveness of the iodine by decreasing the contact time of the povidone-iodine with the skin. The presence of alcohol and other detergents decreases the effectiveness of povidone-iodine. Alcohol-based iodophors, however, have been shown to decrease central venous catheter colonization when compared with aqueous solutions.

The stock solution of povidone-iodine is usually 10%. Dilution of one part stock solution and nine parts water creates the commonly used 1% solution (Protocol 64.1). Either water or an electrolyte solution can be used to dilute the stock solution. Dilution both increases the bactericidal activity and decreases the cytotoxicity, although even at concentrations of 0.5% povidone-iodine can impair tissue fibroblast proliferation. At concentrations greater than 0.05% povidone-iodine can impair lymphocyte blastogenesis and granulocyte and monocyte viability and migration. Given that the most commonly used solution in practice is 1%, it should be kept in mind that at this concentration there may be adverse effects. These effects do not appear to be clinically relevant in most patients, but it may predispose some to delayed wound healing in cases with underlying conditions or chronic wounds requiring repeated contact. Iodophors are more irritating to the skin than bisguanides and can cause contact dermatitis and pruritus at concentrations as low as 0.1%.

Tincture of iodine is a commercially available solution of 2% iodine in 50% ethyl alcohol and is intended for use on intact skin only.

Protocol 64.1 Formulating a 1% Povidone-Iodine Solution

Items Required

- Povidone-iodine stock solution (stock povidone-iodine solution is 10%)
- Water or electrolyte solution

Procedure

- To make 250 ml total volume, add 25 ml of 10% solution to 225 ml of water.
- To make 500 ml total volume, add 50 ml of 10% solution to 450 ml of water.
- Solution can be made using water or an electrolyte solution.

Disinfectants

Hospital surface disinfection, or removal of most of the microbes from a surface, is paramount in preventing the spread of infectious disease, as many bacteria, fungi, and viruses can persist in the hospital environment. Nonenveloped viruses, such as parvovirus, and microorganisms that sporulate can pose a particular challenge, and surface disinfection products must be applied properly to be effective. Disinfectants are chemicals that reduce the microorganisms present on inanimate objects but do not completely eradicate them. Many of the antiseptic agents discussed above can be used as disinfectants, but the inverse is not necessarily true. Disinfectants are often cytotoxic to living tissue, therefore cannot be used as antiseptics. A wide variety of chemical disinfectants are available, and each has a different spectrum of action against pathogens. Factors that determine the spectrum of action include the chemical makeup, concentration, and the surface to which the disinfectant is applied (i.e. presence of organic material or biofilms). It is critical that manufacturers' recommendations are used when applying disinfectants including dilution and surface contact time.

Disinfectants should be applied to surfaces after patients have been in contact with them, including exam room surfaces and cages. They should also be used on any medical equipment that comes into contact with patients such as stethoscopes. The most commonly used disinfectants are further described here (Table 64.2).

Aldehydes

The aldehydes include formaldehyde and glutaraldehyde, which can be used as disinfectants. Glutaraldehyde, when used at a concentration of 2.4% and proper contact time, is considered a high-level disinfectant or chemical sterilant. Because of this property, glutaraldehyde can be used for equipment that requires sterilization, but cannot be placed into an autoclave, such as endoscopes. Glutaraldehyde provides broad-spectrum disinfection and is relatively noncorrosive, and has good activity in the presence of organic material. Glutaraldehyde is toxic and will cause irritation to mucous membranes. Orthophthalaldehyde is also used

Table 64.2 Commonly used disinfectants.

Antiseptic	Spectrum of activity	Disadvantages	Decreased effectiveness
Aldehydes	Broad spectrum, bacterial spores	Irritant to skin	N/A
Halogens/ hypochlorites	Broad spectrum, bacterial spores	Inactivated readily with organic material and soaps. Irritant and corrosive	Organic material, detergents/soaps
Oxidizing agents	Broad spectrum	Corrosive	N/A
Phenols	Broad spectrum	Irritant, toxic to cats	N/A
Quaternary ammoniums	Gram-positive, enveloped viruses. Variable for Gram-negative bacteria, fungi, and protozoa	Inactivated with	Organic material, hard water, soaps/detergents, cotton, iodine, rubber

N/A, not applicable.

for high-level disinfection, is less irritating to mucous membranes, and requires shorter contact times, so is favored over glutaraldehyde.

Halogens/Hypochlorites

Sodium hypochlorite (household bleach) is an alkali that when mixed with an acid releases free chloride and oxygen. The chloride combines with water to form hypochlorous acid. It oxidizes and inactivates bacterial membranes and cytoplasmic enzyme systems, kills bacteria, and liquefies necrotic tissue. It has a broad spectrum of activity against Gram-positive and -negative bacteria, mycobacteria, spores, fungi, enveloped and nonenveloped viruses, and protozoa except for *Cryptosporidium*. It is also effective against biofilms. It has diminished activity in the presence of organic material and cationic detergents. It is corrosive and cytotoxic with a detrimental impact on neutrophils, fibroblasts, and endothelial cells at higher concentrations.

For wound antisepsis, sodium hypochlorite is used as a 0.5% solution (commonly called Dakin's solution) which is made by diluting one part regular laundry bleach with nine parts water (Protocol 64.2). Although some rapid kill occurs, approximately 10 minutes of contact time ensures the destruction of most microorganisms. Bleach is unstable in solution and deteriorates rapidly, a process that is hastened with exposure to metal, high temperatures, light, and acid. Because of this instability, the solution should be made up immediately prior to use and any remaining solution should be discarded.

Protocol 64.2 Formulating 0.5% Sodium Hypochlorite (Dakin's) Solution

Items Required

- Household bleach
- Water

Procedure

1) To make 250 ml total volume, add 25 ml of household bleach to 225 ml of water.
2) Use immediately.
3) Discard any unused solution.

Oxidizing Agents

Hydrogen peroxide (H_2O_2) is a commonly used, foaming wound irrigant that dislodges bacteria and debris by effervescence. It releases oxygen, which causes lipid peroxidation of cell walls. At the high concentrations commonly found in disinfectants it is effective against Gram-positive and -negative bacteria, mycobacteria, fungi, enveloped and nonenveloped viruses, and protozoa; is variably effective against fungi, and is a good sporicide. Significant tissue damage can occur at concentrations of greater than 3%. At the 3% concentration used in antiseptic solutions, it is likely only effective against Gram-positive bacteria, unless the bacteria contain catalase, in which case it is rendered ineffective. Its duration of effect is short-lived since it is not absorbed and has no residual activity.

Accelerated hydrogen peroxide is a proprietary product containing hydrogen peroxide, a surfactant, and an acid resulting in a solution that has shorter contact times compared to H_2O_2 alone. These products are sporicidal and have good activity against non-enveloped viruses such as canine parvovirus. Accelerated hydrogen peroxide is nonirritating and noncorrosive, so has become a popular disinfectant in health care settings.

Potassium peroxymonosulfate, an oxidizing agent, is another high-level disinfectant. When proper concentrations are used, these products can inactivate nonenveloped viruses such as canine parvovirus. The powder concentrations are corrosive and toxic, but the solutions typically used (1%) are less corrosive and less irritating.

Phenols

Phenols are effective antiseptics with a variable spectrum of activity that depends on the formulation. Phenol has a broad spectrum of activity against Gram-positive and -negative bacteria at a 0.1–1% concentration, and mycobacteria and fungi at a 1–2% concentration. It has variable activity against enveloped viruses, is ineffective against spores except at very high concentrations (5%) and has questionable activity against protozoa. Because phenol is highly toxic to tissues, it is not used commonly. It is highly corrosive and extended exposure can cause neurotoxicity.

Quaternary Ammoniums

Quaternary ammonium compounds are cationic surfactants, also known as detergents, that act by disrupting cell membranes, denaturing proteins, and inactivating enzyme systems. They are effective against Gram-positive bacteria and enveloped viruses but have variable effectiveness against Gram-negative bacteria, fungi, and protozoa. They are mycobacteriostatic and sporostatic. They are ineffective against nonenveloped viruses. They are inactivated by hard water, soaps and detergents, cotton, iodine, rubber, and organic material; this list should be considered when applying quaternary ammonium compounds. These compounds are corrosive at higher concentrations, are irritating to membranes, and can cause dermatitis after repeated use.

Sterilization

Sterilization is defined as the use of a physical or chemical process to kill all microorganisms including bacteria, viruses, fungi, mycobacteria, protozoa, and spores. Methods of sterilization include gas, pressurized steam, and liquid. Gas and steam sterilization require the items to be packaged in a specific manner to permit penetration of the steam or gas. All packs to be sterilized, whether by autoclaving or gas, should contain appropriate indicator strips that turn a specific color once the contents have been appropriately sterilized. Indicators should always be checked to ensure that sterility has been achieved prior to use of the pack contents.

Autoclaves

Steam sterilization causes denaturation and coagulation of microorganism proteins by using saturated steam under pressure in an autoclave. Autoclaves are inexpensive, nontoxic, and effective, making them one of the most common methods for sterilization in the small-animal hospital today. Prior to sterilization, all instruments should be cleaned thoroughly with detergent and be free from any organic debris. Jointed instruments should be opened or unlocked to allow steam to penetrate all surfaces appropriately. Avoid overcrowding as this prevents the steam from properly circulating, which can lead to a failed cycle. Cycles are typically complete in under 30 minutes, but times may vary depending on the autoclave and how the instruments are packaged. Once the cycle is complete, allow for roughly 30 minutes for the instruments to dry. After drying is complete, store the instruments in a closed cabinet away from possible contamination.

Routinly clean your autoclave following the manufacturer's recommendations. Biological assessments should be done regularly by autoclaving a bacterial spore-impregnated strip and sending it in for culture to a microbiological laboratory or culturing it in hospital. If bacterial growth is reported, the autoclave should be thoroughly investigated, cleaned appropriately, and retested before use.

Gas Sterilization

Gas sterilization works by destroying the organisms through the alkylation of DNA. It is used to sterilize moisture or heat-sensitive equipment; most items that cannot be autoclaved, such as plastics and endoscopes, can be safely gas sterilized. Gases available include ozone gases, hydrogen peroxide vapor, formaldehyde, or ethylene oxide. Gas sterilized items must be allowed to vent for an appropriate length of time prior to use per the manufacturer's instructions, the entire sterilization process can take 24 hours or more to complete, which is significantly longer when compared with steam sterilization. Caution should be used as the gas is extremely flammable, carcinogenic, and irritating to the mucous membranes and eyes.

Flash Sterilization

Flash sterilization is a modification of steam sterilization and can be performed when an instrument is needed on an emergency basis. Flash sterilization refers to an instrument being sterilized in an open, unwrapped tray without wrapping or packaging that allows for steam to easily surround the instrument and achieve sterilization rapidly. Flash sterilization is not recommended for devices that are going to be implanted as they have been occasionally associated with intraoperative infections. Special handling is required during the removal of the instrument from the sterilizer and during transport to avoid recontamination.

Glutaraldehyde

Glutaraldehyde is considered a chemical sterilant since it has a broad spectrum of activity including being sporicidal. It acts by alkylating microorganism proteins. To ensure it is

sporicidal the solution must be made alkaline. Unfortunately, this leads to a steady loss of activity, which makes the glutaraldehyde solution ineffective after a maximum of 30 days. The shelf life varies depending on the formulation; therefore, the manufacturer's guidelines must be followed. In a 2% glutaraldehyde solution, most bacteria are killed within two minutes of contact; fungi, virus, and mycobacteria within 10 minutes; and spores within three hours. It is noncorrosive and most objects that can be submerged in water may be safely sterilized using glutaraldehyde. Like all chemical disinfectants, it is toxic to cells and irritating to the skin, membranes, and airways, so all materials sterilized with glutaraldehyde must be rinsed thoroughly immediately prior to use.

Bacterial Resistance to Antiseptics and Disinfectants

Intrinsic Resistance

Bacteria can have natural resistance to antiseptics and disinfectants, and they can develop resistance to these chemicals in the same way they develop resistance to antibiotics. Gram-negative bacteria, mycobacteria, and spores have natural resistance. This is primarily due to the presence of a complex cell wall and the inherent difficulty in penetrating the membrane of the organism. Occasionally, the organism has intrinsic properties that allow it to avoid the action of the antiseptic. This intrinsic resistance often means antiseptics are ineffective at concentrations that are safe to be used in patients.

Methicillin-resistant *Staphylococcus aureus* can be resistant to phenols and quaternary ammonium compounds. *P. aeruginosa* are often resistant to antiseptics due to the structure of their outer membrane. More information about these resistant bacteria is found in Chapter 62.

Biofilms

Pseudomonas spp. and *Serratia marcescens* can rapidly develop resistance to chlorhexidine by forming a biofilm. A similar problem has been noted with povidone-iodine. A biofilm is a community of bacteria covered in an extracellular matrix that have strongly adhered to the surface of an object. Bacteria inside biofilms are 1000 times more resistant to antimicrobial agents than when they are not in biofilms. Because of the fact that *Pseudomonas* spp. and *Serratia marascens* can develop a rapid resistance to chlorhexidine, the common practice of having containers of gauze squares soaking in chlorhexidine should not be permitted, nor should large multiuse containers of chlorhexidine-based ointment be used.

Aseptic Techniques

Handwashing

Handwashing plays a vital role in preventing infection in the workplace. Despite this knowledge and continued confirmation that infection is often transmitted by healthcare workers' hands, compliance rates remain poor. Even when handwashing occurs, often not all surfaces of the hands come in contact with the soap or antiseptic. Medical staff should wash their hands before coming on duty, before and after direct or indirect contact with patients, after exposure to any bodily fluids from self or a patient, before and after eating, before and after wearing gloves, before preparing and administering medication, after cleaning cages, and after completing a shift.

The primary action of soap is the mechanical removal of viable transient microorganisms. Regular soap with water should be used if gross contamination is present, and then followed with an antiseptic if needed. Good hand hygiene can also be achieved by using a waterless alcohol-based product. Alcohol-based products are less damaging to the skin and stations can easily be installed throughout the hospital. If an antimicrobial soap is being used, hands should be washed for at least 30 seconds. Multiuse cloth hand towels should be avoided. Artificial nails and nails longer than 0.25 inch have been associated with an increased likelihood of HAIs, so are not recommended. See Chapter 62 for an in-depth discussion of hand hygiene and for a handwashing protocol.

Aseptic Surgical Preparation: Personnel

Surgical hand antisepsis is crucial in reducing the risk of contamination during surgical procedures. All jewelry should be removed and proper surgical attire should be worn, which includes, clean surgical scrubs, a surgical cap over the head, and a face mask. Gross contamination should be removed using soap and water, and debris should be removed from under the nails. Arms should be held upright to allow water and soap to drip toward the elbows thus preventing recontamination of the hands and fingers. Antisepsis can typically be achieved in two to four minutes by using an alcohol–chlorhexdine combination or other antiseptic product. A one- to two-minute scrub with chlorhexidine followed by application of an alcohol-based solution has been shown to be more effective than the typical antiseptic scrub, which has made this the recommended presurgical handwashing technique over the traditional method.

Aseptic Surgical Preparation: Patient

Surgical site infections can occur if proper aseptic preparation is not performed. Most surgical infections are caused by the patient's own bacteria that are introduced into the surgical site from the skin, membranes, or hollow viscera such as intestines during the surgical procedure.

Hair or fur should be clipped immediately before the procedure rather than in advance, since advanced shaving has been associated with an increased incidence of infection. A wide area should be clipped. In the case of wounds or a surgical exploratory celiotomy or thoracotomy, the clip should be wide enough to ensure adequate preparation has been performed in case drains or feeding tubes need to be placed away from the primary surgical site. In traumatic wounds, this clip should extend at least 5 cm circumferentially beyond the known extent of the wound. In other situations, such as vascular catheter insertion sites, care should be taken to ensure that fur is clipped from the area immediately around the site, and that no fur can contact the catheter or insertion site during or after placement.

Gross contamination should be removed prior to using any antiseptic. In heavily contaminated wounds, this may require the use of large volumes of tap water. Tap water has not been shown to increase infection rates or interfere with wound healing. Once this has been completed a surgical preparation with an antiseptic should be performed (Protocol 64.1; see also Chapter 63). In surgical patients, this preparation should be performed using sterile gauze in a sterile bowl, sterile saline, and personnel should be wearing a cap and mask. Concentric circles should be used starting from the location of the incision and working outward toward the periphery. There have been very few studies examining the effects of using different antiseptics on both surgical site colonization and surgical site infection. Chlorhexidine 2% solution is the most commonly recommended antiseptic. It does not have as good in vitro effectiveness as 10% povidone-iodine; however, it has better in vivo effectiveness, especially against *Staphylococci*, as well as a longer residual effect. In addition, iodine-based compounds may cause a hypersensitivity reaction in healthcare personnel.

Summary

Proper selection and use of antiseptics, disinfectants, and sterilization are crucial to the prevention of HAIs. Chlorhexidine is currently the antiseptic documented to have the broadest spectrum of activity and least toxicity. Disinfectants should be chosen for their specific activities against target organisms. New antiseptics are constantly being developed to overcome microbial resistance and reduce toxicity.

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65

Personnel Precautions for Patients with Zoonotic Disease

Sarah Fritz and Christopher G. Byers

One of the inherent risks of working in veterinary medicine is the possibility of contracting a zoonotic disease, also known as a zoonosis. A zoonosis (from the Greek *zoo* meaning animal and *nosos* meaning illness) is defined as a disease of animals transmissible to humans. Owing to the unique proximity of animals and humans in veterinary hospitals, the risk of zoonotic pathogen transmission is higher than for the general population. This chapter covers types of zoonoses that small-animal emergency and critical care personnel may encounter, methods of transmission, prevention methods, and legal and public health issues surrounding zoonoses in veterinary medicine. As there are more than 250 zoonotic organisms known to cause human disease, it is not possible to cover the full spectrum in this chapter; this text addresses some of the zoonotic pathogens known to be transmissible from dogs and cats to humans (Table 65.1). Veterinary professionals should remember there are far greater numbers of zoonoses prevalent in large animal and exotic/special species veterinary work.

Zoonotic Disease Transmission

If an animal is ill with or potentially carrying one of a zoonotic pathogen, they represent the first step in the Centers for Disease Control and Prevention's (CDC) necessary three steps for disease transmission [1]:

1) A source of infection – including clinically ill animals, asymptomatic carriers, or unaffected reservoirs.
2) A susceptible host – due to physical susceptibility (i.e. non-intact skin, loss of cough reflex, or gastric acid reduction) or immunological susceptibility (i.e. immunocompromised individuals, people with underlying disease, pregnant women, and unvaccinated people).
3) A method of transmission – via contact (ingestion, cutaneous, percutaneous, mucus membranes, or fomite transmission); aerosol (on mucus membranes or non-intact skin, inhaled); or vector borne (insects on the animal or insects or rodents that enter the building).

Types of Zoonotic Diseases

Bacterial Diseases

Bacterial diseases are some of the most common zoonotic pathogens seen in small animal hospitals. Most will be instantly recognizable to veterinary personnel.

Bordetellosis

Bordetella bronchiseptica is a Gram-negative bacterium that primarily causes tracheobronchitis in dogs. The patient will often have a harsh, honking cough (kennel cough). These bacteria may cause respiratory disease in cats, but infections are usually subclinical. The pathogen spreads via aerosol transmission. Any patients with suspected bordetellosis should be isolated and staff should follow protective measures against aerosol transmission. Immunocompromised individuals and people with preexisting respiratory disease are most at risk for contracting bordetellosis. The disease usually involves the respiratory tract but has been associated with endocarditis, peritonitis, meningitis, and wound infections in people [2].

Brucellosis

Brucellosis is caused by the Gram-negative bacterium *Brucella canis* in dogs. This disease is usually transmitted through contact with vaginal secretions, placental tissue, semen, and sometimes urine and blood. In dogs and

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Companion website: www.wiley.com/go/burkitt/monitoring

Table 65.1 Major zoonoses in cats and dogs.

Disease	Vector	Method of transmission to humans
Acariasis (mange)	*Sarcoptes scabiei, Notoedres cati*, etc.	Contact
Brucellosis	*Brucella melitensis, B. abortus, B. suis, B. canis*	Aerosol, contact
Bartonellosis (cat scratch fever)	*Bartonella henselae*	Contact
Cryptococcosis	*Cryptococcus neoformans*	Aerosol
Dermatophytosis (ringworm)	*Microsporum* spp., *Trichophyton* spp., *Epidermophyton* spp.	Contact
Echinococcus	*Echinococcus granulosus, E. multilocularis*	Contact
Giardiasis	*Giardia intestinalis, G. lamblia*	Contact
Visceral, ocular, neuro larval migrans (roundworm)	*Toxocara canis, T. cati*	Contact
Leptospirosis	*Leptospira* spp.	Contact, aerosol
Pasteurellosis	*Pasteurella multocida*	Contact
Q fever	*Coxiella burnetii*	Contact, aerosol, vector
Salmonellosis	*Salmonella* spp.	Contact
Staphylococcosis	*Staphylococcus* spp.	Contact
Toxoplasmosis	*Toxoplasma gondii*	Contact
Bordetellosis (kennel cough)	*Bordetella bronchiseptica*	Aerosol
Campylobacteriosis	*Campylobacter jejuni, C. fetus, C. coli*	Contact
Chlamydiosis (mammalian)	*Chlamydophila abortus, C. felis*	Aerosol, contact
Cryptosporidiosis	*Cryptosporidium parvum*	Contact
Dipylidium (tapeworm)	*Dipylidium caninum*	Vector
Ehrlichiosis, anaplasmosis	*Ehrlichia* spp., *Anaplasma* spp.	Vector
Cutaneous larval migrans (hookworm)	*Ancylostoma* spp.	Contact
Leishmaniasis	*Leishmania* spp.	Vector
Listeriosis	*Listeria monocytogenes*	Contact
Plague	*Yersinia pestis*	Vector, contact, aerosol
Rabies	*Lyssavirus*	Contact
Sporotrichosis	*Sporothrix schenckii*	Contact
Streptococcosis	*Streptococcus* spp.	Contact, aerosol
Tularemia	*Francisella tularensis*	Vector, contact, aerosol

humans, the bacterium can be transmitted venereally. Presenting signs in dogs may include spontaneous abortion, epididymitis, orchitis, scrotal dermatitis, or scrotal necrosis in intact males. Discospondylitis and uveitis in dogs of either sex have also been documented [3]. In people, the disease is often subclinical, but symptoms may occur after a one- to two-month incubation period. Clinical illness is manifested as intermittent fever, headaches, chills, weakness, arthralgia, myalgia, weight loss, orchitis/epidydimitis in males, and spontaneous abortion. In rare cases, brucellosis has been linked to sacroilliitis, hepatic disease, endocarditis, colitis, and meningitis [3]. Brucellosis is suspected to be transmissible in breast milk [2]. Brucellosis is a nationally notifiable disease for people in the United States [4].

Bartonellosis

Bartonellosis, commonly known as "cat scratch fever," is caused by the Gram-negative bacterium *Bartonella henselae* (less commonly *Bartonella clarridgeiae*). The disease is transmitted to people primarily through cat bites or scratches but may also be transmitted via flea or tick vector, from a dog bite or scratch, or from saliva contact with broken skin. The feline patient presented to a small-animal clinic will typically be a young kitten, feral, or stray cat, and be a subclinical carrier as disease in cats is uncommon. If the organism does cause clinical signs in cats, the signs are usually mild and self-limiting, and can include pyrexia, uveitis, and lymphadenomegaly. Human disease is also mild in immunocompetant individuals. Symptoms may include papules/pustules at inoculation site, which may

progress to nonhealing wounds with regional lymphadenomegaly, fever, headache, and general malaise. There has been a reported case of optic neuritis stemming from an infection with *B. henselae* [5]. Fatalities have been reported in immunocompromised individuals from infections progressing to bacteremia, meningitis, and hepatitis [5].

Capnocytophaga

Capnocytophaga species live in the mouths of humans, dogs, and cats. They are considered normal oral flora. *Capnocytophaga canimorsus* is the species most commonly implicated in zoonotic infection. Research has shown up to 74% of dogs and up to 57% of cats carry *C. canimorsus* in their mouths [6]. Happily, these Gram-negative bacteria rarely, if ever, cause illness in our companions. *C. canimorsus* may be spread to humans through bites, scratches, and/or after close contact with dogs and cats. The major mode of transmission is via exposure of broken human skin to a pet's saliva. For example, humans with diabetes have been infected after allowing their dog to lick skin ulcers. Based on epidemiological studies in human medicine, 54% of human infections were caused by bites and 8.5% were caused by scratches. Just over 25% of all human cases were reportedly caused simply through close contact with an animal [7].

Infection is considered to be opportunistic. Humans with weakened immune systems are most susceptible to *C. canimorsus* infection. Interestingly, approximately 40% of people are seemingly healthy with no immune system issues [8]. In this patient population, being male, having diabetes mellitus, and being older than 50 years of age have been identified as risk factors for infection. Veterinarians have developed eye infections caused by *C. canimorsus* after tooth fragments hit their eyes during dental procedures. Symptoms of infection often manifest five days after exposure. Most commonly, infected individuals develop flu-like symptoms, including headache, fever, fatigue, skin rashes, muscle pain, difficulty breathing, and abdominal discomfort. Scarily, symptoms can progress to life-threatening proportions within 24 hours and around 30% with septicemia die from infection [8]. Treatment with an appropriate antibiotic is essential.

Coxiella burnetii (Q Fever)

Coxiella burnetti, the causative agent of Q fever, is an obligate intracellular bacterium. This organism has been known to infect a whole host of animals ranging from livestock to birds. In the human population, veterinary and livestock workers are the most at risk as cattle, sheep, and goats are the most common reservoir [9, 10]. The pathogen is commonly found in the placental/amniotic fluid, urine, feces, and milk of infected animals, and is passed via inhalation of dust particles contaminated with these fluids [9, 10]. Infection can also occur through ingesting unpasteurized dairy products. Wind has also been known to carry the pathogen long distances [9].

In animals, abortion and weak offspring are common, however some animals do not show any signs of the disease. Prevention in the animal population includes isolating any animals that have aborted fetuses, disposal of any materials contaminated with fluids from aborted fetuses, and cleaning of contaminated equipment with a phenolic disinfectant [9, 10].

In the human population, *Coxiella* can produce flu-like symptoms, including fever, chills, fatigue, and muscle pain [9]. Treatment involves doxycycline for approximately two weeks. The condition can become a chronic illness lasting several months and becoming potentially life threatening. In these cases, treatment includes doxycycline and hydroxychloroquine [9, 10]. Q Fever has been a nationally notifiable disease in the United States since 1999 [9].

Leptospirosis

Leptospirosis may be caused by any of several serovars of the gram-negative spirochete, *Leptospira interrogans*. The pathogen is transmitted via contact between infected urine or tissue and mucus membranes and/or non-intact skin. The disease is seen primarily in dogs. Although cats are susceptible to infection, they seldom develop clinical symptoms. The patient with leptospirosis may present with a variety of symptoms as the infection may be peracute, acute, subacute, or chronic. The peracute form of the disease results in sudden death with few or no clinical signs. In the acute form, dogs may have pyrexia, icterus, myalgia, vomiting, diarrhea, and peripheral vascular collapse. The subacute form symptoms include pyrexia, anorexia, vomiting, dehydration, polydipsia, and often severe renal disease marked by oliguria or anuria. The chronic form is frequently characterized by pyrexia and renal and/or hepatic failure with no known cause [11].

The majority of cases seen in animal hospitals are subacute and chronic. In humans, leptospirosis may range from asymptomatic infections to flu-like illness with pyrexia, headache, myalgia, nausea, vomiting, diarrhea, abdominal pain, rash, conjunctivitis, and conjunctival hemorrhage. There is also the hepatic-renal form of the disease caused by the serovar *Leptospirosis.icterohemorrhagiae*, and results in jaundice due to hepatocellular dysfunction and renal insufficiency from renal tubular damage; this form has a 20% mortality rate [2]. Rarely, leptospirosis may cause neurologic, respiratory, and cardiac manifestations [11]. A form of leptospirosis characterized by acute renal failure with fever, anorexia, lethargy, vomiting, polyuria, polydipsia, and abdominal pain has been documented in dogs and humans in Wisconsin, Michigan, New York, New Jersey, and Georgia [3].

The leptospirosis vaccine may protect dogs from clinical illness, but vaccinated dogs are still able to acquire the pathogen and shed it in their urine [2]. Leptospirosis is nationally notifiable for humans in the United States [12].

Pasteurellosis

Pasteurella species are Gram-negative coccobacillary bacteria responsible for disease in several animal species, though they are considered host specific [13]. Diseases in animals include gastrointestinal, respiratory, and hemorrhagic illnesses, as well as septicemia. *Pasteurella multocida* is commonly found in the mouths of dogs and cats and is known to cause disease in humans, with involvement in approximately 75% of cat bite and 50% of dog bite wounds [14]. Both localized and systemic infections can occur as a result of bite wounds from both dogs and cats. Localized infection is characterized by inflammation of the tissues immediately surrounding the bite, as well as redness and pain. If left untreated, abscess formation and septic arthritis can occur. Systemic illness, though rare, can include meningitis, sepsis, and pneumonia. Immunocompromised human patients are at risk for development of a fatal form of pasteurellosis [14].

Treatment in human patients includes broad-spectrum antibiotics. Potentiated amoxicillin, concurrent doxycycline and metronidazole (patients with penicillin allergies), concurrent clindamycin and a fluoroquinolone (children) or ceftriaxone (pregnant women) are among the common antibiotic therapies [15].

Prevention in animals involves good husbandry (fresh water, clean litterboxes for rabbits, etc.). Cockroaches have been noted as a potential vector, so good sanitation is important. Prevention in human beings centers around hand hygiene and proper restraint methods to avoid animal bites. Potentially contaminated surfaces may be cleaned with a variety of cleaning products such as phenolic cleaners, 70% ethanol, and glutaraldehyde cleaners [13].

Plague

Plague is caused by the Gram-negative bacterium, *Yersinia pestis*. In the United States, rodents (rats, squirrels, and prairie dogs), and cats are typically infected by fleas or ingestion of the aforementioned rodents. People contract the disease through contact with blood, purulent exudate, or aerosols from infected cats. Dogs may contract the pathogen, but infection is often subclinical and results in mild pyrexia and lymphadenomegaly. In cats and people, plague may present in one of its three classic forms: bubonic, septicemic, and pneumonic.

Bubonic plague is associated with fever, dehydration, lymphadenomegaly, and hyperesthesia. This form, usually acquired by cats ingesting infected rodents, causes regional lymph nodes (submandibular, cervical, and retropharyngeal) to abcessate (buboes) and drain [16].

Septicemic plague may progress from the bubonic form but is not always associated with bubo formation. In this form, the bacteria spread through the blood or lymphatic system to any organ in the body; the lungs and spleen are most commonly affected in cats and people. Clinical signs are due to septic shock and include fever, anorexia, vomiting, diarrhea, tachycardia, weak pulses, hypotension, cold extremities, disseminated intravascular coagulopathy, and leukocytosis [8]. This form is usually rapidly fatal [16].

The pneumonic form of plague may be secondary to bubonic or septicemic forms or may be primarily contracted from aerosolized plague bacteria. Cats do not typically contract primary pneumonic plague, but they have been implicated in the aerosol transmission of pneumonic plague to people [16]. Clinical signs may include fever, cough, hemoptysis, and respiratory distress. Since 1977, the CDC has reported 23 human cases of plague associated with aerosolized pneumonic plague droplets from infected cats. One fifth of these patients died and one quarter were veterinary staff [17]. Plague has caused millions of human fatalities through the ages and is a nationally notifiable disease for humans and animals in the United States [4, 12].

Staphylococcal Infections

Staphylococci are Gram-positive bacteria whose primary habitats are the skin and mucus membranes of mammals and birds. Most staphylococci cause no disease to animals; however, all species are potentially pathogenic [18]. Of particular concern to small animal veterinary personnel is methicillin-resistant *Staphylococcus aureus* (MRSA). This is a nosocomial and/or community acquired human pathogen, reportedly transmissible between small animals and people [18] (see Chapter 62). As of yet, it appears MRSA infections in dogs have been cases of "reverse zoonosis" acquired from humans as opposed to the other way around [18]; still, the possibility of hospital-acquired zoonoses exists and further study is required. MRSA is nationally notifiable for humans in the United States [4].

While *Staphylococcus pseudintermedius* is commonly part of the normal flora of healthy dogs, it is also a significant pathogen, commonly included in skin, ear, urinary, and postoperative infections. Approximately 69–87% of dogs are carriers; it is not well-established in cats [19]. While the prevalence of infection in humans is not as great as *S. aureus*, it has been isolated in healthy owners whose pets are infected, as well as veterinary workers, and can lead to bacterial infections that can become quite severe [19].

Methicillin-resistant *S. pseudintermedius* (MRSP) has been a growing problem within veterinary medicine since the first observation in 1999, particularly in surgical patients. Dogs who carry the pathogen are at increased risk of infection, most commonly following tibial plateau leveling osteotomy surgery [20]. Healthy dogs may also be

asymptomatic carriers of MRSP, leading to a growing concern for zoonosis both for pet owners and veterinary staff, although prevalence of these carriers is relatively low at 1.6–4.6% [19].

Treatment for infections with MRSP typically includes tetracycline antibiotics, with 50% of doxycycline resistant strains susceptible to minocycline. Other antibiotics include amikacin and chloramphenicol, although resistance to these drugs is emerging on the west coast and in the south of the United States [20]. Prevention can be accomplished by keeping wounds covered, washing hands/wearing gloves, and limiting contact with infected patients [20].

Tularemia

Tularemia is caused by the Gram-negative bacterium *Francisella tularensis*. This disease is endemic in temperate regions of the world, where rodents and rabbits are the primary reservoirs. In small-animal medicine, cats are the primary source of infection. Transmission is commonly via bites or scratches but it may also be contracted from aerosolized particles and vectors. Cats may present with a myriad of clinical signs including pyrexia, lymphadenomegaly, splenomegaly, hepatomegaly, oral ulcers, icterus, and panleukopenia [18]. Dogs appear to be relatively resistant to infection. In humans, clinical disease causes flu-like symptoms with fever, headache, and general malaise [2]. The infection may develop into either of the two main syndromes: ulceroglandular or typhoidal. In the ulceroglandular form, ulcerative skin lesions develop at the inoculation site, with regional lymphadenomegaly. The typhoidal form may present as systemic with diarrhea, vomiting, and abdominal pain; or as pneumonic with fever, cough, hemoptysis, and chest pain [2]. Tularemia is nationally notifiable for animals and humans [4, 12].

Fungal infections

Fungal organisms compose an important category of potential zoonotic infection. The more commonly seen fungal infections in small-animal veterinary hospitals include blastomycosis, dermatophytosis, and sprorotrichosis.

Blastomycosis

Blastomycosis is a systemic mycotic infection caused by the fungus *Blastomyces dermatitidis*. This organism is endemic to many areas of the United States, including the Mississippi, Ohio, and Missouri river valleys, the mid-Atlantic states [21]. Most human infections are directly from the environment as they are for dogs and cats; however, transmission has been documented in the veterinary field via inoculation from contaminated sharps during fine-needle aspiration, necropsies, and from the bite of an infected dog [5].

Patients that present clinically ill with blastomycosis commonly have clinical signs from inhaling infective spores. These may include weight loss, cough, dyspnea, anorexia, skin lesions, ocular disease, and lameness. A patient presenting with uveitis and concurrent skin or respiratory disease in an endemic region should raise marked suspicion for blastomycosis [21]. In people, blastomycosis also affects the lungs and may cause fever, chills, sweating, chest pain, coughing, and difficulty breathing. The fungus may also spread systemically affecting skin, bones, meninges, and the genitourinary tract [5].

Dermatophytosis

Dermatophytosis, often referred to as ringworm, is a fungal dermatological disease caused by species of *Microsporum* or *Trichophyton*. Transmission to people is via contact with infected animals and/or asymptomatic carriers. Dermatophytosis is a disease with widely varying signs and imperfect diagnostic tests; personnel should therefore follow precautions in dealing with any animal evincing dermatologic disease. The fungus attacks the follicle and clinical signs often include hair loss, scaling, and crusting. Some animals may have the classic ringlike lesion, but the disease can look like almost any other dermatological disease affecting cats and dogs. Cats are the primary species of concern with regards to zoonotic transmission. People often develop the ring lesions with circular alopecia, scaling, crusting, and ulceration [22]. The disease can be more serious in immunocompromised individuals.

Sporotrichosis

Sporotrichosis is caused by the fungus *Sporothrix schenckii*, which is found in soil throughout the world. Transmission occurs via inoculation with the fungi, and in dogs (particularly hunting or free-roaming dogs) it is thought to arise from contaminated thorns or wood splinters. In cats, transmission is commonly via scratches or bites from other cats; it is seen most often in intact males that are outdoors.

The dog with sporotrichosis will typically have cutaneous or cutaneolymphatic form. Clinical signs of the cutaneous form include nodules in the dermal and subcutaneous layers of the trunk and head that may be ulcerated. Those with the cutaneolymphatic form will have nodules on the distal part of a limb that progress along lymphatic vessels and is associated with regional lymphadenomegaly. In cats, nodules will occur in areas where inoculation occurred and typically resemble non-healing abscesses and may progress to necrosis of the surrounding area. The organism may become systemic in cats and may spread to many organs.

Veterinary personnel are most at risk from cats, as the fungus replicates easily and in large numbers in the exudate from ulcerated nodules, as well as the tissues and feces of infected cats [23]. Most infections in people present as

ulcerative, cutaneous lesions with purulent discharge and localized lymphadenomegaly [23]. Systemic sporotrichosis is a danger to immunocompromised people and has been documented in people with AIDS, alcoholism, diabetes mellitus, and those receiving immunosuppressive medications [24].

Viral Infections

Viruses are another classification of zoonotic disease to which small-animal veterinary personnel are exposed. Within this category, rabies is of paramount concern but there is growing evidence influenza is also transmissible between small animals and humans.

Rabies

Rabies is caused by a *Lyssavirus* in the *Rhabdoviridae* family. These viruses are found nearly worldwide, and all warm-blooded animals are susceptible to infection. Pathogen transmission is nearly always via inoculation with saliva (bite) or contact with saliva and broken skin or mucus membranes. A rabid patient may have a variety of clinical signs and a bite wound history. Commonly, the prototypical manifestations of fury and paralysis may be present but they are not required for definitive diagnosis.

The prodromal phase in dogs and cats may result in pyrexia, apprehension, nervousness, anxiety, avoidance of company, and/or radical behavioral changes from the animal's norm. Licking, biting, or scratching at wound site may be present. In dogs, the furious stage of rabies may develop following the prodromal phase and lasts for up to seven days. Classic signs include irritability, restlessness, photophobia, ataxia, disorientation, generalized seizures, viciousness, and pica. Death usually occurs during seizure activity. Cats have a more classic furious stage with erratic, vicious behavior, tremors, ataxia, and weakness.

The patient with paralytic rabies will develop lower motor neuron paralysis spreading from the infected bite wound. The classic clinical sign in dogs is a hanging jaw with concurrent ptyalism as the animal loses its ability to close its mouth or swallow. This results in death from respiratory failure within a few days [2]. Cats primarily present with paralysis progressing from the infected bite area and in increase in vocalization with a marked change in voice [2].

In people, rabies manifests in the prodromal stage as fever, headache, pain at the bite site, and agitation. The furious phase includes violent behavior, restlessness, anxiety, and seizures that may result in death. The paralytic stage symptoms include the inability to swallow, progressive paralysis, and death due to respiratory failure. The mortality rate for rabies in veterinary and human patients is virtually 100%. Rabies is a nationally notifiable disease for animals and humans in the United States [4, 12].

Influenza

Influenza viruses from the *Orthomyxoviridae* family are infectious to humans and many species of animals, including dogs and cats. In the 1970s, an influenza epidemic in people resulted in the contraction of clinical flu in dogs [25]. Canine influenza virus (CIV) was initially caused by equine influenza A H3N8 virus, and the first reported infections were documented in racing greyhounds in Florida in 2004. Essentially the virus "jumped ship" from horses and is now considered a dog-specific lineage of H3N8. In 2009, there was an outbreak of influenza A (H1N1), also known as "swine flu." The American Veterinary Medical Association confirmed clinical disease in 14 cats and 3 dogs that had been in contact with infected humans [26]. In spring 2015, an outbreak of CIV was noted in several midwestern states, including Illinois, Wisconsin, Ohio, and Indiana [27]. The documented strain, H3N2, was first identified in southern China and the Republic of Korea in 2006, and infectious disease experts thought that this strain was isolated only to parts of Asia. No one yet knows exactly how H3N2 came to the United States.

In cats and dogs, influenza may present with coughing, sneezing, nasal and ocular discharge, anorexia, pyrexia, lethargy, and occasionally secondary pneumonia. To date, influenza is considered a "reverse" zoonotic disease in dogs and cats. This means that they contract the disease from humans as opposed to humans contracting the disease from them. However, influenza does pass back and forth between humans and other animals such as pigs and birds, so the risk of zoonotic transmission is there.

Noroviruses

Norovirus is a common cause of non-bacterial gastroenteritis and is found worldwide. There have been as many as 10 genogroups and 40 genotypes categorized. Noroviruses are highly contagious, found in stools and vomit of infected species [28]. In humans, the virus is spread through contaminated food and drinking water. Human norovirus has the ability to infect a large variety of animal species including companion animals and livestock. These species act as a reservoir for the human virus. It is not clear whether norovirus causes clinical disease in dogs [28]. However, in humans, the virus causes vomiting, diarrhea, headaches, fever, and other flu-like symptoms [29]. These signs will last 24–72 hours, generally. Prevention includes good hand hygiene and disinfecting contaminated surfaces and objects with bleach [27].

Fecally Transmitted Zoonotic Pathogens

Within zoonotic diseases, there is a special subset that is transmitted primarily in fecal matter. These fecally transmitted zoonotic pathogens, *Campylobacter*, *Cryptosporidium*,

Giardia, *Salmonella*, and *Toxoplasma* are some of the most commonly seen in small animal veterinary medicine.

Campylobacter

Campylobacteriosis is most commonly caused in dogs, cat, and people, by the Gram-negative bacterium *Campylobacter jejuni*. Transmission is primarily via the fecal–oral route in veterinary setting. Patients will commonly present with symptoms including diarrhea, vomiting, pyrexia, and anorexia. Clinical disease typically lasts about one week and is most common in puppies and kittens less than six months old. In people, *C. jejuni* is the most common cause of bacterial diarrhea in the United States, with an estimated two million cases a year [2]. The disease causes enteritis with symptoms of fever and malaise, progressing to diarrhea and abdominal pain. The disease is generally self-limiting in healthy adults but has been linked to the development of Guillain–Barre syndrome (GBS) in approximately 1 in 1000 cases [2]. GBS is an immune-mediated myelitis/neuropathy [2]. Another secondary development that has been identified in human C*ampylobacter* infections is Reiter's syndrome, which causes tenosynovitis, skin lesions, uveitis, and urethritis in approximately 7% of cases [2].

Cryptosporidium

Cryptosporidium parvum is a coccidian protozoan that causes intestinal disease in dogs, cats, and humans and is transmitted via the fecal–oral route. Cats with C*ryptosporidium* may have diarrhea, weight loss, tenesmus, blood in the stool, and abdominal discomfort. Disease in cats is typically seen in kittens or in immunocompromised or stressed adults [30]. In dogs, *Cryptosporidium* usually only causes disease in young puppies with concurrent parvovirus or distemper. Symptoms include diarrhea, weight loss, and malabsorption.

In humans, the majority of people will develop mild, self-limiting diarrhea with the disease. However, cryptosporidiosis is a leading cause of life-threatening chronic diarrhea in immunocompromised patients, such as those with HIV [2]. Of primary concern to veterinary staff, oocysts present in fecal matter are highly infectious, highly resistant to environmental deactivation, and there is no routinely successful treatment for infections [2]. *Cryptosporidium* is nationally notifiable for humans in the United States [4].

Giardia

Giardia duodenalis is a protozoan parasite transmitted via the fecal–oral passing of infective cysts. Dogs and cats may present as asymptomatic carriers or evince clinical disease with pale, malodorous, steatorrheic stool, and weight loss [2]. Animals with clinical disease are primarily very young puppies and kittens, or adults that are immunocompromised, stressed, or from kennel situations [2].

In humans, *Giardia* is one of the most common intestinal parasites worldwide [2]. The vast majority of cases stem from contaminated water, however, direct transmission via the feces of infected animals is possible. Symptoms in people may include acute gastroenteritis with diarrhea or a more chronic infection with malabsorptive diarrhea, weight loss, and abdominal pain that waxes and wanes [2]. Giardia is nationally notifiable for humans in the United States [4].

Salmonella

Salmonella enterica is a Gram-negative bacterium with over 2400 pathogenic serovars that cause primarily intestinal disease. *Salmonella* is a fecal pathogen that can survive for a long time in the environment and be transmitted via the direct fecal–oral path, in contaminated food and water, on fomites, via contact with cat saliva, and even aerosolized in dried airborne particles [31]. The majority of affected dogs and cats are subclinical carriers, but when illness does present it is typically an acute enterocolitis with pyrexia, anorexia, lethargy, diarrhea with mucus or blood, abdominal pain, and mesenteric lymphadenomegaly [31]. In some cases, clinical infection can result in septicemia, endotoxemia, and localized organ infection such as *Salmonella* pneumonia [31].

A unique clinical presentation is "songbird fever" in domestic cats. Symptoms include acute pyrexia, anorexia, depression, diarrhea, and vomiting. This illness is named for its suspected link to the predation of migratory birds [31]. In humans, salmonellosis usually follows a 12–36 hour incubation period, and includes symptoms such as headache, fever, vomiting, diarrhea, abdominal pain, nausea, myalgia, and dehydration. Of special concern is the transmission of highly drug resistant strains of *Salmonella typhimurium* DT 104, which has been isolated in dogs and cats and been reported as the causative agent in an outbreak of salmonellosis in cats and people at three small veterinary hospitals [32]. S*almonella* is nationally notifiable for humans in the United States [4].

Toxoplasmosis

Toxoplasmosis is caused by the coccidian protozoa *Toxoplasma gondii*. Domestic cats and other felines are the definitive hosts for this parasite; all other species, including dogs and humans, are intermediated hosts. The parasite causes tissue cysts. Transmission is via oocyte ingestion from cat feces, undercooked meat, or contaminated water. In veterinary hospitals, cat feces are of primary concern. The *Toxoplasma* oocyte is not infectious when first excreted. It takes one to five days depending on temperature and humidity, to sporulate into the infective form.

Once the oocyst has entered its infective phase, it can remain infectious in the environment for 18 months and is extremely hardy and difficult to eradicate.

A cat presenting with clinical toxoplasmosis may have varied symptoms, as the parasite can affect virtually any organ system. The most common systems affected are pulmonary, nervous, hepatic, pancreatic, cardiac, and ocular. Symptoms may include pyrexia, pancreatitis, hepatitis, encephalitis, polymyositis, pneumonitis, uveitis, chorioretinitis, fading kitten syndrome, vomiting, diarrhea, and anorexia [2]. In dogs, disease is uncommon but may result in a rapidly fatal systemic disease involving the gastrointestinal tract, the respiratory system, the neuromuscular system, and the liver [2]. Cats can shed oocytes for up to two weeks following initial infection and have been shown to be susceptible to contracting the parasite again after a previous infection. Cats with latent infections can sometimes reshed oocytes after secondary infections [2]. In people, infection with toxoplasmosis can cause abortion, chorioretinitis, blindness, hydrocephalus, epilepsy, mental retardation, myelitis, and paralysis, fever, malaise, myalgia, lymphadenomegaly, and hepatosplenomegaly [2]. A 2003 study proposed a possible link between toxoplasmosis and schizophrenia [33].

Other Zoonotic Diseases

Despite the low occurrence of two zoonotic disease categories, a chapter on this subject would not be complete without a mention of both vector-borne zoonoses such as anaplasmosis (*Anaplasma* spp.), ehrlichiosis (*Ehrlichia* spp.), leishmaniasis (*Leishmania* spp.), Rocky Mountain spotted fever (*Rickettsia rickettsii*), borreliosis (*Borrelia burgdorferi*), intestinal cestodes, nematodes, and ascarids.

Vector-borne diseases require an insect such as a flea, tick, or mosquito to pass a pathogen from a reservoir species to humans. Most cats and dogs are not definitive hosts for these diseases and so their role in transmission is to bring the shared vectors into close contact with humans. Good insect and rodent control within the hospital and quick response to flea and tick infestation on incoming patients can be preventive.

Intestinal cestodes, nematodes, and ascarids, also known as "worms," are also zoonotic disease-causing agents. Most intestinal parasites, such as the cestodes (tapeworms), *Dipylidium caninum* and *Echinococcus multiocularis* are acquired by humans through the ingestion of fleas, or as in the case of the ascarids and nematodes, *Toxocara cati*, *Toxocara.canis* (roundworms), *Ancylostoma* spp. (hookworms), and *Strongyloides stercoralis*, through the ingestion or skin penetration of infective eggs from contaminated soil. Again, as in the case of vector borne diseases, rodent and insect control as well as immediate treatment of any animal infested with fleas can be preventive. In the case of soil contamination, removing stool from the environment quickly can prevent eggs from developing into the infective stage.

Personnel Protection

Zoonotic disease prevention can start before the patient enters the hospital. Identification of potential zoonotic risk starts with knowledge of the associated clinical signs of disease and methods of transmission of a given disease. Solid knowledge of disease states may allow the technician/nurse or receptionist to identify possible zoonoses based on an animal's symptoms, history, geographic region, and other risk factors, such as age, underlying illness, and proximity to certain high-risk areas or wildlife. The best-case scenario is identification over the telephone and isolation of the incoming animal prior to entering the building. More commonly, rapid isolation of the patient in an examination room once the risk is identified, and limitation of personnel contact, are instrumental in reducing the likelihood of disease transmission. Signs should be used to notify other personnel and clients that an infectious disease may be present and what precautions to take.

Hand hygiene

The single most important precaution veterinary personnel can take to prevent the transmission of zoonotic disease is good hand hygiene [5]. Washing with regular soap and water removes debris and is effective in reducing the number or organisms on the skin. The added use of antimicrobial soaps and hand cleansers kill or prevents the replication of many viruses and bacteria. The use of gel, liquid, or cloth hand sanitizers is helpful in situations where there is no access to handwashing facilities but should primarily be used in conjunction with handwashing and not in place of it.

The proper procedure for hand washing is seen in Protocol 65.1. Soap should be liquid- or foam-based, and not bar soap, as the latter may serve as a reservoir for

Protocol 65.1 Effective Handwashing for Veterinary Personnel

1) Wet hands with running water.
2) Place liquid or foam soap in palms.
3) Rub hands together to form lather.
4) Scrub all surfaces of hands (including between fingers and backs) for 2 minutes.
5) Rinse.
6) Dry with disposable towel.
7) Turn off sink with disposable towel.

bacterial growth. Soap dispensers should be able to be loaded with disposable soap containers and/or be emptied completely, cleaned, and disinfected before being refilled to prevent the colonization of the dispensers with pathogens. When used, hand sanitizers should be rubbed into hands until dry. Veterinary personnel should not wear long fingernails, particularly artificial nails, as they may inhibit adequate hand hygiene. Rings may also harbor organisms that are not killed during hand washing and should be removed during procedures and hand washing.

Barrier Protection

Barrier protection is the next protective measure veterinary personnel should take to prevent the transmission of zoonotic diseases. The most important and regularly used barrier is gloves. Gloves should be worn when handling any animal with unknown history or vaccination status, for handling feces, blood, or other bodily fluids, including during venipuncture and aspiration procedures. They should also be worn when cleaning litter boxes, handling soiled bedding, cleaning any surface in possible contact with bodily tissues or fluids, and when handling any animal with blood or bodily fluid on them. Gloves should be removed immediately after use without touching the external surfaces. Gloves should be removed from the wrist and pulled inside out to prevent hand contamination. Hands should be washed immediately after removing gloves as there may be small defects and/or hands may have been contaminated during glove removal.

Disposable or washable gowns, such as surgery gowns, may be worn when holding or restraining a potentially infectious patient or in any situation where there is a risk of bodily contact with pathogens. Normally permeable gowns are typically used but waterproof gowns should be used in cases where there is a risk of contamination with large volumes of bodily fluids. Any time a barrier gown is worn, gloves should also be worn. Gowns should only be used for one patient at a time and must be removed without touching the contaminated outer surface. To do this, untie the gown with gloved hands, pull away from the body by grasping the chest, again with gloved hands, pull down cuffs on each arm and slide gown off arms and away from the body. Then remove the gloves and wash hands thoroughly in case of accidental contamination. If bodily fluid penetrates a semi permeable gown, remove the gown and contaminated clothing, and shower immediately.

Foot covers and foot baths are useful in reducing the transmission of pathogens on personnel shoes. Disposable surgical booties may be worn and removed just as with gloves to avoid hand contamination. Foot baths are typically composed of a 10% hypochlorite (bleach) solution and should be used when entering and exiting designated isolation areas.

As discussed earlier in this chapter, some pathogens may be transmitted via aerosolization. Facial protection, such as masks and face shields or goggles should be worn when dealing with potential aerosolized pathogens. These protections should also be in place when performing procedures that may cause aerosolization of pathogens, such as dental procedures, wound flushing, abscess draining, nebulization and coupage, suction, lavage, and necropsy. In addition, avoid performing resuscitation by breathing into a patient's mouth, nose, or endotracheal tube; rather one should always use a bag valve mask or mechanical ventilator. Respiratory protection using particulate respirators is not common in small-animal veterinary practice but may have a place if certain diseases such as *Coxiella burnetii*, the causative agent of "Q fever," become more prevalent. Information on respiratory protection is available through the Occupational Safety and Health Administration (OSHA).

Prevention of Transmission

As many zoonotic pathogens are transmitted through bites or scratches, prevention is very important in the veterinary hospital. Knowledge of aggressive tendencies in patients is key to preventing personnel injury. Proper restraint from trained staff, muzzles, heavy gloves, and sedation all play an instrumental part in injury prevention.

There are certain procedures in veterinary hospitals that require full barrier protection. Birth, cesarean sections, and abortions are all considered high-risk events, as some extremely virulent zoonotic diseases may be passed in birthing tissue and fluids. Necropsies are also high-risk procedures, particularly due to the use of saws and drills that may aerosolize zoonotic pathogens. Respiratory protection should be used if any power tools are used on a potentially zoonotic cadaver.

Patients with possible zoonotic diseases should be isolated from other patients, the visiting public, and all personnel except those essential to their care. If possible, these patients should enter the facility via a separate entrance directly into an isolation ward or a dedicated exam room. If a separate entrance is not available, the patient should be transported directly through the waiting room by personnel wearing barrier protection. Cats and small dogs should be transported in a carrier, and larger dogs on a gurney or stretcher. The animal should be moved into a designated isolation area as soon as possible and this area should have a separate entrance from the main patient care area; only personnel needed for zoonotic patient's care should enter the isolation area. Personnel entry should be restricted and monitored, and a record of all staff that have contact with the patient must be made. Signs must be posted at the

entrance to the isolation area to alert staff to the zoonotic disease potential, notify them of necessary precautions to take, and warn away other staff. The isolation area should have its own ventilation system to prevent the aerosolization of pathogens to the rest of the hospital.

Many diseases are transmissible via needlestick injuries. Clinicians should rigorously follow safety guidelines when dealing with all sharps. The primary way of preventing needlesticks is never to recap a needle. Dispose of used needles and syringes in a puncture-proof sharps container. If it is necessary to recap a needle, use the one-handed scoop method or a tool such as a hemostat to hold the needle cap away from your hand. The one-handed scoop method involves placing the cap on a horizontal surface such as a treatment table, holding the syringe, sliding the needle into the cap, and pressing down to tighten on a hard surface. Scalpels come with disposable handles and blades and these are ideal when dealing with potential zoonoses. If a scalpel blade must be removed from a handle, use hemostats to grasp the blade at the base with the sharp edge facing away from that hand and gently work the blade up and off with constant light pressure and wiggling.

After a confirmed or potentially zoonotic patient has left the designated isolation area, the location should be fully cleaned and disinfected to prevent pathogens from living in the environment. Cleaning a potentially contaminated area involves general removal of large waste and debris: ideally a filtered, central vacuum unit should be used to clean an area and remove potentially aerosolized pathogens. If this is not possible, personnel should take all recommended precautions against aerosolized pathogens. After removal of large debris, the area should be cleaned with a disinfectant (see Chapter 64) shown to be effective against the particular pathogen. No one disinfectant is effective against every pathogen. Table 65.2 lists a number of classes of disinfectants and their efficacy against different pathogens. These products should be used according to their manufacturer's instructions for maximum efficacy.

In dealing with potentially contaminated bedding, the fabric should initially be shaken gently over a contained area such as a garbage bag to dislodge potentially overlooked sharps and gross fecal matter. Full barrier protection, particularly gloves should be worn in dealing with all bedding. The bedding should be washed and dried normally. Litter boxes should be emptied into a closed garbage container as soon as they are soiled. Again, the use of disposable litter boxes is ideal, but if regular litter boxes are used, they can be cleaned and disinfected the same way as the overall environment. Food and water bowls can be washed normally with dish detergent if they are nondisposable.

If a bodily fluid from a patient with potential zoonoses is spilled, it should be sprayed with disinfectant and wiped up with absorbent material. All contaminated refuse should be bagged within the isolation area using gloves, and then rebagged in a second garbage bag outside the isolation area using a clean pair of gloves. The bags should be transported out of the hospital to an external garbage area immediately. Medical waste, including sharps and deceased animals should be handled according to OSHA bloodborne pathogen standards [34]. It should be reiterated that no handling, transporting, or cleaning of an area with potential zoonotic contamination should be done without full barrier protection, gloves and gowns minimum, and shoe covers, masks, goggles, face shields, and/or respirators depending upon the suspected pathogen.

Table 65.2 Common disinfectants and their targets.

Disinfectant	Effective against	Variable/limited effective against	Not effective against
Alcohols (isopropyl)	Bacteria, Mycobacteria, enveloped viruses, fungi	Non-enveloped viruses	Spores
Biguinides (chlorhexidine)	Bacteria	Mycobacteria, viruses, fungi	Spores
Hypochlorites (bleach)	Bacteria, Mycobacteria, viruses, fungi	Spores	
Iodine compounds	Bacteria, enveloped viruses, fungi	Mycobacteria, non-enveloped viruses, spores	
Oxidizing agents (hydrogen peroxide)	Bacteria, Mycobacteria, viruses	Spores, fungi	
Phenols	Bacteria, enveloped viruses	Mycobacteria Non-enveloped viruses, fungi	Spores
Quaternary ammonium compounds	Gram-positive bacteria	Gram-negative bacteria, Mycobacteria, enveloped viruses, fungi	Non-enveloped viruses, spores

Vector Control

Vector control is another important way to minimize the potential exposure to zoonotic pathogens. Insects and rodents are attracted to veterinary hospitals and are common vectors of zoonotic disease. Proper facility cleanliness, food storage, prevention of standing water, and blocking points of entry are necessary to prevent these vectors from entering the hospital. If an animal carrying fleas or ticks enter the hospital, they should be separated from other patients, personnel working with them should wear protective clothing, and they should be immediately treated with the appropriate and safe insecticidal medications.

Client Education

Client education is instrumental in prevention of zoonotic disease transmission. Veterinary personnel should take an active role in informing clients of the importance of prophylactic vaccination against diseases such as rabies, bordatellosis, borreliosis (Lyme disease), and leptospirosis. Control of ecto- and endoparasites is also an important preventive measure as is prohibition of predation on reservoir species. All of these steps not only protect the client themselves, but also veterinary personnel who may encounter the client's pet.

Vaccination

Prophylactic vaccination of veterinary personnel is another method of prevention. The CDC Advisory Committee on Immunization Practices recommends that all veterinary personnel who have contact with animals should be vaccinated against rabies. Preexposure prophylaxis is a series of three shots that offer protection against unknown rabies exposure or when post-exposure vaccination is delayed. Rabies titers should be checked every two years and boosted if the titer is too low to offer protection.

Reporting Procedures

In case of possible exposure to a zoonotic disease, a veterinary hospital must have a protocol for documenting and responding to the incident. Most hospitals have reporting procedures for bites or other injuries, and these may be readily altered to include contact with possible zoonotic pathogens. This written documentation should include a list of all personnel involved, specifics of the incident (including the date, time, and location), information on the animal involved, any medical treatment sought, any agencies notified, and follow-up.

Staff Awareness and Training

All methods and guidelines for prevention of zoonotic disease transmission hinge on veterinary personnel being aware of them. Staff training is of paramount importance in every step of zoonotic disease prevention. Frequent training and evaluation, in addition to written hospital guidelines, should be in place so staff are knowledgeable in prevention of zoonotic disease transmission. In addition, hospitals must keep personnel food storage separate from patient food and medicine storage, and prohibit eating, drinking, or smoking in patient care areas.

There is a subset of veterinary personnel that are of particular concern with regards to zoonotic disease transmission. Immunocompromised individuals, such as those with HIV, diabetes mellitus, asplenia, and some congenital abnormalities, as well as pregnant women and those receiving immunosuppressive medications, are at increased risk for contracting zoonotic diseases and experiencing much more severe symptoms. These staff members must be aware of the dangers involved when handling patients, particularly high-risk patients such as ferals, strays, those with unknown vaccination histories, puppies and kittens less than three months of age, animals consuming raw diets, and those with known zoonotic diseases. Ideally, an immunocompromised staff member should alert their employer/manager so that policies may be enacted to minimize their risks. These individuals should also notify their personal physician of the zoonotic risks involved with their employment so familiarity with symptoms and proactive monitoring may be established. Pregnant women face a unique immunocompromised situation. Pregnancy suppresses the body's cell-mediated immunity and may increase a women's risk for contracting certain zoonotic diseases [35].

Legal and Public Health Issues

Another aspect of zoonotic disease in the veterinary hospital is the legal issues involved in potential transmission to veterinary personnel and pet owners. This is a newly evolving field of inquiry, as the liability issues involved have yet to be definitively established. In most cases, veterinary personnel who contract a zoonotic disease from a patient while at work are covered under workers' compensation regulations and state workers' occupational disease acts. However, with the increasing focus on zoonotic disease risks, liability issues have yet to be determined. In 1986, a veterinarian was sued for failure to provide a safe workplace in a case involving the death of a kennel worker from leptospirosis [36]. This case highlights the potential legal issues still to be resolved.

With regard to client risk, the legal ramifications of zoonotic diseases are still evolving. Veterinarians and veterinary staff have an ethical obligation to notify owners of potential zoonotic disease and to advise them to seek information from their personal physicians. There have been malpractice claims filed against veterinarians for human injury and exposure to rabid or potentially rabid animals [37], and such may be the future avenue of legal proceedings for zoonotic diseases.

In terms of public health and zoonotic disease, there are varying guidelines in most states and within the federal government defining which diseases must be reported and to whom. In response to this, the National Academy of Sciences (NAS) presented a report in 2005 calling for the establishment of a federal level, centralized coordinating mechanism for animal health oversight [38]. This body would purportedly organize animal health data for industry, local, state, and federal agencies. Such an oversight body such as the one proposed by the NAS may have facilitated the earlier recognition of West Nile virus in people. In the 1999 outbreak, physicians were diagnosing patients with GBS, meningitis, encephalitis, and aspiration pneumonia [39]. However, a veterinary pathologist at the Bronx Zoo linked the human and animal outbreaks based on the deaths of crows and other birds, allowing identification of West Nile virus as the cause of human illness in these perplexing cases [40].

Summary

The goal of any infection prevention protocol is to substantially lower the risk of disease transmission. It is unrealistic to believe taking rigorous personnel precautions can eradicate all risk of zoonotic disease. In small- animal veterinary medicine, the emergency and critical care staff will encounter patients with zoonotic diseases. By instituting the procedures outlined in this chapter and expanding knowledge of zoonotic diseases, personnel and hospitals can protect themselves, their clients, the public, and their patients against these pathogens.

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Section Nine

Transfusion Medicine

66

Blood Typing and Crossmatching

Sarah Musulin and Kenichiro Yagi

Knowledge of small animal blood types and understanding of precompatibility testing are paramount when making transfusion decisions. Blood group nomenclature is based on the characterization of inherited red blood cell (RBC) antigens located on the surface of the RBC. The prevalence of blood types and RBC antigens varies according to geography and breeds. Precompatibility testing includes blood type determination and crossmatching.

A basic understanding of transfusion immunology terminology is helpful when discussing blood types and compatibility testing. Antibodies can be classified as naturally occurring or immune (inducible). Naturally occurring anti-RBC antibodies are found in individuals that have not been sensitized by previous transfusion. Immune anti-RBC antibodies are found in individuals that have been sensitized by previous transfusion and antigen exposure. Blood group antibodies, whether naturally occurring or immune, can have pathologic effects that result in RBC agglutination and destruction (acute or delayed hemolytic transfusion reaction). Agglutination or hemagglutination refers to the clumping of RBCs secondary to antibodies recognizing surface antigens. Allogenic denotes tissue (e.g. blood cells) that are from the same species. An alloantibody is an antibody specific for an alloantigen that occurs in some members of a species. Alloimmunization is the immune response to foreign antigens after exposure to genetically different cells or tissues from members of the same species. Immunogenicity refers to the ability to provoke an immune response.

Canine Blood Types

The primary canine blood group classification is based on the dog erythrocyte antigen (DEA) system. Newly identified canine RBC antigens have been discovered that do not follow the DEA system, such as *Dal* and *Kai* 1 and 2. Typing sera are available on a limited basis for DEA 1, 3, 4, 5, 7, *Dal*, and *Kai* 1 and 2. For most blood groups, individual dogs exhibit one phenotype for each system; for example, a dog may be DEA 3, 4, and *Dal* positive, and 5, 7, and *Kai* 1 and 2 negative. The DEA 1 blood type is an exception to this, where a dog can be DEA 1(−) or weakly to strongly DEA 1(+). The distribution of DEA 1 in the canine population is nearly equal. DEA 1 is considered the most immunogenic and clinically significant RBC antigen. Naturally occurring anti-DEA 1 antibodies have not been documented in dogs, although sensitization with transfusion does occur. Transfusion of a DEA 1(−) dog with strongly DEA 1(+) blood will lead to the formation of hemolyzing and strongly agglutinating immune antibodies against DEA 1(+) within days and reexposure can lead to an acute hemolytic transfusion reaction. Point of care (POC) typing kits are available to guide DEA 1 type-specific transfusions. Given the clinical relevance of the DEA 1 blood type, its use is highly recommended.

Feline Blood Types

Traditional feline blood group classification is based on an AB system, where a cat is designated type A, B or, rarely, AB. The majority of domestic short- and long-hair cats in the United States are type A, but variability in prevalence exists across breeds and geography. Cats do possess clinically significant naturally occurring alloantibodies necessitating type-specific transfusion of RBC and plasma products. Type A cats have relatively weak or absent anti-B alloantibody titers (generally ≤ 1:32 and often ≤ 1:8), which can cause agglutination and hemolysis [1]. Type B cats have strong, high-titered (1:64 – 1:1024) anti-A alloantibodies, which can result in severe agglutination,

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acute hemolytic transfusion reactions, and neonatal isoerythrolysis [1]. Type AB cats do not possess anti-A or anti-B alloantibodies. In 2007, a novel feline RBC antigen, coined *Mik*, was identified after a hemolytic transfusion reaction occurred in a cat receiving AB-type compatible blood [2]. POC feline blood typing kits are available and are recommended for AB determination.

Blood Typing

The utility of determining blood type in small animals prior to transfusion is multifactorial. Type-compatible transfusions are necessary to improve the likelihood of RBC survival post-transfusion and to avoid sensitization. Blood type should be determined in both blood donors and recipients.

POC blood typing is available for DEA 1, 4, 5, and *Dal* in dogs (Protocols 66.1 and 66.2). More comprehensive canine blood typing and antibody screening is available in specialized and commercial laboratories. Because dogs do not possess naturally occurring anti-DEA 1 alloantibodies, the transfusion of DEA 1(+) cells to a transfusion naïve DEA 1(−) dog does not cause acute hemolysis (≤ 24 hours) but may lead to a delayed hemolytic transfusion reaction after a few days. This principle guides some practitioners to forgo recipient typing in first-time emergent canine transfusions despite the short post-transfusion RBC lifespan and consequential sensitization and incompatibility with future transfusions. DEA 1(−) dogs that have been transfused with DEA 1(+) RBCs are at risk for life-threatening acute hemolytic transfusion reactions with reexposure to DEA 1(+) blood. Because the consequences of DEA 1 mismatched transfusions are severe, type matching of canine transfusions is recommended. If the patient is unable to afford the time to obtain a blood type, the use of DEA 1(−) will prevent sensitization.

In cats, AB blood type determination is necessary to ensure type-specific transfusion of both RBC and plasma products, owing to the presence of clinically significant

Protocol 66.1 Canine (DEA 1, 4, 5, or *Dal*) Blood Type: DMS Card

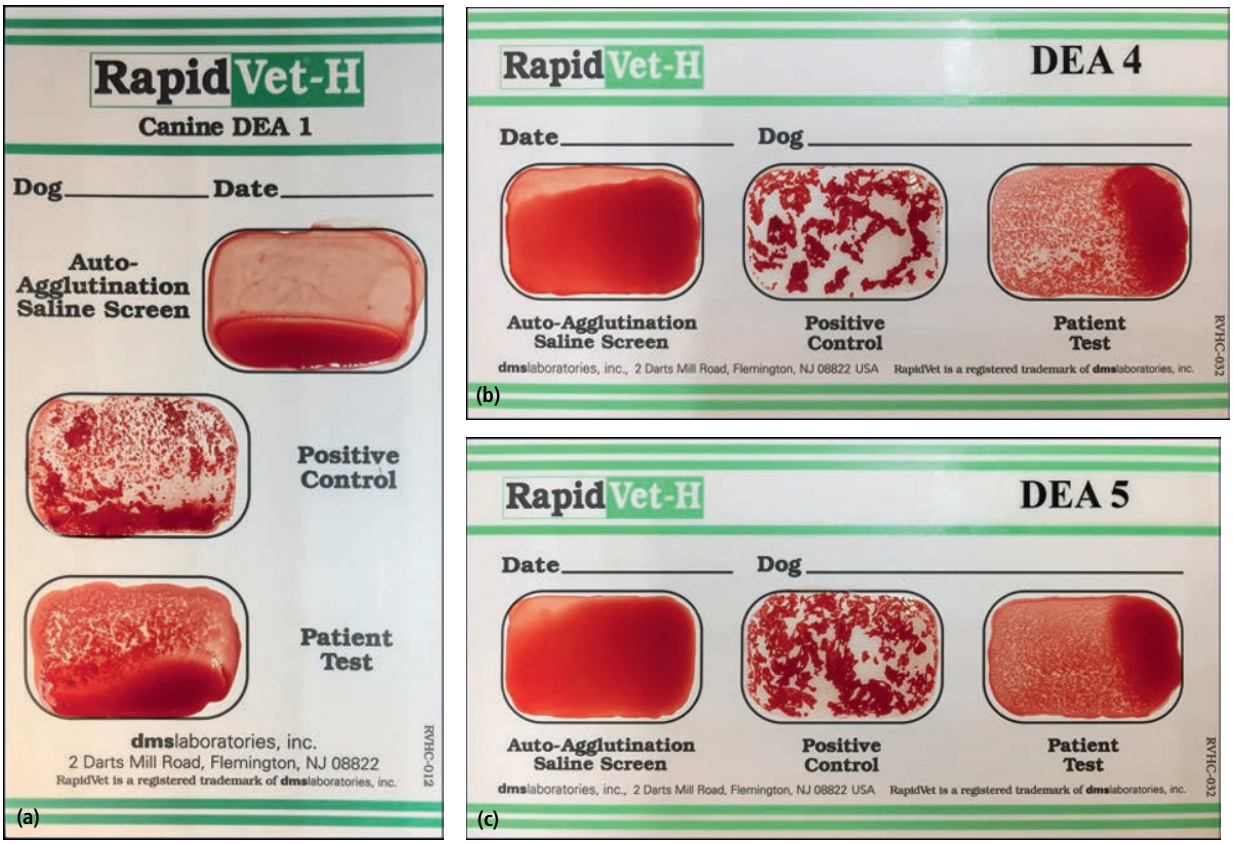


Figure 66.1 (a) Canine DEA 1 test with autoagglutination saline screen, positive control, and patient test wells. (b) Canine DEA 4 test with autoagglutination saline screen, positive control, and patient test wells. (c) Canine DEA 5 test with autoagglutination saline screen, positive control, and patient test wells. *Source:* Courtesy of DMS Laboratories, Inc.

Items Required

- Whole blood (in EDTA) – 150 µl is required for the test, but sample should be large enough for appropriate blood to EDTA ratio
- Rapid-Vet®-H (canine DEA1, 4, 5, or *Dal*) kit containing:
  - 50 µl pipette
  - Wooden stirrer
  - Diluent
  - Agglutination test card

Procedure

The procedures are the same for all four blood type kits.

Autoagglutination Saline Screen

1) Apply one drop of diluent and canine whole blood (in EDTA) to the well.
2) Mix thoroughly with the provided wooden stick for 10 seconds.
3) Rock card for 30 seconds to a slight angle looking for agglutination:
   a) If negative, proceed to typing.
   b) If positive, consider washing cells to retest.

Blood Type Test Wells

1) Apply one drop of diluent into the "Positive Control" and "Patient Test" wells.
2) Apply one drop of canine whole blood (in EDTA) into each well (patient, positive control).
3) Mix each test thoroughly with a wooden stick with downward pressure to agitate the lyophilized material in the well for 10 seconds; use a separate stick or stick end for each well so not to cross-contaminate.
4) Rock the card for one minute being careful to prevent spills outside of the wells; observe for agglutination.

Interpretation

- Autoagglutination saline screen:
  - Hemagglutination – the animal is autoagglutinating; wash cells.
  - No agglutination – proceed with blood type test.
- Positive control:
  - Hemagglutination – the test is functioning as intended.
  - No agglutination – the test is defective; contact manufacturer.
- Patient test:
  - Hemagglutination – the animal is positive for the DEA tested.
  - No agglutination – the animal is negative for the DEA tested.

Notes

- Store at room temperature (20–25°C) for 24 months.
- Agglutination characteristic between the control well and patient well will often look different.
- Fine, granular appearance can occur over one minute into testing and should be disregarded.
- Anemic samples can form pinhead-like aggregations of red cells instead of gross agglutination.

Protocol 66.2 Immunochromatographic (IC) Canine Blood Typing Test

Items Required

- Whole blood (in EDTA, ACD, or CPD) –10 µl is required for the test, but sample should be large enough for appropriate blood to anticoagulant ratio
- Quick Test DEA1 kit containing:
  - Collector strip
  - Buffer solution
  - Quick Test strip
  - Sample well

Procedure

1) Add three drops of buffer solution into sample well.
2) Collect anticoagulated whole blood with collector strip, and then suspend into the buffer solution by gently stirring the collector strip in the solution for seven seconds.
3) Remove the cover on the Quick Test strip and set the exposed membrane strip into the red cell suspension in a vertical position.
4) Allow the red cell solution to migrate up the strip and remove for interpretation in two to five minutes.

Interpretation

- Control: A red line showing at the "C" mark indicates proper function.
- DEA1 negative: a blank space at the "DEA1" mark indicates DEA1 negative (Figure 66.2a).
- DEA1 positive: a red line showing at the "DEA1" mark indicates DEA1 positive (Figure 66.2b).

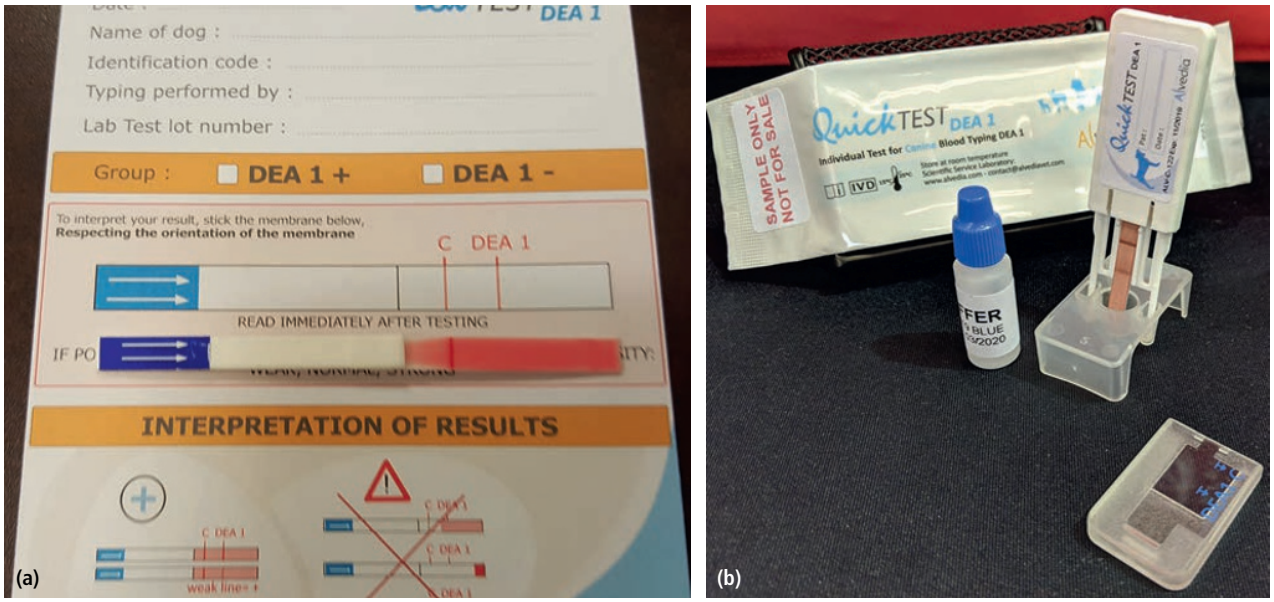


Figure 66.2 (a) Canine LabTest DEA 1 test strip with interpretation kit. This strip shows a DEA 1 negative result. (b) Canine QuickTest DEA 1 kit set up for demonstration. This kit shows a DEA 1 positive result. *Source:* Courtesy of Alvedia, Limonest, France.

Notes

- The IC test is reliable for testing through autoagglutination as the agglutinated cells will be retained at the bottom of the membrane.
- The IC band will be visible through low packed cell volume, although centrifuging red cells to collect packed cells with the collector strip will to help make the band more visible.
- Laboratory test kits containing bulk amounts are also available.

naturally occurring alloantibodies. POC is available for AB typing (Protocols 66.3, 66.4, 66.5, 66.6), but unavailable for *Mik* determination.

Blood typing methods detect visible in vitro hemagglutination of patient RBCs with known RBC antibody or antisera to determine blood type. There are three commercially manufactured tests available for canine and feline blood typing – the tube gel test, the card agglutination assay, and the immunochromatographic cartridge (IC) method. DMS Laboratories manufactures a one-tube gel test for identifying feline AB typing (DMS RapidVet®-H GEL, DMS Laboratories, Inc., Flemington, NJ) (Protocol 66.3). These one-tube gel tests are stored in the refrigerator and are designed to be run on a centrifuge with a fixed-angle rotor. Gel-tube methodology is an agglutination system that uses a reaction tube containing gel and antibody reactive to varying degrees with feline types A, B and AB whole blood. Gel-tube methodology is highly accurate demonstrating a high level of agreement with gold standard laboratory test tube typing [3, 4].

The card agglutination typing kits are available from DMS Laboratories for rapid determination of canine DEA 1, 4, 5, or *Dal* (Protocol 66.1), and feline A, B, or AB blood types (Protocol 66.4). The blood-typing card agglutination assay is based on a visible agglutination reaction when the sample RBC surface antigens interact with a known lyophilized monoclonal antibody impregnated on the typing card. Interpretation of the visible agglutination reaction in an RBC suspension can be subjectively graded on intensity from 0 (no agglutination) to 4+ agglutination. When typing canine blood donors for DEA 1, sensitivity is paramount to prevent misidentification of DEA 1(+) donors as DEA 1(−). Conversely, when typing recipients, maximum specificity in testing is necessary to prevent the administration of DEA 1(+) blood to a DEA 1(−) patient. When interpreting card agglutination tests for donors, sensitivity may be improved by interpreting any agglutination (≥ 1 agglutination) as positive [5]. When interpreting card agglutination tests for recipients, an agglutination cutoff point of ≥ 2+ improves specificity.

Protocol 66.3 Feline (A, B, AB) Blood Type: DMS One-Tube Gel Test

Items Required

- Whole blood (in EDTA) – 50 µl is required for the test, but sample should be large enough for appropriate blood to EDTA ratio
- Fixed-angle rotor centrifuge
- RapidVet®-H GEL feline kit containing:
  - 50 l µl pipettes
  - Blood preparation tube with diluent
  - Positive control gel tube
  - Patient test gel tube (reaction tube)

Procedure

1) Add one drop of blood to the blood preparation tube and gently invert several times.
2) Use a new pipette for steps 3 and 4 (same pipette can be used for both steps).
3) Transfer one drop of blood from the blood prep tube into the positive control tube. Increase to three drops if the animal's packed cell volume (PCV) is less than 15%. Recap without mixing.
4) Transfer one drop of blood from the blood prep tube into the reaction tube. Increase to three drops if the animal's PCV is less than 15%. Recap without mixing.
5) Allow both tubes to incubate at room temperature for five minutes.
6) Centrifuge at settings specified by DMS Laboratories.
7) Examine the tubes to interpret the results.

Interpretation

- Positive control tube: a red line of agglutinated cells at or near the top of the gel column indicates proper function.
- Reaction tube (Figure 66.3):
  - Type A – vast majority of red blood cells (RBCs) are found at the top of the tube.
  - Type B – vast majority of RBC are found at the bottom of the tube.
  - Type AB – RBCs are evenly dispersed within the tube.

Notes

- Store refrigerated at 2–7°C upright for a shelf life of 24 months. If not stored upright, set upright in refrigerator for 10 minutes before use.
- If autoagglutination is present, wash cells.

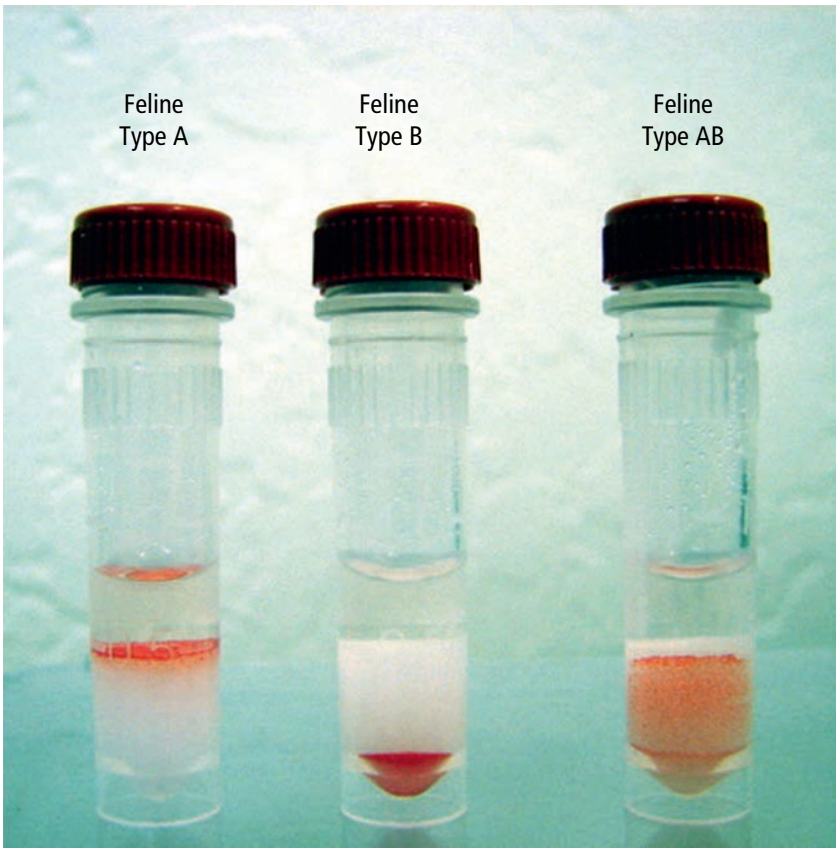


Figure 66.3 Result interpretation guide for RapidVet®-H Feline GEL. *Source:* Courtesy of DMS Laboratories, Inc.

Protocol 66.4 Feline (AB) Card Blood Type

Items Required

- Whole blood (in EDTA) – 150 µl is required for the test, but sample should be large enough for appropriate blood to EDTA ratio
- Rapid-Vet®-H (feline) kit containing:
  - 50 µl pipette
  - Wooden stirrer
  - Diluent
  - Agglutination test card

Procedure

Autoagglutination Saline Screen

1) Apply one drop of diluent and feline whole blood (in EDTA) to well.
2) Mix thoroughly with the provided wooden stick for 10 seconds.
3) Rock card for 30 seconds to a slight angle looking for agglutination.
   a) If negative, proceed to type A and B typing.
   b) If positive, consider washing cells to retest.

Blood Type Test Wells

1) Apply one drop of diluent into each of the two remaining wells labeled Patient Test.
2) Apply one drop of feline whole blood (in EDTA) into each of the wells.
3) Mix each test thoroughly with a wooden stick with downward pressure to agitate the lyophilized material in the well for 10 seconds; use a separate stick or stick end for each well so not to cross-contaminate.
4) Add a *second* drop of diluent to the well labeled type A. *Do not stir* the fluid in this well a second time.
5) Rock the card for two minute or less until hemagglutination has occurred in one of the Patient Test wells. Take care not to cross-contaminate the samples within the wells.

Interpretation

- Blood type A: strong hemagglutination in well labeled type A (Figure 66.4a).
- Blood type B: strong hemagglutination in well labeled type B (Figure 66.4b).
- Blood type AB: strong hemagglutination in wells labeled type A and type B (Figure 66.4c).

Notes

- Store at room temperature (20–25°C) for 24 months.
- Fine, granular appearance can occur over one minute into testing and should be disregarded.
- Severely anemic samples of type A can prevent formation of agglutination and is recommended to be run without using the diluent.

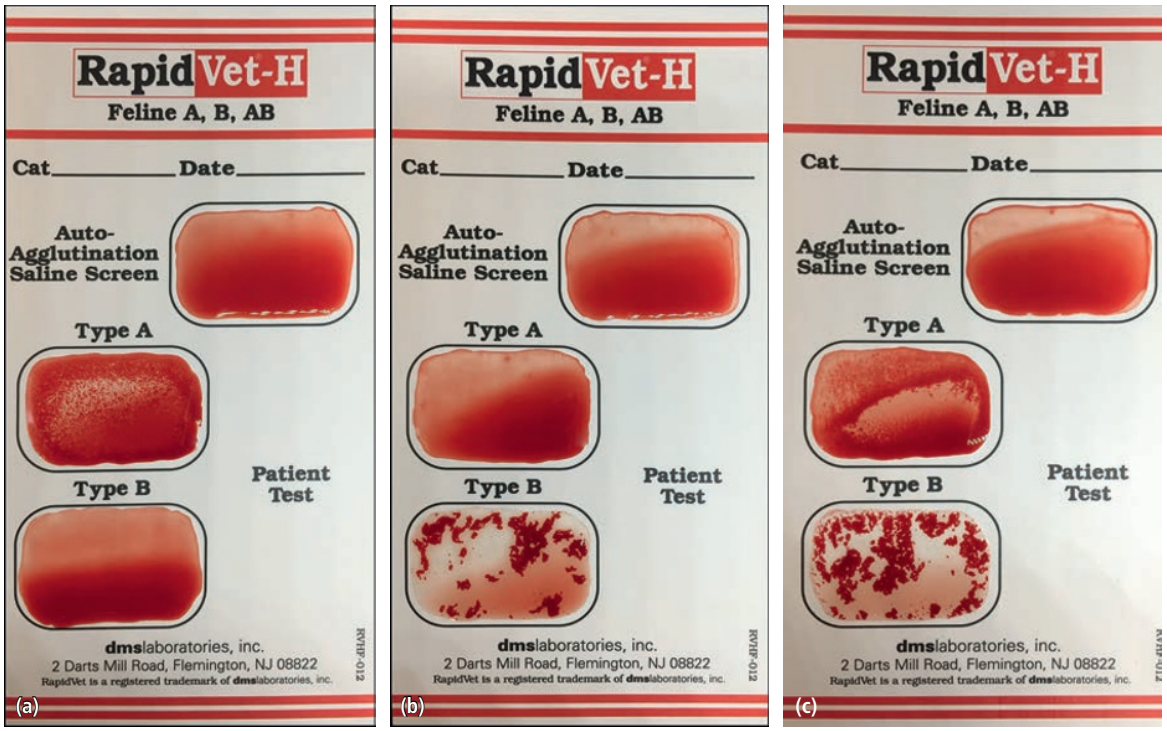


Figure 66.4 RapidVet®-H Feline Agglutination Card Test Kit. (a) Results of type A. (b) Results of type B. (c) Results of type AB. *Source:* Courtesy of DMS Laboratories, Inc.

Protocol 66.5 Immunochromatographic Feline Blood Typing Tests

(Alvedia® QuickTest A+B Kit)

Items Required

- Whole blood (in EDTA, ACD, or CPD) – 10 µl is required for the test, but sample should be large enough for appropriate blood to anticoagulant ratio
- QuickTest A+B kit containing:
  - Collector strip
  - Buffer solution
  - Quick Test strip
  - Sample well

Procedure

1) Add three drops of buffer solution into sample well.
2) Collect anticoagulated whole blood with collector strip, and then suspend into the buffer solution by gently stirring the collector strip in the solution for seven seconds.
3) Remove the cover on the Quick Test strip and set the exposed membrane strip into the red cell suspension in a vertical position.
4) Allow the red cell solution to migrate up the strip and remove for interpretation in two to five minutes.

Interpretation

- Control: a red line showing at the "C" mark indicates proper function.
- Type A: a red line showing at the "A" mark indicates type A (Figure 66.5a).
- Type B: a red line showing at the "B" mark indicates type B.
- Type AB: a red line showing at both the "A" and "B" marks indicates Type AB (Figure 66.5b).

Notes

- The immunochromatographic (IC) test is reliable for testing through autoagglutination as the agglutinated cells will be retained at the bottom of the membrane.

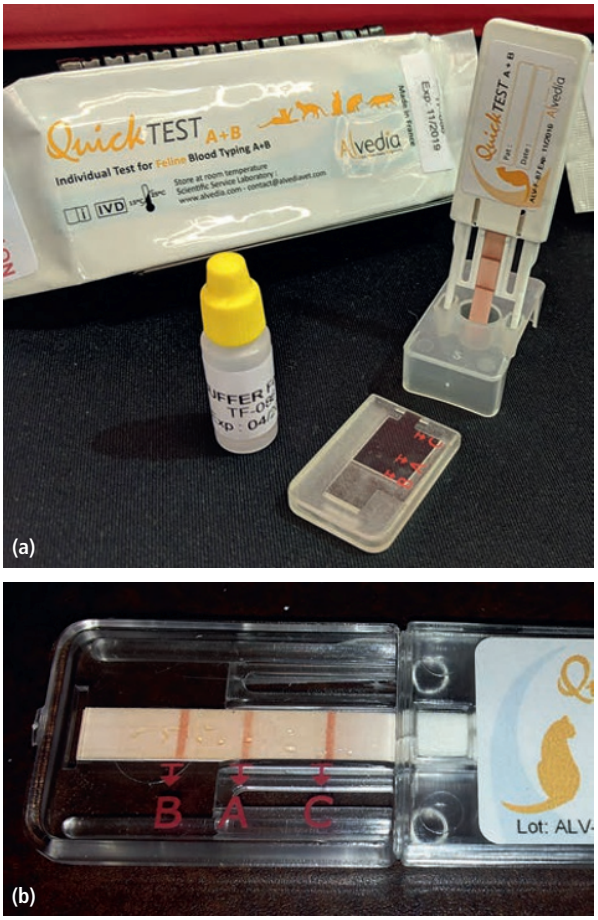


Figure 66.5 (a) Feline QuickTest A+B kit set up for demonstration. This kit shows a type A result. (b) Feline QuickTest A+B kit. This kit shows a type AB result. *Source:* Courtesy of Alvedia, Limonest, France.

- The IC band will be visible through low packed cell volume, although centrifuging red cells to collect packed cells with the collector strip will help make the band more visible.
- Laboratory test kits containing bulk amounts are also available.

Protocol 66.6 DMS Laboratories RapidVet®-H IC Feline Test Kit

Items Required

- Whole blood (in EDTA) – 30 µl is required for the test, but sample should be large enough for appropriate blood to EDTA ratio
- RapidVet-H immunochromatographic (IC) feline kit containing:
  - Red-top blood preparation tubes
  - Buffer solution
  - Pipettes
  - IC test device

Procedure

1) Pipette one drop of blood into the blood preparation tube, replace cap, and invert several times.
2) Using a new pipette, place three drops of diluted blood from prep tube into the sample port and allow several seconds to absorb.
3) Place three drops of buffer solution into the same sample port.
4) Rest the device on a flat surface and record test results based on visible lines formed after 5–10 minutes.

Interpretation

- Control: a red line showing at the "Control" window indicates proper function. Use a new kit and contact the manufacturer if no line is visible.
- Type A: a red line showing at the "Type A" window indicates type A (Figure 66.6a).
- Type B: a red line showing at the "Type B" window indicates type B (Figure 66.6b).
- Type AB: a red line showing at both the "Type A" and "Type B" window indicates type AB (Figure 66.6c).

Notes

- The IC test is reliable for testing through autoagglutination as the agglutinated cells will be retained at the bottom of the membrane.
- The IC band will be visible through low packed cell volume, although centrifuging red cells to collect packed cells with the collector strip will help make the band more visible.

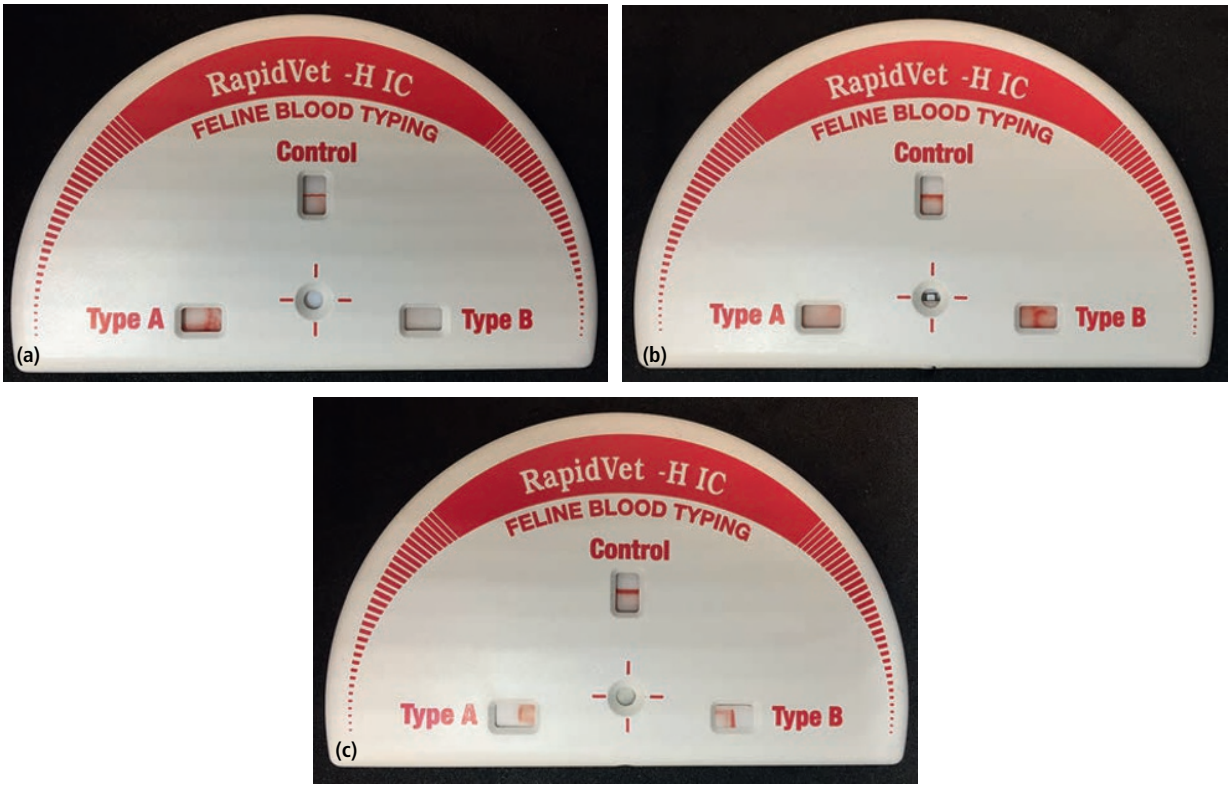


Figure 66.6 RapidVet®-H IC feline blood typing kit. (a) This test shows a type A result. (b) This test shows a type B result. (c) This test shows a type AB result. *Source:* Courtesy of DMS Laboratories, Inc.

The most recent technology in POC blood typing is the IC method. IC kits are available for DEA 1 (Protocol 66.2) and feline AB typing (Protocols 66.5 and 66.6). RBCs in an anticoagulated (EDTA) whole blood sample are mixed with a provided buffer solution and allowed to diffuse across a membrane containing monoclonal antibodies against a known RBC antigen (canine DEA 1 or feline AB) creating a visible line when positive for blood type. The IC methodology is reliable in patients with autoagglutination as agglutinated RBCs will be retained at the starting end of the membrane whereas non-agglutinated RBCs will migrate to the distal end of the dipstick membrane. In patients exhibiting autoagglutination, it is necessary to serially wash serum separated RBCs prior to performing the gel-tube and card agglutination methodologies for accurate results.

Crossmatch Considerations

Crossmatch testing determines in vitro pretransfusion compatibility between recipient and donor blood to ensure recipient safety and maximize transfusion product efficacy. The major crossmatch assesses for recipient plasma alloantibodies against donor RBC alloantigens. A major crossmatch may be used prior to administering any RBC-containing transfusion product, such as whole blood or packed RBCs (pRBCs). The minor crossmatch assesses for donor plasma alloantibodies against recipient RBC alloantigens. A minor crossmatch may be considered when transfusing any plasma containing product, such as whole blood or fresh frozen plasma, although minor crossmatches are rarely performed in veterinary medicine. Selected blood donors should have no history of prior transfusion and associated alloimmunization. For blood donors with unknown histories, antibody screening can be performed prior to admittance into the donor program. Because dogs do not possess naturally occurring alloantibodies associated with acute hemolysis, minor crossmatching is not indicated with the transfusion of plasma products [6]. The transfusion of AB-type specific plasma products from feline donors with no history of transfusion avoids the need for minor crossmatching.

The objective of crossmatch technology is to assess for hemolysis and/or hemagglutination. A positive crossmatch test exposes visible hemolysis and/or hemagglutination consistent with incompatibility. A negative test lacks hemagglutination (macroagglutination ± microagglutination depending on crossmatch methodology) or hemolysis and implies compatibility. Crossmatching is performed to prevent acute immune-mediated hemolytic transfusion reactions, but delayed hemolysis and other transfusion reactions cannot be predicted. The identification of acute hemolysis during in vitro crossmatching implies that acute hemolysis of donor cells would occur. The consequences of various degrees of agglutination with no hemolysis on in vitro crossmatching are unclear, although an immune reaction such as delayed hemolysis and shortened post-transfusion viability should be considered. When possible, the administration of fully compatible blood is recommended. Crossmatching evaluates the current immunological state of a recipient and should be performed just prior to transfusion.

A major crossmatch is necessary for all cats and dogs that have been previously transfused with an appropriate time interval for alloantibody formation. In dogs sensitized by transfusion, alloantibody formation has been demonstrated at four days [7] aligning with the recommendation to ensure crossmatch compatibility in dogs that have been previously transfused four or more days prior. In cats sensitized by transfusion, alloantibody formation has been demonstrated as early as two days post-transfusion (range 2–10 days, median 5 days) of AB-type compatible blood [8]. Further research is indicated to determine the optimal timing for post-transfusion crossmatching in cats.

The necessity of determining crossmatch compatibility in transfusion-naïve dogs and cats is debated. As dogs lack naturally occurring hemolyzing alloantibodies, namely anti-DEA 1 alloantibodies, an acute hemolytic transfusion reaction has never been described in a dog receiving a first-time transfusion. The Association of Veterinary Hematology and Transfusion Medicine (AVHTM) Transfusion Reaction Small Animal Consensus Statement (TRACS) suggests that major crossmatching may not be necessary in transfusion-naïve dogs [9]. Naturally occurring alloantibodies against other canine blood types (e.g. DEA 3, 5, and 7) have been identified with no evidence of acute hemolysis. The clinical significance of these naturally occurring canine alloantibodies is unknown as well as the ability of various crossmatching technologies to identify their presence, especially in low levels. The decision can also differ between dogs that are known to be transfusion-naïve as opposed to those with unknown transfusion histories. The discovery of clinically significant naturally occurring anti-*Mik* alloantibodies in some cats suggests the utility of assuring crossmatch compatibility in AB-type matched transfusion-naïve cats. Several contradictory studies have investigated pretransfusion compatibility in transfusion naïve cats, as well as outcomes in crossmatched versus non-crossmatched transfusions in naïve cats. The AVHTM statement suggests that major crossmatching be performed in addition to AB typing in transfusion-naïve cats [9].

Crossmatching Methods

Crossmatch testing is more time-consuming and technically challenging than blood typing. A variety of laboratory and POC crossmatching methods are available. In veterinary medicine, there is a lack of standardization in

crossmatch test procedures and test result interpretation. The standard tube agglutination crossmatch procedure is a laborious method used in many university veterinary laboratories. Tube agglutination crossmatching requires experienced trained personnel, such as licensed clinical pathology technologists, for optimal and consistent results (Protocol 66.7). Blood samples are incubated at body temperature (37°C) in the tube agglutination crossmatch procedure, while some institutions also evaluate at 25°C to evaluate for cold agglutinating antibodies (i.e. cold agglutinins). Because the tube agglutination crossmatch method is time consuming and reliant on highly experienced personnel, it has been replaced by gel column technology in many human blood banks as the gold standard. Gel column technology is easy to perform and standardized but requires specialized centrifuge equipment and specially prepared gel column test cards, often reserved for larger clinical laboratories. Canine and feline gel column test crossmatch kits (Alvedia®, Limonest, France) are available that require the Hettich EBA 270 centrifuge.

POC crossmatch testing kits available in veterinary medicine are RapidVet-H Crossmatch Test Kit (DMS

Protocol 66.7 Major Tube Crossmatch Procedure

Includes major crossmatch, recipient autocontrol, ± donor autocontrol.

Items Required

- Recipient whole blood (EDTA tube); ± recipient serum
- Donor whole blood (EDTA tube or stored blood bag segment, i.e. pigtail)
- Centrifuge (capable of 1000× G or 3400 rpm)
- 37°C incubator (heat block)
- 12 × 75 mm test tubes
- Disposable pipettes
- 0.9% or phosphate-buffered saline (NaCl)
- Timer
- Microscope, microscope slides, coverslips (optional)

Procedure

1) Centrifuge blood and separate plasma/serum from red blood cells (RBCs). Label tubes "Recipient RBCs," "Recipient serum/plasma," "Donor RBCs," "Donor plasma." If crossmatching to multiple donors, label tubes accordingly.
2) Prepare a suspension (~3%) of washed *donor* red cells in 0.9% saline:
   a) Washing – add around 3 ml saline to a small amount of RBCs (100 µl or around 2–3 drops), mix, and centrifuge (1000× G or 3400 rpm for 60 seconds). Decant the saline and repeat three times, filling the tube with saline, mixing, centrifuging and decanting leaving RBCs in tube.
   b) Preparing 3% suspension – add 3.2 ml of saline to washed RBC pellet. Mix thoroughly. Trained laboratory personnel may prepare suspension visually to approximate the appearance of red Kool-Aid®. See Figure 66.7a for appropriate RBC suspension color.
3) Prepare a suspension (~3%) of washed recipient red cells in 0.9% saline.
4) Label a tube for each donor (red cell suspension) being tested:
   a) Using a pipette, place two drops of recipient's plasma or serum to each tube.
   b) Using a pipette, add one drop of donor RBC suspension to each tube.
5) Label a tube recipient autocontrol:
   a) Using a pipette, place two drops of recipient's plasma or serum to the tube.
   b) Using a pipette, place one drop of recipient RBC suspension to the tube.
6) Label a tube donor autocontrol (performed if using donor EDTA sample):
   a) Using a pipette, place two drops of donor's plasma or serum to the tube.
   b) Using a pipette, place one drop of donor RBC suspension to each tube.
7) Mix all tubes gently.
8) Cover each tube or the entire group with plastic or Parafilm® to prevent condensation from entering any of the tubes.
9) Incubate all tubes at 37°C for 15–30 minutes.
10) Centrifuge (1000× G or 3400 rpm) the tubes for 15–20 seconds.
11) Read each tube and grade according to the chart below. Reactions should be read in a well-lit area, preferably with a white background to maximize visualization. A 3 × 5 magnification lens can be used, if needed.

Grading

1) Remove one tube from the centrifuge at a time, taking care not to dislodge the red-cell button.
2) Note the color of the supernatant. Free hemoglobin (red-tinged supernatant) greater than the amount in the original blood sample denotes hemolysis. Hemolysis is considered a positive reaction.
3) While viewing the tube and red cell button, gently shake the tube back and forth to dislodge the red cell button.

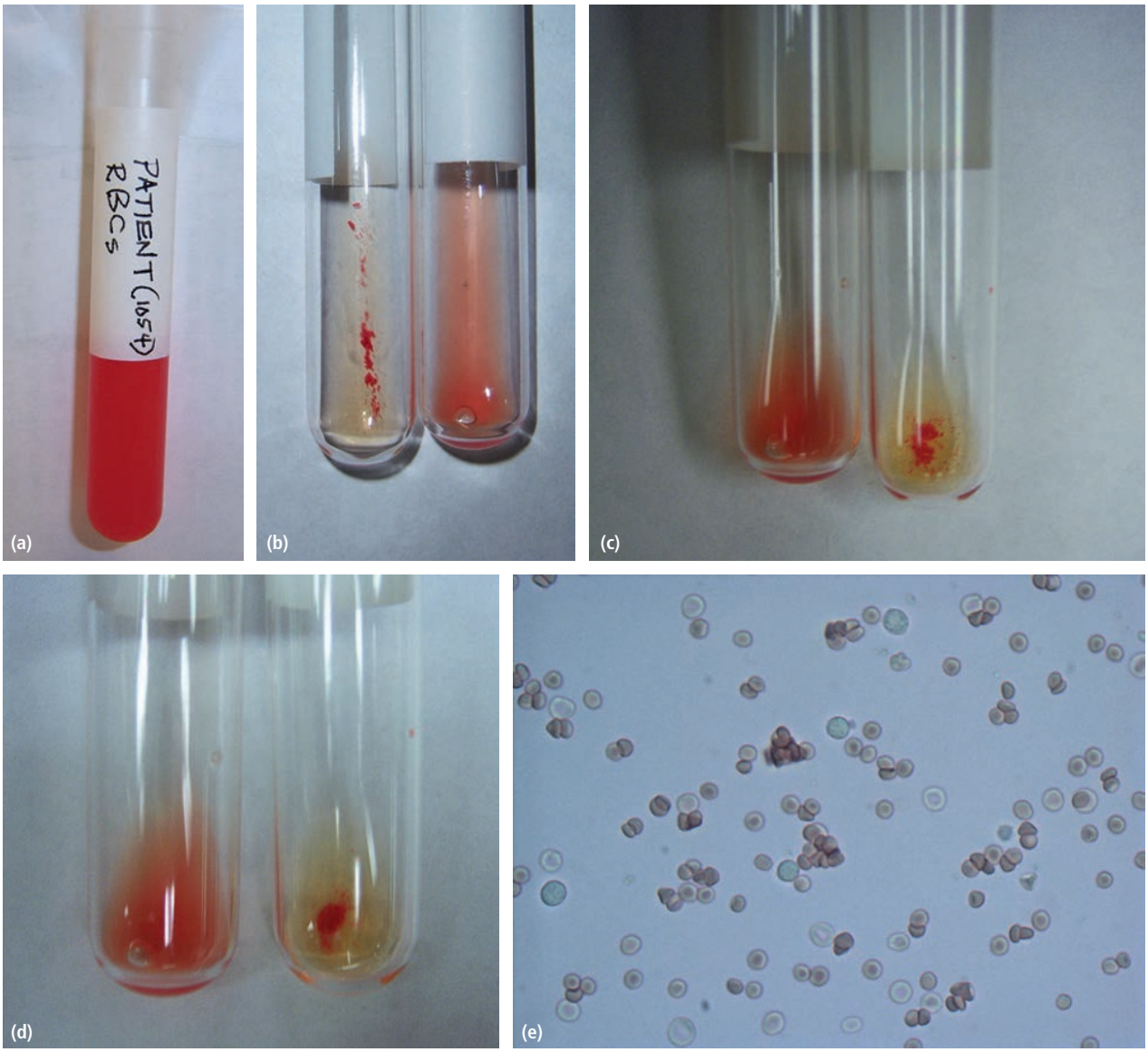


Figure 66.7 (a) Appropriate red blood cell suspension color. (b) A 2+ incompatible reaction is occurring in the tube on the left and the tube on the right (patient autocontrol) reveals no hemagglutination. (c) 3+ incompatible reaction in tube on right. Patient autocontrol (tube on left) is negative. (d). 4+ incompatible reaction in tube on right. Patient autocontrol (tube on left) is negative. (e) Positive microscopic hemagglutination, 40×.

4) Observe the manner in which the red cells leave the button.
5) Microscopic evaluation (optional): For samples lacking obvious macroscopic (visual to the naked eye) hemagglutination, a drop of the resuspended RBC-plasma mixture is placed on a microscope slide followed by a coverslip and evaluated at 40×.
6) Grade the reaction based on the following criteria:

Result	Description
Negative	No hemagglutination
Negative	Rouleaux (see below)
Positive	Hemolysis
Positive microscopic	Negative macroscopic Red cell aggregates (look like clusters of grapes, Figure 66.7e, rather than stacks of coins)

Result	Description
Weak positive	Minimal hemagglutination
1+	Small red cell aggregates
2+	Small and large red cell aggregates (Figure 66.7b)
3+	Many large red cell aggregates (Figure 66.7c)
4+	One solid button of red cells (Figure 66.7d)

Rouleaux

Suspected rouleaux ("stacked coin" appearance) must be confirmed by saline replacement as follows:

1) Recentrifuge the tube containing suspected rouleaux.
2) Remove the residual serum/plasma.
3) Resuspend the cells using two drops of 0.9% saline.
4) Centrifuge for 15 seconds.
5) Read and grade reaction as above. If hemagglutination persists, it suggests true incompatibility. If hemagglutination resolves, it confirms rouleaux.

Note: True red-cell antigen–alloantibody reactions will not disperse with the addition of saline.

Tube/test	Result	Interpretation
Recipient Autocontrol	Positive	Patient has formed autoantibodies against self RBCs; these autoantibodies may also cause hemagglutination of donor RBCs in vitro and/or in vivo. Recommend finding a compatible donor or the "least incompatible" donor (hemagglutination in major is less than in autocontrol).
Recipient autocontrol	Negative	No detectable autoantibodies.
Major (recipient)	Positive	Recipient of intended whole blood or pRBCs has alloantibodies against an antigen(s) on the donor RBC surface.
Major (recipient)	Negative	Recipient of intended whole blood or pRBCs is compatible with donor RBCs.
Rouleaux	Present	Hemagglutination that disperses following saline replacement suggests an increase in plasma proteins is causing RBCs to aggregate loosely. This is not thought to be of clinical relevance in transfusion of red blood cells.

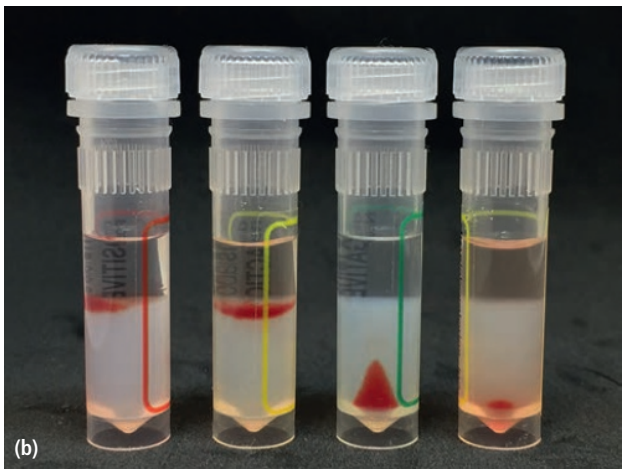
pRBC, packed red blood cells.

Laboratories) and Alvedia's IC crossmatch tests (Quick Test XM Canine®, LabTest XM Canine®, EmMa XM Feline®, LabTest XM Feline®, Alvedia, Limonest, France). The RapidVet-H Crossmatch Test is a modified gel-tube agglutination test where after room temperature incubation and centrifugation, the gel allows compatible RBCs to filter to the bottom of the tube, but traps agglutinated RBCs at the top or the near the top of the gel (Protocol 66.8). The DMS website provides complete instructions with illustrations, centrifuge specifications and photo identifiers of compatible and incompatible results. The Alvedia IC crossmatch cartridge tests (Protocol 66.9) are similar in methodology to their blood typing kits. Donor RBCs and recipient plasma are combined, allowed to incubate, washed three times and then the washed RBC pellet is combined with a second buffer solution and allowed to wick-up a membrane impregnated with anticanine antiglobulins in a detector line area. The anticanine antiglobulins can detect immunoglobulins G and M, and C3 complement components bound to donor RBCs, and trap these RBCs within the detector line, indicating incompatibility. A reported advantage of the IC methodology is reliability in patients with autoagglutination as agglutinated RBCs will be retained at the starting end of the membrane whereas non-agglutinated RBCs will migrate to the distal end of the dipstick membrane [10].

Veterinary studies comparing POC crossmatch kits with the laboratory standard tube agglutination crossmatch procedure as gold standard suggest that POC crossmatch kits lack sensitivity and may result in false negatives and the inadvertent administration of incompatible blood [11–14]. Conversely, it may be that the tube agglutination

Protocol 66.8 Companion Animal Crossmatch Test: RapidVet®-H





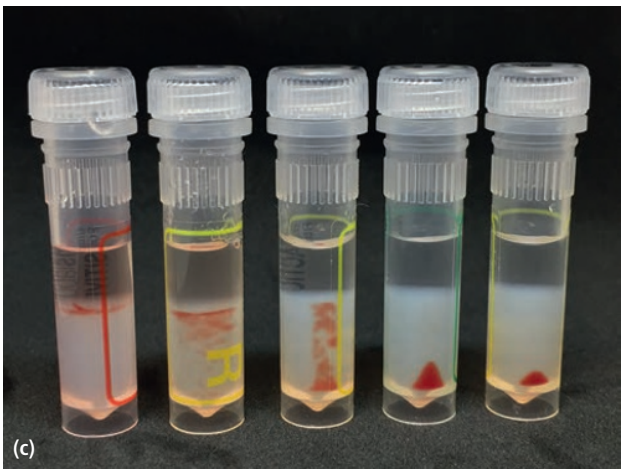


Figure 66.8 (a) RapidVet®-H Canine Crossmatch kit. (b) RapidVet-H Canine Crossmatch kit. Positive control (first tube, red line), positive reaction from a sensitized dog (second tube, yellow line), negative control (third tube, green line), negative reaction from a transfusion naïve dog (fourth tube, yellow line). (c) RapidVet-H Feline Crossmatch Kit Positive control (first tube, red line), A blood with B serum (second tube, yellow R), B blood with A serum (third tube, yellow line), negative control (fourth tube, green line), A blood with A serum (fifth tube, yellow line). *Source:* Courtesy of DMS Laboratories, Inc.

Items Required

- Recipient serum or plasma – 1.0 ml
- Donor blood sample – 0.1 ml (100 µl) EDTA anticoagulated whole blood *or* 0.05 ml (50 µl) packed red blood cells (pRBCs)
- Centrifuge (centrifuge list provided by DMS Laboratories with speed and time required specifications)
- RapidVet-H kit containing:
  - Test stand containing seven tubes
  - One pipette bag
  - Instructions, procedure diagram, photo result identifier, centrifuge specifications list, report card

Procedure

1) Gel tubes should remain upright at all times. A clean pipette must be used for every step to prevent contamination.
2) For each donor being tested, remove one test stand containing seven tubes, one pipette bag and one report card.
3) Write donor name/identification on all seven tubes.
4) Write recipient name/identification on yellow-top reaction tube and clear-top reaction gel tube (yellow-bordered labels).
5) Insert blue-top blood preparation tube upright into the well provided in the test stand.

6) Pipette donor sample – two drops (100 µl) whole blood *or* one drop (50 µl) pRBCs – to a blue-top blood preparation tube; cap tightly and gently invert several times to mix thoroughly. Place upright in the test stand.
7) Pipette four drops (200 µl) recipient plasma or serum to yellow top reaction tube.
8) From blue-top blood preparation tube, using a clean pipette for each transfer:
   a) Transfer two drops (100 µl) to yellow-top reaction tube. Replace cap, tighten, and gently invert several times to mix thoroughly.
   b) Transfer two drops (100 µl) to green-top negative control tube. Replace cap, tighten, and gently invert several times to mix thoroughly.
   c) Transfer two drops (100 µl) to red-top positive control tube. Replace cap, tighten, and gently invert several times to mix thoroughly.
   d) Incubate: let all tubes stand for five minutes at room temperature (20–25°C/68–77°F).
   e) Transfer one drop (50 µl) from yellow-top reaction tube to clear-top reaction gel tube (yellow-bordered labels). Cap tightly.
   f) Transfer one drop (50 µl) from green-top negative control tube to clear-top negative gel tube (green-bordered labels). Cap tightly.
   g) Transfer one drop (50 µl) from red-top positive control tube to clear-top positive gel tube (red-bordered labels). Cap tightly.
   h) Place gel tubes in centrifuge and spin for a cumulative G-force of 6500 (refer to crossmatch centrifuge list within kit or at DMS Laboratories (http://rapidvet.com) for speed and time settings for common models).
   i) Interpret and report results.

Tube	Result	Interpretation
Negative control tube	Negative	The negative control gel tube should demonstrate a collection of red blood cells at the bottom of the gel column (Figure 66.8b,c)
Positive control tTube	Positive	A red line of agglutinated red blood cells at or near the top of the gel column indicates proper function (Figure 66.8b,c)
Reaction tube	Negative	The vast majority of the red blood cells are at or near the bottom of the gel matrix and no firm line of agglutinated cells remains at the top of the gel (Figure 66.8b,c); a negative reaction suggests the donor is compatible
Reaction tube	Positive	The reaction tube demonstrates agglutinated red blood cells at or near the top of the gel column (Figure 66.8b,c); a positive reaction indicates an incompatible donor

Notes

- Store upright at room temperature for a shelf life of 24 months.
- A large number of cells suspended without a firm line of cells at the top is likely due to incompatibilities to something other than DEA1 (such as other DEA types).

Protocol 66.9 Immunochromatographic Canine Crossmatch Test – Quick Test XM Canine (Alvedia, Limonest, France)

See also Figure 66.9a (video tutorial available at: http://alvedia.com/movie-procedure-chromatography-tests).

Items Required

- Recipient plasma – EDTA, ACD, or CPD anticoagulants only; do not use heparin
- Donor whole blood – EDTA tube or stored blood-bag segment (pigtail) in ACD or CPD
- Centrifuge
- Timer
- Quick Test XM canine kit containing (Figure 66.9b):
  - One XM Quick Test
  - Two blood collector strips
  - One blue top buffer 1
  - One green top buffer 2
  - One wash buffer
  - One test tube (1.2 ml)
  - One empty well plastic stand
  - One pipettes (1 drop = 40 µl)
  - Instructions

Procedure

Takes around 25 minutes.

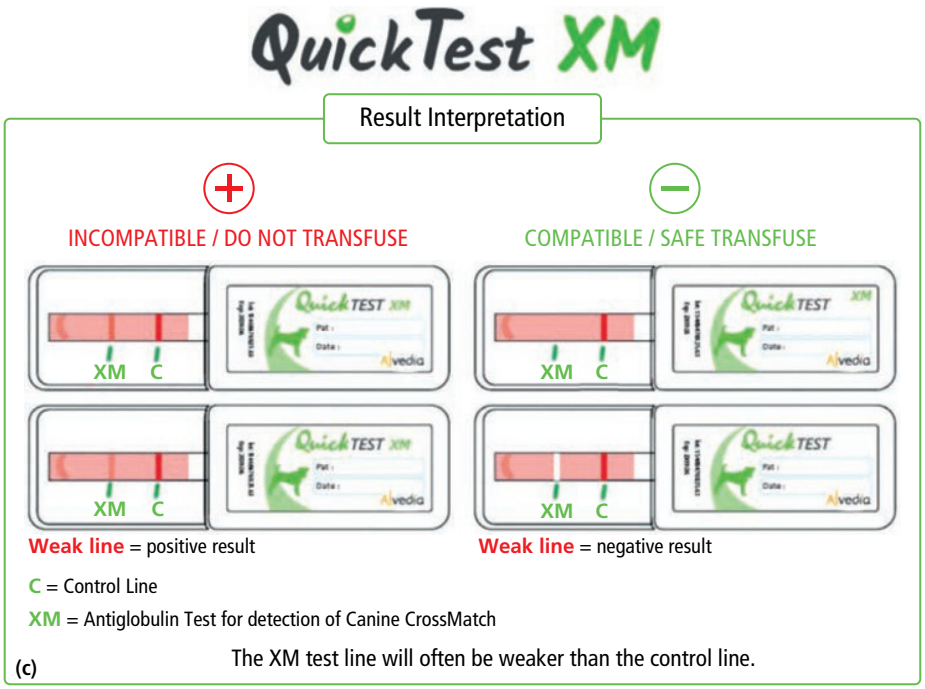
1) Preparation of blood samples:
   a) Donor EDTA tube – centrifuge EDTA blood tube (5 minutes at 1000× G). Discard the plasma to collect pRBCs. Donor blood-bag segment – collect packed red blood cells (pPRBCs).



(a)



(b)



(c)

Figure 66.9 (a) QuickTest XM test kit. (b) QuickTest XM test kit content. (c) QuickTest XM result interpretation guide. (d) QuickTest XM. Top – negative result (crossmatch compatible). Middle – strong positive result (crossmatch incompatible). Bottom – weak positive result (crossmatch incompatible). *Source:* Courtesy of Alvedia, Limonest, France.



(d)

Figure 66.9 (Continued)

b) Recipient plasma – centrifuge blood tube (5 minutes at 1000× G). Collect plasma.
2) Add four drops of buffer 1 in the test tube
3) Collect donor pRBCs with the blood collector strip, and then suspend into the buffer 1 solution by gently stirring the collector strip in the solution for seven seconds. Discard collector strip.
4) With the pipette collect the recipient plasma and transfer three drops in the test tube. Discard pipette and mix gently.
5) Incubate at room temperature for 10 minutes
6) Washing procedure – fill the test tube up to 1.2 ml with wash buffer. Mix the suspension three times minimum.
7) Centrifuge at 1000× G for two minutes. Be sure to counterbalance in centrifuge.
8) Discard the supernatant. The RBC pellet must stay at the bottom of the test tube.
9) Second washing procedure – fill the test tube up to 1.2 ml with wash buffer. Mix the suspension three times minimum to resuspend completely.
10) Centrifuge at 1000× G for two minutes. Be sure to counterbalance in centrifuge.
11) Discard the supernatant. The RBC pellet must stay at the bottom of the test tube.
12) Third washing procedure – fill the test tube up to 1.2 ml with wash buffer. Mix the suspension three times minimum to resuspend completely.
13) Centrifuge at 1000× G for two minutes. Be sure to counterbalance in centrifuge.
14) Using a new pipette, discard the entire supernatant (a few RBCs may be captured when pipetting the supernatant). Keep washed RBCs pellet to be used immediately for XM test procedure. Discard pipette.
15) Add three drops of buffer 2 into the empty well of the plastic stand.
16) Collect the RBCs pellet with the blood collector strip (~10 µl).
17) Dip the blood collector strip into the well to mix with buffer 2 solution, gently stirring the collector strip in the solution for seven seconds. Discard collector strip.
18) Remove the cover of the XM Quick Test strip and set the exposed membrane strip into the red cell suspension well in a vertical position.
19) Wait 5–10 minutes to allow complete migration up the membrane strip and remove for interpretation. You must see the Control line (C).
20) Read results (Figure 66.9c,d).

crossmatch procedure is too sensitive and identifying clinically irrelevant incompatibilities [11]. Further work is needed to determine what grade of tube agglutination crossmatch incompatibility is predictive of in vivo incompatibility and post-transfusion RBC viability. Continued research determining the accuracy of POC and laboratory crossmatch methods in predicting clinically relevant incompatibilities is warranted.

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67

Blood Transfusion

Julien Guillaumin and Kristin Kofron

Blood transfusion is common in small-animal practices, especially in multispecialty or tertiary centers. It is important for both clinicians and technicians to be familiar with the various types of blood products available, as well as their indications and adverse effects. Blood components can be used to improve tissue oxygen delivery with red blood cells (RBC), to replace blood proteins (e.g. coagulation factors, albumin) with plasma, or to replace blood volume in an exsanguinating patient [1–3]. Platelet products, used to replace depleted platelet stores, are used less commonly.

Blood component transfusion is privileged in blood banking. Separating fresh whole blood (FWB) in its various components allows for better individual use, improved storage and fewer adverse effects (Table 67.1) [4]. In dogs, the most common reason for using packed red blood cells (pRBC) is hemorrhage, and for plasma it is coagulopathy [5, 6]. In some circumstances, blood can be delivered as FWB, therefore providing all components at the same time.

In cats, both component therapy and FWB transfusion is common, although it is less documented than in dogs. Pretransfusion testing, especially the correct blood type and blood cross-matching, is important to limit the risks of transfusion reactions [7]. Reactions can vary from life-threatening hemolysis in cases of incompatible transfusions, especially in cats, to mild febrile reactions [4, 8].

Packed Red Blood Cells

Definitions

pRBC is the most commonly transfused blood product. pRBC is a refrigerated (2–6°C) stored product obtained after FWB fractionation. Storage medium consists of acid-citrate-dextrose, which allows for a 21 days shelf life or citrate-phosphate-dextrose-adenine, which allows for a 28–30-day shelf life. Other additives (e.g. Optisol®, Adsol®) can be added as a source of energy, increasing the shelf-life to 35 or 42 days, respectively [4].

Indications

The main indication for pRBC transfusion is to treat anemia and improve oxygen delivery. Oxygen delivery is cardiac output and arterial oxygen content (CaO_2), expressed as:

$$1.34 \times \left[\text{Hb} \right] \times \text{SaO}_2 + 0.003 \times \text{PaO}_2 \text{ g / dl}$$

where Hb = hemoglobin, SaO_2 = saturation of hemoglobin with oxygen in arterial blood, PaO_2 = partial pressure of oxygen in arterial blood.

Using normal values for each component, CaO_2 is equal to approximately 20 g/dl, with 19.7 g/dl being the oxygen present at the RBC surface, and only 0.3 g/dl being the dissolved oxygen. Thus, the RBC is the most important part of oxygen *transportation* in the blood, whereas PaO_2 becomes the most important part of oxygen *diffusion* to the cells, for use in cellular ATP formation. Transfusion of RBC also increases blood volume and can improve oxygen delivery by increasing stroke volume and cardiac output. In a retrospective study on over 3000 canine cases, the most common reason for pRBC transfusion was hemorrhage (68%), with neoplasia (36%) being the leading reason for hemorrhage before trauma (13%) and surgical blood loss (12%). Hemolysis was the major cause of anemia in those dogs (16%), with immune-mediated hemolytic anemia accounting for 90% of these cases [6].

The decision to transfuse is based on clinical signs [9]. If the anemia is chronic, compensatory mechanisms develop, aimed to maintain energy production. It includes maintenance of normal blood volume (through the renin-angiotensin-aldosterone and antidiuretic hormone

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Companion website: www.wiley.com/go/burkitt/monitoring

Table 67.1 Types of blood products, and their components, available in veterinary medicine.

Blood product	Contains
Fresh whole blood	Red blood cells, white blood cells, plasma, platelets
Packed red blood cells	Red blood cells, plasma
Fresh frozen plasma	Clotting factors, albumin, immunoglobulins, plasma
Frozen plasma (> 1 year or > 6 hours)	Clotting factors (except FV and FVIII), immunoglobulins, albumin plasma
Cryoprecipitate	Factors VIII, XIII, von Willebrand factor, fibrinogen (factor I)
Cryopoor plasma	Factors other than VIII, XIII, I, and vWF, including vitamin K-dependent factors
Platelet-rich plasma or platelet concentrate	Platelets, plasma

systems), improvement of PaO_2, local vasoconstriction directing blood flow to the most important organs, decreasing activity to lower oxygen consumption, and changes in cellular metabolism toward anaerobic ATP production (i.e. hyperlactatemia).

Clinicians can use various criteria to determine a patient's need for a pRBC transfusion [9]. First, age and comorbidity of the patient, as it can be related to the ability to develop compensatory mechanisms or increased oxygen consumption. Second, the chronicity of the anemia, directly related to the development of compensatory mechanisms, should also be considered.

Presence of clinical signs related to the anemia is a common criterion for clinicians to decide whether a transfusion is needed. However, it is a challenging criterion, as 100% of anemic patients will have clinical signs, varying from mild (e.g. lethargy, pale mucous membranes) to more severe (e.g. syncope, severe tachycardia, hyperlactatemia). With the risks associated with pRBC transfusion, it should be done when clinical signs are more severe, for example in a failure in the maintenance of energy production, usually represented by hyperlactatemia. Presence of blood loss and hypovolemia is different than the euvolemic, hemolytic anemia patient. Euvolemic patients have more risks of fluid overload when transfused. Hypovolemic bleeding patients benefit the most from a blood transfusion to expand the blood volume and provide oxygen carrying capacity. Several criteria are used in the decision to transfuse, the cause of anemia (e.g. the transfused RBC will likely be destroyed by a hemolytic patient), the risks associated with transfusion, including screening availability (i.e. blood type and crossmatch) or history of previous transfusion, and the presence or absence of a regenerative response, as the absence of regenerative response makes the patient at risk for more prolonged periods of anemia.

Listed as the last criteria on purpose is the hemoglobin (or hematocrit) level. In critically ill humans, pRBC transfusions are recommended at a hemoglobin level of 7 g/dl (hematocrit of 21%), decreased for the hemoglobin of 10 g/dl (hematocrit of 30%) rule, unless the patient has clinically significant heart disease, severe hypoxemia, or acute hemorrhage [10, 11].

Dose

Studies showed that dogs receiving a pRBC transfusion have an average hematocrit of 18% [6]. The usual goal of transfusion is to abate clinical signs, and reach a safer hematocrit, usually between 25% and 30%. Therefore, a hematocrit increase of around 10 points is usually needed. The current belief is that 1.5 ml/kg is required to increase 1 point hematocrit [12], which is a higher volume that the previous dogma that 1 ml/kg will increase 1 point of hematocrit. Clinicians may also use the following formula to predict a volume of pRBC (ml) to transfuse:

$$\text{Volume} = \text{body weight (kg)} \times \text{blood volume (90 ml)} \times [(\text{desired PCV} - \text{recipient PCV})/\text{donor PCV}]$$ [12].

If using FWB, the calculation uses 2–3 ml/kg FWB to increase hematocrit by 1 point.

Blood Types and Pretransfusion Testing

Eight dog erythrocyte antigens (DEAs) exist, with DEAs 1.1, 1.2, and 7 being clinically significant. It has been shown that the DEA 1 group is a continuum from negative to strongly positive antigen expression, meaning that previously typed DEA 1.2-positive and 1.3-positive are just DEA 1-positive [13]. The rule of thumb in canine pRBC transfusion is that dogs do not have naturally occurring antibodies against DEA 1 blood type, but they will develop anti-DEA 1 antibody when in contact (i.e. transfused) with DEA 1-positive RBCs. Those antibodies will cause agglutination and hemolysis if DEA 1-positive blood is transfused again. Put in simple words, dogs may have a "free pass" on the first transfusion of RBCs. However, dogs have naturally occurring antibodies for some DEA types that may result in delayed reaction (e.g. DEA 3, DEA 4, DEA 5, and DEA 7).

Testing for canine blood types other than DEA 1.1 is controversial and complicated by reagent availability. A new common blood type named Dal has been identified in Dalmatians, with a presumed prevalence of 20%. Dalmatians lacking the Dal antigen are likely at risk of acute and delayed hemolytic reactions [14]. Although initially described in the Dalmatian breed, the prevalence of

Table 67.2 Prevalence of blood types and naturally occurring antibodies.

DEA group	Population prevalence (%)	Presence of naturally occurring antibody	Transfusion significance
1[a]	62	No	Acute hemolytic reaction (<12 hours)
3	6	Yes (20%)	Delayed reaction (i.e. decreased RBC survival)
4	98	No	None
5	23	Yes	Delayed reaction (i.e. decreased RBC survival)
6	98–99	No	Unknown
7	45	Yes (20–50%)	Delayed reaction (i.e. decreased RBC survival)
8	40	No	None

[a] Previously known as 1.1 and 1.2. The blood group system DEA 1 is a continuum from negative to strongly positive antigen expression, meaning that previously typed DEA 1.2+ and DEA 1.3+ appears to be DEA 1+ [13].
DEA, dog erythrocyte antigen; RBC, red blood cell.
Source: Adapted from Hale [16].

dogs lacking the Dal antigen (i.e. Dal negative) and risking a transfusion reaction, is actually higher in Shih Tzu (almost 60%) and Doberman Pinscher (over 40%), with other small breeds such as Lhasa Apso and Bichon Frise between 20% and 30% Dal negative [15]. The prevalence of blood types and naturally occurring antibodies presence is shown in Table 67.2. No anti-Dal alloantibodies were detected in plasma of the 23 Dal-negative dogs tested without prior transfusion history in a 2017 study [15]. It is recommended that the blood type of all donors and recipients be known prior to transfusion (for at least DEA 1) so type-specific blood can be administered.

With the findings regarding the Dal antigen, and although we used to say that dogs could get a "free pass" for their first transfusion, clinical practice is changing. In a 2017 study, it was shown that 17% of 148 of transfusion-naïve dogs were incompatible with one or two of the three potential donors. Interestingly, the change in hematocrit after transfusion was significantly higher in dogs whose blood had been cross-matched (12.5 ± 8.6%) compared with dogs whose blood was not cross-matched (9.0 ± 4.3%) [17]. These findings suggest that incompatibility can still happen in DEA 1-compatible patients.

It is important to recognize that blood typing and cross-matching are answering two different questions. Blood typing technically only answers the question, "what is the patient's DEA 1 status?" We cannot tell whether a patient will develop strong anti-DEA 1 antibody if transfused with DEA 1-positive blood leading to a strong, acute hemolytic reaction if transfused again with DEA 1-positive blood.

Cross-matching is done by mixing the RBCs of the donor with the plasma of the recipient (major cross-match) or the RBCs of the recipient with the plasma of the donor (minor cross-match) and observing for agglutination [4]. Cross-matching answers the question regarding current compatibility between a specific donor and a specific recipient blood type "profile." Because of current concerns regarding Dal antigen and delayed hemolytic reactions, it may be prudent to both blood type and cross-match canine patients, regardless of whether they have already received a blood transfusion, as recommended in cats.

Cats have three major blood types A, B, and AB. A is the most common blood type (95–99% of cats in the United States). Type B is less common in domestic breeds but is seen with more frequency in other breeds (e.g. Devon Rex, British Shorthair) or in other geographic locations (e.g. Turkey). Cats have naturally occurring circulating alloantibodies targeting the blood type they do not carry. Type B cats have more antigenic anti-A antibodies, and transfusion of a type B cat with A blood could result in severe hemolytic transfusion reaction. Type A cats have fewer antigenic anti-B antibodies, so transfusion of a type B blood to a type A cat may result in lower lifespan of the transfused cells. Another erythrocyte antigen, Mik, has also be described and can result in acute hemolytic reaction due to circulating alloantibodies. Owing to the risk of reaction, it is recommended that all cats should be blood typed before transfusion. Because no test is available for detecting the Mik antigen, a cross-match is also recommended before transfusing a cat, even if the cat has not been previously transfused.

In Practice

pRBC are administered through a 170–210 μm filter, available in commercial blood delivery sets. pRBC are usually transfused over four hours in normovolemic patients, but can be transfused much faster, even as a bolus, in hypovolemic patients, such as the ones actively bleeding and exsanguinating. Blood should not be given concurrently with hyper- or hypotonic solutions. Calcium-containing solutions (e.g. lactated Ringer's solution) should also be avoided, as calcium may bind to citrate and "neutralize" the anticoagulant in the fluid line. It has been shown that it is preferable to use gravity instead of fluid pump to better preserve transfused cells [18].

Adverse Effects and Reactions

Transfusion reactions are usually divided in between acute immune-mediated transfusion reactions and nonimmune-mediated transfusion reactions (Table 67.3).

Table 67.3 List of transfusion reactions, clinical signs, and appropriate therapy.

Reaction	Type	Clinical signs	Treatment
Hemolytic	II hypersensitivity	Vomiting, hypotension, tachycardia, tachypnea, pyrexia	Stop the transfusion, treat symptomatically, intravenous fluids to promote diuresis
Anaphylactic	I hypersensitivity	Urticaria, pruritus, often with plasma products	Stop the transfusion, administer antihistamines or a small dose of steroids
Anaphylactic shock	I hypersensitivity	Cardiovascular collapse, dyspnea, seizures	Stop the transfusion, treat symptomatically with intravenous fluids, epinephrine
Leukocytes and platelet sensitivity		Increase in body temperature of at least 1°C	Stop the transfusion, may be restarted at a lower rate

Nonimmune-mediated reactions include hemolysis (e.g. inappropriate collection, storage, or administration), bacterial contamination, transfusion-associated circulatory overload, hypothermia (i.e. in case of rapid transfusion of inappropriately warmed products), citrate toxicity (causing hypocalcemia), transfusion-related immunomodulation, or transfusion-associated acute lung injury (TRALI), which has been described in humans. Although TRALI is the leading cause of transfusion-related morbidity and mortality in humans, it is caused by antibodies directed against human neutrophil antigens present in the plasma of predominantly multiparous female blood donors. It is thus unclear whether other species can develop TRALI. Leukoreduction (the removal of white blood cells, WBC, from pRBC) has been shown to decrease inflammation markers post-transfusion in experimental dogs, but not presently in clinical patients [19, 20].

Acute transfusion reactions to pRBC are documented in 15% of cases in a retrospective study of 136 dogs, with fever and vomiting documented in 53% and 18%, respectively [21].

Neonatal isoerythrolysis is possible in cats, but its presence in dogs necessitates a previously sensitized (i.e. transfused) bitch with an incompatible sire [22, 23].

Finally, the age of the transfused pRBCs may be a variable affecting the risk of transfusion-related hemolysis. In a retrospective study of 210 dogs, the age of stored pRBC products was associated with increased risk of transfusion-related hemolysis, but not with fever [24].

Plasma Products

Definitions

Fresh frozen plasma (FFP) is plasma separated from pRBC after FWB donation, and frozen within six to eight hours of collection or stored for less than one year at 30 degrees C or lower. Frozen plasma is FFP stored for more than one or two years, or frozen after six to eight hours of collection. Frozen plasma is thought to be depleted of co-factors V and VIII, although this human dogma has been challenged in veterinary medicine [25]. The maximum storage length is five years for fresh frozen or frozen plasma [4]. FFP can be further divided in cryoprecipitate and cryopoor plasma, because different factors have different freezing points. Cryoprecipitate is made by partially thawing FFP at 4 degrees C for 24 hours and removing the supernatant, which leaves the semisolid cryoprecipitate, containing factor VIII, fibrinogen and von Willebrand factor (vWf) [4]. The supernatant is cryopoor plasma, also known as cryosupernatant or cryodepleted plasma. Cryoprecipitate is indicated for bleeding associated with vWf deficiency or hemophilia A, as well as hypofibrinogenemia cases such as the exsanguinating patient, whereas cryopoor plasma is listed for use in vitamin K antagonist rodenticide toxicities and hypoalbuminemia [4]. Albumin therapy is covered in Chapter 68.

Indications

The major indication for FFP transfusion is coagulopathies (e.g. anticoagulant rodenticide intoxication, hemophilia A or B). FFP transfusion is considered to be inappropriate for treating increased clotting times without evidence of clinical bleeding (including for a minor procedure), or for α-macroglobulin replacement in pancreatitis, or antithrombin replacement in disseminated intravascular coagulation [4, 26]. A secondary indication is species-specific albumin replacement.

Dose

Considering that clotting times become increased when 70–80% of clotting factors are missing, and that a 10–20 ml/kg of FFP will provide a 10–20% replacement of clotting factors, the rule of thumb is to transfuse 10–20 ml/kg of FFP to patients with clinical bleeding due to a coagulopathy. When FFP is used as an albumin replacement

fluid, the dose is 20–50 ml/kg/day, or approximately 1 ml/kg/hour (depending on reference and formula used). It may predispose some patients to volume overload, and there is an increased cost of hospitalization. When FFP transfusion is provided in exsanguinating patients, where it is recommended to provide a one to one ratio of pRBC to plasma products [27]. In this case, the volume transfused may reach a total of approximately 20–40 ml/kg. The dose of cryoprecipitate is 12–20 ml/kg (corresponding usually to 1 unit /10 kg). The dose for cryopoor plasma is 10–20 ml/kg.

In Practice

After thawing in (ideally) a warm water bath at 37 degrees C (which usually takes around 35–45 minutes), FFP can be administered to patients over four hours in normovolemic patients, or faster if hypovolemia is present. If plasma product is used as a constant-rate infusion, for example for albumin replacement, it can be given as a rate of 1–2 ml/kg/hour [28].

Several methods have been described regarding thawing FFP. Although FFP has been thawed using a regular microwave oven using approximately 15 cycles of 10 seconds, and has been shown to have similar activated partial thromboplastin times, one-stage prothrombin times, concentrations of fibrinogen, factor VIII coagulant activity, and vWf antigen levels to aliquots thawed in a warm water bath, it is not a preferred method of thawing [29]. One study has shown that a running water bath had the shortest thaw time (i.e. 15 minutes) compared with a dry plasma thawer or warm water bath, while maintaining levels of clinically similar hemostatic proteins [30]. Finally, one study has shown that using commercial microwave plasma defrosters allows thawing in three to five minutes while maintaining clinically relevant activities of clotting factors and fibrinogen concentration, although no control group was used and some measurements of factor VIII activity fell below the reference interval [31].

Finally, the hemostatic activity of liquid phase (i.e. refrigerated) canine plasma has been investigated [32]. This product is used in humans with catastrophic hemorrhage needing massive transfusion. The study showed that refrigerated storage resulted in significant decreases in the activity of all clotting factors, although no values were outside of the reference interval. Importantly, none of the bacterial cultures at days 7 and 14 yielded growth [32].

Adverse Effects and Reactions

Transfusion reaction to plasma has been documented in a retrospective study of more than 300 canine plasma transfusion. The only adverse effects were fever, pruritis, and anxiety. Adverse effects were noted in less than 1% of cases [5].

Platelets

Definition

Platelets are extremely fragile, and their viability may be compromised with manipulation. The gold standard in human medicine is therefore platelet concentrate harvested from FWB kept at room temperature under constant agitation for up to five days [33]. The American Association of Blood Banks currently recommends as a cut-off point greater than 5.5×10^{10} platelets/unit of random donor platelets (i.e. 50–60 ml) [34]. The US Food and Drug Administration (FDA) decreased the expiration date for room-temperature stored platelets from seven to five days in 2016, except when the platelets are stored in a container approved by FDA for seven-day storage and the individual platelets units are tested for bacterial detection [35]. Although a platelet-concentrate product is commercially available in veterinary medicine, because of the nature of the need for platelet concentrate many veterinarians mostly rely on FWB, fresh platelet-rich plasma (PRP) or fresh platelet concentrate [36]. Any fresh product must be used within six to eight hours.

Platelets can be harvested following a PRP protocol (i.e. hard and soft spin) or a buffy coat technique [37]. In veterinary medicine, the comparison of both methods showed that the PRP-derived platelet concentrate had lesser white blood cell and pRBC contamination and superior platelet function compared with the buffy coat technique [37]. Platelets can also be harvested by pheresis (a standard of $> 3 \times 1011$ platelets/unit of apheresed platelet concentrate, with a volume around 300 ml) [38]. During the platelet pheresis procedure, blood is removed from the donor through an extracorporeal circuit, anticoagulated and separated into components by centrifugation, allowing production of a platelet concentrate, while the other blood components are returned to the donor. The advantages of platelet concentrates prepared by apheresis in comparison with PRP or from a unit of FWB are greater platelet yield and negligible pRBC and WBC contamination [36]. Apheresis has also been used in veterinary medicine [39–42]. Preservation of platelets using cryopreservation or freeze–dry cycle (i.e. lyophilized) are being investigated. A lyophilized canine platelet product is commercially available but its hemostatic activity is unknown [41]. In humans, it appears that some cryopreserved or lyophilized platelets may preserve some hemostatic activity through microparticles [43].

Table 67.4 Platelet products that are available in veterinary practice, approved by the US Food and Drug Administration in humans and current alternatives to fresh platelet products, either available commercially or in development.

Product	Storage	Advantages	Disadvantages
Fresh whole blood	6–8 hours	May be the only option; should have good post-transfusion platelet recovery and function.	Donor availability Lack of time for donor testing Short shelf-life
Fresh platelet rich plasma or platelet concentrate	5–7 days	Good post-transfusion platelet recovery (dog, 80%), survival (dog, half-life 3.8 days), and function; FDA approved in humans; commercially available in veterinary medicine	Short shelf-life Limited availability Risk of bacterial proliferation during room temperature storage
Cold stored platelets	Up to 3 days	Decreased risk of bacterial proliferation; do not require bacterial testing; can be stored with agitation	May be cleared more rapidly compared to fresh platelets FDA approval restricted only for the resuscitation of actively-bleeding trauma patients
Canine lyophilized platelets Stable Plate RX™	Shelf life is 1 year	Long term storage; commercially available (BodeVet Inc., Rockville, MD)	Reduced post-transfusion platelet recovery (dog, 49%) and half-life (dog, 2 days); impaired *in vitro* function, although evidence of hemostatic efficacy in vivo
Cryopreserved platelets	Shelf life not available on package insert	Long-term storage; commercially available (Animal Blood Resources International, Stockbridge, MI)	Lack of preclinical or clinical data
Synthoplate™	Unknown	Long-term storage; not species specific (synthetic nanoparticles); commercially available (Haima Therapeutics, Cleveland, OH)	Lack of preclinical or clinical data; not available for sale

Various platelet products characteristics are presented Table 67.4 [36]. There are minimal clinical data regarding the use of fresh platelet products in veterinary medicine.

Indications

Historically, many physicians have transfused platelets to maintain platelet count above 20×10^9/l, with the belief that this level was required to prevent spontaneous bleeding [44]. However, there is difficulty in defining an optimal transfusion trigger because major hemorrhage is rare, even at very low platelet numbers (i.e. $< 5 \times 10^9$/l), minor clinical bleeding is difficult to quantify, and platelet counts are less accurately determined at the low platelet numbers found in severely thrombocytopenic patients, making the distinction between 5×10^9 and 20×10^9/l challenging [44]. Unfortunately, even in human medicine, there is no clear consensus of clinical indications for *prophylactic* platelet transfusion, only guidelines [38, 44, 45]. There is little evidence for the effectiveness of *therapeutic* platelet transfusions or the optimal dose when a patient with thrombocytopenia is actively bleeding [45].

There is also no consensus in veterinary medicine, partly because platelet transfusion is challenging in veterinary medicine. Most clinicians recommend platelet transfusion for severe, life-threatening bleeding due to thrombocytopenia, with intracranial bleeding or pulmonary bleeding being the principal indications [36, 46]. Prophylactic platelet transfusion is rare in veterinary medicine. Also, in clinical practice, bleeding secondary to thrombocytopenia is caused in the vast majority of cases by immune thrombocytopenia, where it is unclear whether transfused platelets will survive [46]. However, in dogs with immune thrombocytopenia experiencing uncontrolled or life-threatening bleeding (e.g. suspected bleeding into the brain, myocardium, or lungs), platelet transfusions may provide short-term hemostasis despite a negligible increase in platelet count following transfusion [36].

Platelet numbers commonly quoted as a transfusion trigger that put the veterinary patient at risk for spontaneous bleeding are $5–20 \times 109$/l [36, 46]. It is worth noting that the common transfusion trigger of 20×109/l comes from a case report/small case series in human cancer patients from 1962 [47]. Besides the platelet count, the presence of active bleeding (e.g. dropping hematocrit, visible bleeding) in life-threatening places (e.g. lungs, brain), or invasive procedures are indications for platelet transfusion [4, 36, 48].

In a veterinary study investigating the use of lyophilized platelets, dogs were eligible to receive platelet products if they had a platelet count of less than 70×109/l and evidence of active bleeding [41]. In a retrospective study on

the use of cryopreserved platelet concentrate in 43 dogs, indications were retrospectively divided between prophylactic (i.e. $< 10 \times 10^9$/l in absence of other risks factor for bleeding, or $< 20 \times 10^9$/l if other risks factors such as disseminated intravascular coagulopathy, sepsis, or elevating clotting times) and therapeutic (i.e. $< 50 \times 10^9$/l prior to surgery or $< 60 \times 10^9$/l with major bleeding requiring RBC transfusion) [49].

Dose

Most consensus recommendations quote 1 unit of platelet concentrate/10 kg body weight as their platelet dose, which the author assumed consisted of a pooled unit (i.e. four to six whole blood units) of whole blood-derived platelets, containing greater than 4×10^{10} platelets/pooled unit, for a final volume of 300 ml, or one apheresis platelet unit, containing greater than 3×10^{10} platelets/unit, also for a final volume of 300 ml [38, 44].

For a "typical" human being of 70 kg/2 m^2, the low dose corresponds to a 3×1010 platelets/10 kg, and the high dose would be around 10×1010 platelets/10 kg. Dosage recommended by the package insert for the lyophilized platelet product StablePlate Rx™ is 3×1010 particles/10 kg.

In therapeutic platelet transfusion, the goal may not be a platelet increment but an improvement in clinical bleeding [48].

In Practice

As many in veterinary medicine rely on FWB for platelet transfusion, it requires the availability of a blood donor and blood typing and/or cross-matching precautions, as pRBC will also be transfused. The product should be transfused rapidly after donation and procession, usually within six to eight hours after the blood donation. As the dose is the equivalent of 1 unit/10 kg of platelet concentrate, which comes from 1 unit FWB, then the dose of FWB is 1 unit/10 kg, or around 40 ml/kg. This represent both an unsurmountable number of blood donors for a large dog, but also increases the risk of blood overload for the patient. Finally, the clinician should expect no increase in platelet count following a FWB transfusion, and sometimes a decrease due to hemodilution, as the platelets will be immediately consumed during hemostatic processes. However, a decrease in clinical bleeding should be expected if the transfusion is successful. In humans, 1- and 24-hour increments are usually measured.

Reactions

In humans, acute transfusion reactions associated with platelet transfusion are three times more frequent than with pRBC transfusion [50]. In humans, 2% of the platelet transfusions were associated with a severe reaction defined as increase in temperature of more than 2 degrees C, shaking, chills, extensive urticaria, dyspnea, cyanosis, or bronchospasm [51]. In veterinary medicine, a canine study using lyophilized platelet concentrate, 14% of the lyophilized groups had a possible mild transfusion reaction, including a 2-degree F (1°C) rise in rectal temperature, sinus tachycardia, and one episode of emesis. Thirteen percent of the dogs receiving fresh platelets had a mild transfusion reaction, one dog developed urticaria and periorbital swelling and one experienced emesis. No delayed transfusion reactions were recognized [41].

The main risks of platelet transfusion are the role of leukocytes and platelets in acute inflammation, as well as bacterial contamination. Febrile, nonhemolytic transfusion reactions, defined as a temperature increase of greater than 1 degree C associated with transfusion and without any other explanation, are often accompanied by chills or rigors and occur with a reported frequency of up to 38% of platelet transfusions [34, 36].

Because platelet concentrate is stored at room temperature, contaminating bacteria introduced during phlebotomy or, less likely, during blood processing or transient donor bacteremia, may rapidly proliferate with administration of the contaminated unit potentially causing transfusion-associated sepsis. A study investigating more than 100 000 platelet units found 50 contaminated units. Forty-two of those were transfused, resulting in 16 septic transfusion reactions, including 1 fatality [52]. However, since then, interventions such as leukodepletion, the use of male donor plasma, irradiation and bacterial screening, have significantly reduced the risk of harm from platelet transfusions [53].

Alternatives to Platelet Transfusion

Owing to the lack of availability of platelet transfusion, clinicians have been relying on pRBC or plasma transfusion as alternatives. The rationale for use of pRBC is that anemia increases bleeding times, which can be corrected with reversal of the anemia [36, 54]. Proposed mechanisms for the role of pRBC in hemostasis include the relocation of platelets from the center of the blood vessel toward the vessel wall, improving contact between platelets and endothelial cells; improvement of platelet function through release of ADP and increased production of thromboxane, as well as scavenging of endothelial cell nitric oxide (inhibiting platelet function) [54]. It has been suggested that a PCV above 20% could be an appropriate target [55].

The use of FFP, as source of microparticles, has also been advocated [56]. This strategy can be implemented for anemic and/or hypoproteinemic patients, especially with

non-life-threatening bleeding secondary to thrombocytopenia. This includes surgical or gastrointestinal bleed. The use of desmopressin (1-deamino-8-D-arginine vasopressin, abbreviated DDAVP), as a platelet function stimulator, and the use of antifibrinolytics such as aminocaproic acid and tranexamic acid, has also been advocated [57].

Preservation of platelets using cryopreservation or lyophilization have been investigated in both veterinary and human medicine. A commercial dimethyl sulfoxide-stabilized frozen canine platelet concentrate is available [53], although in vitro studies indicated that there was a decrease in platelet quantity and function as well as an increase in platelet activation during the freeze-and-thaw process [58, 59]. However, it is possible that frozen platelets retain some hemostatic activity through microparticles [43]. Ng et al. retrospectively reported the clinical use of the commercially available cryopreserved PC in 43 dogs and compared it to 43 control dogs [49]. Although there was a statistically significant increase in platelet count after transfusion and the transfusion was well tolerated, the cryopreserved platelet concentrate was not found to be effective in improving clinical bleeding or increasing survival compared with the control group [49].

A lyophilized canine platelet concentrate, Stable Plate RX™ (BodeVet, Rockville, MD), is also commercially available. The improvement of preparation and fixation of lyophilized platelets have led to a renewed interest in such product. Lyophilized platelets has been tested in rabbit, dogs, swine, and baboon models, and seemed to improve hemostasis [60]. A multicentric pilot study tested a lyophilized canine platelet product that is different from the commercially available lyophilized platelet concentrate in 22 dogs and compared it with 15 dogs receiving fresh platelets. The actual measured total platelet counts for the lyophilized platelets used for that clinical trial material ranged from 52 to 57 $\times$ 10^9/units. The dosing regimen provided approximatively 3.3 $\times$ 10^9 lyophilized platelets/kg. There was no difference between groups in all outcome variables (i.e. transfusion reaction rates, the need for additional transfusions, 24-hour bleeding scores, hospitalization time, survival to discharge, or 28 days' survival) [41]. The manufacturers' recommended dose of StablePlate RX is 3 $\times$ 10^9/kg, equivalent to one 8 ml vial for 5 kg. A 2020 clinical trial enrolled 88 dogs with bleeding associated with thrombocytopenia and found that StablePlate RX was clinically not inferior to dimethyl sulfoxide (DMSO) frozen platelets [61]. Early trends indicated that StablePlate RX may reduce bleeding score in patients and show an increased one-hour post-infusion platelet count compared with DMSO platelets, although the DMSO platelets are higher than StablePlate RX at 24 hours (no statistics available). A lyophilized human platelet product, Stasix™ from Entegrion, has been tested in an uncontrolled bleeding model in pigs and prevented blood loss and improved survival [62]. However, the testing and manufacturing of this product has been halted. In an experimental model of splenectomized canines on cardiopulmonary bypass with prolonged bleeding times, infusion of lyophilized platelets showed consistent and persistent lowering of bleeding times compared with control [63].

Finally, a synthetic platelet nanoparticle (SynthoPlate™, Haima Therapeutics, Cleveland, OH) is being investigated for both human and animal use. This non-species-specific nanotechnology has been shown to reduce bleeding in a porcine model of hemorrhagic shock and in a murine model of thrombocytopenia [64, 65].

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68

Administration of Other Biological Products

Jennifer E. Prittie and Jasmine De Stefano

The administration of biologic products to veterinary patients has become common practice in emergency and critical care. Indications include treatment of anemia, coagulopathy, hypoalbuminemia, immune-mediated disease, snake envenomation, various intoxications, and occasionally local thrombosis. While these products can have a positive impact on morbidity and mortality in selected candidates, their use is not without risk. Acute allergy, the most severe form of which is anaphylaxis, and delayed hypersensitivity are the most adverse reactions to administration of these products. Bleeding is an additional specific complication that can occur subsequent to pharmacological thrombolysis.

Detrimental effects from biologic product use can be minimized through thoughtful selection of candidate and product, appropriate administration practices, and careful monitoring to ensure early recognition and treatment of adverse reactions. Red blood cell (RBC) and plasma products are reviewed in Chapter 67. Other routinely administered biologic products, their uses, recommended administration protocols, and associated adverse effects are covered in this chapter. Among the products discussed are vaccines, human and canine albumin, human intravenous immunoglobulin (hIVIG), and thrombolytic agents. Specific immunoglobulin therapies for snake envenomation, tetanus, and digitalis glycoside toxicosis are also summarized. Those biologic products less frequently used in small animal emergency and critical care (e.g. bacterial extracts or toxoids, erythropoietin, and arsenic compounds) are beyond the scope of this chapter and are not discussed.

Vaccines

Vaccines are killed, living attenuated, or living virulent microorganisms given to increase or produce immunity to a disease. These are biologic products widely administered to animals, both to protect the individual patient from disease and as a component of herd medicine.

Adverse Reactions

Immune-mediated vaccine reactions in veterinary medicine are widely appreciated. In particular, inactivated viral vaccines contain cell growth media products (e.g. bovine or egg protein), stabilizers (e.g. gelatin) and/or adjuvants (alum) that function as nontarget antigens [1]. While small amounts of immunoglobulin (Ig)G produced against these antigens are usually harmless, IgE-mediated reactions, or type I hypersensitivity reactions, can result in serious clinical manifestations. Type I hypersensitivity reactions, also termed acute hypersensitivity, allergy, or anaphylaxis, occur when patient/recipient mast cell IgE molecules are cross-linked by biologic product (vaccine, in this case) antigen. Mast cell activation and degranulation results in the release of various preformed vasoactive substances from the mast cells, such as proteases, serotonin, and histamine. Leukotrienes, prostaglandins, and thromboxane are subsequently released following activation of the arachidonic acid cascade. Clinical signs associated with systemic release of these mediators and the resultant systemic inflammation, increased vascular permeability and peripheral vasodilation include pruritus, facial swelling, erythema, urticaria, vomiting and diarrhea [2].

It has been shown that the severity of acute hypersensitivity vaccine reactions is higher in dogs with higher IgE levels against vaccine stabilizers and adjuvants [3]. Anaphylaxis represents the most severe form of acute hypersensitivity, and in addition to cutaneous and gastrointestinal signs, is manifested by respiratory distress and cardiovascular collapse secondary to fluid extravasation and maldistribution of blood flow [4]. While anaphylaxis

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can occur subsequent to exposure of any foreign substance, vaccines are one of the most common causative antigens in veterinary patients, along with venoms, antimicrobials, blood components, and contrast agents [4, 5].

Acute hypersensitivity reactions are frequently reported following administration of other biologic products. These products contain white blood cells (WBCs), RBCs, hemoglobin (Hb), platelets, clotting factors, albumin, immunoglobulins, and/or animal-derived lipids, which can act as antigens in the recipient animal. Hypersensitivity is particularly likely upon repeat exposure to an antigenic product (i.e. after prior sensitization). Previous exposure to a foreign substance primes the body's immune system, and the body's secondary (called "anamnestic") response involves recruitment of preformed antibodies and augmentation of the original humoral immune response [2].

Administration Protocols, Monitoring, and Interventions

Management strategies for type 1 hypersensitivity reactions are similar across antigenic sources and are outlined in Table 68.1. These strategies involve stopping any biologic product infusion and parenteral administration of antihistamines. Product infusion can sometimes be subsequently reinstituted with careful monitoring (e.g. blood products; hIVIG). Anaphylaxis is a medical emergency that require aggressive intravenous (IV) fluid resuscitation and administration of epinephrine for α_1- (vasoconstriction and blood pressure restoration), β_{1-} (positive inotropy and chronotropy) and β_2-adrenergic (bronchodilation) actions. Possible ancillary therapies include administration of antihistamines, glucocorticoids, and bronchodilators [4–8]. The use of glucocorticoids for acute immune-mediated hypersensitivity is controversial [9–15]. These drugs inhibit nuclear factor κB, which reduces production of several proinflammatory cytokines, decreases transcription of cyclooxygenase-2 (which converts arachidonic acid to its more active mediators of inflammation), and inhibits release of histamine and serotonin from mast cells [9–15]. However, these anti-inflammatory effects are delayed by several hours, likely limiting their usefulness during life-threatening allergy. Given their theoretical uses, various specific steroid agents are still incorporated into veterinary treatment algorithms for hypersensitivity reactions (Table 68.1) [4, 5, 9].

One additional "therapy" for acute hypersensitivity associated with vaccination is avoidance. Certain states allow measurement of serum antibody titers as a substitute for rabies vaccination, for example. Alternatively, potential vaccine reactivity may be assessed via intradermal skin testing. To this end, 0.1 ml of vaccine is injected under the furred skin of the lateral thorax. Concurrent sterile saline and histamine injections can serve as negative and positive controls, respectively. Formation of a wheal at the site 15–20 minutes later might indicate increased risk for IgE-mediated vaccine-associated hypersensitivity [1].

Table 68.1 Therapies for Type 1 Hypersensitivity Reactions.

Type of reaction	Therapeutic considerations
Allergy	Diphenhydramine, 2–4 mg/kg IM
	± Dexamethasone sodium phosphate, 0.125–0.5 mg/kg IV, IM
	Methylprednisolone sodium succinate, 30 mg/kg IV
Anaphylactic/ anaphylactoid reaction	Epinephrine (1 : 1000), 0.01 mg/kg IM (can repeat every 5–15 minutes); alternatively, if shock present, as slow IV CRI, 0.05 µg/kg/minute
	Diphenhydramine, 2–4 mg/kg IM
	Famotidine, 0.5 mg/kg IV
	Ranitidine, 0.5–2.5 mg/kg IV
	Dexamethasone sodium phosphate, 0.125–0.5 mg/kg IV
	Methylprednisolone sodium succinate, 30 mg/kg IV
	Aminophylline 5–10 mg/kg IM or slow IV
	Intravascular volume expansion
	Vasopressors as needed: norepinephrine, 0.25–2 µg/kg/minute

CRI, constant rate infusion; IM, intramuscular; IV, intravenous.

Another immune-mediated vaccine reaction is the Arthus reaction, which is a local type III hypersensitivity reaction. It is characterized by activation of complement, local vasculitis, and deposition of antigen–antibody complexes at the injection site within the first few days of vaccination [1]. While this particular reaction is typically self-limiting, immune complexes (biologic product antigen and recipient antibody) that form from administration of other biologic products can cause serious illness.

Systemic deposition of these immune complexes that form within the vascular space may result in vasculitis, synovitis, arthralgia, myalgia, fever, lymphadenopathy, neuritis, and glomerulonephritis (serum sickness). This syndrome typically follows antigen exposure by one to three weeks. The treatment for serum sickness is largely supportive. Plasmapheresis may be considered for severe cases [2, 3].

Other vaccine reactions reported in veterinary literature, including feline vaccine-associated fibrosarcoma, vaccine-induced disease exacerbation, and autoimmune disease (e.g. immune-mediated hemolytic anemia, IMHA), are not discussed in this chapter [1].

Hypersensitivity reactions seen in association with biologic product administration are summarized in Table 68.2.

Table 68.2 Classification of Hypersensitivity Reactions.

Immune reaction	Mechanism	Clinical manifestations	Timing
Type I (IgE-mediated)	Binding of antigen-IgE complex with mast cells and release of inflammatory mediators	Fever, facial swelling, pruritus, urticaria, vomiting, diarrhea, anaphylaxis	Minutes to hours following administration of biologic product
Type II (cytotoxic) AHTR	Interaction between specific IgG or IgM antibodies and red cell-surface antigens	Intravascular hemolysis: fever, vomiting, dyspnea, hypotension, hemoglobinemia/uria Extravascular hemolysis: fever, jaundice, decline in PCV	Minutes to hours following RBC transfusion
Type II (cytotoxic) DHTR	Interaction between specific IgG antibodies and RBC surface antigens	Extravascular hemolysis: Hyperbilirubinemia/bilirubinuria, decline in PCV	3–21 days post-RBC transfusion
Type III (immune complex); serum sickness	Deposition of antigen–antibody complexes in tissues with complement activation and inflammation	Fever, vasculitis, arthralgia, myalgia, lymphadenopathy, glomerulonephritis	1–3 weeks post-infusion of biologic product

AHTR, acute hemolytic transfusion reaction; DHTR, delayed hemolytic transfusion reaction; Ig, immunoglobulin; PCV, packed cell volume; RBC, red blood cell.

Type II hypersensitivity reactions occur specifically when administration of RBC products to a genetically dissimilar recipient of the same species (an allogenic transfusion) results in an immune response directed at donor RBC surface antigens. This immune response results in RBC destruction (hemolysis).

Two main categories of hemolysis exist: extravascular and intravascular. Extravascular RBC destruction is mediated by the mononuclear phagocytic system and results in decreased life expectancy of transfused cells but no significant clinical patient decline. Acute intravascular hemolysis is the most dangerous RBC product hypersensitivity reaction and occurs secondary to donor–recipient incompatibility or previous sensitization (in dogs). Further characterization of this type of transfusion reaction, including clinical signs and recommended therapies are discussed elsewhere [15].

Albumin Solutions

Albumin is the major osmotically active protein in the body and is responsible for 80% for the plasma colloid osmotic pressure (COP) and preservation of intravascular volume (Chapter 58). Albumin's additional roles include maintenance of endothelial integrity, mediation of coagulation, scavenging of toxic compounds, transportation of exogenous and endogenous substances, and inhibition of oxidative injury. Hypoalbuminemia is common in critically ill patients with systemic inflammation and is due to fluid shifts from the intravascular space to the interstitium, gastrointestinal and renal losses, and decreased production as a negative acute phase protein. The consequences of hypoalbuminemia in this patient population include enteral feeding intolerance, hypercoagulability, poor wound healing, and multiple organ failure. Additionally, serum albumin concentration is inversely related to mortality in both human and veterinary patients [16–18].

Despite the well-documented detrimental effects of hypoalbuminemia, the merits of albumin transfusion in critically ill patients remain unclear. Studies evaluating the safety and efficacy of transfusion with albumin products have yielded conflicting results, and a survival benefit associated with their administration has yet to be documented in any specific patient population [19–21]. Correction of underlying cause of hypoalbuminemia and provision of nutrition remain fundamental goals of treatment in all sick patients. The use of artificial colloids to support COP and maintain IV volume has come into question in sick patient cohorts. Several randomized controlled trials in sick people have demonstrated the development of coagulation disorders, acute kidney injury (AKI), and increased mortality following synthetic colloid administration [22–24]. Administration of hydroxyethyl starch (10% 250/0.5/5 : 1) has also been reported in a single retrospective veterinary study to increase the risk of AKI and death in dogs [25].

Given the paradigm shift away from use of artificial colloids for maintenance of COP and IV volume, supplementation of albumin may be prudent in select patients. Patient cohorts include those with severe continuing fluid losses and resultant IV volume depletion and/or those with significant peripheral and organ edema and associated organ dysfunction.

Concentrated Human Serum Albumin Solution

Historically, the only readily available source of species-specific albumin was plasma transfusion, limitations of which include cost, availability, and the potential for volume overload and other adverse transfusion-related effects. An alternative and more effective means of affecting serum albumin concentration is infusion of 25% human serum albumin (HSA) produced from fractionation of human plasma (25% Human Serum Albumin, Octapharma USA Inc., Hoboken, NJ). However, while pharmaceutical HSA does increase albumin concentration, this product has no proven survival advantage and is associated with significant complications in veterinary patients [26–30]. The most clinically relevant adverse effects are related to the highly antigenic nature of HSA. As a foreign protein, HSA elicits immune responses in both critically ill and healthy dogs. Anti-HSA IgG antibodies have been demonstrated in some dogs with no prior exposure to HSA and in most dogs following a single HSA infusion (typically two to six weeks after transfusion) [29].

Adverse Reactions

Reported acute reactions in canine HSA recipients include mild type 1 hypersensitivities, and less commonly, hemodynamic collapse and signs of hypovolemic shock that characterize anaphylactic responses [27, 28, 30]. These reactions are documented following both initial and repeat exposure to HSA. More recently, occurrence of serum sickness has been documented in canine HSA recipients [28]. Facial and peripheral edema, vomiting and inappetence, urticaria, joint effusion and lameness, acute kidney failure, and death several days to weeks following HSA infusion are reported. The more severe clinical signs attributable to serum sickness occur in healthy dogs. This dichotomy may be related to immunocompetence and normal serum albumin concentrations in these recipients [29].

Administration Protocols

Risk assessment, careful recipient selection, and close patient monitoring are paramount to safe administration of HSA. Reasonable goals for HSA administration are to increase serum albumin to 2.0–2.5 g/dl and/or COP to 14–20 mmHg, respectively.

Preparation Prior to administration, the product is inspected for turbidity or discoloration, and if they are present, the product is discarded. Human albumin is administered within four hours of opening the vial to decrease potential of bacterial contamination. A vented delivery set may be used as HSA is supplied in a glass vial, or the contents can be aseptically transferred to a buretrol for administration. Transfusion filters are routinely used with HSA to filter macroaggregates that may form in solution, but according to the manufacturer, a filter is not required. The product may be directly administered or diluted with crystalloids [26, 28, 30].

Dosage Dosages are based on either a calculated albumin deficit or an extrapolated empirical dosage of 2–5 ml/kg of 25% HSA. Albumin deficit can be estimated using the following equation [31]:

$$10 \times \left(\text{serum albumin desired} - \text{serum albumin of patient}\right) \times \text{body weight}\left(\text{kg}\right) \times 0.3 = \text{albumin deficit} \quad (68.1)$$

Monitoring Baseline vitals are obtained prior to HSA administration, and frequent monitoring of perfusion parameters, including capillary refill time (CRT), heart rate, pulse rate and quality, respiratory rate and effort, and temperature, is required throughout transfusion. Infusion rates are typically started at a decreased rate and slowly increased to full rate if no sign of reaction occurs (Figure 68.1). Signs of acute hypersensitivity reaction warrant cessation of transfusion or decrease of transfusion rate. Emergency management of these reactions is outlined in Table 68.1. Owing to the hyperoncotic nature of HSA solution, volume overload may complicate therapy, and close monitoring of respiratory parameters and volume status is also indicated.

After HSA administration, reevaluation of the patient's interstitial edema status, serum albumin concentration, and/or COP will determine transfusion efficacy. While significant continuing protein losses may interfere with reaching target albumin level, repeat administration of this foreign protein is ill advised.

Canine Albumin

Animal Blood Resources International have developed a lyophilized 98% pure canine albumin by pooling source plasma from their donors (Lyophilized Canine albumin, 5 g. Animal Blood Resources International, Stockbridge, MI). Advantages over HSA include species specificity and decreased risk of transfusion adverse effects.

Adverse Reactions

In one study in which canine albumin was administered to healthy Beagles weekly for one month, no adverse reactions or anti-canine albumin antibody formation were documented up to five weeks post-infusion. In a single clinical study in dogs with septic peritonitis, albumin level, COP, and blood pressure increased following infusion of canine albumin. No adverse reactions were documented [32]. However, as with any biologic product, acute type I hypersensitivity is possible from administration of canine albumin and has been personally observed by the authors.

Date:	Technician Initiating Transfusion:
	Transfusion Product:
	Donor/Unit ID:
	Product Volume:
	Full rate: 1/2 rate: 3/4 rate:
	Start Time: End Time:
	Total Volume of Transfusion:
	Pre PCV TS Post PCV TS

If there is a significant change in vitals at ANY time please notify the DVM.

Time	Time Increment	Time due	Actual time done	RR	HR	Rate of Infusion	Temp	Rate ml/hr	Volume infused
Pre transfusion	0								
If no change with 15 minute vitals, continue at 1/2 transfusion rate									
15 minutes post start	15								
If no change with 30 minute vitals, increase to 3/4 tranfusion rate									
30 minutes post start	15								
If no change with 45 minute vitals, increase to full tranfusion rate.									
45 minutes post start	15								
60 minutes post start	15								
75 minutes post start	15								
90 minutes post start	15								
120 minutes post start	30								
150 minutes post start	30								
180 minutes post start	30								
240 minutes post start	60								
300 minutes post start	60								
360 minutes post start	60								

Transfusion must be completed within 6 hours or discard remainder of blood

Figure 68.1 Biologic product transfusion log.

Administration Protocols

Close patient monitoring is paramount to the safe administration of canine albumin. Reasonable goals for administration are to increase serum albumin to 2.0–2.5 g/dl and/or COP to 14–20 mmHg, respectively.

Preparation Lyophilized canine albumin A is stored at 39.2–42.8°F (4–6°C). According to the manufacturer, this biologic product can be reconstituted with 0.9% NaCl or 5% dextrose in water.[2] Varying concentrations can be achieved with dilution (Table 68.3). A more hypertonic bolus (e.g. 16%) may be preferable in hypovolemic patients to achieve acute volume expansion and maintenance of effective circulating blood volume.

Dosage Optimal dosing for canine albumin has not been determined. Various dosing regimens for veterinary patients have been published and are outlined in Box 68.1. Once reconstituted, the manufacturer recommends administration within six hours. However, a 2020 veterinary study demonstrated no bacterial growth and a stable albumin concentration when 5% canine albumin in saline was stored for 24 hours at 4°C [33].

Monitoring Hypersensitivity and volume overload are potential adverse reactions secondary to administration of canine albumin. Monitoring of the patient during and following product administration is as outlined above with HSA. Repeat transfusions are unlikely to cause immune reactions due to the species-specific nature of the product. However, a history of allergy to either human or canine albumin is a contraindication for its use. The manufacturer recommends a maximum daily dose of 2 g/kg.

Table 68.3 Albumin Reconstitution Recommendations.

Desired albumin concentration (%)	Amount of diluent (ml)
16	30
10	49
5	100

Box 68.1 Canine Albumin Dose Recommendations

Recommended canine albumin dose

Albumin deficit (grams) = 10 × (desired albumin – patient albumin) × patient weight (kg) × 0.3 [31]

Albumin dose (ml of 5% solution) = patient weight (kg) × 90 ml/kg × (desired albumin – patient albumin) × 0.2 dl/g [conversion factor for 5% albumin]

450 mg/kg to raise the serum albumin by 0.5 g/dl

800–884 mg/kg over 6 hours [32]

Human Intravenous Immunoglobulin

hIVIG is fractionated from plasma pooled from 1000–10 000 blood donors. This product is composed of at least 90% biologically active immunoglobulin (Ig)G and smaller amounts of IgE, IgA, IgM, and IgD. The immunomodulating properties of hIVIG have proven beneficial in a variety of human immune-mediated disorders, including systemic lupus erythematosus, myasthenia gravis, pure RBC aplasia, immune-mediated neutropenia, vasculitis, and toxic epidermal necrolysis [34, 35].

The mechanisms of action of hIVIG are complex but are postulated to include phagocyte Fc receptor blockade and resultant decreased phagocytic activity of mononuclear cells, modulation of T-cell function (inhibition of cytotoxic T cells), neutralization of harmful autoantibodies, clearance of IgG, and attenuation of complement-mediated damage and proinflammatory cytokines [34].

Commercially available hIVIG has been used with some success in dogs and cats affected with IMHA, immune-mediated thrombocytopenia, myasthenia gravis, sudden acquired retinal degeneration syndrome, myelofibrosis, erythema multiforme, and a variety of other immune-mediated dermatologic disorders (e.g. toxic epidermal necrolysis, Stevens–Johnson syndrome) [36–40].

Adverse Reactions

Adverse effects in human patients following hIVIG administration are uncommon, affecting <15% of patients, and are typically limited to fever and malaise. More serious effects reported include hypotension during infusion, AKI, aseptic meningitis, hemolytic anemia, thromboembolic events, and anaphylaxis in patients with IgA deficiency. Volume overload and resultant pulmonary edema and transfusion-related acute lung injury have also been reported [34, 36].

Similarly, complications associated with hIVIG administration in veterinary patients are infrequently reported. Dogs with IMHA treated with this product have developed transient thrombocytopenia (60%) and thromboembolism (71%), but these findings may be unrelated to hIVIG and instead associated with the patients' underlying diseases [36–40]. Vomiting was reported following infusion of hIVIG in a healthy dog [40]. Other potential complications are related to the product's hyperoncotic nature (i.e. volume overload, especially in patients with underlying cardiovascular disease) and antigenicity, which may result in acute or delayed hypersensitivity reactions. The potential for sensitization in veterinary patients has been postulated. This may preclude safe repeat infusion of this product.

Administration Protocols

Preparation

Lyophilized hIVIG (10%; Gammagard S/D, Hyland Division, Baxter Healthcare, Westlake Village, CA) products can be stored at room temperature for up to 24 months prior to rehydration [41]. A variety of diluents may be used for reconstitution; these vary with product and manufacturer, but include sterile water, 0.9% NaCl, and 5% dextrose. Rehydration is recommended over 15–20 minutes, and gentle handling of the product during this process is advisable to prevent foaming. Refrigeration is required following reconstitution, and the product must be administered within 24 hours to reduce risk of contamination [41].

Immunoglobulin is administered via a designated peripheral or central venous catheter. Most hIVIG manufacturers advocate the use of an in-line filter during transfusion; filter size recommendations vary. The product is administered at room temperature for optimal patient comfort [41].

Dosage

The optimal dosage of hIVIG in veterinary patients is unknown. Historical doses of hIVIG in animals range from 0.5 to 1.5 g/kg [36–40]. In five dogs with immune-mediated thrombocytopenia, a low-dose therapy (0.28–0.34 g/kg) was used and was deemed efficacious [38].

Monitoring

An infusion period of 6–12 hours is recommended in human patients to ensure safe delivery; hang times of 4–8 hours are reported in veterinary patients [36–40]. In all species, transfusion is initiated at a slow rate (0.01 ml/kg/minute) and gradually increased every 30–60 minutes to a maintenance rate not to exceed 0.08 ml/kg/minute. Fluid-sensitive patients may not tolerate high fluid rates. Frequent monitoring during administration includes CRT, heart and pulse rates and quality, respiratory rate and effort, blood pressure, and temperature (Figure 68.1). Signs of acute hypersensitivity warrant prompt, temporary cessation of the infusion and antihistamine administration (Table 68.1). Most patients tolerate reinstitution and completion of the transfusion at a slower infusion rate.

Specific Immunoglobulin Therapy

Very few small animal intoxications exist for which a specific antidote can be provided. Three such intoxications are poisonous snake envenomation, tetanus, and intoxication with digitalis glycosides. In each case, the antidote provided is a specific immunoglobulin that targets the toxicant, limiting host toxicity via immunomodulation.

Snake Envenomation

Approximately 20 species of snakes native to the United States are venomous. Most small animal envenomations occur in the southwestern part of the country, and are attributable to *Crotalidae* species, or pit vipers (rattlesnakes, cottonmouths, and copperheads). Of the pit vipers, the Eastern diamondback rattlesnake is responsible for most human fatalities annually. Less frequently, envenomations are due to *Elapidae* (coral snakes) [42–45].

Snake venom consists of mixtures of enzymatic proteins (hylauronidase, collagenase, proteases, and phospholipases) that cause local tissue injury and vasculitis, enhancing spread of the venom. Other proteins and enzymes, including fibrinolysins and thrombin-like enzymes, result in defibrination (depletion of fibrinogen and fibrin) and friable clot formation, respectively. The end result is coagulopathy characterized by bleeding propensity. Victims of snake bites are also at risk for AKI and for neurotoxicosis associated with neuromuscular blockade, the latter typically caused by *Elapidae* envenomation. Lethality following envenomation is associated with the smaller low-molecular-weight polypeptides in the venom that result in increased capillary permeability, intravascular volume depletion from third spacing of fluids, and hypovolemic shock [42–45].

While supportive measures such as administration of IV fluids, analgesics, and antibiotics (for documented concurrent bacterial infection) are the standard of care for victims of snake bites, administration of antivenom plays the most important role in morbidity and mortality reduction in many of these animals. A mortality reduction of 36% was observed comparing dogs that were treated with antivenom with those who were not in one study [46]. The two approved antivenom products in the United States for treatment of rattlesnake bites in human patients are Antivenin™ crotalid polyvalent (ACP; Boehringer Ingelheim Vetmedica, St. Joseph, MO) and CroFab® *Crotalidae* polyvalent immune antigen-binding fragment (Fab; Boston Scientific, Marlborough, MA) (Table 68.4) [45]. ACP is an antiserum composed of equine gammaglobulins. This product is rich in antibodies that target the venoms of all endemic pit viper species, but in addition contains allergenic equine protein contaminant. CroFab has approximately five times the potency of ACP, and is more efficacious than ACP for treatment of crotalid-induced neurotoxicity [45, 47]. CroFab is also less allergenic than ACP, as it is pure Fab immunoglobulin, and as such contains less contaminant than ACP antivenom [45, 47, 48]. A potential drawback to CroFab is its shorter half-life, which may allow for recurrence of the effects of unneutralized venom and may necessitate repeat dosing [48, 49]. While a multicenter trial evaluating the efficacy of CroFab in

Table 68.4 Comparison of Crotalid Antivenoms.

Property	Antivenin[a]	CroFab[b]
Source	Horse	Sheep
Immunoglobulin	Immunoglobulin G	Fab fragment
Potency	+	+++
Antigenicity	+++	+
Initial dose	10–50 ml (1–5 vials)	4–6 vials
Clearance/need for repeat administration	+	+++
Approximate cost	$250/vial	$3500/vial
Licensure	Human, animal	Human

[a] Antivenin™ (Boehringer Ingelheim Vetmedica, St. Joseph, MO);
[b] CroFab® (Boston Scientific, Marlborough, MA).

veterinary patients is underway, ACP is currently the only product licensed for use in animals in the Western hemisphere.

Adverse Reactions

Adverse effects associated with antivenom therapy are uncommon, and most are related to patient hypersensitivity to foreign proteins in the antiserum. Mild, self-limiting type 1 hypersensitivity reactions have a reported prevalence of 14–56% and 4% in humans and dogs, respectively [45, 48–52]. In two reports of rattlesnake envenomation in dogs, ACP therapy resulted in type 1 hypersensitivity in 1/22 and 0/23 patients, respectively [51, 52]. Clinical signs referable to allergy in affected dogs were limited to facial swelling and pruritus. Anaphylaxis has not been reported in veterinary patients. The reported incidence of serum sickness in human snake bite victims treated with ACP or CroFab is 18–86% and 16%, respectively [45]. A single case of serum sickness resulting from ACP administration to a dog following *Crotalus adamanteus* envenomation has been reported [53]. The dog developed fever, anorexia, lethargy, and generalized pitting edema three days after ACP therapy. The dog made a full recovery.

The antivenom formulated from hyperimmune equine serum (IgG) that was available for small animal victims of systemic elapid snake envenomation (associated with one documented acute anaphylactic reaction in a dog) is no longer available [54, 55]. Coralmyn® (Instituto Bioclon, Mexico City, Mexico) is a polyclonal antivenom F(ab') [2] product available in Mexico and obtainable in the United States with a US Department of Agriculture importer's permit. Although this antivenom has less extraneous protein than its predecessor, acute and/or delayed hypersensitivity reactions may complicate therapy [56].

Administration Protocols

Antivenom therapy is recommended for victims of snake bites with worsening local injury, clinically significant coagulopathy, or systemic signs of shock. The recommended initial dosage of antivenom varies with amount of venom injected, time elapsed since envenomation, severity of clinical signs, and patient size.

Preparation Skin testing for prediction of allergic response may be performed by injecting a 1 : 10 dilution of antivenom at 0.02 ml subcutaneously. A positive reaction, manifested by a wheal surrounded by erythema, occurs within 30 minutes [56]. A negative skin test does not guarantee that a severe reaction will not take place, and the time it takes to perform the test delays prompt administration of the antivenom (the sooner the product is administered, the more effective it is) [57, 58]. Routine skin testing is therefore not recommended.

Antivenom is reconstituted with the provided diluent. It should be swirled but not shaken, and the vial should be warmed to body temperature to facilitate reconstitution, which usually takes 10–15 minutes [57]. The reconstituted antivenom is diluted at a ratio of one vial to 100–250 ml of isotonic crystalloids, adjusting the volume to prevent fluid overload in at-risk patients [56]. The initial dosage is administered IV over 30 minutes.

Dosage The suggested starting dose ranges from one to five vials. Redosing depending on product and clinical efficacy may be necessary. Antivenom is costly and financial constraints may need to be considered before administration [57]. A CRI protocol has recently been reported using CroFab for North American rattlesnake bites, where rates of infusion varied from two to four vials per day, for 6–14 days post-envenomation. Continuous infusion appeared safe and effective [59].

Monitoring Careful patient monitoring during infusion is paramount for early detection of allergy or anaphylaxis (Table 68.1). Frequent measurement of tissue swelling and reassessment of coagulation status will help to determine the need for antivenom redosing. The dose may be repeated as needed every two hours for clinically significant coagulopathy or if local swelling worsens despite therapy [43].

Tetanus

Tetanus is an acute toxigenic illness that occurs subsequent to infection with the spore-forming bacillus *Clostridium tetani*. In dogs and cats, the source of the infection is typically a contaminated penetrating wound, and clinical signs of illness occur 5–10 days following inoculation of the organism.

C. tetani spores produce two toxins, tetanolysin and tetanospasmin, the latter of which is responsible for the neurologic signs associated with infection. The toxin travels up peripheral nerves and hematologically to the central nervous system and inhibits the release of the inhibitory neurotransmitters gamma aminobutyric acid (GABA) and glycine. Loss of neuronal inhibition of skeletal muscle and autonomic dysfunction ensue.

Clinical signs associated with infection include severe muscle rigidity and cranial nerve deficits (e.g. facial muscle spasms – risus sardonicus, lockjaw – trismus, third eyelid protrusion, and dysphagia). Additionally, disinhibition of the sympathetic nervous system and excessive catecholamine levels may result in tachycardia and hypertension [60–62].

Specific immunoglobulins that target unbound tetanospasmin in the blood are available but their efficacy remains unproven [62, 63]. Two products exist for treatment of tetanus in veterinary patients: Tetanus Antitoxin (equine; Zoetis, Parsippany-Troy Hills, NJ) and Tetanus Antitoxin Behring (MSD Animal Health, Rahway, NJ). The Zoetis product has been used off-label in small animals; the latter product is not recommended for use in cats but has been used without complication in dogs [56, 60, 62–64].

Adverse Reactions

As both products are comprised of foreign proteins, hypersensitivity reactions are possible during and following administration. While mild, self-limiting hypersensitivities have been documented in case series, anaphylaxis has not [62, 64].

Administration Protocols

Preparation Historically, intradermal skin testing was widely practiced to predict local hypersensitivity. However, recent reports have demonstrated both false-positive and false-negative results, calling into question this routine practice [62].

Dose Recommended antitoxin dosage varies widely from 100 to 1000 units/kg, with doses of up to 1900 units/kg reported in dogs [56, 64]. Antitoxin may be administered intravenously, intramuscularly, or subcutaneously. Other reported dosing regimens include 1000 units at the wound site or 1–10 units intrathecally. However, intrathecal administration has been shown to increase morbidity and mortality in human patients and is not currently recommended [60].

Monitoring Patients receiving antitoxin are monitored closely for signs of acute hypersensitivity, and emergency drugs (Table 68.1) should be readily available.

Digitalis Glycosides

Digitalis glycosides (digoxin) are occasionally prescribed by practitioners for cardiovascular disease in veterinary patients. These agents exert positive inotropic effects via dose-dependent inhibition of the cardiac Na-K-ATPase pump, and subsequent increased intracellular calcium and force of cardiac contraction (Figure 68.2). Antiarrhythmic effects are mediated through an increase in parasympathetic tone and include decreased sinus rate, depressed impulse conduction through the atrioventricular (AV) node, prolonged AV refractory period, and decreased automaticity of specialized atrial fibers. These effects make glycosides appropriate agents for management of supraventricular tachycardias and systolic dysfunction.

These agents have low therapeutic-to-toxic ratios, and inadvertent overdose can be fatal. Extracardiac effects associated with digitalis glycoside toxicosis are common and include depression and inappetence, nausea, vomiting, and diarrhea. More importantly, overdose can result in a variety of cardiac conduction disturbances (atrioventricular block and bundle branch block), atrial and ventricular arrhythmias, and life-threatening hyperkalemia with resultant cardiac dysrhythmias.

Treatment of toxicosis may include gastric decontamination, IV fluid therapy, administration of cardioprotective drugs (calcium and antiarrhythmic agents), and cardiac pacing. Additionally, in cases of life-threatening toxicosis in human patients and sporadically in veterinary case reports, digoxin-specific antibody is administered [65–69].

Digibind® (digoxin immune Fab, ovine; GlaxoSmithKline LLC, Research Triangle Park, NC.) is ovine-derived,

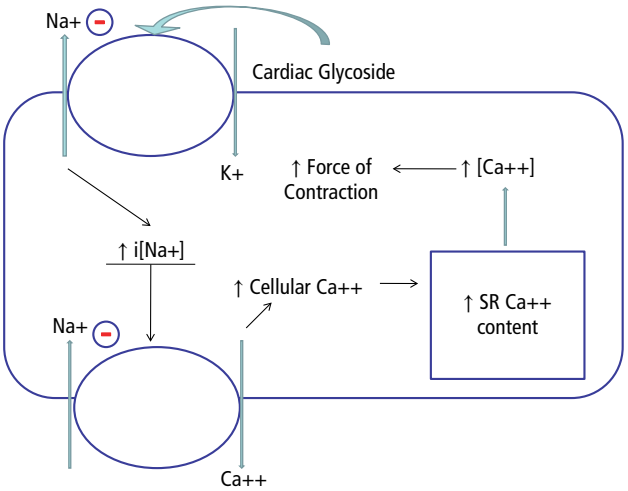


Figure 68.2 Mechanism of action of cardiac glycosides. Cardiac glycosides inhibit the cardiac Na+/K+ATPase pump. This inhibition leads to an accumulation of intracellular Na^+, decreased Na^+/Ca^{2+} exchange and a resultant increase in intracellular Ca^{2+}. Higher intracellular Ca^{2+} concentration leads to increased force of ventricular contraction.

digoxin-specific Fab antibody fragment with a reported efficacy of greater than 80% in patients with refractory, life-threatening digitalis glycoside toxicosis [67].

An additional intoxication for which Digibind might be beneficial is following ingestion of *Nerium oleander* (oleander). The toxic metabolites of this ornamental plant are steroidal glycosidic cardenolides, cardiac glycosides, and rarely cardiac arrhythmias have been reported in association with its consumption by small animals [70].

Adverse Reactions

Potential adverse effects associated with Digibind administration are infrequently reported in human reviews, but include allergic reactions (< 1%), severe acute hypokalemia from reversal of pump paralysis (4%), and exacerbation of congestive heart failure [65–68]. No detrimental effects have been reported in the few veterinary cases to which this therapy has been applied. However, the foreign nature of the Fab fragments (ovine-derived) imparts the potential for more serious allergic or anaphylactic reactions, and repeat dosing is currently not recommended in veterinary patients.

Administration Protocols

Preparation Digibind (38 mg digoxin immune Fab) is diluted with 4 ml sterile water to achieve a concentration of 9.5 mg/ml. If not used immediately, storage for up to four hours at 35.6–46.4 degrees F (2–8°C) is acceptable. The reconstituted product can be further diluted with 0.9% saline to achieve a desired volume; the dose is administered through a 0.22 μm filter [66, 69]. It may be given as a bolus in emergency situations, but an infusion over 1–2 hours is recommended when possible [66].

Dosage Determination of appropriate Digibind dose is based on the digoxin body load, which is estimated by either the serum concentration or the amount ingested [66]. The serum digoxin concentration obtained eight hours after the last dose is incorporated into Eq. (68.2):

$$\mathrm{DBL} = \frac{\text{Serum glycoside concentration}\left(\text{ng/ml}\right) \times^{*} V \times \text{body weight}\left(\text{kg}\right)}{1000} \quad (68.2)$$

where DBL = digoxin body load and *V = volume of distribution (5.6 l/kg for digoxin and 0.56 l/kg for digitoxin). The dose of Digibind is then calculated using Eq. (68.3):

$$\left[DBL/0.5mg\ per\ vial\right] = \#\ of\ 38mg\ Digibind\ vials\ to\ administer \quad (68.3)$$

where DBL = digoxin body load [66, 69]. Each 38 mg vial of digoxin-specific Fab fragments binds approximately 0.5 mg digoxin or digitoxin. The reader is referred to the product package insert for alternate dosing strategies.

Thrombolytic Agents

Endothelial injury, alterations in blood flow, and hypercoagulable states increase the risk for thrombosis (clot formation). These risk factors (known together as Virchow's triad) are associated with many conditions common to critically ill veterinary patients, including but not limited to sepsis, systemic inflammatory response syndrome, hyperadrenocorticism, diabetes mellitus, cardiomyopathy, neoplasia, protein-losing nephropathy or enteropathy, IMHA, bite wounds, snake envenomation, polytrauma, heat stroke, and burns. The end results of thrombosis are maldistribution of blood flow (which may cause signs of shock), tissue ischemia, and hypoxia.

The fibrinolytic system is responsible for clot dissolution through the formation of the enzyme plasmin. Plasmin is derived from the inactive proenzyme plasminogen via the action of the body's plasminogen activators. Once generated, plasmin is responsible for fibrinolysis (fibrin degradation and subsequent clot lysis) (Figure 68.3).

Thrombolytic agents augment fibrinolysis via conversion of plasminogen to plasmin and have been used in the treatment of thrombi in both human and veterinary patients. Available agents include streptokinase, urokinase-type plasminogen activator, and tissue-type plasminogen activator (tPA). Streptokinase is a bacterial protein produced by hemolytic species of *Streptococci* that indirectly activates plasminogen to plasmin. Because of its bacterial origin, this protein can elicit an immune response in the recipient, particularly in patients with previous β-hemolytic streptococcal infection. Circulating antibodies to streptokinase limit the efficacy of the agent (necessitating a loading dose initially) and may result in hypersensitivity reactions. Reported signs in human patients include pruritus, rash, hypotension, and bradycardia, more commonly following rapid infusion and thought to be associated with histamine and/or bradykinin release. Additional detrimental effects associated with streptokinase administration are systemic

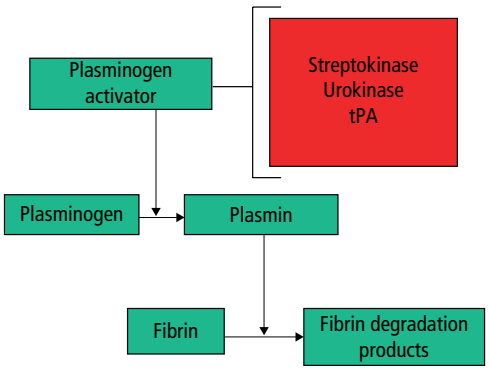


Figure 68.3 Initiation of fibrinolysis.

hemorrhage due to activation of fibrin-bound (thrombus-associated) and circulating plasminogen, and ischemia–reperfusion injury when blood flow to ischemic areas is reestablished. Reperfusion of such areas leads to systemic release of reactive oxygen species, metabolic acidosis, and hyperkalemia.

tPA is secreted by endothelial cells and circulates in the blood. Human recombinant tPA is derived from mammalian cell tissue culture, and hypersensitivity reactions are possible in veterinary patients, as are systemic bleeding and reperfusion injury. However, as tPA is more specific for thrombus-associated plasminogen (as opposed to circulating plasminogen), severe hemorrhage is less likely than with streptokinase. Urokinase is found in the urine and produced from human neonatal kidney cells in tissue culture. This enzyme has clot specificity in between that of streptokinase and tPA. This product has been infrequently administered to veterinary patients and carries risks similar to other thrombolytic agents.

Indications for thrombolytic agents in human patients include massive pulmonary embolism complicated by right heart failure and/or hypotension, and greater than 50% obstruction of a major pulmonary artery. Contraindications include acute myocardial infarction, intracranial hemorrhage, head trauma, and ischemic stroke [71–73]. Specific indications and contraindications are not established for veterinary patients.

Despite lack of established indications, thrombolytic agents have been used in dogs and cats with clots of the arterial and venous circulation. No survival advantage has been documented associated with their systemic administration, and reported adverse effects including fever, hyperkalemia, significant hemorrhage, and acute death are common [74–79]. There are isolated case reports documenting successful use of thrombolytics (tPA) more locally to address catheter-related thromboses and urinary bladder blood clots without adverse consequences [80, 81]. Most recently, administration of tPA directly at the site of either arterial or venous clots (catheter directed thrombolysis, CDT) has been described by veterinary interventional radiologists [82].

Adverse Reactions

The most common complication of thrombolytic agents is hemorrhage, which may manifest as a dropping packed cell volume (PCV) in association with hematuria, gastrointestinal or oral mucosal bleeding, bleeding from catheter insertion sites, dyspnea, or changes in neurologic status [74]. Metabolic acidosis and electrolyte abnormalities such as hyperkalemia associated with tissue reperfusion are common complications of thrombolytic therapy and can be fatal. Allergic reactions and anaphylaxis are not well documented in the veterinary patient, but result in pruritus, rash, and fever in humans [71].

Administration Protocols

Dosage Alteplase (Cathoflo® Activase® 2mg, Genentech, San Francisco, CA) is tPA and is reconstituted with 2.2 ml sterile water and swirled, not shaken, until dissolved. The solution can be further diluted with 0.9% saline or 5% dextrose to achieve a concentration of 0.5 mg/ml as desired. Without preservatives, sterility cannot be ensured greater than eight hours following reconstitution.

A published IV dose for tPA in veterinary patients is 0.5 mg/kg over 30–60 minutes (not exceeding 9 mg), with repeat doses up to three times within 24 hours [82]. For local infusion protocols, the reader is referred to the included references [80, 81].

Monitoring The patient receiving thrombolytic therapy requires intensive monitoring and nursing care. Catheter-associated bleeding may be subtle and go undetected until a substantial volume of blood is lost, as hemorrhage can migrate down fascial planes or be covered by catheter wraps [76]. Direct manual pressure may be applied to a hemorrhaging catheter site, and bleeding patients may require therapy with blood components. Frequent assessment of mucous membrane color and CRT, respiratory rate and effort, heart rate, pulse quality, blood pressure, and temperature can aid in detection of continuing bleeding. Continuous electrocardiogram (ECG) monitoring is indicated as rising serum potassium causes a progression of ECG changes. Narrow, peaked or "tented" T-waves, flattening and disappearance of the P waves, and widening of the QRS complexes typify the cardiotoxicity associated with progressive hyperkalemia. Tachypnea is nonspecific and may indicate a metabolic acidosis with respiratory compensation, pain, pulmonary thromboembolism, or hemorrhage.

Summary

Most veterinarians would consider biologic products as an essential component of their armamentarium. These agents are indicated for a variety of clinical syndromes in critically ill patients but do carry significant risks. Safe administration of biologic products involves appropriate patient selection, awareness of associated adverse effects and timely interventions if an untoward reaction is suspected. Implementation of evidence-based guidelines and standardized hospital protocols for administration of these products are recommended.

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69

Blood Banking

Marie K. Holowaychuk and Kenichiro Yagi

Introduction

The caseload of patients requiring blood transfusions is growing as more pet owners seek advanced care for their animals. As such, emergency and specialty hospitals are required to have a supply of blood products available. Alternatively, blood donors may be on standby for collection of blood when urgent needs arise. Hospital teams must therefore identify commercial or other sources of animal blood products and must possess appropriate means to store those products. For those hospitals choosing to use blood donors, protocols must be established for donor selection and screening, blood processing, and maintenance of quality assurance for donors and recipients. Meeting the transfusion needs of patients with an appropriate supply of blood products can be extraordinarily challenging and rewarding for the veterinary team. Because there are currently no regulations for acquiring blood products for in-hospital use, each hospital is responsible for establishing their own protocols and remaining diligent in evaluating the sources of their blood products.

Obtaining Blood Products

Because the demand for blood products is inconsistent and unpredictable, and since blood products have a finite shelf life, many emergency and referral hospitals choose to meet blood product demands with a combination of products obtained from commercial blood banks, other local hospitals, or in-hospital collections.

Commercial Blood Banks

There are several commercial blood banks located in large metropolitan areas that can be used as sources for blood products (Box 69.1). These blood banks tend to have on-site donors or a large pool of established donors. Because commercial blood banks are not regulated in most jurisdictions, hospitals must exert due diligence to ensure that the quality of the blood products obtained is satisfactory. Blood banks can be asked to provide information regarding the maintenance of on-site donors, donor screening protocols, strategies for preservation of donor health and prevention of vector-borne disease, plans for on-site donors once they retire, and steps to sustain the health and supply of community-acquired donors. It is also appropriate to confirm whether there is a consistent supply of blood products, whether the product with the longest shelf life is always shipped, and the anticipated delivery once an order is placed, including additional fees assessed for overnight or expedited shipments [1].

Before and after a blood product is shipped, it is the responsibility of the receiving hospital to ensure that the product was collected, stored, and shipped appropriately, and remains viable. Questions related to method of collection, sterile procedure, leukoreduction protocol, and preservation additives are necessary to determine blood product sterility and viability. Upon shipment, the temperature of the shipping container and time spent in transit are important to note. For example, packed red blood cells (PRBCs) are typically shipped overnight or within two business days and should be shipped at refrigerated temperatures (33.8–42.8°F; 1–6°C). Alternatively, plasma products must arrive frozen and are typically shipped on dry ice

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Box 69.1 A Selection of Commercial Blood Banks Located in Canada and the United States

- Animal Blood Bank (California)
- Animal Blood Bank DBA (Michigan)
- Animal Blood Resources International (Michigan)
- Best Friends Blood Bank (Georgia)
- Blue Ridge Veterinary Blood Bank (Virginia)
- BodeVet (Maryland)
- Buddies for Life Canine Blood Bank (Michigan)
- Canadian Animal Blood Bank (Manitoba)
- Canine Blood Bank of Central Iowa (Iowa)
- Hemopet (California)
- HemoSolutions (Colorado)
- LifeStream Animal Blood Bank (Ontario)
- Nine Lives Blood Services (Michigan)
- North American Veterinary Blood Bank (Virginia)
- Northwest Veterinary Blood Bank (Washington)
- Rocky Mountain Blood Services (Colorado)
- St. Louis Blood Bank (Missouri)
- Sun States Animal Blood Bank (Florida)
- The Pet Blood Bank (Texas)
- The Veterinarians' Blood Bank (Indiana)
- Twin Cities Animal Blood Bank (Minnesota)

overnight. The blood bank must be able to provide information related to any adverse events encountered during collection, processing, or storage of the product.

Other Hospitals

Nearby specialty, emergency, or teaching hospitals may also serve as sources of blood products for hospitals with an emergent or urgent need. Products obtained from other hospitals may have been originally sourced from commercial blood banks or collected in-hospital. The same recommendations apply in that the hospital requesting the blood product must assess the product's quality in terms of sterility and viability as outlined above. Unfortunately, other hospitals may elect not to sell or give blood products to requesting hospitals if their own supply is low or their anticipated need is high. Some other hospitals would rather the requesting hospital refer the patient so that they can receive the transfusion in their own hospital.

In-Hospital Collection

Blood collected from animals in hospital can replenish stores of blood products or meet urgent or emergent needs for whole blood or component transfusions. Some emergency or referring hospitals may choose only to perform in-hospital collections during circumstances when commercial or other hospital sources of blood products are unavailable or insufficient to meet emergent need. Regardless of the situation, the hospital must establish protocols for donor selection and screening, precollection screening, and collection procedures, to assure the quality of the blood product and safety of the donor.

Donor Sources

Most hospitals that perform in-hospital collections do so using donors procured from different sources such as employee-owned animals, client-owned animals, or animals specifically acquired for in-hospital donation (i.e. from research colonies or breeders). Unfortunately, securing donors that can be used on-call for urgent or emergent purposes tends to be more difficult than those who are scheduled for donation every three to six months. Therefore, it is important to establish with the employee or client the extent of their ability to volunteer on an on-call basis and to have several contact numbers at which they can be reached. Alternatively, some hospitals acquire animals that are fostered by hospital employees and are designated as on-call donors [1].

Canine Donors

Selection and Screening

Canine donors must be selected and screened judiciously to protect donors and recipients from harm. A complete history, comprehensive physical examination, blood tests, and bloodborne pathogen screening are recommended before collecting blood for transfusion. Canine donors should be between one and eight years of age, apparently healthy, and have a history that rules out prior transfusions, acute or chronic illness, and current pregnancy. Dogs without a known history (e.g. rescues) can be considered at the discretion of the attending veterinarian who must weigh the risks and benefits of using blood collected from a dog that is not an ideal donor [2]. Dogs previously used in breeding programs can still be used as donors, given that pregnancy does not sensitize dogs to erythrocyte antigens [3]. A calm temperament is preferred, such that restraint in lateral or sternal recumbency can be achieved without sedation. An owner that is reliable, responsible, and committed is also best to ensure honest responses to history questions and dedication to the program.

A lean body weight of at least 50 lbs (22.7 kg) is recommended for standard collections of 450 ml of blood (the volume collected with most human blood collection systems used for canine donors). Alternatively, dogs that weigh less can be used to collect a smaller volume of blood for whole blood transfusion. Most dogs can tolerate having 11–19 ml/kg of blood collected with minimal cardiovascular compromise [2]. However, careful attention should be

paid to small donors to ensure that they do not exhibit cardiovascular or other complications. A physical exam must elicit no abnormalities and detection of ectoparasites should prohibit donation [4].

A complete blood count and serum biochemistry profile should yield normal results and must be repeated annually. Additional screening tests, such as urinalysis and fecal parasite testing, can be performed at the discretion of the attending veterinarian. Blood typing is also recommended to ascertain whether the donor is dog erythrocyte antigen (DEA) 1 positive or negative. Additional screening for DEA 4 and 7 is available through commercial laboratories; however, most donor pools are limited such that maintaining a pool of DEA 1 and 7 negative dogs can be difficult. Therefore, most in-hospital donor programs only test for DEA 1 positive or negative status and administer type-specific transfusions whenever possible. It is important to characterize DEA 1 weak positive donors as DEA 1 positive (rather than negative). This is because transfusion of DEA 1 weakly positive blood to DEA 1 negative recipients will result in alloimmunization of the negative recipient [5]. Given the tendency for most dogs to express Dal and Kai antigens (like the majority of dogs who are DEA 4 positive) [6] and that testing for those antigens is not widely available, antigen testing in donors outside of DEA is uncommon.

It is imperative that canine donors also be screened for any vector-borne diseases that have the potential for transmission from the donor to the recipient within the blood product. Testing recommendations are derived from an American College of Veterinary Internal Medicine (ACVIM) consensus statement, which denotes minimal and optimal standards for screening canine donors [4]. The testing performed by the attending veterinarian will vary depending on the breed and geographical location, as well as test cost and availability. The consensus panel recommends annual testing of canine donors with the potential need to test more often for some pathogens in endemic regions or in donors that have repeated exposure to risk factors such as ticks [4]. Minimum standards recommend that canine donors test negative for *Anaplasma phagocytophilum, Anaplasma platys, Babesia canis vogeli, Babesia gibsoni, Bartonella henselae, Bartonella vinsonii* var. *berkhoffi, Ehrlichia canis*, and *Mycoplasma haemocanis*. Optimal standards include additional tests for *Brucella canis, Trypanosoma cruzi, Neorickettsia risticii, Leishmania donovani, Hepatozoon canis, and other Bartonella and Ehrlichia* spp. when indicated [4].

Precollection Screening

A dog's eligibility for donation is screened upon acceptance into the program and again prior to blood collection. This includes confirming the dog's recent health history, obtaining owner consent, performing a physical examination including measurement of vital signs, and measuring hematologic parameters. A form asking questions pertaining to illness, medications, travel, vaccinations, and preventatives is strongly advised. Dogs receiving a short course of medication(s) for an acute illness should be healthy for at least two weeks after the medications are finished before donating. Dogs should also receive routine ectoparasite prevention as recommended for the geographical region. The form should also outline the risks of donation and complications to monitor for afterwards and request consent from the owner/employee. A packed cell volume (PCV) and total solids are minimum requirements to assess hematologic status and should be at least 35% (preferably higher than 40%) and between 6.0–9.0 g/dl (60–90 g/l), respectively. Visual inspection of the plasma should also be performed and discolorations (e.g. hemolysis, icterus, or lipemia) investigated [7].

Collection Supplies and Equipment

Various combinations of collection containers and anticoagulant solutions can be used for the collection depending on the volume of blood collected; 450 ml collection sizes are most often used in dogs, with the appropriate amount of citrate-based anticoagulant. Acid citrate dextrose (ACD) and citrate phosphate dextrose adenine (CPDA) are commonly used for whole blood collection, while citrate phosphate dextrose (CPD) in conjunction with additive nutrient solutions (such as AS-1, AS-3, AS-5, and SAGM) are used for preparation of PRBC.

Single-chambered collection bags are used for the collection of whole blood (Figure 69.1). Multichambered bags, available in double, triple, or quadruple bag versions, are used for component separation (Figure 69.2). Historically, the availability of these bags has fluctuated based on changes driven by demand in the human blood banking industry. Blood collection bags with leukoreduction filters



Figure 69.1 Single chambered 450-ml blood collection bags filled with CPDA-1 used for canine blood collection.



Figure 69.2 Triple-chambered 450-ml blood collection bags filled with CPD and separate bag with AS-5 as nutrient solution.

are also available, although the benefits of leukoreduction among canine transfusion recipients are still being investigated [8]. For now, the practice of leukoreduction is at the discretion of the hospital and blood bank.

For collection of blood volumes less than 450 ml, 250 ml bags with CPDA-1 may be available. Alternatively, the use of syringes with 1 ml citrate-based anticoagulant for every 7 ml of blood collected can be used as an open-collection system that is unsuitable for storage but can be used for immediate transfusion.

In addition to the container and anticoagulant for blood collection, several instruments and pieces of equipment are needed. A blood tube stripper is used to apply pressure on the blood tube through rollers that push the blood into the blood bag once the collection has concluded. An atraumatic tubing clamp will allow for temporary clamping of the blood tubes without causing damage during the collection process. A tubing heat sealer will allow for permanent closure of blood tubing by melting and fusing the plastic to form a seal and can be a handheld (Figure 69.3) or benchtop device. Aluminum loops with crimpers can be a substitute for heat sealing, but they are not as stable and can result in accidental uncrimping during handling. Hospitals might also opt to use vacuum collection chambers with a suction machine, or blood collection mixers. For hospital performing component separation, a refrigerated large volume centrifuge, plasma extractor, and blood tube welders are recommended.

Collection Procedure

The collection process should be conducted in the least stressful manner for the donor. The use of positive reinforcement, stress-free handling, maintaining a calm environment, and staff expertise in reading the donor's stress level and preventing negative experiences is vital. Gather all supplies (Box 69.2) and secure a quite environment to perform the blood collection.

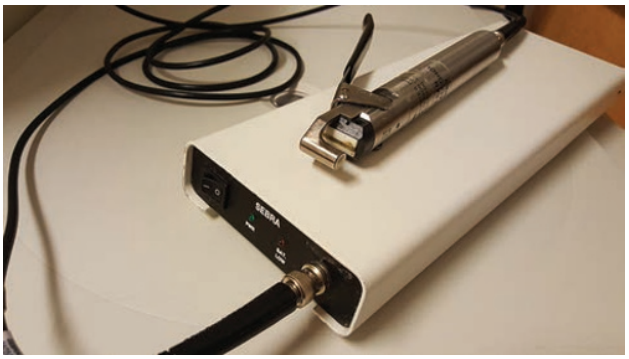


Figure 69.3 Handheld heat tubing sealer.

Box 69.2 List of Supplies Required for Canine Blood Collection [7]

- Topical anesthetic cream
- Collection system:
  - If whole blood, single-bag collection system
  - If component processing, multiple-bag collection system
- Gram scale
- Clippers and #40 blade
- Surgical scrub and gauze
- Blood tubing clamp
- Blood tubing stripper
- Heat sealer or metal sealing clip
- Treats/cookies
- Vacuum chamber[a]
- Suction machine[a]
- Blood collection mixer[a]

[a] Optional equipment

Canine donors are often able to donate without any sedation. The blood banking community prefers these "volunteer" donors as the perception is that the donor is not being forced to donate and it removes any concern about sedatives in the blood that is collected. Sedation might be considered at small doses to introduce donors to the collection process or in emergency situations where donors with desirable blood types can only donate with sedation. A combination of mu-agonist opioids and benzodiazepines are used most, while alpha-2 agonists are a less favorable option due to cardiovascular compromise. Oxygen supplementation is recommended for donors that are sedated during collection.

Administration of subcutaneous fluids post-donation is commonplace. Intravenous (IV) catheters can be placed in donors to facilitate fluid administration, although this is

not routine since the standard collection volume is unlikely to lead to hypotension.

Collection of the blood requires two to three team members to perform the tasks of restraint, venipuncture, and monitoring of the collection (for detailed steps, see Protocol 69.1). The external jugular vein is use for blood collection. The positioning that allows the donor and the phlebotomy team to stay comfortable for the duration of

Protocol 69.1 Canine Blood Collection Procedure [7]

1) Place blood collection system on the gram scale.
   a) If using the vacuum-assisted collection:
      i) Place the collection bag in the chamber, hanging it from the clip on the lid.
      ii) Close the lid with the tubing positioned exiting the notched portion of the chamber, leaving slack in the tubing within the chamber to prevent occlusion of the tubing.
      iii) Connect the vacuum chamber to the suction machine and set it at 50 mmHg. Lightly push up on the lid to confirm a negative pressure seal. A light push on the lid above the notch and tubing is often needed to form a seal. Turn the suction machine off once this is confirmed.
      iv) The chamber and bag assembly are placed on the gram scale.
   b) If using gravity-assisted collection, simply place the collection bag on the gram scale.
2) Temporarily clamp the tubing to prevent air from entering the bag when the needle cap seal is broken.
3) Turn on scale. Tare the scale with the collection system to zero.
4) Have the assistant restrain the donor comfortably in lateral or sternal recumbency.
5) Clip the venipuncture site in a gentle manner. Confirm adequate margins and location of shaving by occluding the jugular below the venipuncture site.
6) Inject lidocaine intradermally if topical analgesic cream was not applied during the pre-donation preparation.
7) Prepare venipuncture site by scrubbing gently.
8) Remove the needle cap and perform the venipuncture.
9) Release the clamp from the tubing and confirm blood flow (Figure 69.4). If using vacuum-assisted collection, turn on the suction machine using pressures ⩽101.6 mmHg. If there is no flow, check for occlusion of the tubing and reposition the needle as needed.
10) Monitor the progress of the blood collection via the gram scale reading (Figure 69.5) while periodically mixing the blood until the target weight is met (450 ml = 474 g). If blood flow is disrupted, troubleshoot by adjusting the needle or removing occlusions.
11) Once the desired amount is collected, reclamp the blood line close to the needle and remove the needle from the donor. Apply pressure digitally or with a pressure wrap to the venipuncture site.
12) Promptly use the line stripper to move the blood in the tubing into the collection bag and then gently rock the bag to mix the blood with the anticoagulant.
13) Allow blood to fill back into a length of tubing so that aliquots ("pigtails") can be made. Use metal sealing rings or a heat sealer to create several 2–4 inch (5–10 cm) segments.
14) Label the unit with blood type, donor identification, collection date, type of blood product, and expiration date.



Figure 69.4 Canine blood donor in lateral recumbency during blood collection. Note the gentle handling with light pressure applied to the muzzle.



Figure 69.5 The volume of blood entering the bag is measured through weight conversion during the donation.

the collection should be used to maximize the chances of a successful collection. The donor is typically positioned on an elevated surface such as a treatment table if gravity collection is used, while the donor can remain on the ground if vacuum chamber collection sets are used.

The venipuncture site is clipped and prepped with surgical scrub with care taken to prevent irritation of the skin. After clipping of the hair but before scrubbing, the use of topical anesthetic cream (e.g. liposomal lidocaine) will reduce the discomfort caused by venipuncture and is recommended with the appropriate contact time necessary for it to take effect. Applying the cream with piece of gauze and a light bandage wrap around the neck at the time of predonation examination and bloodwork typically allows enough contact time.

A decisive puncturing of the skin and vein without the need to redirect the needle will minimize pain and the chance of coagulation of blood during collection. Collection of the blood is continued until the desired volume of blood is collected. When collecting blood with a bag, a scale is used to weigh the amount of blood collected, calculated at a weight of 1.053 g/ml. A typical 450 ml collection will measure 474 g of blood, although 405–495 ml (426–521 g) is acceptable. During collection, the collection bag is rocked to mix the blood with anticoagulant as it enters the bag. As the blood is collected, the donor is monitored closely for any signs of hypotension or hypovolemia and discomfort. Once the desired collection weight is reached, the blood tubing clamp is placed on the tubing to cease the flow and the needle gently removed from the donor as pressure is applied to the venipuncture site. The mixing and monitoring of the collection weight can be automated using a blood collection mixer, which functions as a scale and rocker simultaneously, with many having the additional feature of clamping the line when the desired weight is reached.

As soon as the needle is removed from the donor, the blood within the tubing should be stripped and pushed into the blood bag with a tubing stripper to allow mixing with the anticoagulant before clotting occurs. Once the blood is mixed, a length of the blood tube can be allowed to refill to create several 10-cm length blood aliquots or "pigtails" (the tubes are marked at the appropriate lengths) and sealed to provide blood samples for crossmatching or quality control testing. The blood bag is then taken for immediate processing or stored.

Post-donation care of the donor involves applying gentle pressure on the venipuncture site for at least five minutes to allow for hemostasis. The donor is continuously monitored for signs of hypotension or hypovolemia. The donor is then provided with water and treats and given praise to provide positive reinforcement for the donation process. If fluids are administered, that can be performed any time after the conclusion of the donation. Once the donor is fully recovered without complications, they can be sent home with a plan to follow up with the owners after 24 hours to ensure an uneventful recovery at home.

Feline Donors

Selection and Screening

Feline donors must be selected and screened carefully to protect donors and recipients from harm. A complete history, comprehensive physical examination, blood tests, and bloodborne pathogen screening are recommended before collecting blood for transfusion. Feline donors should be between one and eight years of age, apparently healthy, and have a history that rules out prior transfusions or pregnancies and current or chronic illness. Indoor-only cats are preferred, whenever possible. Cats without a known history (e.g. strays) can be considered at the discretion of the attending veterinarian who must weigh the risks and benefits of using a donor who may have a history that could put the recipient at risk [9]. An agreeable temperament is ideal; cats that are stressed by car rides and trips to the hospital should only be used as donors if necessary. Conscious (i.e. without sedation) blood collections are increasingly being performed from cats, with similar success rates compared to sedated collections, suggesting that behavior and personality should be prioritized when selecting a feline donor [10]. Having a reliable, responsible, and committed owner is also advantageous to ensure compliance with the program.

A lean body weight of at least 4.5 kg (ideally 5 kg) is recommended for standard collections of 60 ml of blood [11]. Alternatively, cats that weigh less can be used during extenuating circumstances but the volume collected must be decreased to 12 ml/kg or less to avoid cardiovascular compromise [12]. A comprehensive physical examination must be performed to rule out a heart murmur of gallop rhythm, as well as any other abnormalities. An echocardiogram is recommended for all feline donors in which a murmur or gallop rhythm has been auscultated [13]. Alternatively, some feline donor programs may elect to screen all cats with an echocardiogram prior to donation, given the number of cats without murmurs that have structural heart disease, which should automatically exclude cats as donors [14]. Visualization of ectoparasites should disallow donation due to risk of exposure to vector-borne disease [4].

A complete blood count and serum biochemistry profile should yield normal results and must be repeated annually. Additional screening tests such as urinalysis and fecal parasite testing can be performed at the discretion of the attending veterinarian. Blood typing is also recommended to ascertain whether the donor is type A, B, or C (also known as AB) [15]. Unfortunately, screening for additional antigens such as Mik are not currently available.

It is imperative that feline donors also be screened for any vector-borne diseases that have the potential for transmission from the donor to the recipient within the blood product. Testing recommendations are derived from an ACVIM consensus statement, which denotes minimal and optimal standards for screening feline donors [14]. The consensus panel recommends annual testing of feline donors with the potential need to test more often for some pathogens in endemic regions or in donors that have repeated exposure to risk factors (e.g. ticks) or in donors who access the outdoors and have potential exposure to other untested cats. Minimum standards recommend that feline donors test negative for *A. phagocytophilum, B. henselae, Mycoplasma haemofelis,* feline leukemia virus, and feline immunodeficiency virus. Optimal standards include additional tests for *A. platys*, other *Bartonella* spp., *Cytauxzoon felis,* and *E. canis* when indicated [4].

Precollection Screening

A cat's eligibility for donation must be screened before acceptance into the program and again prior to each blood collection. This includes confirming the cat's recent health history, obtaining owner consent, performing a physical examination including measurement of vital signs and cardiac auscultation, and measuring hematologic parameters. A form asking questions pertaining to illness, medications, travel, vaccinations, and preventatives is recommended. Cats receiving a short course of medication(s) for an acute illness should be healthy for at least two weeks after the medications are finished before donating. Cats should also receive routine ectoparasite prevention as recommended for the geographical region. The form should also outline the risks of donation and complications to monitor for afterwards and request consent from the owner/employee. A PCV or hematocrit are minimum requirements to assess hematologic status and should be at least 30–35%. Performing in-hospital testing for feline leukemia virus and feline immunodeficiency virus is also recommended prior to each blood collection if the donor has contact with other cats or access to the outdoors [12].

Collection Supplies and Equipment

Collection supplies required for feline blood collection vary depending on the chosen protocol (Box 69.3). For the basic task of blood collection, a semiclosed collection system that includes a 21–19 gauge butterfly catheter attached to a 60-ml syringe and small collection bag that has been sterilized is most often used. The system is considered "semiclosed" since sterility is broken to add the anticoagulant just prior to blood collection. Some hospitals choose to use an open system using a butterfly catheter and collecting blood into three 20-ml syringes with a single venipuncture. While a semiclosed system is likely to provide a smaller chance of bacterial contamination, whole blood collected by an open system and stored for up to 35 days has shown acceptable levels of hematologic and biochemical changes and no bacterial contamination [16]. Closed collection systems can be custom made using tubing welders to add anticoagulant to a smaller bag and attaching an apheresis needle to the bag under a laminar flow hood. Blood collections using custom made closed systems have similar success rates, hematologic parameters, and bacterial contamination rates compared to blood collections using open systems, but the use of a vacuum collection set significantly shortens the collection time [17].

Collection Procedure

Unless a custom closed collection system is used, the collection process starts with the addition of anticoagulant to the collection system just prior to the collection (for detailed instructions, see Protocol 69.2).

Box 69.3 List of Supplies Required for Feline Blood Collection [12]

- Sedation suitable for the donor[a]
- Clippers and #40 blade
- Aseptic scrub for the peripheral intravenous (IV) catheter[a] placement and jugular venipuncture
- Topical anesthetic cream
- Open blood collection system:
  - 3 × 20-ml or 6 × 10-ml syringes
  - Sterile syringe caps
  - 21-gauge or 19-gauge butterfly catheters (extras in case needed)
- Semiclosed collection system: if not using an open system
- Anticoagulant
- Ocular lubricant
- Endotracheal tube(s) and laryngoscope
- Oxygen source
- Blood pressure and pulse oximetry monitoring equipment
- 250-ml or 500-ml bag isotonic crystalloid fluid
- IV fluid administration set
- For subcutaneous fluids: 19-gauge butterfly catheter or 18-gauge needle
- For IV fluids:[a]
  - 22-gauge IV catheter
- Materials to secure the IV catheter in place
- Flush for the IV catheter
- Fluid administration pump

[a] Optional equipment

Protocol 69.2 Feline Blood Collection Procedure Using a Semi-Closed System [12]

1) Fill the 60 ml syringe with the chosen anticoagulant (1 ml/7 ml intended collection volume) through one of the injection ports using aseptic technique. Prime the three-way stopcock and butterfly catheter with the anticoagulant (Figure 69.6).
2) If needed, administer the sedation and allow it to take effect. If desired, place an intravenous (IV) catheter.
3) Gently position the donor in either lateral, sternal, or dorsal recumbency, apply ocular lubrication to both eyes, and have an assistant extend the neck.
4) Clip the fur at the venipuncture site. Using the skin preparation materials and surgical scrub technique, clean the skin over the jugular vein (Figure 69.7).
5) Pass the syringe to the second assistant while the phlebotomist holds the butterfly catheter.
6) When the position of the jugular vein is confidently determined, perform the venipuncture (Figure 69.8).
7) Request that the assistant with the syringe very gently create a small amount of negative pressure by pulling the plunger to approximately the 1 ml mark. This will allow blood to flow into the catheter if the needle is positioned within the vessel.
8) Draw the blood into the syringe while rocking the syringe.
   a) Care must be taken not to cause collapse of the vein or a "flutter," as both will restrict blood flow and promote coagulation. A "flutter" occurs when the bevel of the needle gets drawn against the vessel wall. The phlebotomist will feel the "flutter" as the collection line will vibrate, and the vessel will visibly collapse. Reducing the negative pressure on the syringe can alleviate both situations.
9) Once the entire 60 ml has been drawn, turn the three-way stopcock off to the syringe (Figure 69.9).
10) Remove the needle and apply pressure to the venipuncture site for a minimum of 5 minutes.
11) Turn the three-way stopcock open to the bag from the syringe and push the blood into the bag gently so as not to damage the red blood cells (Figure 69.10).
12) Using metal sealing rings or a heat sealer, seal off the tubing from the three-way stopcock, leaving three 2–4 inch (5–10 cm) segments.
13) Label the bag appropriately, including the date of collection, donor information, blood type, product type, and expiration date.
14) Provide IV or subcutaneous/. fluids to the donor and reverse sedation if indicated.

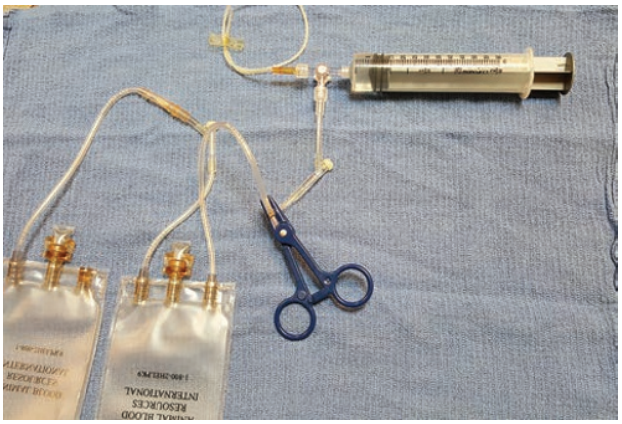


Figure 69.6 Semiclosed collection system with a double-chamber blood bag set attached, primed with anticoagulant to ready for blood collection.



Figure 69.7 Feline donor in lateral recumbency with the venipuncture site prepared.



Figure 69.8 Venipuncture being performed with the butterfly catheter attached to the semiclosed system for feline blood collection.

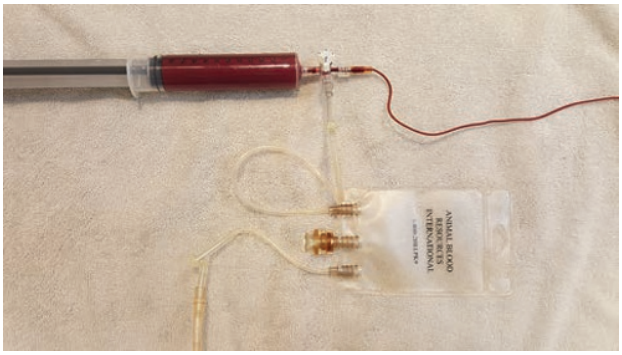


Figure 69.9 Full collection of blood totaling 60 ml in the collection syringe of the semiclosed system.



Figure 69.10 Collected blood being pushed gently into the blood bag through the three-way stopcock.

Box 69.4 Sedation Protocols used for Feline Donations

Option A:
- Midazolam 0.2 mg/kg intravenous (IV)
- Ketamine 3 mg/kg IV
- Butorphanol 0.1 mg/kg IV

Option B:
- Midazolam 0.25 mg/kg intramuscular (IM)
- Ketamine 5 mg/kg IM
- ± Butorphanol 0.2 mg/kg IM

Option C:[18]
- Alfaxalone 2 mg/kg IM
- Butorphanol 0.2 mg/kg IM

Unlike dogs, feline blood donors are commonly sedated to allow for a stress-free collection. Various sedation protocols are used, with combinations of butorphanol, midazolam, ketamine, and/or dexmedetomidine reported (Box 69.4). Alfaxalone and butorphanol administered intramuscularly result in adequate sedation and equal quality of recovery compared to a dexmedetomidine and butorphanol combination [18] and is the typical sedation combination of choice. When sedation is used, monitoring the blood pressure, and administering oxygen supplementation is recommended. While anesthetic induction agents such as propofol or inhalant anesthetics have been used historically, they are no longer recommended due to concerns for hypotension.

An IV catheter can be placed in feline donors to facilitate post-donation fluid administration and to provide venous access should there be any emergent complications that occur during the donation. Alternatively, fluid administration can be provided subcutaneously at the discretion of the attending veterinarian. The supplies gathered will vary depending on the method of fluid administration.

A quiet environment and the use of topical anesthetic cream is essential for all feline donations. Minimizing the amount, frequency, and stress caused by handling, maintaining a calm environment, and staff expertise in reading the donor's stress level and preventing negative experiences is vital. Collection of the blood requires two to three team members to perform the tasks of restraint or positioning, venipuncture, handling of the collection syringe, and monitoring of the donor. During collection, the donor can be positioned laterally, sternally, or ventrally to perform venipuncture of the external jugular vein.

The venipuncture site is clipped and prepped with surgical scrub with care taken to prevent irritation of the skin. The same considerations for application of topical anesthetic cream in dogs should be applied to cats. A decisive puncturing of the skin and vein without the need to redirect the needle will minimize pain and chances of coagulation of blood during collection. Gentle suction on the syringe and rocking the syringe gently to mix the incoming blood with the anticoagulant are continued throughout the collection. While the blood is being collected, the donor is monitored closely for any signs of hypotension or hypovolemia and discomfort. Once the desired collection volume (typically a total of 60 ml with the anticoagulant included) is reached, the needle is gently removed from the donor as pressure is applied to the venipuncture site. The blood is then pushed into the collection bag with some left in a length of blood tubing to create several blood aliquots.

Post-donation care of the donor involves applying gentle pressure on the venipuncture site for at least five minutes to allow for hemostasis. Sedative agents can be reversed to reduce recovery time and if it will not cause added stress to the donor. The donor is continuously monitored in a quiet environment for signs of hypotension or hypovolemia until fully recovered. If subcutaneous fluids are used, they should be administered prior to the sedatives wearing off.

If IV fluids are given, 10 ml/kg of isotonic crystalloids over 0.5–3 hours is recommended [12]. Once the donor has fully recovered without complications, food and water can be offered. The donor can then be discharged with a plan to follow up with the owners after 24 hours to ensure an uneventful recovery at home.

Processing and Storage of Blood Products

Processing

The benefits of component therapy have led to a widespread practice in veterinary medicine. Because of this, commercial blood banks have had a difficult time keeping up with demand and some veterinary practices are choosing to collect and process their own components. While there is no regulation that prevents hospitals from processing their own blood components, the hospital must take into consideration the resources, expertise, and effort required to produce blood products of proper quality. The simplest form of blood component separation to achieve in-hospital is the separation of whole blood into PRBCs and plasma. Separation of blood into other components is beyond the scope of this chapter and readers are encouraged to consult other resources [19].

Separation Supplies and Equipment

A multichambered collection bag is required for component processing, otherwise the supplies and equipment used for blood collection do not differ between products administered as whole blood or components. In addition to the instruments and equipment mentioned above, the hospital will require a refrigerated centrifuge with rotor cups that can accommodate the 450-ml multichambered collection bags, as well as a plasma extractor to separate the blood components.

There are two types of centrifuges: benchtop and floor models. The benchtop centrifuge has the advantage of using less physical space to store and operate, however, it often has physical limitation in the relative centrifugal force (RCF) it can create because of the shorter rotor length and motor strength. It will also have limits as to the number of rotor cups it can hold. A floor model is physically larger but has the advantages of achieving a higher RCF, accommodating a larger number of rotor cups, and tends to be more physically stable during operation (Figure 69.11). Modern centrifuges can program temperature, acceleration, and deceleration settings with preset memory, and can include features such as balance stabilization and imbalance alarms.

A plasma extractor is a device that presses the plasma into one of the satellite bags after separation by the centrifuge. Manual plasma extractors use a spring-loaded panel



Figure 69.11 A large volume, refrigerating centrifuge for blood processing.



Figure 69.12 Plasma extracted using a manual plasma extractor.

that presses against a second surface to move the plasma out vertically into the satellite bag and requires close attention during operation (Figure 69.12). Automated plasma extractors are also available, which reduce the manual operation required to separate plasma from the red blood cells, although at significantly higher expense.

Separation and Processing Protocols

If there will be a delay in processing the whole blood into components, the blood should be stored at refrigeration temperatures (33.8–42.8°F; 1–6°C) unless there is consideration for using the whole blood as a source of platelets, in which case room temperature (68–75.2°F; 20–24°C) storage is appropriate. The processing of whole blood into

PRBCs and plasma should be conducted within 24 hours (maximum) of collection.

The first step in component separation is to centrifuge the whole blood to separate it into cellular components and plasma. The RCF, temperature, and duration of spin vary depending on the desired product (see Protocol 69.3 for detailed steps and settings).

Packed Red Blood Cells Plasma is extracted from whole blood to obtain PRBCs. Historically, 20% of the plasma was intentionally left with the PRBCs to serve as a nutrient solution, but with the advent of additive solutions that contain combinations of adenine, glucose, and mannitol to serve as storage nutrients, as much of the plasma as possible is extracted. The additive solution is added approximately at 1 ml solution per 2 ml RBCs, resulting in a 55–70% PCV depending on the donor's PCV and the amount of plasma extracted. The PCV of the processed PRBCs should be measured and written on the label to facilitate accurate dosing.

Protocol 69.3 Blood Component Processing Protocols for Packed Red Blood Cells and Plasma [19]

1) Turn the centrifuge on and allow it to cool down to a target temperature of 33.8–42.8°F (1–6°C).
2) Weigh the whole blood collection, including satellite bags, and record the weight.
3) Place the whole blood collection into the centrifuge bucket. Ensure that the placement within the bucket meets recommendations of the the centrifuge manufacturer (e.g. place toward the outside of a circular centrifuge bucket).
4) Fold the attached bags and lines securely into the bucket next to the unit. If using a set that contains a leukoreduction filter, ensure that it is tucked away so that it will not cause damage to the unit while centrifuged.
5) Weigh the filled centrifuge buckets and select the heaviest bucket to balance all others against. The centrifuge buckets must be accurately balanced. Suitable balances include pieces of tubing, unused blood collection bags filled with saline, weights provided by manufacturers, or other soft plastics or rubbers.
6) Check that all the buckets swing freely in their axis.
7) Centrifuge the unit(s) following the appropriate protocol.
   a) Hard spin: 5000 g, 7 minutes (for PRBCs and plasma)
   b) Soft spin: 2000 g, 3 minutes (typically used for platelet products)
8) Prepare all other equipment: plasma extractor, tube sealer, tube stripper, and scissors.
9) At completion of the centrifugation, carefully inspect the units to ensure that the plasma is clear.
10) Carefully remove the bucket from the centrifuge, gently remove the unit, place it in the prepared plasma press, and gently release the pressing plate.
11) Snap the seal at the top of the blood collection pack to allow plasma to run into the satellite bag.
12) For manual plasma presses, use thumb and index finger or an atraumatic tube clamp to pinch the line shut while stopping the pressing plate from further plasma expression. Raising the plasma bag to the same level as the RBCs can also assist to prevent further fluid transfer.
13) Seal the line between the RBCs and plasma using the available method. Ensure that the seal allows both the plasma and red cells to remain sterile once physically separated from each other.

Additional instructions for PRBCs:

1) Snap the seal between the additive solution and concentrated red cells.
2) Suspend the additive solution and allow the fluid to run onto the concentrated RBCs.
3) Remove the additive solution bag if not required, using the method of tube sealing available and leaving a tubing segment long enough to later generate several sub78segments.
4) Gently mix to resuspend the red cells in the additive solution.
5) Use a tube stripper to strip the line content into the bag. Repeat three or four times to ensure a representative sample of the final RBC product remains in the line.
6) Subdivide the tubing into segments or "pigtails" using the available method of tube sealing.
7) Remove a "pigtail" for measurement of final PCV and record value.
8) Weigh the unit if desired to establish approximated volume.
9) Label the bag, then store in the refrigerator.

Additional instructions for plasma:

1) Label the bag and weigh if desired.
2) Place in a freezer at −0.4°F (−18°C) or colder, or a blast freezer. Lay the unit flat and in single layers to freeze as rapidly as possible.

Plasma Products Plasma is traditionally considered fresh frozen plasma when separated and frozen within eight hours of collection and contains viable levels of all coagulation factors. It is considered frozen plasma if separation occurs longer than eight hours after collection, which affects the activity of unstable coagulation factors. There is, however, evidence supporting plasma frozen within 24 hours of collection having minimal changes in coagulation factor activity and hemostatic protein content, possibly extending the length of time before plasma must be frozen for its maximum benefits [20].

Leukoreduction The removal of white blood cells can be performed before or after processing using leukoreduction filters. Because white blood cells can release cytokines during storage, preprocessing leukoreduction seems to have additional benefits in reducing the cytokine concentration and subsequent inflammatory response by recipients upon transfusion [21, 22]. Blood collection bags with leukoreduction filter attachments (Figure 69.13) are available and allow for filtration of the blood shortly after the collection of blood and before component separation. Alternatively, post-processing leukoreduction can be performed cage side prior to administration of the blood product.

Storage

Refrigerator

The shelf life of whole blood and PRBCs varies depending on the anticoagulant and nutrient solution used (Table 69.1). PRBCs and whole blood should be stored in a refrigerator designated only for blood products (Figures 69.14 and 69.15). Household refridgerators have traditionally been used for blood product storage; however, they are not designed to maintain narrow temperature ranges. Refrigerators designed for use in laboratories or blood banks are recommended. A designated refrigerator will ensure unnecessary opening and closing of the door, which can lead to temperature fluctuations. The refrigerator must have temperature monitoring capabilities that include a minimum–maximum thermometer inside the refrigerator, digital readout, and chart recorder, or a temperature probe attached to a recording device. Independently operated temperature monitoring devices can also be used and are especially helpful should a power outage occur. Temperature monitoring devices using insulating liquid mimicking thermal consistency of blood enables an accurate measurement of the temperature the blood bags are experiencing during storage (Figure 69.16). Alarms should be set such that personnel can be alerted about harmful temperature deviations that could damage stored products.

Table 69.1 Shelf life of blood products [19].

Blood product	Shelf life
Red blood cell products (33.8–42.8°F; 1–6°C):	
Whole blood	CPDA-1: 28 days
Packed red blood cells	CPDA-1: 20–21 days
	CPD w/AS-1: 35 days
	CPD w/AS-3 or AS-5; 35–37 days
	CPD w/SAGM: 42 days
Plasma products (≤ 0.4°F; ≤ 18°C):	
Fresh frozen plasma	1 year
Frozen plasma	5 years

CPD, citrate phosphate dextrose; CPDA, citrate phosphate dextrose adenine.
Source: Mansell and Boller 2016.

Freezer

Freezers designed for research specimens or blood banks are also recommended for plasma product storage over traditional refrigerator-freezer combinations, to maintain the temperature between −0.4°F (−18°C) to −22°F (−30°C; Figures 69.15 and 69.17). Freezing of plasma products is traditionally performed at −0.4°F (−18°C); however, there

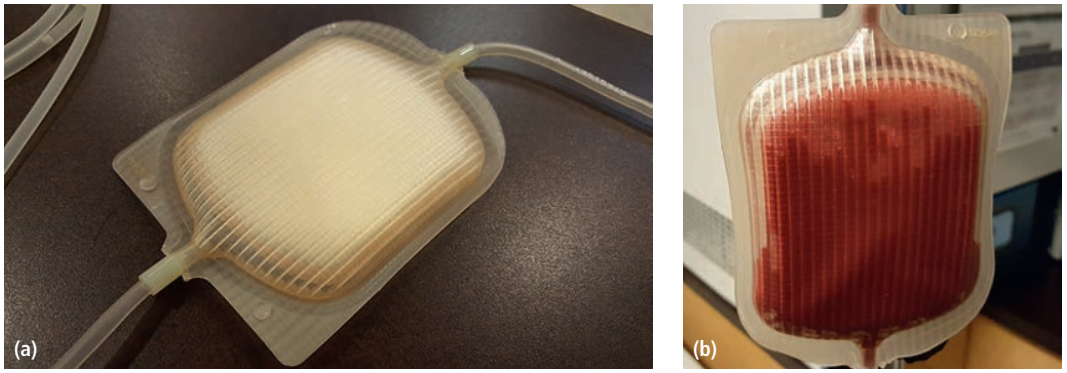


Figure 69.13 (a) Prestorage leukoreduction filter incorporated in blood collection bag tubing. (b) Prestorage leukoreduction filter in use.



Figure 69.14 Inside view of a benchtop blood storage refridgerator.



Figure 69.16 Insulating liquid container installed inside the blood storage fridge to accurately measure temperature.



Figure 69.15 Benchtop blood storage refrigerator (left) and plasma freezer (right).



Figure 69.17 Inside view of a benchtop plasma freezer.

are recommendations from human blood banking to freeze plasma products at −22°F (−30°C) or colder to better preserve coagulation factors [19].

Quality Assurance

Quality assurance in the context of blood banking is the maintenance of a desired level of quality of a blood product and occurs by carefully attending to each stage of the collection, processing, and storage of the blood product. As such, standard operating procedures are recommended and must include protocols for blood collection, blood processing, maintenance of storage equipment, maintenance

of blood product logs, and assessment of the quality of blood products prior to administration.

Maintenance of Equipment

Centrifuge

The hospital's blood product centrifuge must be inspected regularly for wear and tear such as cracks or stress fractures on the rotor. The centrifuge should also be kept clean and dry without the use of corrosive or abrasive solvents, and it should be periodically serviced and calibrated according to its level of use [19].

Refrigerator

Daily temperature checks should be a routine part of maintenance and periodic recalibration is recommended to confirm the set temperature is accurate. The refrigerator should also be routinely wiped clean with appropriate cleaning agents and dust removed from filters, condensers, and motors [19].

Freezer

Freezer maintenance is as described above, with the addition of periodic defrosting and cleaning of door seals to prevent excessive buildup of ice. The freezer designated for blood product storage should be temperature monitored as described above, so that defrosting and refreezing cycles are detected. Otherwise, two methods are available to detect unintentional thawing of frozen products. A rubber band may be placed around a plasma bag prior to freezing and then removed once the product is frozen. If the indentation on the product disappears during storage or transport, the product has been thawed. Alternatively, an air bubble can be left within the plasma product after which the product is frozen while lying flat, such that the bubble remains in the middle of the top side of the bag. The plasma should then be stored upright and if the bubble moves to the top of the bag, this is an indication that the product has thawed [19].

Maintenance of Blood Product Logs

Hospitals must maintain a blood product usage log that that includes the date a blood product was received from another source or collected in the hospital, the source or donor from which it was obtained, and whether adverse events were noted during its administration.

Blood Product Quality Control

All items used for blood product collection and storage should have sterility certificates attached and must be used within any designated expiration dates. Blood products should not be used from donors if their screening tests are incomplete. Aliquots of blood from donors can be stored long term for repeat testing should a recipient develop a transfusion-acquired infection.

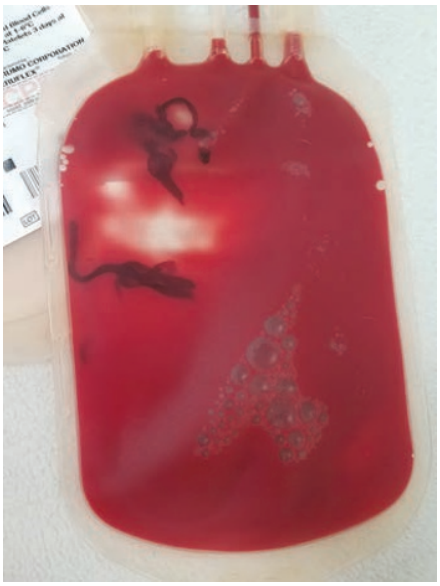


Figure 69.18 A bag of packed red blood cells containing visible blood clots, and must not be administered. *Source:* Courtesy of Laura Call, LVT.

All blood products must be visually inspected prior to administration. PRBC or whole blood units that appear discolored (e.g. dark purple to black, green) or contain visible clots and/or fibrin strands (Figure 69.18), must not be used and should have a bacterial culture submitted prior to being discarded. The presence of cell aggregates that appear as white clumps that do not dissipate with gentle rocking also indicate a red cell product should not be used. However, small white specks that dissolve with warming of the blood are more likely to be lipid aggregates, which do not typically require disposal [23]. Percentage hemolysis can also be calculated to quantify the degree of red cell degradation (Eq. 69.1). Units with hemolysis greater than 1% should not be administered [24].

$$\text{Total hemolysis of the unit} = (100 - \text{Hct}) \times \text{plasma Hemoglobin (g/dL)} / \text{total Hb (g/dL)} \quad (69.1)$$

Plasma units may appear lipemic, indicated by a milky appearance, which typically occurs with blood collected from donors postprandially (Figure 69.19). The presence of lipemia does not compromise the efficacy or safety of the product and transfusion of lipemic products remains acceptable [23]. Because not all plastics are suitable for frozen storage, cracks or breaks in the frozen product bag should be noted and the product discarded (Figure 69.20).



Figure 69.19 Lipemia is noted within the plasma during separation. *Source:* Courtesy of Anna Bakirova, Chance Bio, Moscow.



Figure 69.20 A blood product contains a visible crack and must be discarded. Courtesy of Anna Bakirova, Chance Bio, Moscow.

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Section Ten

Nursing Care of Specific Populations

70

Care of the Patient with Intracranial Disease

Marie K. Holowaychuk and Charlotte E. Donohoe

Animals with intracranial disease may have any number of underlying disorders such as traumatic brain injury (TBI), idiopathic epilepsy, meningitis, encephalitis, or neoplasia. Signs associated with the disease will vary and the patients will differ in terms of the intensity of the nursing care they require. For example, one patient may be a dog hospitalized for observation after cluster seizures, while another may be a cat recovering from a craniotomy to remove a brain tumor. Regardless of the underlying disease or severity of signs, the goal for all patients with intracranial disease is to preserve their brain function. This is achieved by ensuring adequate cerebral blood flow (CBF) by maintaining a normal cerebral perfusion pressure (CPP) of 50–60 mmHg [1]. In the normal or uninjured brain, pressure autoregulation maintains a constant CBF despite a wide range of mean arterial blood pressures (MAP, 50–150 mmHg) [1]. When MAP is maintained within that range and CPP increases, cerebral blood vessels constrict to decrease overall cerebral blood volume and intracranial pressure. However, as MAP decreases to below 50 mmHg, CBF decreases and is dependent on MAP [1]. In addition to MAP, CPP is affected by changes in ICP (Eq. 70.1). Elevations in ICP will decrease CPP, resulting in worsening of brain function [1]. The aim in all patients hospitalized for intracranial disease is to ensure stability of cardiovascular parameters, namely, blood pressure, and most importantly, to prevent elevations in ICP.

$$\text{CPP} = \text{MAP} - \text{ICP} \tag{70.1}$$

ICP may increase for many reasons, including the underlying disease process itself. For example, masses such as tumors or abscesses, cerebral edema or hemorrhage following a traumatic injury, or hydrocephalus due to a congenital malformation, are all space-occupying lesions that can cause elevations in ICP. Because a rigid nonexpandable skull surrounds the brain, if there are pathologic increases in intracranial tissue or fluid, the ICP will increase because of the additional intracranial volume (Figure 70.1) [2]. If surgery is not an option to remove the structural abnormality contributing to the increase in intracranial volume, other treatments must be employed. These might include the administration of steroids to reduce tumor-associated inflammation in patients with neoplasia or to reduce the production of cerebrospinal fluid (CSF) in patients with hydrocephalus. Alternatively, mannitol or hypertonic saline may be given to decrease ICP (Box 70.1). Ultimately, the goal with these treatments is to reduce intracranial tissue or fluid volume.

Intracranial volume and thus ICP can also be increased due to physiologic increases in blood volume. These physiologic alterations can be monitored and addressed by veterinary technicians/nurses, to prevent increases in ICP. For example, increases in venous blood volume might occur due to jugular venous obstruction, a head-down position, or increases in central venous pressure [2]. Therefore, to decrease ICP, it is important that patients with intracranial disease are carefully positioned so as not to place pressure on the jugular veins or increase blood flow to the head. Additionally, increases in arterial blood volume and subsequently ICP can occur due to systemic hypertension, hypoventilation, hypoxemia, or increases in cerebral metabolic rate due to seizures, agitation, or elevations in body temperature [2]. Thus, it is imperative that patients with intracranial disease maintain normal blood pressure, ventilation, and oxygenation, and that seizures are treated quickly and any temperature elevations are eliminated. Clearly, the veterinary technician or nurse has the particularly important task of monitoring for changes that could lead to ICP elevations and, if possible, preventing some of the physiologic changes that can increase ICP.

Because these physiologic changes can occur very quickly, resulting in rapid deteriorations in a patient's status, animals with intracranial disease must be constantly and closely monitored. Therefore, it is imperative that

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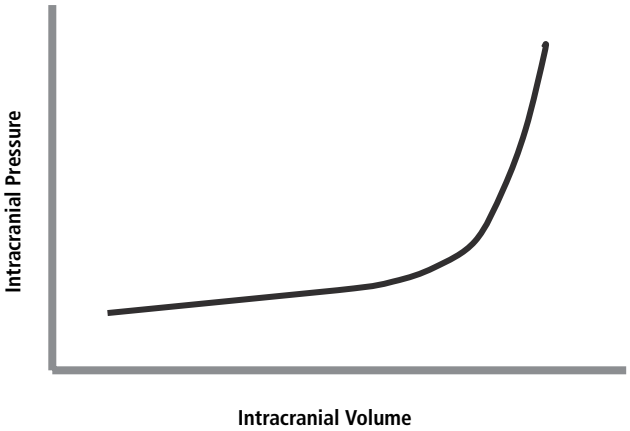


Figure 70.1 Relationship between intracranial pressure (ICP) and volume. At low intracranial volumes, increases in intracranial volume produce minimal changes in ICP. However, small increases in intracranial volume will eventually cause dramatic increases in ICP.

these patients receive 24-hour monitoring. This ensures that nursing care and appropriate monitoring is provided continuously, so that changes in a patient's status can be detected quickly and treated appropriately. Many of the monitoring devices or treatment modalities required for patients with intracranial disease are available only at 24-hour or specialty hospitals. For these reasons, to provide the best care possible it is recommended that patients with intracranial disease are transferred to a facility capable of providing 24-hour monitoring and nursing care.

Serial Neurologic Examinations

Patients with intracranial disease require constant monitoring, including serial neurologic examinations. Because the patient's condition can change very quickly, it is

Box 70.1 Medications [3]

Mannitol

Mechanism of action

- Increases the osmotic gradient between the vasculature and brain causing fluid to move out of the brain and into the blood.

Storage

- Room temperature for lower concentrations.
- Incubator for higher concentrations (>15%) to avoid crystal formation.
- Bottles stored at room temperature can be heated in a warm water bath or incubator up to 140°F (60°C) to dissolve crystals, but should be cooled to body temperature prior to administration.
- Discard if unsuccessful in dissolving the precipitate.

Administration

- Dose: 0.5 g/kg IV:
  - Draw up using a filter needle (Monoject™ filter needle, Kendall Healthcare, Mansfield, MA) or administer with an inline filter (Hemo-Nate® filter, Gesco International, San Antonio, TX).
  - Give slowly via a syringe pump or slow push (over 15–20 minutes).
  - Can be repeated every 6–8 hours as needed.
  - Constant rate infusions are not recommended.
  - Use a dedicated line (i.e. do not mix with other medications).
- Monitor:
  - Urine output
  - Electrolytes (sodium, potassium) and osmolality
  - Respiratory rate and effort (risk of fluid overload)
  - Blood pressure
- Cautions:
  - Hypovolemia
  - Severe dehydration
  - Pulmonary edema
  - Hyperosmolar states (e.g. ethylene glycol toxicity).

Hypertonic saline (7–7.5% NaCl)

Mechanism of action

- Increases the osmolality of the blood to cause water to shift from the brain into the intravascular space.

Administration

- Dose: 3–5 ml/kg [4]:
  - Typically drawn into a syringe and administered via syringe pump or slow push (over 15 minutes).
  - Administer in conjunction with isotonic crystalloids.
  - Constant rate infusions are not recommended.
- Monitor:
  - Urine output.
  - Electrolytes (sodium and chloride).
  - Respiratory rate and effort (risk of fluid overload).
  - Blood pressure and heart rate (transient hypotension and vagally mediated bradycardia can occur during rapid administration).
- Cautions:
  - Significant hypernatremia or hyperchloremia
  - Intravascular overload
  - Dehydration
  - Repeated doses can cause phlebitis (give via a central line if available).

Box 70.2 Serial Neurologic Examinations

Normal Findings

- Alert and responsive to the environment
- Normal pupillary light reflex
- Normal pupil size bilaterally
- Normal gait and normal spinal reflexes

Mild Intracranial Disease

- Depressed or inappropriate mentation
- Slow pupillary light reflexes
- Normal pupil size bilaterally
- Paresis

Moderate Intracranial Disease

- Stuporous/semicomatose but responsive to noxious (i.e. painful) stimuli
- Absent pupillary light reflex
- Miotic (i.e. pinpoint) pupils
- Recumbency ± extensor rigidity of the forelimbs

Severe Intracranial Disease

- Comatose and unresponsive to noxious stimuli
- Absent pupillary light reflex
- Mydriatic (i.e. dilated) pupils
- Recumbent ± loss of muscle tone and spinal reflexes
- Loss of facial sensation or gag reflex

important that the neurologic status is assessed frequently to detect changes as they arise. The veterinary technician/nurse must contact a veterinarian immediately if the patient's neurologic status is deteriorating, or if the results of the neurologic examination are difficult to interpret.

Neurologic examination findings will vary depending on the severity of the intracranial disease and can include mild to pronounced neurologic deficits. It is especially important that the technician/nurse coming on shift immediately assesses any patients with intracranial disease to obtain a baseline from which to compare subsequent examinations. If an initial examination is not performed, observations by the technician/nurse might be assessed as "normal" for that patient when in fact they may have progressed from previous evaluations. Such a circumstance could result in a devastating failure to detect deterioration in a patient's status in a timely manner.

The patient should be assessed frequently to determine its level of consciousness, pupillary light reflexes (PLRs), pupil size, posture, and reflexes (Box 70.2). Almost all patients with intracranial disease will have an abnormal level of consciousness varying from a mildly altered mentation to a coma. If the patient is sedated, it may be difficult to assess its level of consciousness. Regardless, patients receiving sedative medications should be intermittently "woken up" (i.e. sedation briefly stopped) to enable proper assessment of their mentation and level of consciousness (Box 70.3). Normally, patients with mild intracranial disease will still have periods of alertness and responsiveness to their environment. As the severity of intracranial disease progresses, patients will become increasingly altered and less capable of responding to their environment.

Box 70.3 Assessing Mentation and Level of Consciousness

- Normal: alert and responsive to the environment.
- Altered: abnormal or decreased response to environmental stimuli.
- Stuporous/semicomatose: responsive only to painful stimuli.
- Comatose: unresponsive to painful stimuli.

Additionally, some patients will exhibit an inappropriate response to environmental stimuli, such as excessive vocalization. Patients that progress to a stuporous or semicomatose state will be less responsive to visual or auditory stimuli in their environment until they are responsive only to painful (i.e. noxious) stimuli. Finally, an animal that is comatose is unresponsive to all environmental stimuli including repeated noxious stimuli [5].

It is important when assessing patients with intracranial disease never to assume that a patient is "sleeping," and for that reason not assess its mentation. A patient that has otherwise been responsive to environmental stimuli may progress into a semicomatose to comatose state, and if one assumes that the patient is "sleeping," their condition may continue to deteriorate without intervention. Thus, patients should always be awakened every one to two hours if they appear to be sleeping, to assess their level of consciousness accurately and ensure that it is not deteriorating.

It is also important that PLRs and pupil size are assessed in patients with intracranial disease on a frequent basis. PLR is tested by shining a bright light into the pupil and

assessing for constriction of the pupil (direct reflex) [5]. The opposite pupil should constrict at the same time (consensual reflex). It is not necessary to assess the consensual reflex if the direct PLR is present in both eyes [5]. Generally, a critically ill patient with mild elevations in ICP will have normal PLRs that will progress to slow or completely unresponsive pupils in patients with moderate to severe elevations in ICP [6]. In addition to alterations in the PLR, patients with moderate to severe elevations in ICP will also exhibit changes in their pupil size. While patients with mild elevations in ICP will have normal to slightly miotic (i.e. constricted) pupils, patients with moderate to severe elevations in ICP will have progressively altered pupil sizes ranging from miotic or pinpoint pupils to unilateral or bilateral mydriatic (i.e. dilated) pupils that are completely unresponsive to light [6]. Miotic or mydriatic pupils in a patient with intracranial disease and previously normal-sized pupils represents an emergency and a veterinarian should be contacted immediately.

Ideally, patients with intracranial disease that are not sedated should have their posture and reflexes assessed frequently. Patients with mild intracranial disease will exhibit a normal posture, gait, and reflexes. As the severity of intracranial disease worsens, patients will exhibit varying degrees of paresis (weakness) progressing to full recumbency [6]. Additionally, patients may exhibit other abnormalities including circling, a head tilt, decerebellate rigidity, or decerebrate rigidity. Decerebellate rigidity is characterized by opisthotonus (extension of the head and neck), with the forelimbs extended [5]. Animals exhibiting decerebellate rigidity also typically have flexed hips and normal mentation. In contrast, patients with decerebrate rigidity have a stuporous or comatose mentation that is associated with opisthotonus and extension of all limbs [5]. Finally, a patient with severe intracranial disease may have a complete loss of muscle tone and decreased to absent spinal reflexes. The simplest spinal reflex to test in patients with intracranial disease is the withdrawal reflex. This reflex is elicited by applying a noxious stimulus to the tested limb by pinching the nail bed or toe with fingers or a hemostat [5]. The normal reflex is contraction of the flexor muscles and withdrawal of the tested limb.

Cranial nerve reflexes should also be assessed, the easiest of which include the gag reflex and facial sensation. To assess the gag reflex, stimulate the pharynx with a finger to elicit a gag [5]. An absent gag reflex may be due to excessive sedation if the patient is receiving sedatives; however, in the absence of sedating medications, loss of the gag reflex represents severe intracranial disease. The loss of a gag reflex is especially concerning because it puts the patient at risk for aspiration since the patient cannot normally swallow and protect its airway. Therefore, if a patient has lost its gag reflex, a veterinarian should be contacted immediately, and endotracheal intubation considered. Facial sensation is assessed by touching the medial or lateral canthus of the eye to cause a blink (palpebral reflex), stimulating the nasal mucosa with a pen or hemostats to cause withdrawal of the head, or pinching the skin of the face with a hemostat and observing for a blink or facial twitch on that side [5]. Loss of facial sensation also indicates severe intracranial disease, and if it is a new finding, the veterinarian should be contacted immediately.

While a patient with intracranial disease might seem "stable" and its condition is unlikely to change, patients with intracranial disease can deteriorate rapidly and without warning. Therefore, the importance of frequent and thorough neurologic examinations cannot be stressed enough, with the goal of detecting subtle changes in a patient's neurologic status before they have progressed to a state in which the changes are irreversible.

Nursing Care

Managing hospitalized patients with intracranial disease requires intensive monitoring and special attention to the animal's comfort. A substantial component of nursing care for patients with intracranial disease is avoiding further brain injury as a result of increased ICP. Superior nursing care for these patients relies primarily on the ideal setup of the patient's cage or kennel to reduce the risk of further brain injury. By selecting a cage that is free from unnecessary stimulation but easily visible for constant monitoring, th technician/nurse can quickly recognize any change in a patient's mentation that may be indicative of an increase in ICP. Whether the patient is active or recumbent will also guide the arrangement of the animal's cage or kennel. Appropriate bedding should be selected that provides adequate cushioning, allows the patient to be kept clean, and optimizes the comfort of the patient.

The cage location for a dog or cat with intracranial disease can play a major role in reducing unnecessary stimulation. Environmental stressors can induce an unsafe and prolonged increase in ICP. Ideally, these patients should be placed in a location free from loud noises, such as barking dogs. Avoid selecting a cage in an area with heavy foot traffic, and place signs on or near the cage alerting staff to keep loud noises at a minimum. If auditory stimulation cannot be minimized simply by cage location, cotton can be placed in the patient's ears if tolerated. To avoid misinterpretation of decreased response to auditory stimuli or sending the animal home with cotton in its ears, always put white tape on the patient's head and write "cotton in ears" on the tape (Figure 70.2).



Figure 70.2 A Bulldog is intubated following treatment for cluster seizures and has cotton in his ears to prevent auditory stimulation. An end-tidal CO_2 monitor is attached to the end of the endotracheal tube for continuous capnography.

Visual and tactile stimulation should also be reduced. Select a cage far away from bright lights in a darker corner of the intensive care unit. A cage isolated from others can reduce the number of technicians/nurses, doctors, and other animals passing by that could disturb the patient. Reduce tactile stimulation by clustering treatments together, which will minimize the frequency of entering the cage or kennel throughout the day. For example, treatments such as examination of PLRs, auscultation, and venipuncture can be conducted when the patient is due to be turned.

While reduction of stressors is important in cage selection, it is also important to keep in mind that the patient needs to be monitored closely. Technicians/nurses should be able to identify any change in the animal's level of consciousness, as this is one of the first signs of an increase in ICP. Other parameters such as the patient's respiratory rate and character, blood pressure, heart rate and rhythm, pulse oximetry, end-tidal CO_2 ($ETCO_2$), and body temperature can all be monitored without disturbing the patient and can provide additional indications that a patient's condition is changing. Patients with intracranial disease are also prone to have seizures and therefore must be in a location



Figure 70.3 A Chihuahua with vestibular disease is surrounded by padding and blankets to prevent it from injuring itself when it rolls.

in which they can be readily monitored and accessed quickly if necessary. Additionally, it is important that treatment orders are already available if the patient experiences a seizure, so that the animal can be treated immediately rather than after a veterinarian is called.

The safety and comfort of patients with intracranial disease are controlled by a carefully designed cage or kennel. Depending on the disease affecting the patient, the animal may be active or recumbent. Active or moving patients, such as those with vestibular signs that are circling or rolling, or animals experiencing generalized seizures will benefit from a cage that is heavily padded on all sides. A cradle can also be made using rolled towels or foam padding on either side of smaller dogs and cats, to protect them from injury (Figure 70.3). Keep in mind that a patient surrounded by extra bedding or moving excessively may develop hyperthermia; therefore, more frequent monitoring of body temperature may be warranted in these patients. A patient who continuously circles in the kennel or cage should not have any unnecessary fluid lines or monitoring cords in their path. Coiled intravenous fluid administration sets prevent lines from lying on the cage floor and getting tangled and can be mounted to the ceiling of the cage (Figure 70.4).

Recumbent patients are those who are stuporous or comatose, heavily medicated, sedated for mechanical ventilation, or recovering from surgery. These animals require more padding, such as a thick mattress, to limit pressure on joints and prevent the formation of decubital ulcers. Nonambulatory patients should ideally be kept in sternal recumbency or, if lateral, should be turned frequently (every four to six hours) to prevent atelectasis of the dependent lung [7]. Patients in sternal recumbency should still have their rear limbs or hips rotated.



Figure 70.4 A coiled fluid administration set is used on a cat to prevent tangling of the fluid lines with excessive movement or circling.

Physical rehabilitation is an important part of the patient's recovery and should be started as early as possible. After consultation with a rehabilitation practitioner, the nursing team can pair exercises with regular treatment times to minimize sleep disruption and any handling associated stress. Muscle strength rapidly deteriorates with lack of use. Weight bearing is important for maintaining muscle strength and joint health. With an appropriate exercise plan, the nursing team can minimize muscle atrophy, strengthen postural muscles, reduce the risk of muscle contractures, and provide pain relief for the recumbent patient [8].

Passive range of motion (PROM) exercises and massage of limbs should be performed to reduce edema and stimulate blood flow to the extremities (see Chapter 51 for more information regarding how to perform PROM exercises). Gentle stretching of all joints in limbs can be performed during PROM exercise times and will also be of benefit once the patient is mobile again.

Assisted standing exercises activate unused muscles and allow joints to briefly bear weight. Small patients can be held and helped to stand by supporting under the sternum and the pelvis. Larger patients can be supported by placing



Figure 70.5 A tetraparetic dog benefits from standing exercises on an inflatable therapy peanut. *Source:* Courtesy of Ontario Veterinary College.

an exercise peanut between their legs (Figure 70.5), by using a sling, or by using a hoist. This exercise has the added benefit of allowing the lungs to be in a neutral non-dependent position for brief intervals. Treatment using other modalities such as neuromuscular electrical stimulation can be employed to minimize muscle atrophy in patients that are recumbent for extended periods [9]. Ideally, rehabilitation treatments should be incorporated into the patient's daily care routine at regular intervals.

It is important to keep inactive patients clean and to prevent urine scald by frequently checking for urine and providing bedding that can wick away moisture. Intermittent urinary catheterization or placement of an indwelling catheter may be necessary in patients that are unable to void on their own and when bladder expression is not possible or contraindicated.

Another concern for the recumbent patient with intracranial disease is the positioning of the patient's body. By using slanted grates or boards, or additional towels and bedding, the patient's forelimbs and head should always be elevated at a 30–45 degree angle. This positioning maximizes CSF outflow and cerebral venous drainage, thus alleviating risks of ICP elevation [10]. Additionally, many of the comatose or sedated patients have a loss of protective reflexes, such as a gag reflex. Positioning the patient on an incline can reduce the risk of aspiration if the patient vomits or regurgitates [11]. When placing the patient's cranial end in this elevated position, obstruction of the jugular veins should be prevented. Placing extra blankets or bedding under the patient's forelimbs, neck, and head to angle the cranial portion of the body on an incline can accomplish this most easily (Figure 70.6).

Bedding must be distributed carefully, with even placement under the patient, not just under the neck. Uneven



Figure 70.6 A Maltese Terrier recovers after being hit by a car and sustaining head and cervical trauma. Note that the dog is positioned on a 30–45 degree incline to help prevent elevations in intracranial pressure or aspiration.

placement can position the patient in such a way that jugular blood flow is compromised. Occlusion of the jugular veins for venipuncture, or catheterization of the jugular veins should be avoided. If bandaging of the cervical region is mandatory, bandage placement must be done with care to avoid the possibility of compromised jugular flow.

Patients with intracranial disease frequently require oxygen therapy for treatment of hypoxemia. It is important to be cautious regarding the route of oxygen supplementation (see Chapter 24). Although administration of oxygen via nasal cannulae is relatively easy, it is imperative not to stress the animal or cause it to sneeze during cannula placement as this could lead to elevations in the patient's ICP and subsequent deterioration of the neurologic status. For that reason, veterinarians may elect to provide oxygen supplementation via flow-by, face mask, oxygen cages, or hoods.

Pain assessment is an important aspect of nursing care often carried out by the technician/nurse. Among patients with intracranial disease, those most likely to exhibit pain due to this condition are those with TBI. Unfortunately, assessment for pain in all patients with intracranial disease can be difficult given that patients are often heavily medicated, sedated, or exhibiting inappropriate mentation. This is an important task for the technicians/nurses because pain or anxiety that is untreated can lead to elevations in ICP that can worsen the patient's condition [2]. Therefore, if the patient exhibits any signs associated with pain, a veterinarian should be notified immediately so that appropriate analgesics can be administered. Fentanyl, a short-acting opioid, is typically the drug of choice because it provides immediate effective analgesia and can be titrated to effect [12]. Additionally, because it is short-acting, the infusion can be discontinued if assessment of neurologic function is required. Similarly, its effects can be reversed with naloxone if immediate evaluation of the neurologic status is desired.

Nutrition is an important aspect of nursing care for patients with intracranial disease. Many patients maintain a normal appetite; however, not all can safely be given food and water without supervision. The technician/nurse must use discretion when providing nutrition by mouth and must ensure that patients have an appropriate gag reflex and that their level of consciousness enables them to swallow normally. Patients should be placed in sternal recumbency if they are not able to sit or stand on their own. If there is any uncertainty, a gag reflex should be tested to ensure that the patient is able to swallow. Next, a small amount of water can be offered via bowl or syringe, to ensure that the patient is able to drink normally and does not cough. If the patient can drink water, food can be offered. It is recommended that those patients that are laterally recumbent be maintained elevated on a 30–45-degree incline postprandially to prevent aspiration.

Fluid therapy is also required in patients with intracranial disease to ensure normovolemia and normal blood pressure and to minimize electrolyte abnormalities. Fluids are typically administered intravenously at a maintenance rate unless the patient has abnormal continuing losses such as with a fever. Patients receiving steroids may be polyuric and may have higher fluid requirements. Similarly, patients that have received mannitol or hypertonic saline will also have increased fluid requirements. Fluids should be administered until the patient is able to eat and drink its recommended maintenance requirements. The adequacy of fluid administration can be determined by monitoring daily body weight. Urine output can be measured in patients with indwelling urinary catheters; urine production is expected to be 1–2 ml/kg/hour.

Patient Monitoring

It is extremely important to monitor patients with intracranial disease closely to ensure that blood pressure, oxygenation, ventilation, and temperature are normal (Table 70.1). Because alterations in these findings can contribute to further brain injury, it is important that any abnormalities are identified immediately. Blood pressure should be measured regularly, especially in patients with moderate to severe intracranial disease. Hypotension, as indicated by a systolic blood pressure (SBP) less than 100 mmHg or a MAP less than 80 mmHg, can lead to decreased cerebral perfusion in patients with elevated ICP [12]. The patient's normal response to a decrease in cerebral perfusion is to vasodilate its cerebral vessels, which can lead to an increase

Table 70.1 Desired parameters for monitoring patients with intracranial disease.

Measure	Parameter
Blood pressure	80 mmHg < MAP < 110 mmHg
Pulse oximetry	SpO_2 > 95%
Heart rate	Within normal limits for the species and size of the patient
Respiratory rate	10–20 breaths/minute
Temperature	98.5–100.5°F (37–38°C)
Blood gases	PaO_2 > 80 mmHg, 35 mmHg < $PaCO_2$ < 45 mmHg, 40 mmHg < $PvCO_2$ < 50 mmHg
End-tidal CO_2	$ETCO_2$ 40–50 mmHg
Plasma lactate	< 2.5 mmol/l
Blood glucose	80–120 mg/dl (4.4–6.6 mmol/l)

in ICP. Likewise, elevations in blood pressure such as a MAP greater than 120 mmHg are undesirable because that can lead to reflex cerebral vasoconstriction, thus decreasing cerebral perfusion [13]. If blood pressure measurements are outside the normal range (SBP < 100 or 120 mmHg MAP < 80 mmHg), the technician/nurse should contact a veterinarian immediately.

In patients with severe intracranial disease, a Cushing's reflex can occur and typically is a near-death event. It is characterized by systemic hypertension that occurs as the patient mounts a final effort to maintain cerebral perfusion in the face of an elevated ICP [2]. The patient's heart rate may decrease dramatically in a vagal response to the hypertension. Thus, bradycardia in conjunction with systemic hypertension represents a life-threatening emergency in a patient with intracranial disease, and the veterinarian should be contacted immediately.

Pulse oximetry should also be performed regularly to assess oxygenation in patients with intracranial disease. Hypoxemia, as indicated by oxygen saturation (SpO_2) less than 95% can hasten the progression of brain damage. If a reliable SpO_2 measurement is less than 95%, the technician/nurse should immediately initiate oxygen supplementation and contact a clinician [13]. If the patient is intubated or has a nasal cannula in place, an $ETCO_2$ monitor can be applied to provide a noninvasive method of measuring ventilation. Adapters are available for both endotracheal tubes and nasal oxygen lines.

$ETCO_2$ correlates with partial pressure of carbon dioxide in arterial blood ($PaCO_2$); therefore, an elevation in $ETCO_2$ greater than 50 mmHg is suggestive of hypoventilation. Hypoventilation can have serious consequences in patients with intracranial disease, as it can lead to cerebral vasodilation and elevations in ICP. Therefore, if increased $ETCO_2$ is identified by the technician/nurse, the veterinarian should be contacted as manual or mechanical ventilation may be indicated.

Blood gas monitoring is another method by which the patient's oxygenation and ventilation can be assessed. Monitoring of arterial blood gases is especially helpful in patients with intracranial disease that are stuporous or comatose or have an abnormal respiratory pattern. A PaO_2 less than 80 mmHg indicates hypoxemia, and if this is identified during patient monitoring, oxygen supplementation should be provided immediately as mentioned above. Likewise, an ideal $PaCO_2$ is in the low end of the normal range given that hypercapnea intensifies ischemic damage to the brain. A $PaCO_2$ greater than 50 mmHg indicates hypoventilation and is of concern in a patient with intracranial disease. Partial pressure of carbon dioxide in venous blood ($PvCO_2$) is typically 5 mmHg higher than $PaCO_2$ [14] and can be used as a guideline when arterial samples are not available. Hypercapnea or hypoventilation is an indication that intervention with mechanical ventilation may be required, and the veterinarian must be notified regarding the change in patient condition.

Should an animal need to be intubated for manual or mechanical ventilation, it is imperative that the intubation be efficient and effective to avoid coughing or gagging that could further increase the patient's ICP [3]. Once the patient is intubated and manually ventilated, continuous monitoring of CO_2 via blood gases or $ETCO_2$ is crucial. Overventilation of patients with intracranial disease can be detrimental, as decreases in $PaCO_2$ to less than 25 mmHg will cause cerebral vasoconstriction and reduced CPP [15]. In an emergent situation, hyperventilation can be used to reduce ICP by reducing CBF; however, if the patient is stable and being maintained with manual or mechanical ventilation, a target of 35–45 mmHg for $PaCO_2$ (40–50 mmHg for $PvCO_2$) should be the goal.

It is also important that patients with intracranial disease maintain body temperature within the normal range. Patients with TBI or those undergoing intracranial surgery can have impaired thermoregulation [3]. Ideally, temperature should be monitored continuously in the recumbent patient with the placement of a rectal probe, and appropriate measures to maintain the temperature in the normal range should be employed. An esophageal probe is another option for monitoring the temperature of intubated patients. Hyperthermia is associated with a poor outcome in patients with intracranial disease, as it can increase CBF and lead to elevations in ICP [16]. As such, steps should be taken to externally cool intracranial disease patients with elevated body temperatures. Hypothermia decreases the cerebral metabolic rate and is thought to be neuroprotective; however, controlled hypothermia is not routinely practiced in veterinary medicine and is not recommended at this time.

Because most patients with intracranial disease are not highly mobile, and because their condition can change from moment to moment, continuous electrocardiogram monitoring is beneficial and recommended. This allows for rapid identification of changes in heart rate as well as early detection of arrhythmias such as ventricular premature contractions, ventricular tachycardia, sinus tachycardia, sinus bradycardia, or atrioventricular block; all of which can be seen in patients with intracranial disease.

In addition, patients with intracranial disease should have electrolytes, plasma lactate, and blood glucose monitored regularly for diagnostic, treatment, and prognostic purposes. Electrolyte derangements, especially sodium, are common in patients with intracranial disease that receive medications to reduce ICP. In patients receiving repeat doses of these medications, frequent monitoring of electrolytes is recommended. Hyperlactatemia is often detected in patients with intracranial disease and can be caused by hypoperfusion, hypoxemia, seizures, or tremors [17]. Hyperlactatemia has also been associated with some forms of neoplasia and seems to occur frequently in dogs with brain tumors, the mechanism of which is unknown at this time [18]. Concentrations of plasma and CSF lactate are also higher among dogs with intracranial disease compared with healthy dogs [19].

Blood glucose monitoring is an important aspect of nursing care in intracranial disease patients. Hypoglycemia causes changes in clinical signs including mentation and can lead to seizures. Because seizures can also signify worsening of neurological status, regular monitoring of blood glucose is essential to clarify the cause of alterations in patient status and provide treatment as indicated.

Stress and inflammatory responses are both significant causes of hyperglycemia in patients with TBI. Hyperglycemia post TBI leads to increases in proinflammatory cytokine concentrations, electrolyte abnormalities, edema, ICP and, potentially, brain herniation [20]. Blood glucose monitoring and management strategies are important roles for the veterinary team as hyperglycemia is associated with poor outcome in TBI patients [21]. Studies indicate that higher blood glucose is associated with more severe head trauma and that more profound hyperglycemia is associated with a poorer prognosis in TBI patients [20]. In addition, the association between hyperglycemia and poor outcome is more prominent with persistent hyperglycemia than in patients that exhibit hyperglycemia briefly after injury [20].

Advanced Monitoring Techniques

Electrophysiologic techniques such as electroencephalography (EEG) are available primarily at teaching institutions and specialty hospitals. EEG provides a graphic recording of the electrical activity of the brain and is helpful for determining the presence and type of cerebral disease [22]. The amount of voltage, speed of activity, and presence or absence of "spikes" (very fast activity) indicates inflammatory or degenerative changes and seizure activity [22]. The EEG can change over time with progression or resolution of the disease; therefore, patients are sometimes monitored continuously (Figure 70.7). The EEG varies with the level of consciousness, and drugs such as sedatives, tranquilizers, and anesthetic agents can also alter results [22]. An investigation of wireless video EEG was able to clarify whether unusual behavioral events were seizures in unsedated dogs [23] and may also be considered for hospitalized patients with intracranial disease.

ICP monitoring is used frequently in human hospitals but only rarely in the clinical veterinary setting. Measurement of ICP can be useful in patients in which neurologic signs cannot detect deterioration or improvement in neurologic status due to the loss of other observable neurologic functions or because the patient is sedated or anesthetized. Available methods for invasive ICP monitoring include the use of an intracranial bolt or screw attached to a fluid-filled system with a pressure transducer or a fiberoptic transducer [24]. Complications of invasive ICP monitor placement include focal edema, hemorrhage, parenchymal injury, and infection [24]. Miniaturized silicon ICP transducers inserted in subdural locations are easier to operate, better tolerated, and less prone to adverse effects and have been minimally investigated in healthy dogs [25].

Noninvasive methods for monitoring ICP have been sparsely investigated and include optic nerve sheath diameter (ONSD) measurements, magnetic resonance imaging (MRI), and transcranial Doppler ultrasound. Ultrasonographically measured ONSD is positively



Figure 70.7 A Boxer is continuously monitored with an EEG while undergoing mechanical ventilation.

associated with epidural ICP measurements in healthy dogs and holds promise for patients with intracranial disease [26]. MRI abnormalities [27] and MRI-obtained measurements of ONSD [28] might also provide evidence to support the presence or absence of ICP elevations in dogs with intracranial disease. Alternatively, transcranial Doppler ultrasound is a tool increasingly used to diagnose ICP elevations. In dogs with neurologic signs, transcranial Doppler ultrasound examination of the basilar artery has been performed under anesthesia for MRI and an increased ratio of systolic to diastolic mean velocity associated with MRI findings of suspected intracranial hypertension in dogs with intracranial disease [29].

Summary

- The goal for all patients with intracranial disease is to protect brain function by preserving cerebral perfusion and preventing elevations in ICP.
- Steps must be taken to ensure that temperature, oxygenation, ventilation, and blood pressure are maintained within the normal range and that pain, agitation, and seizure activity are treated expeditiously.
- Patients with intracranial disease can deteriorate rapidly; therefore, continuous monitoring by veterinary technicians/nurses at a 24-hour hospital is recommended.
- Serial neurologic examinations including assessment of the level of consciousness, PLR, pupil size, posture, spinal reflexes, and cranial nerves is recommended.
- Veterinary technicians/nurses must ensure that patients with intracranial disease are housed in an environment that has minimal visual and tactile stimuli but that enables continuous monitoring and easy access if emergency treatment is required.
- Patient comfort is imperative and might necessitate the use of a padded cage and thick bedding to prevent further injury.
- Patients with intracranial disease should be positioned on an incline with minimal pressure on the jugular veins to reduce ICP and prevent aspiration.
- Patients with intracranial disease require intensive care that often includes fluid therapy, nutritional support, oxygen supplementation, physical rehabilitation, and pain management.
- Monitoring of blood gas values, electrolytes, plasma lactate, and blood glucose is important as abnormalities can necessitate treatment and might worsen the prognosis of patients with intracranial disease.
- Advanced monitoring techniques such as EEG, ICP monitoring, advanced imaging, and other newly researched modalities may be considered in certain settings.

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71

Care of the Burned Animal

Steven Epstein

Introduction

Burns in cats and dogs are rare presentations to the emergency room and can caused by chemical, electrical, radiation, or thermal induced injury. Thermally induced burns is the most common type of injury and have been reported in small animals from fires, heating pads, boiling water, stoves, radiators, automobiles, and sun exposure in high environmental temperatures [1, 2]. Burned animals often have both immediate needs (fluid therapy, pain control, and potential treatment of smoke inhalation), as well as long-term needs (metabolic derangements and wound care) that can require prolonged treatment and nursing care resulting in a large financial commitment from owners.

Classification of Burns

Burn wounds can be classified two different ways, and likely the combination of the two will yield the most information about the patient. First, burns can be classified as to the depth of the injury. These systems use either first- through fourth-degree terminology, which is considered outdated, or directly classify the wound depending on depth of the injury. A combination of these classification schemes is summarized in Table 71.1 with examples shown in Figure 71.1.

Classifying burn wound by depth provides the healthcare team and owner with an idea of the length of time it may take the wounds to heal. First-degree wounds tend to heal rapidly (around one week) and do not require daily interventions as they heal by re-epithelization. Second-degree wounds that only involve the superficial part of the dermis tend to heal in one to two weeks, while those that involve the deeper aspects of the dermis need two to four weeks to heal. Scarring after healing should be expected for all wounds that have dermal or deeper involvement. Third- or fourth-degree wounds are wounds that often need surgical interventions and because of this healing times can vary greatly. It is important to note that it may be difficult to classify burn wounds on presentation as the degree of damage may not be readily apparent and it may take up to 72 hours for a burn wound to fully declare the extent of the injury.

The other method of classifying burn wounds involves determining the total body surface area (TBSA) affected. This can be done in conjunction with depth analysis to help determine whether the patient is likely to have systemic effects. Patients with either epidermal or epidermal and superficial dermal wounds are unlikely to have systemic effects, while those with deeper wounds have a higher likelihood of developing systemic metabolic derangements. When calculating TBSA, it is useful to separate out the area that has superficial injury (first-degree), from that which has a deeper component, as it is only the deeper components that are relevant to quoted prognosis and expected complications. Human patients with more than 20% of TBSA burned are considered as severe burn injuries and the American Burn Association recommends that they are managed at a burn center [3]. Although no burn centers exist in veterinary medicine, a referral to a multispecialist 24-hour care facility should be considered if more than 20% of TBSA is seen.

To estimate the TBSA, there are two different methods that can be used, with the simplest being the "rule of nine." For this technique, which is based on an adult human, the body is split into areas of approximately 9%. The head and neck compromise one set of 9%, each thoracic limb is 9%, each pelvic limb is 18%, and the dorsal and ventral trunk

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Table 71.1 Classification of burn wounds by depth.

Degree	Depth	Skin layers involved	Clinical appearance of skin
First	Superficial	Epidermis only	Erythematous and dry
Second	Superficial, partial thickness	Epidermis and superficial dermis	Erythematous and either moist or blisters, hair follicles spared
Second	Superficial, deep thickness	Epidermis and deep dermis	Often blackened or yellowed skin color, hair follicles destroyed
Third	Full thickness	Entire epidermis and dermis	Often brown and leathery or blackened, eschar may be present
Fourth	Full thickness with muscle, tendon or bone involvement	Entire epidermis and dermis	Same as above



Figure 71.1 Images of different wound depths in cats from the recent California wildfires. (a) Superficial or first degree burn over the right eye. (b) Superficial partial thickness or second-degree burn of foot pads. (c) Full thickness or third-degree burns of foot pads. (d) Full thickness with bone involvement, or fourth-degree burns. The black arrows point to the distal aspect of the third phalanges of that digit.

each make up 18%. This sums to 99% with 1% left for genitalia and perineum. To use this technique, the healthcare team would assess what fraction of each area is estimated to be burned, then they are all summed up to achieve an estimated TBSA. This technique is easy to use, although it is recognized that body shape, condition score and experience of user can affect results in humans [4] and this technique has not been validated in cats or dogs.

An alternative method has been developed is using a resuscitation burn card. This is achieved by cutting a piece of plastic or other device to the size of a standard credit card, as the area of this card is 45 cm^2. On one side of the card, a weight to body surface area conversion chart is printed. This resuscitation burn card is then placed over the various parts of the body that are burned to get an estimated number of cards that would be needed to

completely cover the burn area. Alternatively, a standard-sized plastic credit card can be used (8.5 × 5.3 cm). The patient is then weighed, and a body surface area calculated in square meters (m^2) or read from the burn card. The TBSA is then calculated by the formula (Eq. 71.1):

$$TBSA\ burned\ (\%) = \frac{number\ of\ cards\ to\ cover\ burn\ area \times 0.45}{calculated\ body\ surface\ area\ (m^2)} \quad (71.1)$$

This method overcomes the limitations of the "rule of nine" and can be applied to either cats or dog of any body condition score.

Medical Considerations

In treating a burned cat or dog, there are both immediate (need for fluid resuscitation, evaluation for smoke inhalation, pain management) and later-stage (ocular and cardiac health, nutritional assessment) considerations for the clinician when a severe burn injury has occurred.

Following a burn of over 20% TBSA, there are multiple metabolic derangements that can occur, with hemodynamic effects being one of the most urgent to address. With this degree of epidermal/dermal injury, it is not surprising that the body may lose large quantities of protein-rich fluid, as well as having a severe inflammatory insult resulting in intravascular volume depletion. This can lead to hypovolemic shock at presentation or can occur anytime during the first 72 hours following the burn injury. Initial treatment of shock should be treated with standard doses of isotonic crystalloids or synthetic colloids. If the patient does not present with hypovolemic shock, and the burn injury has just occurred, it is recommended to give 2–4 ml/kg per percentage of TBSA affected of an isotonic crystalloid in the first 24 hours to combat the development of shock due to future fluid losses. Commonly, half of the calculated amount is given in the first six to eight hours, "front-loading the fluid plan." For the following 24–48 hours when fluid exudate may be large, the patient should be reassessed frequently to determine its fluid need. Serum albumin concentrations may be tracked and if hypoalbuminemia develops with concurrent interstitial edema of non-burned areas, synthetic colloids should be considered.

Patients with severe burn injuries may present with mild to extreme pain. When burn injuries reach the deep dermis or are third-degree burns, the tissue is often no longer painful as nerve endings are also damaged. However, the depth of the burn should not preclude appropriate analgesia while the clinician is completing their evaluation of the patient. Typically, full mu opioid agonist medications, such as hydromorphone, morphine or methadone, are used in the initial period. After a full evaluation of the patient and a longer period for pain assessment, the decision for what level of analgesia is needed can be made. In severe cases, constant rate infusions (CRIs) of full mu opioid agonists such as fentanyl, with or without adjunctive CRIs of ketamine or dexmedetomidine, may be needed to control pain. (see Chapters 47 and 48). Alternatively, if patients are not acting as if they are in pain, a partial mu opioid agonist such as buprenorphine can be considered in the initial period.

Patients who have been burned in a house or wildfire may also experience complications of smoke inhalation. Smoke inhalation injury can cause airway obstruction from the nasal cavity to the small airways, develop pneumonitis from particulate matter inhalation or pneumonia, or present with carbon monoxide or cyanide toxicity depending on the material that burned. Airway obstruction in the upper airways can occur from direct thermal injury or the particulate matter inciting an inflammatory reaction and usually occurs in the first 24 hours. Tracheal and small-airway obstruction can result from thermal injury or (trachea) or from pseudomembranous casts that migrate into the lower airways with necrosis of the airways being a possible sequela [5].

Carbon monoxide toxicity is one of the major sources of early morbidity and mortality in fire victims [6]. It is produced when combustion of carbon containing material occurs. Significant quantities of carbon monoxide can be inhaled in short periods and as the affinity for hemoglobin is 200–250-fold greater than oxygen, tissue hypoxia can rapidly result from a left-shifted oxyhemoglobin saturation curve. Carbon monoxide can also directly depress central nervous system function and indirectly injures the brain by increasing nitric oxide production, facilitating white blood cell migration into brain with resulting leukoencephalopathy among multiple other poorly characterized mechanisms.

For most veterinarians, diagnosing carbon monoxide toxicity is difficult. A tentative diagnosis is based on compatible history with a decreased mentation plus or minus hyperlactatemia. Standard pulse oximeters are not able to detect carbon monoxide in blood; however, specialized pulse co-oximeters can accurately detect carboxyhemoglobin [7]. If arterial blood gases are measured, the resulting partial pressure of oxygen in arterial blood is typically normal unless concurrent pulmonary dysfunction has already resulted. The gold standard would be direct measurement of carboxyhemoglobin levels in blood via co-oximetry, which is rarely an option for veterinarians. Because of this difficulty, any animal who has been in a house or wildfire that presents acutely after the fire should be given oxygen supplementation. The rationale for this is because the half-life of carboxyhemoglobin is 250 minutes for the victim breathing room air. This is reduced to

40–60 minutes with inhalation of 100% oxygen. In one study of dogs in a kennel fire that received oxygen therapy (via nasal canula or oxygen cage), those that had oxygen administration had a significantly reduced carboxyhemoglobin compared with those that did not [8]. Although the higher the fraction of inspired oxygen achieved, the more quickly the carboxyhemoglobin leaves the blood, the clinician must weigh the risks of anesthesia and intubation for 100% oxygen against the benefits of that step. If the patient initially recovers, delayed neurologic effects may occur days to week later from the injury that may or may not be reversible with time [9, 10].

Once the patient has been stabilized for the above medical considerations, the focus can shift to wound care and later stage medical issues. Patients that have burn wounds to the face or have sustained injuries in a fire should have careful evaluation of their eyes including evaluation of eyelid closure and application of fluorescein stain to look for corneal ulceration at the time of admission. It has been documented that humans with facial burns have abnormal tear film predisposing to future ulcer formation [11], and anecdotally at the UC Davis William R. Pritchard Veterinary Medical Teaching Hospital, a large proportion of the cats treated in various California wildfires had a presumed qualitative deficiency in their tear film in addition to corneal ulcer formation. Because of this concern for a qualitative tear film deficiency in patients with facial burns, the clinician should consider treating all animals sustaining significant burn injuries from fires (house or wildfire) with a topical ocular lubricant that contains hyaluronic acid. The mucinomimetic properties of hyaluronic acid have been shown to improve tear film quality and promote corneal health, two properties that can help prevent corneal ulcer formation. If there are burn wounds to the face of the patient, special attention should be paid to determining whether the patient has lagophthalmos, which will predispose them to ulcer formation (Figure 71.2). If present, an ointment-based tear film replacement should be used, either instead of or together with a lubricant that contains hyaluronic acid, given the beneficial increased corneal contact time with ointment use. The owners should be advised that when a burn-related eyelid wound heals, eyelid contracture is possible, which may lead to the need for future surgical intervention if lagophthalmos subsequently worsens.

Another organ system that can develop dysfunction if the burn injuries are due to a fire is the cardiovascular system. A patient may develop hypovolemia from fluid transudation from wounds, but cardiac changes and thrombi formation may occur as well. Significant cardiovascular effects secondary to thermal burn injury and accompanying smoke inhalation have been demonstrated



Figure 71.2 A cat with burn wounds over the left eye that led to lagophthalmos and subsequent corneal ulceration.

in human populations and experimental animal models [12, 13]. A 2020 clinical report of 51 cats with burns from a wildfire showed that 18 had evidence of myocardial thickening on echocardiogram and 16 had spontaneous echocardiographic contrast and thrombi formation [14]. Careful auscultation for murmurs, gallops, or arrhythmias should occur daily, and if they develop, judicious use of fluid therapy is indicated to avoid a fluid overload state, as these animals may be predisposed to developing heart failure. If any cardiac abnormalities are auscultated, performing an echocardiogram is warranted to look for myocardial dysfunction and spontaneous contrast, a predisposing factor for blood clot formation. Based on these results, antithrombotic use may be warranted to prevent cardiac or arterial thrombi; however, this needs to be balanced against the need for surgical intervention from the patient's wounds.

As burn wounds are often associated with a hypermetabolic state and protein loss through exudation, careful daily assessment of nutritional intake is warranted. As this hypermetabolic state is associated with muscle catabolism and loss of muscle mass, adequate intake of protein is key for patient health. If the nasal cavity has been injured from thermal injury of inhaled gas, this is likely to reduce the patients sense of smell and interest in food and a feeding tube is more likely to be needed in these patients. If the animal is not eating their metabolic energy requirements for two consecutive days, placement of a feeding tube should be considered. Either nasogastric or esophageal can be placed depending on the health of the nasal cavity, if burn wounds are present over the neck, and the risk of anesthesia in deciding which location of feeding tube to place.

Principles of Burn Wound Management

General Recommendations

As the skin represents a barrier to infection, when it is burned this normal barrier function no longer exists. Special care to prevent a nosocomial infection is therefore indicated and good hand hygiene is critical. Hands should be washed before handling any burn patient and clean examination gloves should be worn. If a full physical examination is being performed, gloves should be changed between handling the wounds and the rest of the animal to prevent bacteria that flourish in areas like the armpit and groin to be transmitted to the wounds. Stethoscopes should be cleaned with alcohol prior to use to prevent them acting as a fomite. If there are large surface-area burns that are deep partial or full thickness, a barrier gown should be considered when handling the wounds.

Strict hygiene requirements should also occur when handling any invasive device (intravenous catheters, feeding tubes, etc.) to help prevent nosocomial infections. These consist of all ports being wiped with alcohol prior to injections, fresh examination gloves before handling devices, and daily inspection of the site for signs of inflammation with removal if present.

If the burn wounds are on the limbs, mobility is likely to be decreased in these animals. Careful notation of body position every four hours is indicated with patients who are not moving being turned at that time frame. Checking for eliminations every four hours and making sure the animal is not soiled can help to prevent infections and fecal or urine scald developing due to decreased mobility. Passive range of motion exercises can be considered depending on where the burn wounds are located.

Wound Management

On initial evaluation of wounds, the patient should be heavily sedated or anesthetized to allow for a thorough evaluation. If the patient is presented acutely after the burn, the skin should have cool water (45–65°F; 7–18°C) applied for 15–30 minutes to dissipate any residual heat and prevent further injury. Ice should not be applied as this can cause excessive vasoconstriction and worsen the extent of tissue damage. After the wounds have been cooled, or if this step was not necessary, the initial evaluation should include a thorough clipping of hair around the area to fully evaluate the extent of the wound.

Specific recommendations for wound management will depend on the depth of the wound, but the general principles involve initial daily evaluation, debridement of necrotic tissue, and bandaging of the wound in between evaluations. Superficial burns often only need initial clipping of the hair and covering of the wound to keep it moist until healed. Superficial partial thickness burns should be cleaned with a chlorhexidine solution and then bandaged. There is no consensus on the optimal concentration of chlorhexidine to use, with the range of 0.05–4% being used in human medicine [15]. The author recommends a dilute solution of no more than 0.1% chlorhexidine. Following cleaning and debridement, silver sulfadiazine ointment can be applied with a bandage covering it until healed.

Both deep partial and full thickness wounds should be surgically debrided of dead tissue and debris at initial evaluation in addition to topical cleaning of the wound. If there doubt about the viability of the tissue, it should be left until the next day's evaluation, as it may take two or three days for the tissue to declare its viability. In human medicine, eschars are recommended to be removed as they may be covering evidence of infection and delay wound healing. However, if there is no evidence of surrounding tissue inflammation or seepage from the edges of the area, the author has successfully managed patients with large eschars intact until the wound contracted and reepithelized under the eschar when more aggressive surgical management was not an option. This prevented the need for daily sedation and bandage changes and the eschar acted as a biologic bandage. It should be acknowledged, however, that delayed primary closure after eschar removal produces better functional and cosmetic results than those achieved if the escharotomies are allowed to close by secondary intention in humans [16].

Surgical excision or grafting are recommended for burn wounds that are estimated to take longer than two to three weeks to heal with conservative management in human medicine. This is because if the wounds are left to heal on their own with conservative management, patients experience longer hospital stays, increased pain, and decreased function of the area. In veterinary medicine, skin grafting is not always an option. Even though some deep partial- and full-thickness wounds are large and not expected to heal in that time frame, conservative management with bandage changes may be the best option. Conservative management involves daily sedation and evaluation initially and the application of topical antimicrobial agents until the wound closes by second intention or graft application is later possible.

Which topical antimicrobial agent to use has been the subject of much research. A 2017 Cochrane review came to the conclusion that it was often uncertain whether topical antimicrobial agents were associated with any difference in healing, infections, or other outcomes when compared with each other [17]. Moderate to high certainty findings from this review include:

1) Moderate certainty that burns treated with honey are probably more likely to heal over time compared with topical antimicrobials.

2) High certainty evidence that treating burns with honey reduced mean times to healing in comparison with non-antibacterial and non-conventional treatments.
3) Moderate certainty evidence that burns treated with nanocrystalline silver dressings probably have a slightly shorter mean time to healing than those treated with petroleum jelly gauze.

As there is still a large degree of variability in results, veterinarians can use silver sulfadiazine as the topical antimicrobial agent of choice or consider the use of medical grade honey products.

Systemic antimicrobial therapy is not recommended for management of routine burn wounds but should be reserved for where there is evidence of infection present at the wound. Signs of infection include change in wound color, increased exudate, increased pain, increased wound depth, or early separation of eschars. If a wound infection is suspected to be present, the tissue should be cultured, and a susceptibility performed to enable the appropriate antimicrobial choice. Gram-positive infections are more likely to colonize the wound in the first five days, with Gram-negative pathogens two to four days later. There is a multitude of organisms that may be the cause of the infected wound with *Staphylococcus* spp., *Streptococcus* spp., *Enterococcus* spp. being the most common Gram-positive pathogens, and *Pseudomonas aeruginosa* and *Acinetobacter baumannii* being the top two Gram-negative pathogens, followed by the Enterobacterales family [18]. Because of this wide range of pathogens, broad-spectrum intravenous antimicrobial therapy should be considered pending culture results when a wound infection is thought to have occurred.

With time, most burn wounds that do not become infected will heal well with appropriate therapy. For poorly healing or large burn wounds, referral to a board-certified surgeon for optimal care should be considered.

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72

Care of the Environmentally Injured Animal

Michael S. Lagutchik, Rufus W. Frederick, and James O. Barclay

This chapter addresses emergent management of dogs and cats with environmental injuries, including cold-induced injury (frostbite and hypothermia), heat-induced injury, nonsurgical management of open wounds and necrotic tissues, and open fractures. The chapter emphasizes specialized nursing guidelines critical to improve likelihood of successful outcome.

Cold-Induced Injury

Cold-induced injuries include local or regional injuries (i.e. nonfreezing and freezing injuries of extremities) and generalized cold injury (i.e. systemic hypothermia). Hypothermia is very common in veterinary practice, although the true incidence is unknown [1]. Frostbite is uncommon in dogs and cats and tends to be related to geographic location (i.e. freezing climates), use of the animal (e.g. as sled dogs), or in neglect cases.

Nonfreezing Injuries

Nonfreezing injuries typically involve the extremities; they occur despite the tissue not actually freezing and are commonly caused by prolonged exposure to cold. With nonfreezing injuries, extremities (ear pinnae, paws, tail tip, scrotum) are exposed to cold temperatures above freezing for prolonged periods (>12 hours), causing intense erythema of the skin, pain, and pruritus. If skin is exposed to damp conditions or submerged and exposed to cold, tissue edema and maceration may also develop.

Treatment of nonfreezing cold injuries involves removing the animal from the cold environment and passively warming the affected tissues slowly. Passive warming of nonfreezing injuries can be accomplished by moving the patient to a warm room and gently wrapping the patient or affected body part in warm blankets or towels.

Freezing Injury

Freezing injury or "frostbite" is the development of cold injury in which tissues actually become frozen, with crystallization (ice formation) of tissue and cell water [2]. Frostbite develops at environmental temperatures below 32°F (0°C) and primarily affects the distal extremities, ears, nose, scrotum, and tail. Frostbite varies in severity from superficial (first-degree frostbite) to deep injury (fourth-degree frostbite) [2].

Clinical signs of superficial frostbite include a gray to white, waxy appearance of affected skin (first degree); blistering of affected skin may also be present (second degree). Clinical signs of deep frostbite include involvement of the entire epidermis, either without subcutaneous tissue involvement (third degree) or with subcutaneous tissue involvement, to possibly include muscle and bone (fourth degree). Tissues affected with deep frostbite may be black and friable. In all cases of frostbite, pain may be intense, especially during rewarming of tissues.

Management of patients with freezing injury is detailed in Protocol 72.1. Treatment of frostbite involves rapid warming of affected tissues, comprehensive management of concurrent problems, analgesia, and protection of affected tissues [3]. Careful warming of frozen tissues is critical to avoid further trauma and pain. Provide systemic analgesia, as frostbite is extremely painful. Antibiotic use is not generally recommended [3]. Aseptically aspirate large blisters that develop [3]. In some cases, open wounds, infected wounds, or necrotic wounds may develop in frostbitten tissue.

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Protocol 72.1 Management of Freezing Injury (Frostbite)

1) Treat whole-body hypothermia (Protocol 72.2), trauma, or shock.
2) Provide systemic analgesia.
3) Warm frozen tissues gently and slowly, using one of two methods:
   a) Immerse in a water bath that is 104–108°F (40.3–42.6°C) for at least 20 minutes or until thawing has occurred.
   b) Wrap with warm, wet towels for 15–20 minutes, changing the towels every 5 minutes.
   c) Do not use dry heat or rub or massage tissues to warm tissues.
4) Protect affected tissues
   a) Apply loose protective bandages.
   b) Minimize movement (confine to a cage).
   c) Apply an Elizabethan collar to prevent self-trauma.
5) Aseptically aspirate large blisters that develop.
6) Manage open, infected, or necrotic wounds (Protocol 72.4).

Hypothermia

Hypothermia is defined as a core body temperature that is subnormal and is subdivided into primary and secondary hypothermia [1, 3–6]. Core temperature refers to the temperature of the blood in the pulmonary artery. In veterinary patients, rectal or esophageal temperature measurements are more practical, even though they may be lower than core temperature in many instances. Primary hypothermia is caused by exposure to low environmental temperatures; secondary hypothermia has multiple causes, to include secondary to trauma, toxicity, underlying illness, anesthesia, surgery, and other factors. This differentiation is important, as patients with primary hypothermia can apparently tolerate much more severe hypothermia than patients with secondary hypothermia [4] and complications are more commonly reported in patients with secondary hypothermia at significantly closer-to-normal temperatures than patients with primary hypothermia [4]. Regardless of the type of hypothermia, the mechanisms that lead to hypothermia are excessive heat loss, decreased heat production, or both. Primary hypothermia is classified as mild (90–99°F; 32.5–37.5°C), moderate (82–90°F; 28.0–32.5°C), severe (68–82°F; 20.2–28.0°C), or profound <68°F; (<20.2°C) [1, 4, 5]. Secondary hypothermia is classified as mild (98–99.9°F; 37.0–37.5°C), moderate (96–98°F; 35.8–37.0°C), severe (92–96°F; 33.6–35.8°C), or profound <92°F; (<33.6°C) [1, 4, 5].

Complications Related to Hypothermia

Hypothermia can cause many complications [1, 3–6]. The duration and severity of hypothermia, type of hypothermia (primary or secondary), and the patient's ability to adapt directly affect which complications develop and how severe those complications become. The clinician must recognize that potential problems are common and anticipate them, regardless of the patient's type or severity of hypothermia.

Hyperglycemia is common in mild and moderate hypothermia; specific measures to reduce blood sugar are seldom necessary. Hypoglycemia can develop in severely hypothermic patients, and dextrose supplementation may be necessary. Hypokalemia is common in mild to moderate hypothermia, and supplementation may be necessary. Hyperkalemia is reported in severe hypothermia; specific measures may be necessary to reduce the potassium concentration if potassium is greater than 7–8 mmol/l. Metabolic and respiratory acidosis are reported in most types and degrees of hypothermia; these typically correct with fluid therapy and patient warming. Glucose, electrolyte, and venous blood gas measurements should be performed at least every 6–12 hours initially.

Hemostatic defects are common. Patients are commonly in a hypocoagulable state with prolonged clotting times. Platelet abnormalities are also noted. Patients with mild hypothermia typically have increased platelet aggregation, whereas patients with severe hypothermia have decreased platelet aggregation. Serial platelet counts and coagulation testing to screen for abnormalities should be performed upon admission and every 6–12 hours afterward until patients are stable.

Tachycardia and hypertension are common in mild to moderate hypothermia. As hypothermia worsens, hypotension and bradycardia develop, and other cardiac arrhythmias may occur. For these reasons, continuous electrocardiogram (ECG) and blood pressure monitoring is recommended until the patient stabilizes. It is recommended [3] to avoid giving drugs, to include antiarrhythmic agents, until the body temperature is above 86–90.3°F (30–32°C), as drugs are believed ineffective at temperatures below this.

Measures to correct hypothermia can actually cause complications to develop, such as afterdrop and rewarming shock [4, 5]. Careful warming and close monitoring are essential. Afterdrop is the continued decrease in the patient's core temperature as warming is provided and is caused by the return of cold peripheral blood to the central circulation. To prevent afterdrop, it is important to warm the patient's chest and abdomen, not the extremities. Rewarming shock develops with excessively rapid warming and is due to the sudden development of systemic vasodilatation. This vasodilatation causes hypotension at a

Protocol 72.2 Management of Hypothermia

1) Warm rapidly but carefully.
   a) Increase the body temperature by 2–4°F (0.06–0.13°C)/hour.
   b) Warm to a temperature of 98.5°F (37.2°C) and then cease use of all warming methods except passive warming.

Mild Hypothermia

Adequate blood volume:

1) Warm using passive surface warming (wrap patient in blankets or towels; hospitalize in warm environment).

Moderate to Severe Hypothermia

Mild hypothermia with inadequate blood volume:

1) Warm using active surface warming (use of externally applied heat sources such as forced-air devices, warm water bottles, heating pads or lamps, or dryers).
2) Apply heat to the thorax and abdomen, and not the extremities.
3) Continue passive warming as above.

Severe to Profound Hypothermia

1) Warm using active core warming (warm inhaled air provided by endotracheal tube, warm peritoneal or pleural lavage, warm IV fluids, and urinary bladder and rectal lavage with warm fluids).
2) Perform active and passive warming as above.
3) Provide cardiovascular support:
   a) Provide IV fluids at relatively moderate rates (2–3 times maintenance rates, or 2–10 ml/kg/hour) until normothermic.
   b) Once resuscitated and stabilized, provide maintenance IV fluids.
   c) Provide oxygen supplementation for severe to profound hypothermia to reduce risk of cardiac arrhythmias.
4) Anticipate and manage complications:
   a) Monitor ECG continuously, and treat arrhythmias as directed, but do not treat until body temperature is 86–90.3°F (30–32°C).
   b) Monitor for glucose, electrolyte, and acid–base abnormalities every 6–12 hours.
   c) Monitor platelet count and coagulation parameters every 6–12 hours.
   d) Provide and reassess analgesia.
   e) Perform continuous or intermittent blood pressure monitoring, lactate clearance, serial body weight, changes in mentation, and urine output to monitor for rewarming shock.
   f) Perform continuous temperature measurement, to monitor for correction of hypothermia and afterdrop.

time when the circulatory system may not be able to react. The systemic hypotension is aggravated by the increased metabolic demand that develops as hypothermic patients are rewarmed, which increases the demand for perfusion. To prevent or reduce rewarming shock, intravenous (IV) fluid therapy must be provided and volume status, systemic blood pressure, and tissue perfusion must be monitored carefully. Other possible problems are due to changes in regional blood flow (e.g. development of cerebral edema, cerebral ischemia), reperfusion of tissue beds (e.g. development of systemic reperfusion injury, pancreatitis), and other factors (e.g. pneumonia, pulmonary edema, acute respiratory distress syndrome) [4].

Management of patients with hypothermia is detailed in Protocol 72.2. Hypothermic patients must be warmed rapidly but carefully, and with anticipation for possible complications. Cardiovascular support (principally IV fluid therapy), management of coexisting problems, and prevention of rewarming complications are necessary [5]. The recommended rate of rewarming is to increase the core body temperature by 2–4°F (0.06–0.13°C)/hour [4–6]. Treatment recommendations are provided in the protocol.

In summary, patients with cold-induced injuries require intensive management. Rapid but careful rewarming of the patient is critical. Minimizing complications and providing supportive care are essential.

Heat-Induced Injury

Heat-induced injury, primarily seen in dogs, develops when a patient accumulates a severe heat load that cannot be dissipated rapidly enough to prevent injury, exceeding the capability of the patient to compensate [7–15]. There are three types of heat-induced injury in veterinary patients based on the severity of the resulting injury: mild (heat stress), moderate (heat exhaustion), or severe (heat stroke) [7–9].

Classification of Heat-Induced Injuries

Heat stress is characterized by development of excessive thirst, discomfort associated with physical activity, and sodium and chloride abnormalities, but with controlled

panting (i.e. the patient can control or reduce panting when exposed to a noxious inhalant such as alcohol) [7, 14, 15]. Heat exhaustion is present when the signs of heat stress are present, as well as weakness, anxiety, and uncontrolled panting (i.e. the patient cannot reduce panting when exposed to a noxious inhalant) [7]. Heat stroke is present when signs of heat exhaustion are present, with any degree of central nervous system (CNS) abnormality [7]. The most common CNS abnormalities include changes in mentation and level of consciousness (e.g. obtunded, stupor, coma), seizures, abnormal pupil size, cortical blindness, head tremors, and ataxia [8]. Heat stroke is a life-threatening condition. It often leads to widespread multiple organ injury with risk of progression to multiple organ dysfunction syndrome. No specific body temperature defines heat stroke in veterinary patients; however, temperatures as low as 105.8°F (41.3°C) have been associated with pathology [16]. Studies have established that heat directly induces tissue injury and that the severity of tissue injury and cell death is a function of the degree and duration of hyperthermia [17]. Retrospective veterinary studies report multiple serious complications and high fatality rates in heat stroke patients despite proper treatment [18, 19].

Causes of Heat Injury

Heat injury is a rise in core body temperature due to physical activity or endogenous sources (exertional heat stroke), or exposure to increased ambient temperature or exogenous sources (classical or environmental heat stroke) [7–9, 11, 17]. In veterinary patients, the cause of heat stroke is often a combination of exertional and environmental factors, to include high ambient temperatures, confinement in unventilated or poorly ventilated areas, and high ambient humidity. A retrospective study [18] conducted at a veterinary teaching hospital found large-breed dogs and brachycephalic dog breeds were more likely than smaller breeds to present for heat stroke. Additionally, studies [18, 20] show the mean environmental temperature on the days when animals presented with heat stroke was significantly increased when compared with average temperatures.

Initial Management Considerations

Mentation in heat injured patients can range from alert and responsive to stuporous or comatose. Patients presenting in stupor or coma are in imminent danger of death. Some patients with heat stroke present actively seizuring[8]. They may be hypothermic, hyperthermic, or normothermic on presentation, based on cooling measures initiated by the owner or handler and length of time since onset of heat stroke.

A common cause and frequent finding in patients with heat injury is partial or intermittent airway obstruction. The obstruction can be due to anatomical conditions or foreign objects. Conditions such as elongated soft palate, stenotic nares, everted laryngeal saccules, narrow trachea, or laryngeal paralysis are predisposing factors to heat injury [10]. Although pulmonary artery temperature measurement is the gold standard for evaluating core body temperature, rectal temperature compares well and can be performed with little risk or expense. Note that rectal temperature may lag behind core body temperature by up to 15 minutes [14, 15, 21]. In an abnormal state of hyperthermia, the initial physiological response is to move blood to surface vessels to maximize conductive cooling. This phase will generally include renal and splanchnic vasoconstriction, peripheral vasodilatation, and an increased cardiac output [11]. Over time, if the body temperature remains high, splanchnic and renal vasoconstriction will eventually fail, creating conditions favorable for venous pooling and hypovolemia or distributive shock [10].

Emergency management of hyperthermic patients is shown in Protocol 72.3. Emergency interventions must be performed immediately to optimize outcome [7, 9, 10, 13, 16, 19, 20, 22, 23]. If the patient is intubated, the airway must be protected during the cooling phase to prevent aspiration of running water. Supplemental oxygen therapy is necessary; the method of oxygen delivery used should not interfere with cooling efforts during initial care. Avoid oxygen cages and oxygen masks that can create increased humidity and prevent maximal heat dissipation.

Patients presenting with heat injury with a rectal temperature above 105°F (40.9°C) require emergency cooling measures. Numerous cooling methods are described; no one technique has been shown to be superior or ideal [16]. Use a combination of cooling methods for optimal care. The rate of cooling should be as rapid as possible until the rectal temperature is 105°F (40.9°C) [7]. The most practical, most expedient, and most rapid method to reduce body temperature is to soak the patient to the skin with room-temperature water [8]. The patient can be placed under running water in a well-drained tub or run, submerged partially in a tub of water, or sprayed with a hose. The key is to soak the entire patient as rapidly as possible, and to soak through the hair coat to soak the skin thoroughly.

Intravenous fluid therapy is essential, unless there are specific contraindications, using room-temperature fluids [8]. Adequate circulating blood and plasma volume are required to maximize heat dissipation by conduction [17]. Additionally, IV administration of room-temperature fluids reduces core body temperature.

Use additional cooling methods such as directing fans on the patient to increase surface cooling, remembering that it is essential that the skin be thoroughly wet for fans to be

Protocol 72.3 Management of Heat-Induced Injury

1) Triage the patient as for other types of injury or illness: carefully assess mentation, airway and breathing, circulation, and body temperature.
   a) Establish and protect the airway if apneic.
   b) Provide supplemental oxygen therapy.
   c) Establish at least one IV catheter and resuscitate for shock and persistent hypotension.
2) Emergently cool the patient if rectal temperature is >105°F (>40.9°C), cooling as rapidly as possible until the body temperature is 105°F (40.9°C).
   a) Soak the patient to the skin with copious amounts of room-temperature water.
   b) Administer room-temperature IV fluids at rates necessary to combat shock or persistent hypotension.
   c) Direct fans on the patient to facilitate surface cooling.
   d) Reduce the room temperature, if possible.
   e) Avoid cold IV fluids, iced water baths, or surface cooling with ice water or ice packs.
3) Reduce the rate of cooling once the patient's body temperature is <105°F (<40.9°C) to avoid rebound hypothermia.
   a) Cease cooling with water and dry the hair/skin.
   b) Remove fans.
   c) Return room temperature to normal.
4) Provide supportive warming once the patient's temperature has been reduced to 103°F (39.8°C).
   a) All active cooling efforts must cease.
   b) Continue temperature monitoring.
   c) Actively warm the patient to prevent rebound hypothermia if temperature is at or below 100°F (38°C).
5) Monitor for and treat concurrent problems.
   a) Monitor blood pressures, lactate clearance, urine output, mentation, and other measures of perfusion to check for shock hypotension. Continue IV fluid therapy as directed by the attending veterinarian.
   b) Monitor blood glucose and venous blood gas analyses every 6–12 hours. Maintain normoglycemia with supplemental dextrose.
   c) Monitor arterial blood gas analysis or pulse oximetry and capnography to assess oxygenation and ventilation. IV fluids should be supplemented with dextrose as needed to maintain normoglycemia.
   d) Monitor for occult or active bleeding and petechiae and ecchymoses; perform coagulation tests every 6–12 hours and serial complete blood counts every 12–24 hours to screen for thrombocytopenia and coagulopathies.
   e) Monitor the ECG continuously to detect cardiac arrhythmias, especially ventricular arrhythmias. Provide specific anti-arrhythmic therapy as directed for hemodynamically unstable patients.
   f) Monitor for vomiting and diarrhea, provide excellent nursing care to maintain patient hygiene, provide gastrointestinal protectants as directed, and provide enteral or parenteral nutrition as directed.
   g) Monitor urine output hourly and assess creatinine every 12–24 hours. Place and maintain urethral catheters as directed.
   h) Monitor respiratory rate hourly, perform thoracic auscultation at least every 4 hours, and perform thoracic radiographs if pulmonary edema or other abnormalities are suspected. Perform arterial blood gas analysis or pulse oximetry and capnography every 4–6 hours initially, then as needed, to assess oxygenation and ventilation. Consider mechanical ventilation if the patient cannot oxygenate or ventilate adequately.
   i) Monitor mentation and level of consciousness, vision, gait, and postural responses, and monitor for seizures.

effective. Manage patients in an air-conditioned environment. Cooling of carotid arteries (e.g. by ice packs placed over the neck) induces vasodilatation, which may increase cerebral perfusion, based on studies in people [24]. Future veterinary studies may reveal positive therapeutic modalities that include advanced brain hypothermia.

Although debatable and poorly researched, avoid cold IV fluids, iced water baths, and surface cooling with iced water or ice packs because these cause peripheral vasoconstriction with sustained increase in core temperature; cause shivering, generating more internal heat; and promote capillary sludging, which contributes to coagulopathy [8, 11, 17, 21, 24, 25]. Placing isopropyl alcohol on the footpads is commonly done, but is generally ineffective because the paw pads have such a small surface area. Wetting a large percentage of the pet's surface area to the skin with alcohol is required for adequate cooling, but risks combustion, and should not be performed [8].

Once the patient's body temperature is lower than 105°F (40.9°C), reduce the rate of cooling to avoid rebound hypothermia. Discontinue ancillary cooling measures and dry the patient's skin. Supportive warming is necessary once the patient's temperature has been reduced to 103°F (39.8°C) or below. At this point, cease all cooling efforts, monitor temperature frequently, and be prepared to actively warm the patient to prevent rebound hypothermia [10].

Although warming a patient with a temperature of 103°F (39.8°C) may seem counterintuitive, anticipate a period of rebound hypothermia. Additionally, the delay between rectal temperature and true core temperature probably means that the true core temperature may be lower [21].

Monitor for and Treat Concurrent Problems

Patients with heat injury often present in shock and may develop sustained hypotension. IV fluid therapy should be continued not only to cool the patient but also to maintain adequate tissue perfusion. In some cases, fresh frozen plasma or albumin transfusions may be indicated to treat concurrent problems such as disseminated intravascular coagulation (DIC) or hypoalbuminemia. Continuous or intermittent blood pressure measurement, lactate clearance, clinical assessment of perfusion, and assessment of volume status should be monitored over time.

Glucose, acid–base, and electrolyte abnormalities are common due to shock and reduced tissue perfusion. Blood glucose measurement and venous blood gas analyses, to include measurement of blood lactate concentration, are necessary, generally at intervals of every 6–12 hours, depending on the severity of the derangements. If concurrent pulmonary abnormalities are present, arterial blood gas analysis (or surrogates such as pulse oximetry and capnography) may be necessary to optimally assess oxygenation and ventilation. IV fluids should be supplemented with dextrose as needed to maintain normoglycemia.

Patients with heat injury are at an increased risk for developing hypercoagulable and consumptive coagulopathic states (e.g. DIC) [8, 16]. Be prepared to run the full spectrum of coagulation testing. In many cases, thrombocytopenia will develop in the first 24 hours; observe for clinical signs (e.g. petecchiae, ecchymoses) and perform serial complete blood counts at least daily. Additionally, careful monitoring for signs of clotting abnormalities (e.g. hematoma formation, intracavitary bleeding, epistaxis, hematuria) is necessary throughout the hospital stay [17], and coagulation testing should be considered every 6–12 hours until the patient is stabilized. It is common for these patients to require blood component therapy [16].

Heat injury patients are at increased risk for development of cardiac arrhythmias, especially ventricular arrhythmias. Continuous or intermittent ECG monitoring is necessary to identify arrhythmias. Specific antiarrhythmic therapy may be indicated if the patient develops hemodynamic instability due to the arrhythmia.

Patients suffering from heat injury often will have concurrent vomiting and diarrhea. In some cases, the diarrhea will be hemorrhagic [16]. Hemorrhagic diarrhea is common and can create husbandry challenges. Hygiene is critical, and bedding should be changed as needed, long hair shaved, tails wrapped, and patients bathed. Gastrointestinal protection agents may be administered [12]. Nutritional support may be necessary during the hospital stay, to include enteral or parenteral feeding [16].

Renal insufficiency may develop. Anticipate placing a urinary catheter with a closed collection system to monitor adequacy of urine output or perform intermittent urethral catheterization. Urine production should be maintained at 1–2 ml/kg/hour.

As heat injury often induces multiple organ dysfunction, careful monitoring of the respiratory system is necessary. During initial resuscitation and for the next 12 hours, the respiratory rate should be monitored hourly, thoracic auscultation should be performed at least every four hours, and thoracic radiographs should be taken if pulmonary edema is suspected. Arterial blood gas analysis or surrogates should be performed as needed to assess oxygenation and ventilation, as discussed above. Mechanical ventilation may be necessary if the patient cannot oxygenate or ventilate adequately.

Generally, CNS abnormalities resolve with mild or moderate cases of heat injury. However, severe heat injury may cause cerebral edema and necrosis, and thus careful monitoring is necessary. Cortical blindness usually resolves but may take several days. Persistence of altered mentation, ataxia, tremors, or seizures strongly suggest increased intracranial pressure; specific therapy may be necessary (e.g. mannitol infusion).

A study in 40 dogs [23] demonstrated that 90% of dogs presenting with heat stroke had increased peripheral nucleated red blood cells (nRBC) at presentation, with a cutoff point of 18 nRBC/100 leukocytes corresponding to a sensitivity and specificity of 91% and 88%, respectively, for death. Dogs with nRBC above this cutoff point were also significantly more likely to have life-threatening complications such as kidney failure and disseminated intravascular coagulopathy. Thus, rapidly screening for the presence of nRBC may be useful to confirm clinical suspicion of heatstroke, and guide aggressiveness of therapy and monitoring.

A severity scoring system [22] has been validated in dogs with heat stroke that may prove useful to gauge severity of injury and prognosis based on key clinical and laboratory parameters noted within the first 24 hours of admission. Parameters useful to measure include heart rate, blood glucose, and coagulation tests. Clinically, the presence of obesity, acute collapse, shock, seizures, altered mental status, coagulopathy, acute kidney injury, and acute lung injury are documented risk factors for death.

In summary, patients with heat injury require rapid reduction in body temperature and intensive management. Targeted temperature reduction using thorough soaking of the skin with water is followed by cessation of cooling

measures at an appropriate temperature to prevent rebound hypothermia. Intensive monitoring and supportive care will minimize development of complications.

Open Wounds and Necrotic Tissue

Wounds commonly result from animal bites, motor vehicle or other trauma, and are classified as contaminated or dirty or infected wounds, depending on the length of time since injury. Contaminated wounds are those less than six hours old, and dirty or infected wounds are those six or more hours old, generally with obvious exudate or infection [26]. Wounds are often noted in conjunction with potentially life-threatening injuries; triage and management of more severe concurrent injuries takes precedence.

Considerations in Wound Management

Protocol 72.4 details the management of open wounds and necrotic tissue. Wound management goals are to create a healthy wound bed with adequate blood supply to support repair and without contamination or necrotic tissue that impedes healing and increases infection risk [26–28]. Wounds may require frequent evaluation and care. Many wounds must be managed as bandaged open wounds before definitive surgical repair. Management recommendations are provided in the protocol. Wound lavage is

Protocol 72.4 Management of Open or Necrotic Wounds

1) Triage the patient for life-threatening problems:
   a) Provide appropriate resuscitation and stabilization before wound management.
   b) Apply direct pressure followed by a temporary pressure bandage to stop active hemorrhage at wound sites.
2) Manage potential local and systemic infection:
   a) Collect and submit samples for microbial culture and sensitivity testing, preferably before starting antibiotic therapy.
   b) Initiate empiric antibiotic therapy within the first 6 hours of the wound's development, or as soon as possible thereafter.
   c) Culture the wound if obvious infection develops during any phase of wound management, the wound fails to heal normally, or systemic signs of infection develop.
3) Provide initial wound management:
   a) Provide effective analgesia or anesthesia based on wound severity, location, and other factors.
   b) Apply sterile water-soluble lubricant to the wound bed. Then clip the hair generously around the wound.
   c) Gently cleanse the skin around the wound, but not the wound bed, with surgical scrub.
   d) Gently lavage the lubricant and gross contaminants from the wound using sterile saline or lactated Ringer's solution.
   e) Debride grossly necrotic and nonviable tissues carefully using aseptic technique and sharp dissection in accordance with hospital policy:
      i) Do not mass ligate tissues or use cautery excessively.
      ii) Do not damage, transect, or ligate major blood vessels (unless actively hemorrhaging) or nerves, as these are crucial to maintain effective blood flow and innervation distally.
   f) Lavage the wound to remove particulate debris and reduce bacterial contamination:
      i) Thoroughly lavage the wound bed with at least 1 l for this final lavage.
      ii) Lavage under pressure, using a lavage pressure of 7–8 mmHg: use a 16-gauge hypodermic needle attached to an IV administration set attached to a 1 l bag of saline pressurized to 300 mmHg using a pressure cuff.
   g) Bandage the wound:
      i) Apply a primary layer to provide mechanical debridement initially, using a wet-to-dry bandage of sterile gauze sponges saturated with sterile saline, gently wrung to eliminate excessive moisture, and applied directly to the wound.
      ii) Apply several dry gauze sponges over the primary layer.
      iii) Apply a secondary layer over the primary layer, using cast padding or roll cotton ± splints to provide support.
      iv) Apply a tertiary layer of nonadherent conforming bandage (e.g. VetWrap®), adhesive bandage (e.g. Elastikon®), or both, using light compression.
4) Provide daily wound care, using appropriate analgesia, sedation, or anesthesia.
   a) Change bandages at least once daily, but more frequently if heavy discharge is present or the bandage is soiled or partially removed by the patient.
   b) Lavage the wound as above at every bandage change.
   c) Debride the wound as above at every bandage change.
   d) Apply a new bandage as above; change the primary layer to a nonadherent dressing once a healthy granulation bed is formed.

essential, ideally under specific pressure [29]. One modality to consider after emergency management of the wound is negative pressure wound therapy [26].

Antibiotic Use in Patients with Open or Necrotic Wounds

Systemic antibiotic use is ideally based on culture results. Empiric antibiotics are often used, based on expected microbial presence [29, 30]. Many contaminated wounds, with proper management, can be converted to clean-contaminated wounds, and systemic antibiotics may not be indicated. Most dirty/infected wounds require antibiotic use.

Open Fractures

Fractures are generally not considered life-threatening emergencies. Open fractures, in which the skin and subcutaneous tissues overlying a fracture are injured and expose fracture ends and fragments to external contamination, are significant and risk increasing morbidity and mortality because they provide access to microbial organisms that can cause local infection (i.e. osteomyelitis, cellulitis) and serve as a nidus of infection for systemic infection [27, 31, 32]. Thus, proper management of open fractures is an important part of the overall management of trauma patients. Early and proper management of open fractures is critical, and open fractures should be treated as a medical emergency, once more pressing problems are addressed. The majority of fractures are caused by motor vehicle trauma, but gunshot, entrapment injury, and blunt trauma from non-motor vehicle causes are also common. Recognize the intense forces involved in trauma that cause fractures, and address the overall patient for life-threatening injuries, first.

Management recommendations for open fractures are shown in Protocol 72.5. While providing initial resuscitation, quickly remove gross contaminants from the fracture site, but do not attempt to clip hair or clean open wounds at this stage. Do not attempt to reduce open fractures. Cover the fracture and wound with sterile nonadherent dressing and apply a light bandage to protect open wounds and exposed bone from further contamination during initial patient resuscitation; fractures are not placed in an attempt to stabilize or immobilize the fracture at this time. Culture open fracture sites as soon as possible after presentation and before antibiotic use if possible. The majority of open fractures are infected on admission, and in the majority of cases, the organism cultured on admission is the same organism involved in later infections [29, 31, 33]. Administer antibiotics as directed, focusing on use of IV antibiotics based on likely contaminants. Never withhold antibiotic therapy in any patient with an open fracture. Address pain with appropriate analgesia; reassess every four to six hours. Manage soft-tissue injuries over the fracture site appropriately to optimize outcome (Protocol 72.4). Once the patient is stable, immobilize the fracture if possible, to minimize pain, improve function, and prevent further injury to the neurovascular bundle and bone. Consider spoon splints, lateral plastic splints, or Robert Jones bandages. It may be better to leave the fracture alone than to apply an immobilizing device incorrectly. If an immobilizing device is not applied, apply a sterile wet-to-dry bandage to open fractures (Protocol 72.4). Always use a wet dressing over open fractures to keep soft tissues and bones moist for optimal healing. Change bandages at least once daily, based on degree of strike-through, soiling, or loosening.

In summary, patients with open fractures require careful management, with utmost attention to identifying and treating life-threatening problems first. Protect wounds and immobilize fractures initially. Provide subsequent appropriate antibiotic therapy, effective analgesia, daily wound care, and supportive care until definitive repair.

Protocol 72.5 Management of Open Fractures

1) Address life-threatening problems first:
   a) Use a standardized approach to trauma management.
   b) Focus on the "ABCs" of initial trauma patient management.
   c) Protect the open fracture site:
      i) Do not attempt to reduce any bone protruding at the fracture site.
      ii) Remove any large gross contaminants from the wound (e.g. leaves, rocks, stick fragments), but do not attempt to clip the hair or cleanse the wound at this point.
      iii) Cover the fracture and wound with sterile nonadherent dressing and apply a light bandage.
2) Prevent bacterial infection, provide analgesia, and promote normal healing pending surgical fracture repair:
   a) Culture open fracture sites as soon as possible after presentation and before antibiotic use if possible.
   b) Administer antibiotics as directed by the attending veterinarian.
   c) Never withhold antibiotic therapy in any patient with an open fracture.

d) Provide appropriate analgesia; reassess pain every 4–6 hours using a pain scoring system.
e) Manage soft tissue injuries over the fracture site appropriately (Protocol 72.4).

3) Manage orthopedic injuries once the patient is stabilized:
a) Immobilize the fracture, if possible, to minimize pain, improve function, and prevent further injury to the neurovascular bundle and bone.
b) If an immobilizing device is not applied, apply a sterile wet-to-dry bandage to open fractures (Protocol 72.4). Change bandages at least once daily, based on degree of strike-through, soiling, or loosening.

4) Monitor patients with open fractures:
a) Focus on the overall status of the patient.
b) Recognize common complications due to open fractures:
i) Assess pain frequently and provide appropriate analgesia.
ii) Base continued antibiotic use on the initial culture and sensitivity results.
iii) Monitor the open fracture site at least daily for evidence of local infection and monitor the patient frequently for evidence of systemic infection.
iv) Assess adequacy of immobilization, if applied, and monitor for complications of immobilizing devices (e.g. chafing, distal swelling, pain, skin wounds, tissue maceration).

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Blood Glucose Monitoring and Glycemic Control

Erica L. Reineke

In animals, blood glucose concentration is maintained within a very narrow range due to a dynamic balance between production, storage, and release of glucose. Quantitively, glucose is the most abundant carbohydrate that exists in the circulation and serves as the principal fuel for peripheral tissues except during prolonged fasting [1]. Glucose comes from intestinal absorption through digestion of carbohydrates, from breakdown of glycogen, or from production of glucose via precursors such as lactate, pyruvate, amino acids, and glycerol. When blood glucose concentration rises, the anabolic hormone insulin is secreted from the β-cells in the pancreas [2]. Ultimately, the effect of insulin is to lower blood glucose concentration by causing increased transport of glucose into cells, where it is used to form energy or stored in the form of glycogen. In addition, insulin also decreases the production of additional glucose by inhibiting gluconeogenesis and glycogenolysis. When hypoglycemia develops, there is increased secretion of the counter regulatory hormones glucagon, catecholamines, cortisol, and growth hormone. These hormones increase blood glucose concentration through inhibition of peripheral glucose uptake, increasing hepatic glycogenolysis and gluconeogenesis, and inhibition of insulin secretion. The maintenance of a normal blood glucose concentration depends on appropriate hormone secretion in response to changing concentrations in addition to normal hepatic glycogen synthesis, glycogenolysis, and gluconeogenesis [2]. Abnormalities in any of these physiologic functions can lead to either high or low blood glucose levels.

Abnormalities in Glucose Homeostasis

Abnormalities in glucose homeostasis (low or high blood glucose concentrations) occur commonly in the small animal patient. Normal blood glucose concentration is 53–117 mg/dl (2.9–6.5 mmol/l) in the resting state in adult dogs and 57–131 mg/dl (3.1–7.2 mmol/l) in adult cats [3]. These values may differ slightly depending on the clinical laboratory in which concentration is measured. In general, puppies and kittens may have slightly higher resting blood glucose concentration compared with adult animals [4]. The appearance of clinical signs related to altered blood glucose concentration depends on the absolute glucose concentration, and also on the duration, degree, and rate of decline or rise of glucose.

Hypoglycemia

Clinically relevant hypoglycemia occurs when the blood glucose concentration reaches less than 50 mg/dl in both dogs and cats. Clinical signs of hypoglycemia primarily manifest as cerebral dysfunction such as behavioral changes, ataxia, collapse, seizures, stupor, and coma [5–7]. These clinical signs occur because the brain, unlike most other tissues in the body, has an obligatory need for glucose for the production of ATP [8]. The brain has limited glycogen store, so it relies on hepatic glycogen breakdown to supply the glucose it requires for normal function. Other clinical signs of hypoglycemia that may occur in the small animal patient, such as pacing, vocalizing, restlessness, shaking and trembling, likely result from activation of the adrenergic system in response to impending hypoglycemia [6].

When neuroglycopenia, or hypoglycemia of the central nervous system, occurs, the reduction in cerebral ATP production results in dysfunction of the membrane-associated Na-K-ATPase pumps. The result of this pump failure is cell swelling and release of excitatory neurotransmitters such as glutamate and aspartate, which ultimately results in the clinical signs associated with cerebral dysfunction. Severe and prolonged hypoglycemia can lead to neuronal cell death

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[9, 10]. Early recognition and emergency treatment of hypoglycemia is thus essential to prevent permanent neuronal damage. In a recent single-center study of 660 dogs, hypoglycemia (<80 mg/dl) was present at emergency admission in 9% of dogs and was associated with increased mortality [11].

Hypoglycemia may occur in the small animal patient due to decreased production of glucose by the body or increased uptake. Common causes of hypoglycemia include juvenile and toy breed hypoglycemia, insulinoma or iatrogenic insulin overdose, hepatic failure, sepsis, and xylitol intoxication.

Hyperglycemia

Hyperglycemia is considered to be present when the blood glucose concentration exceeds 117 mg/dl in dogs. In cats, hyperglycemia is less well defined but is usually considered to be present when the concentration exceeds 130 mg/dl. When only mild elevations occur, clinical signs are generally absent. However, with severe elevations, clinical signs can include increased thirst and urination, dehydration, alterations in mental status, and coma.

Since glucose contributes to the osmolality of the blood, it is capable of causing the movement of water between body compartments. Hyperglycemia results in fluid shifting from the intracellular compartment into the intravascular space resulting in cellular shrinkage [12]. Once the glucose concentration in the blood exceeds the renal transport maximum, glucosuria will occur. The result is an osmotic diuresis, which may potentiate dehydration and hypovolemia in animals who are unable to drink water to compensate for these fluid losses [13]. In addition, chronic hyperglycemia and hyperosmolality induces the formation of osmotically active idiogenic osmoles in the brain. The purpose of idiogenic osmoles is to protect the brain against cerebral dehydration by preventing water movement from the brain into the blood (for more information, see Chapter 58) [14].

In addition to its effects on water distribution in the body, hyperglycemia can also contribute to the suppression of the immune system. Hyperglycemia has been found to alter cytokine release from macrophages and to impair phagocytosis and free radical production from leukocytes [15, 16]. Finally, hyperglycemia can contribute to an overall proinflammatory and prothrombotic state because it may incite leukocyte release of proinflammatory cytokines and activation of the coagulation system [15, 16].

Documented causes of hyperglycemia in veterinary patients include diabetes mellitus, hyperadrenocorticism, acromegaly, stress, and pancreatitis. There are also iatrogenic causes such as the administration of glucose-containing fluids, parenteral nutrition, and glucocorticoids. In addition, hyperglycemia has been documented to occur in critically ill animals including those with head trauma, sepsis, and congestive heart failure [17–20].

Hyperglycemia that occurs secondary to critical illness in nondiabetic patients has been termed stress hyperglycemia or diabetes of injury. Stress hyperglycemia likely results from a combination of low or normal insulin concentrations, increased counter regulatory hormone secretion, peripheral tissue insulin resistance, and deranged hepatic autoregulatory mechanisms [16]. Initially, this was considered to be an adaptive response of the body during illness or injury to maintain an energy supply to non-insulin-dependent tissues such as the brain and immune system [21]. However, a large body of evidence in the human medical literature suggests that even moderate hyperglycemia can contribute to both morbidity and mortality in critically ill people with severe brain injury, trauma, burns, sepsis, myocardial infarction, and stroke [22–28].

As seen in people, hyperglycemia has been documented to occur in critically ill veterinary patients, and evidence is accumulating that it is also associated with worse outcomes. In a study of dogs and cats sustaining head trauma, hyperglycemia at admission was associated with worse neurologic disease but not outcome [17]. In a different study evaluating dogs and cats presenting to an emergency service with congestive heart failure, hyperglycemia at admission was found to be associated with a worse outcome [20]. Critically ill cats admitted to an intensive care unit were found to have a higher median blood glucose concentration (183 mg/dl, range 51–321 mg/dl) than healthy controls and found to have similar hormonal derangements as critically ill people [29]. Similarly, 16% of critically ill dogs admitted to an intensive care unit were found to have hyperglycemia. In this study, nonsurvivors had significantly higher blood glucose concentrations than survivors, had longer hospitalization, and more septic complications [19]. Finally, in the single-center study of 660 dogs mentioned above, hyperglycemia (>120 mg/dl) was found in 40% of dogs at hospital admission and was associated with a higher mortality compared with dogs that had normal blood glucose concentration [11]. Despite accumulating evidence that hyperglycemia is common in critical illness, it is still unknown whether these increases in blood glucose concentration actually have detrimental consequences in our patients or whether hyperglycemia is instead a reflection of disease severity.

Blood Glucose Monitoring

As derangements in blood glucose concentration are common, blood glucose measurements and serial blood glucose monitoring should be performed on all critically ill patients. For patients presenting on an emergency basis, a blood glucose measurement should be taken at the time of presentation to the veterinary hospital, especially if clinical signs

such as altered mental status, seizures, or coma are present. Hospitalized patients should have blood glucose measurements performed at least once daily or more often depending on the underlying disease process or administration of therapies, such as dextrose-containing fluids or insulin, which are known to affect blood glucose concentration.

The method by which glucose is measured and the type of blood drawn (arterial, venous, or capillary) may lead to differing glucose measurements. It is therefore important to become familiar with the various methods available to measure glucose concentration and possible confounding issues a veterinary technician and clinician might encounter.

Blood glucose measurements can be performed on whole blood, serum, or plasma. Glucose measurements performed on plasma or serum are approximately 12–13% higher than whole blood measurements [30]. This is because the water content of red blood cells (73%) is less than that of plasma (93%), and because glucose is freely diffusible between plasma and erythrocytes. The greater water content of plasma means that the glucose concentration per unit volume of plasma is higher than in whole blood [30]. In addition, as the water content of whole blood is the sum of plasma water and red blood cell water, glucose concentration depends on the hematocrit of the sample. For example, in severe anemia, whole blood glucose and plasma glucose concentration will be nearly equal. With rising hematocrits, the disparity between whole blood and plasma glucose concentration increases [31, 32]. In general, the difference in glucose concentration between plasma and whole blood is minor (<20% difference) and typically does not affect clinical decision making. However, in human medicine, to avoid any potential clinical misinterpretation, it has been suggested that only glucose concentration in plasma be reported. The current recommendation is to apply the constant factor of 1.11 for the conversion between concentration of glucose in blood and the equivalent concentration in plasma [31]. No similar recommendations currently exist in veterinary medicine.

Measured glucose concentration may also vary depending on the location of the blood draw. For example, venous blood samples will give slightly lower results than capillary samples. However, in fasting subjects the glucose values obtained in arterial, capillary, and venous samples are practically the same. Differences are seen primarily after meals when glucose uptake by the periphery may be rapid, resulting in arterial and capillary glucose samples that may exceed the concentration from venous samples [28].

Glucose concentration, if not measured immediately, will decrease after blood collection due to continuing glycolysis by red blood cells and leukocytes. Thus, delay may lead to an artifactual lower measurement [29]. Addition of preservatives, such as sodium fluoride, may help to avoid or delay this loss [28]. Another option for avoiding artifactually low measurements is to separate plasma promptly from the cellular components in blood and measure the plasma glucose concentration instead.

Methods of Measuring Glucose Concentration

Glucose measurements are based on one of three enzyme systems – glucose oxidase, glucose-1-dehydrogenase, or hexokinase – which cause glucose conversion [31, 33]. These enzymatic reactions are either colorimetrically detected through reflectance or absorbance photometry, or amperometrically (electrochemically) detected. For example, glucose oxidase catalyzed reactions result in the production of gluconic acid and hydrogen peroxide. In colorimetric detection systems, hydrogen peroxide reacts with various hydrogen donors to produce a color change that is proportional to glucose concentration. This color change is measured using a reflectance photometer that converts that reflected light to an electronic signal for digital display [34]. Amperometric detection systems use the glucose oxidase or glucose-1-dehydrogenase enzymes. In amperometric measuring devices, an electrical current is produced from the reaction that is directly measured. Finally, for systems using the enzyme hexokinase, NADH (nicotinamide adenine dinucleotide) reacts with a dye to produce a color change detected by the machine [34].

Instruments Used to Measure Glucose

Blood glucose measurements are typically performed either in a central laboratory or cage-side using portable devices. As pathologic changes in glucose concentration require immediate intervention, blood glucose measuring devices need to provide rapid and accurate results. Unfortunately, reporting of central laboratory measurements of blood glucose concentration may take too long to be clinically useful. Therefore, cage-side glucose measuring devices are typically used in the clinical setting. In addition to providing rapid results, these cage-side portable blood glucose measuring devices also use minimal volumes of blood, thereby helping to limit anemia that may result from frequent blood sampling.

Common instruments currently used to rapidly evaluate blood glucose concentrationin small animals include portable blood glucose meters (PBGM) and point of care (POC) analyzers such as the i-STAT® (Abbott Point of Care Inc., Princeton, NJ), the NOVA Stat Profile® (NOVA Biomedical, Waltham, MA), or the RAPIDPoint® systems (Siemans Medical Solutions, Malvern, PA). Point-of-care refers to any laboratory test performed outside a clinical laboratory by nonlaboratory personnel [35]. It is important for the veterinarian and technician to understand and recognize potential interferences that may exist with glucose measurements obtained by these devices, particularly with PBGMs.

Portable Blood Glucose Meters

Many PBGMs are available from different manufacturers to measure blood glucose concentration. These devices were originally developed to allow for self-monitoring of blood glucose by people with diabetes mellitus. The benefits of blood glucose measurements provided by PBGMs include rapid results, ease of use, minimal blood requirements, and cost effectiveness. PBGMs use test strips that consist of an enzyme-impregnated reagent pad that is either sponge-like or covered with a mesh or membrane. When a drop of blood is applied to the surface, plasma soaks through into the reagent layer and red cells are retained on the surface [36]. Depending on the particular device, it may use colorimetric or amperometric detection systems to measure glucose concentration. PBGMs developed for use in people report the blood glucose concentration as a plasma equivalent by applying an internal equation to allow for more accurate comparisons with laboratory measurements.

The accuracy of PBGMs has been extensively evaluated in the human literature. In several human studies, large variations in the accuracy of different PBGMs in different glycemic ranges have been found [37–39]. Other factors such as hematocrit, inadequate sample volume, drugs, partial pressure of oxygen (PO_2), and user error have also been documented to affect the accuracy of glucose measurements obtained with PBGMs [40–43]. User errors most commonly cited to affect the accuracy of measurements obtained by PBGMs include failure to maintain the meter properly, incorrect techniques or operating procedures, or failure to follow instructions for meter use. Table 73.1 is a summary of some of the variables that may affect measurements obtained by PBGMs.

Table 73.1 Variables that may affect blood glucose concentration measurements obtained with a portable blood glucose meter.

Variable	Effect on glucose measurement
Hematocrit:	
Increased hematocrit (polycythemia or dehydration)	Decreases
Decreased hematocrit (anemia)	Increases
Drugs:	
Dopamine	Increases
Mannitol	Increases
Acetaminophen	Increases
Oxygen tension (PaO_2):	
Hyperoxia ($PaO_2 > 100$ mmHg)	Decreases
Hypoxia	Negligible
Small sample size (< 3 µl)	Decreases

Increases in hematocrit decrease whole blood glucose measurements and vice versa. In PBGMs, this may be caused by mechanical impedance of plasma diffusion into the reagent layer of the strip at higher hematocrits, resulting in slower diffusion of glucose and hence lower glucose measurements [44]. In patients with anemia, artifactually increased measurements may be obtained, which may mask hypoglycemia.

Substances such as drugs may also affect glucose measurements obtained by PBGMs. In a study evaluating the effect of 30 different commonly used drugs on glucose concentration measured by seven different PBGMs, the investigators found that ascorbic acid (vitamin C), acetaminophen, dopamine, and mannitol were all found to interfere with glucose measurements. Acetaminophen increased glucose measurements on several PBGMs [45]. This effect may be clinically relevant in small animals when acetaminophen ingestion can lead to hepatic failure and hypoglycemia. Dopamine falsely increased glucose measurements in some devices primarily at high drug concentrations, as did the drug mannitol [45].

Partial pressure of oxygen, pH, and temperature have also been evaluated to determine their effects on blood glucose concentrations obtained with PBGMs. When oxygen tension is high (partial pressure of oxygen in arterial blood, PaO_2, > 100 mmHg) investigators have found falsely lowered glucose readings in glucose oxidase systems [46]. This may be relevant in patients receiving high concentrations of supplemental oxygen, such as those under general anesthesia. Conversely, lower oxygen tensions ($PaO_2 < 40$ mmHg) had a negligible effect on glucose concentration. Blood pH, on the other hand, has not been shown to be a major source of error at a pH range of 6.8–7.84 [47, 48]. Finally, some data suggest that cold temperatures may adversely affect the accuracy of glucose measurements [49, 50].

Multiple studies evaluating different PBGMs have been published in the veterinary literature [51–55]. However, the number of commercially available PBGMs is constantly increasing and older models are continually being replaced by newer ones, making some of these previous investigations obsolete. These previous veterinary studies have found that PBGMs results may consistently overestimate, underestimate, or accurately reflect a laboratory measurement depending on the PBGM being evaluated. The AlphaTrak® (Abbott Laboratories, Abbott Park, IL), a PBGM marketed for dogs, did not consistently provide results that were either higher or lower than the laboratory values in one study. Even though the measurements obtained by the PBGMs were different from the reference laboratory values, they were unlikely to adversely affect clinical decision making [54]. In the most recent study investigating the Accu-Chek® Performa (Roche Diagnostics Corp, Indianapolis, IN) it was found that whole blood,

serum, or plasma blood glucose measurements were strongly correlated with laboratory measurements for dogs and cats [55].

Moreover, despite pitfalls that may be encountered when using PBGMs, they will continue to be a valuable tool to measure blood glucose in the veterinary setting. To prevent user error, it is important to always follow the manufacturer's instructions on maintenance and calibration of the device. In addition, whenever a new PBGM is obtained by a veterinary hospital, results should be compared with laboratory measurements to ascertain whether these results are consistently higher or lower than expected. Finally, if a patient is identified as hypoglycemic on a PBGM, the clinician should be notified; however, treatment may be recommended only if the patient is exhibiting clinical signs consistent with low blood glucose concentration. Alternatively, a comparison laboratory measurement of blood glucose concentration could be obtained [54]. Recently the American Society of Veterinary Clinical Pathologists released guidelines for the use of PBGMs in veterinary clinical practice. The reader is referred to another source for additional recommendations [56].

Point of Care Analyzers

The use of POC analyzers is becoming increasingly common in veterinary clinical practice. Similar to PBGMs, these analyzers require a minimal volume of blood and results are rapid. The i-STAT is the only POC analyzer that has been evaluated in dogs thus far [57]. The i-STAT measures glucose amperometrically through the glucose oxidation reaction. In one study, investigators found that the i-STAT provided accurate glucose concentration results that varied from the reference method by only 15% and did not result in altered clinical decision making [52].

To the author's knowledge, the accuracy of the NOVA Stat Profile and RAPIDPoint in measuring glucose concentration has not yet been evaluated in small animal patients. However, both of these devices have been validated for use in people and are likely to provide results consistent with laboratory methods. Both the NOVA Stat Profile and RAPIDPoint use an electrode that is covered by a three-layered membrane in which glucose oxidase is immobilized. As glucose flows through the membrane, it reacts with glucose oxidase, resulting in the generation of hydrogen peroxide. The instrument is calibrated using an aqueous glucose standard solution [36]. Similar to measurements obtained by PBGMs, hematocrit may affect blood glucose concentration values obtained by this method [58].

Newer Glucose Monitoring Devices

In the past 20 years, continuous glucose monitoring systems (CGMSs) that measure interstitial glucose concentration have received intense interest in both human and veterinary medicine as an attractive method by which to monitor patient glucose concentrations. Interstitial glucose concentration has been extensively studied and found to mimic blood glucose concentration in a variety of species including rats, rabbits, dogs, and people [59]. This knowledge has led to the development of a CGMS that measures interstitial glucose concentration within the subcutaneous space. The CGMS has been approved by the US Food and Drug Administration (FDA) for use in human diabetic patients and has resulted in significant improvements in glycemic control [60].

There are now a number of different CGMSs available. The two most common systems in use in veterinary medicine include the Medtronic Guardian™ (Medtronic Diabetes, Northridge, CA) and the FreeStyle Libre® (Abbott, Abbott Park, IL). Both systems consist of a recording device and a flexible electrode glucose sensor. The sensor is implanted in the subcutaneous space via a spring-loaded insertion device and is wirelessly connected to a small monitor or mobile device that can be worn by the patient or placed in a cage. The sensor contains an electrode covered by a glucose diffusion limiting membrane. When glucose flows onto the membrane in the electrode, it is oxidized to hydrogen peroxide by glucose oxidase. Glucose is then determined amperometrically for glucose concentrations of 40–400 or 500 mg/dl, depending on the device. The Medtronic CGMS measures interstitial glucose concentrations every 10 seconds, and an average value is recorded by the device every 5 minutes whereas the FreeStyle Libre measures interstitial glucose concentrations every 15 minutes. Because changes in blood glucose concentrations are related to changes in interstitial glucose, the CGMS can be used to estimate levels from the interstitial measurements. The Medtronic Guardian system must be calibrated with at least two to three blood glucose measurements during a 24-hour period whereas the FreeStyle Libre does not require calibration.

Since its development, the CGMS has been used in clinically normal animals, as well as in diabetic dogs and cats [61–63]. In 1999, Rebrin et al. found that subcutaneous interstitial glucose sensing accurately mimics plasma glucose irrespective of changes in plasma insulin in experimental dogs. However, in this same study, it was shown that rapid changes in blood glucose concentrations result in slower changes in interstitial glucose concentration, with a time delay between changes in blood and interstitial glucose concentrations of typically less than 10 minutes [59]. Clinical veterinary studies have since been performed to evaluate the accuracy of the CGMS in stable diabetic dogs and cats. The investigators found that there was significant correlation (dogs, $r = 0.81$; cats, $r = 0.82$) between the CGMS device and blood glucose concentration [62, 63]. Two veterinary studies have evaluated the use

of a CGMS in diabetic ketoacidotic dogs and cats. The results of these studies also found that the CGMS provides clinically accurate estimates of blood glucose concentrations and that these measurements were not affected by tissue perfusion or metabolic variables, body condition score, or degree of ketosis [64, 65].

The benefit of using a CGMS device is that they allow clinicians to identify fluctuations in blood glucose concentrations as they are occurring in the patient. Additionally, these real-time devices allow the clinician to avoid placing catheters for the sole purpose of blood sampling or performing repeated venipuncture to measure blood glucose concentrations, both of which can contribute to morbidity in the patient by contributing to patient stress, catheter complications, and anemia in small patients. The CGMS is useful in patients in which repeated phlebotomy is either contraindicated due to underlying disease or cannot be performed. Compared with intermittent glucose monitoring, continuous monitoring of interstitial glucose concentration may help to identify rapid changes, which may be missed in patients in which blood glucose is measured intermittently. This may be important in diabetic patients that are having blood glucose curves plotted to evaluate insulin dose. The addition of alarms that can notify technicians and clinicians of either dangerously high or low glucose excursions make these devices even more clinically useful. Many veterinary hospitals currently use CGMSs as the main method of monitoring estimated blood glucose concentration, especially in critically ill diabetic patients receiving insulin infusions that require frequent glucose monitoring.

Several disadvantages of the CGMS include the initial cost for the device, the cost of the sensors, and the need to obtain blood glucose measurements for calibrations in the Medtronic Guardian CGMS. Another potential disadvantage of the interstitial glucose monitoring system is the time delay for rapid changes in glucose concentrations to be reflected in the interstitial space. Therefore, data obtained from these devices should be interpreted cautiously when large changes in glucose concentrations are suspected. In these situations, evaluating glucose concentration trends over time may be more clinically useful. Finally, these devices have not been fully investigated for clinical accuracy in patients with hypoglycemia.

Placement of the CGMS in dogs and cats is a simple process. Figure 73.1 shows the placement of the Guardian RT device in a patient. First, a small area of fur is clipped, usually just caudal to the shoulder blades. The area should not be cleaned with alcohol, as this may interfere with the adherence of the sensor. The sensor is then placed into the spring-loaded insertion device. The insertion device is placed against the patient's skin and the sensor is discharged. The transmitter is then attached to the sensor, and both are covered with a clear adherent bandage. After an approximately two-hour initialization period, the Guardian CGMS device will begin to continuously display estimates of blood glucose once it has been calibrated with data from a measurement. The CGMS will then need to be recalibrated every 8–12 hours with either a PBGM or POC analyzer measurement of blood glucose. If there is any concern that inaccurate estimates are being obtained by the CGMS (either extremely high or low readings), the patient's blood glucose should be checked directly. If this measurement is different from the CGMS readings, the CGMS should be recalibrated. If the problem persists, the sensor should be removed and replaced.

In contrast with the Guardian CGMS, the FreeStyle Libre has a one-hour initialization period after placement of the sensor. No calibrations are required and interstitial glucose measurements are taken every 15 minutes and only displayed when the monitor or mobile device is passed over the sensor.

Glycemic Control

Glycemic control implies the maintenance of blood glucose within a clinically acceptable range. This is achieved through the administration of dextrose, insulin, or medications known to raise or lower blood glucose. Patients in which interventions are required to maintain clinically acceptable blood glucose concentrations should be monitored frequently to avoid complications associated with hypo- and hyperglycemia. Frequent monitoring is particularly important in the short- and long-term management of diabetic patients, in which glycemic control is achieved through adjustments in insulin therapy.

The benefits of glycemic control in people with diabetes mellitus include a reduction in mortality, as well as a reduction in diabetes-related complications such as blindness, kidney failure, and heart failure [66]. Insulin is also used in critically ill people who develop hyperglycemia during hospitalization [67–69]. The use of insulin therapy to control critical illness-associated hyperglycemia has yet to be evaluated in veterinary medicine.

Treatment of Hyperglycemia

The treatment of hyperglycemia in veterinary medicine depends on the underlying disease process. For example, stress hyperglycemia in cats associated with patient struggling can result in blood glucose concentrations as high as 613 mg/dl, with or without glucosuria [70]. Since stress hyperglycemia can last 90–120 minutes, one should wait at least three hours before retesting or instituting treatment for hyperglycemia if stress hyperglycemia is suspected.

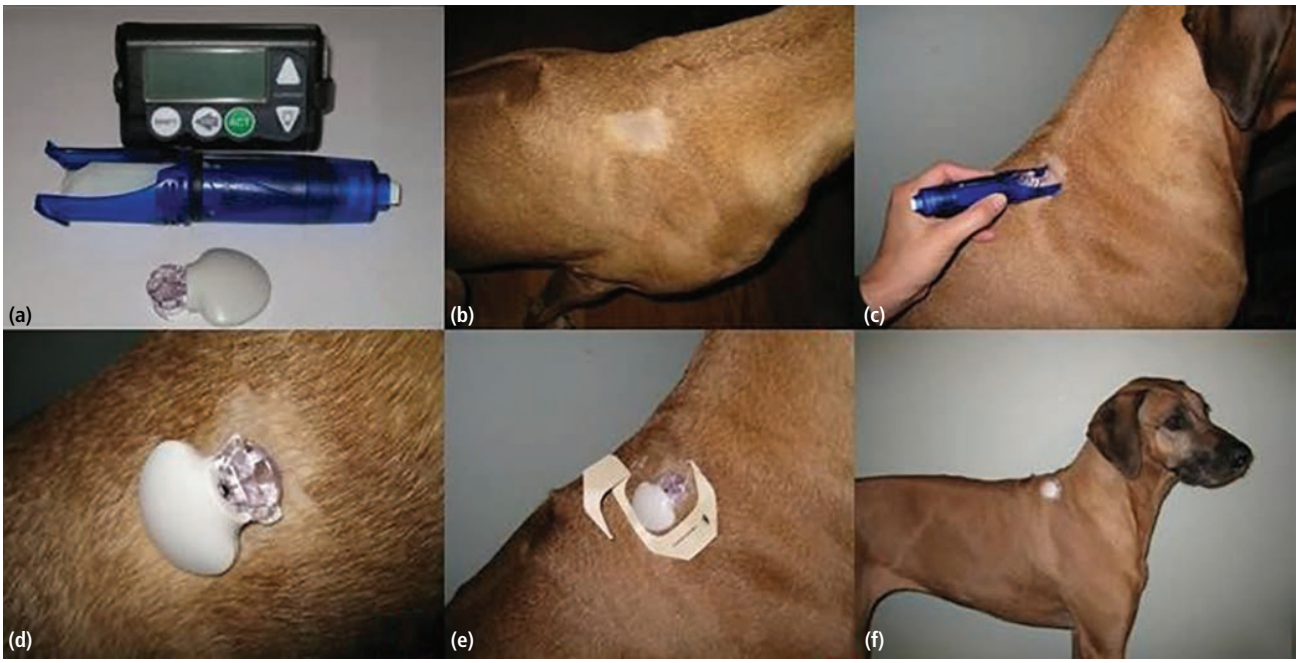


Figure 73.1 Placement of Medtronic Minimed Guardian® RT continuous glucose monitoring system. (a) Equipment: monitor, spring-loaded sensor insertion device for accurate placement, blood glucose sensor with transmitter attached. (b) Small area of clipped fur just caudal to the scapula. (c) Using the spring-loaded device for sensor insertion. (d) Sensor in place with transmitter attached. (e) Clear adherent dressing being placed over sensor and transmitter. (f) Dog wearing the sensor with transmitter attached.

Hyperglycemia caused by diabetes mellitus requires administration of the hormone insulin. Insulin promotes peripheral glucose uptake, inhibits lipolysis and release of free fatty acids, decreases glycogenolysis, and increases glycogenesis [2]. Insulin is available in several forms with differing durations of action. Typically, regular crystalline insulin, in which the duration of action is approximately three to eight hours when given intramuscularly (IM) or one to four hours when given intravenously (IV), is administered during critical illness until the patient is eating and drinking. Regular insulin is usually administered either IV as a constant rate infusion (CRI) or intermittently IM. Other newer, short-acting insulins, such as lispro (Humalog®, Eli Lilly) and aspart (Novolog®, Novo Nordisk) have been recently evaluated in a small number of dogs with diabetic ketoacidosis [71, 72]. In these studies, both aspart and lispro administered as a CRI were found to be safe and effective as compared to a CRI of regular insulin [71, 72].

Insulin may also be used to treat persistent hyperglycemia that results from veterinarian-directed interventions. The administration of dextrose-containing fluids such as parenteral nutrition and the administration of medications that are known to affect blood glucose, such as vasopressors or steroids, can cause hyperglycemia.

When persistent hyperglycemia cannot be explained by either diabetes mellitus or veterinarian-directed interventions, the patient should be reevaluated by either the veterinary technician or clinician for worsening of the underlying disease condition or the development of complications. This may involve performing a physical examination, hemodynamic monitoring, and diagnostic tests. Persistent hyperglycemia should be definitively established prior to initiating insulin therapy.

Intravenous Insulin Constant Rate Infusion

Table 73.2 is an example of an IV insulin infusion chart. The infusion is prepared by adding regular insulin (2.2 U/kg) to 250 ml of 0.9% NaCl [73, 74]. It is initially administered at a rate of 10 ml/hour in a line separate from that used for fluid therapy [73]. At least 50 ml of the insulin-containing fluid should be run through the drip set and discarded prior to administering it to the patient because insulin adheres to plastic and glass; this 50 ml preflush helps saturate the interior of the administration set with insulin.

Generally, the placement of two separate IV catheters is recommended: one catheter for insulin and an additional catheter for IV fluids and blood sampling. Separate catheters are recommended to avoid starting and stopping the insulin infusion during blood sampling for glucose monitoring. In addition, if IV fluids containing dextrose are being administered, blood sampling for glucose monitoring should be done from a separate catheter to avoid falsely elevated blood glucose measurements that may occur from contamination with dextrose-containing fluids. A multi-lumen catheter, either placed centrally or peripherally, may also be used. The most distal lumen of the central

Table 73.2 Constant Rate Infusion of Regular Insulin.

Blood glucose concentration (mg/dl)	Rate of administration of insulin solution (ml/hour)[a]	Dextrose added to maintenance intravenous fluids (%)[b]
> 250	10	None
200–250	7	2.5%
150–200	5	2.5%
100–150	5	5%
< 100	Stop insulin infusion	5%

[a] Solution comprised of regular insulin at a dose of 2.2 U/kg for dogs, or 1.1–2.2 U/kg for cats, added to 250 ml 0.9% NaCl or lactated Ringer's solution. Adjustments to rate of insulin therapy are made based on measurements of blood glucose.
[b] These intravenous fluids are administered separately and in addition to the insulin infusion when needed to compensate for continuing losses, dehydration, and to maintain intravascular volume in the patient.
Source: Adapted from Macintire DK. Treatment of diabetic ketoacidosis in dogs by continuous low-dose intravenous infusion of insulin. J. Am. Vet. Med. Assoc. 1993;202:1266–1272.

catheter is typically reserved for blood sampling. A separate peripheral catheter, in addition to the central catheter, should be maintained for the administration of dextrose-containing IV fluids. This is to avoid contamination of the blood sample being obtained for glucose monitoring with dextrose-containing IV fluids that could artifactually increase the measured blood glucose concentration. If the maintenance of a separate peripheral catheter is not possible or the administration of greater than 5% dextrose solutions necessitates administration through the central catheter (see section on treatment of hypoglycemia below), a large presample should be obtained (5–10 ml of blood) prior to checking the blood glucose. If there is any question about the measured sample (i.e. suspicion of a falsely elevated level) even with the large presample, a direct venipuncture blood sample should be obtained for comparison.

Blood glucose concentration should be monitored every one to two hours in animals on insulin CRI, and changes in insulin rate should be made based on each measurement. Intravenous insulin is typically administered until the patient can be switched to a longer-acting insulin product in patients with diabetes mellitus or when insulin administration is no longer needed to maintain glycemic control.

Intramuscular Insulin Techniques

When given IM, regular insulin can be administered hourly or less frequently depending on the technique chosen. The hourly technique involves the IM administration of regular insulin and measurement of blood glucose every hour. The usual initial dose for the IM insulin protocol is 0.2 U/kg of regular insulin. After one hour, the blood glucose is checked, and if it exceeds 250 mg/dl, 0.1 U/kg regular insulin is administered IM. This protocol is repeated on an hourly basis as long as the level exceeds 250 mg/dl. Once the level drops below 250 mg/dl, regular insulin is administered IM every four to six hours or subcutaneously every eight hours if the hydration status is adequate. In patients with diabetic ketoacidosis, a 2.5–5% dextrose-containing infusion is initiated at this time [75]. Box 73.1 gives instructions for calculating a dextrose infusion. Once the blood glucose has reached an acceptable clinical range, it is monitored less frequently. The goal of insulin therapy is to cause a gradual decline in the concentration, preferably at a rate of about 50–75 mg/dl/hour [76]. Subcutaneous administration of insulin is not recommended in the initial treatment of critically ill diabetic patients, as dehydration could lead to erratic insulin absorption.

Regular insulin can alternatively be administered intramuscularly every four to six hours at 0.25 U/kg. This is less labor-intensive compared with the hourly administration of regular insulin. Following the first dose of insulin, blood glucose is checked hourly but then the frequency of monitoring can be decreased depending on the duration of insulin action. An hourly decline of 50 mg/dl is ideal. If the concentration is dropping too rapidly, the veterinarian should be notified and subsequent insulin dosages may be decreased.

Initiation of Longer-Acting Insulin

Once the patient is eating and drinking, intensive insulin plans are usually replaced by longer-acting insulin therapy.

Box 73.1 Calculation of a Percent Dextrose Solution Using 50% Dextrose

C1 × V1 = C2 × V2 where C = the concentration of a solution and V = the volume

(Dextrose 0.5) × (Volume of 50% dextrose required, ml) = (Desired strength of solution) × (Volume of infusion, ml)

$$\frac{\text{Desired Strength of Solution}}{\text{50\% Dextrose}} = \frac{\text{Volume of Dextrose needed (ml)}}{\text{Volume of Infusion (ml)}}$$

Consider making 1 l of a 2.5% dextrose solution

1. Convert percentage to decimals
2. $\frac{\text{0.025 Dextrose}}{\text{0.50 Dextrose}} = \frac{\text{X ml}}{\text{1000 ml}}$

   0.025 (1000 ml) = 0.50 (X ml)
   X = 50 ml of 50% dextrose
3. Add 50 ml of 50% dextrose to 950 ml (if you do not remove the 50 ml from the total volume, the solution will be slightly less than 2.5% dextrose)

Consider making 500 ml of a 5% dextrose solution

1. Convert percentage to decimals
2. $\frac{\text{0.05 Dextrose}}{\text{0.50 Dextrose}} = \frac{\text{X ml}}{\text{500 ml}}$

   0.05 (500 ml) = 0.50 (X ml)
   X = 50 ml of 50% dextrose
3. Add 50 ml of 50% dextrose to 450 ml

Several intermediate-acting and long-acting insulin products are available, including neutral protamine Hagedorn (NPH) insulin, purified porcine insulin zinc, protamine zinc insulin, and glargine insulin.

Purified porcine insulin zinc (Vetsulin®, Merck Animal Health, Rahway, NJ) is the only FDA-approved insulin for use in both dogs and cats. Glargine insulin (Lantus, Aventis Pharmaceuticals, Bridgewater, NJ) and detemir (Levemir®, Novo Nordisk Inc., Plainsboro, NJ) are newer insulin analogues being used in the management of diabetic companion animals. Twice daily subcutaneous administration of both glargine and detemir have been investigated in diabetic dogs and cats and the studies suggest that it is safe and effective in the management of diabetes mellitus [77–81]. Detemir is a more potent insulin in dogs and starting insulin doses are much lower than for other insulin products. Table 73.3 lists available insulin products and current initial insulin dose recommendations in dogs and cats.

Table 73.3 Insulin types and initial dosing recommendations.

		Recommended initial dose	
Insulin	**Concentration**	**Dog**	**Cat**
NPH	U-100[a]	0.25–0.5 U/kg every 12 hours	1 unit/cat every 12 hours
Porcine Zinc (Lente)	U-40[b]	0.5 U/kg every 12 hours	1–2 U/cat or 0.25–0.5 U/kg every 12 hours[c]
PZI	U-40[b]	Not recommended	0.4 U/kg or 1 U/cat every 12 hours
Glargine	U-100[a]	0.5 U/kg every 12 hours	0.25–0.5 U/kg every 12–24 h
Detemir	U-100[a]	0.02–0.13 U/kg every 12 hours or 1 U/dog/day[d]	0.25 U/kg every 12 hours if BG < 360 mg/dl 0.5 U/kg every 12 hours if BG > 360 mg/dl

BG, blood glucose; NPH, neutral protamine Hagedorn; PZI, protamine zinc insulin.
[a] U-100: 100 units/ml
[b] U-40: 40 units/ml
[c] A recommended starting dose is 0.25 U/kg twice daily if the blood glucose concentration (BG) is 216–342 mg/dl, and 0.5 U/kg twice daily if the BG is > 360 mg/dl. Alternatively, a dose of 1 U/cat twice daily for cats weighing less than 4 kg and 1.5–2.0 U/cat twice daily for cats weighing > 4 kg can be used to initiate therapy.
[d] Only one study evaluating the effect of detemir in diabetic dogs currently exists in the veterinary literature. These dogs were given an initial starting dose of 1 U/dog. Bacause of its potency, dogs may be a greater risk for hypoglycemia. Fracassi F, Corradini S, Hafner M et al. Detemir insulin for treatment of diabetes mellitus in dogs. J Am Vet Med Assoc 2015;247(1):73–78.

Specific Considerations when Administering Insulin

Insulin is commercially available in 40, 100, and 500 U/ml concentrations, which are designated U-40, U-100, and U-500, respectively [82]. Depending on the concentration of the insulin being administered, there are specific syringes, also labeled U-40, U-100, and U-500, that should be used. For example, glargine insulin, a U-100 insulin, should be administered only with a U-100 syringe. It is important to always check that the label on the insulin syringe matches the concentration of the insulin being administered.

Insulin syringes are also available in different sizes including 1, 1/2, and 3/10 cc (1, 0.5, and 0.3 ml) with different gauge needles. Lines on the insulin syringes are units. For example, 1 cc insulin syringes are generally marked with a line for every two units, that is for 2, 4, 6, and 8. The 1/2-cc syringe is marked with a line for every unit. The 3/10-cc syringe is marked with lines for every half or one unit.

Insulin should always be stored in a refrigerator, as freezing and heat inactivate insulin in the bottle. The exceptions to this rule are glargine and detemir, which may be stored at room temperature but away from direct heat and light once opened. Unopened glargine and detemir should be stored in the refrigerator. Once opened, whether refrigerated or not, glargine should be discarded after 28 days and detemir after 42 days according to the manufacturer. Currently, it has also been a common recommendation that other insulin preparations be discarded after one month. However, many veterinarians allow use of an opened bottle of insulin that is refrigerated for up to three months before discarding. However, if clinical signs recur in a previously well-controlled diabetic patient, loss of activity of the insulin could be the cause, and the insulin bottle should be replaced.

Prior to drawing insulin into the insulin syringe, the bottle should be gently rolled between the palms of the hands (15–20 times) to allow for uniform resuspension of the insulin within the liquid. Failure to mix the insulin properly could decrease its effectiveness. It is important not to shake the insulin bottle, as this could create small air bubbles within the vial leading to inaccurate dosing.

Always administer the type of insulin as designated by the treatment orders. In other words, Vetsulin® (Merck Animal Health) should not be substituted for NPH, as each insulin product will have a different duration of action and effect in each individual patient.

Glucose Monitoring

When insulin is administered in the treatment of hyperglycemia, blood glucose is monitored closely to prevent hypoglycemia. During treatment of a patient with hyperglycemia, the patient should be observed closely for neurologic signs, such as changes in mentation and seizure activity, which could indicate hypoglycemia. If neurologic signs develop, blood glucose should be checked.

Treatment of Hypoglycemia

Acute hypoglycemia requires immediate therapy because the longer the hypoglycemic episode lasts, the greater the potential for irreversible brain damage. Patients that are presented on an emergency basis or that develop clinical signs attributable to hypoglycemia during hospitalization are often treated with an IV bolus of 50% dextrose (0.25–0.5 g/kg). 50% Dextrose is very hyperosmolar and ideally it is diluted to less than 10% with sterile water if given through a peripheral IV catheter. This dose of dextrose is often repeated as indicated by serial blood glucose measurements or until clinical signs are resolved. Dextrose and dextrose-containing fluids should never be given subcutaneously.

Following a bolus of dextrose, the patient may be placed on a CRI of 2.5–5% dextrose in crystalloid solutions. To make 1 l of a 2.5% dextrose solution, 50 ml of 50% dextrose can be added to 950 ml of fluids (total volume of 1 l). To make 1 l of a 5% dextrose infusion, 100 ml of 50% dextrose is added to a 900 ml of fluid (remove 100 ml of fluids from a 1 l bag). See Box 73.1 for instructions on how to calculate a percentage dextrose infusion.

Both 2.5% and 5% dextrose infusions may be administered through a peripheral venous catheter. However, when higher concentrations of dextrose (> 5% solution) are needed to maintain a normal blood glucose concentration, higher concentrations of dextrose (> 5% solution) may be administered through a central line. This is because glucose infusions greater than 5% are irritating to the vascular endothelium and may result in thrombophlebitis [83]. Administering higher concentrations of dextrose solutions in a larger vein will help to decrease the incidence of thrombophlebitis. Alternatively, rather than increasing the concentration of the dextrose infusion, the rate (ml/hour) of dextrose administration may be increased. This will allow for increased delivery of dextrose to the patient. Finally, 5% dextrose in water may also be administered IV to supplement dextrose but should be given cautiously, as profound hyponatremia can develop if large volumes are administered. The goal of glucose supplementation is usually to maintain the blood glucose within the normal physiologic range, as hyperglycemia can have deleterious consequences.

In addition to IV dextrose supplementation, hypoglycemic patients are often fed as soon as possible, which will help to support blood glucose concentration. However, feeding may not be recommended in patients with hypotension, low body temperature, vomiting, or other conditions common in urgently and critically ill animals.

If seizure activity is present in a patient with low blood glucose and does not resolve despite attaining euglycemia, anticonvulsant therapy such as diazepam (0.25–0.5 mg/kg), midazolam (0.2 mg/kg), or levetiracetam (20–30 mg/kg) should be administered intravenously [84]. Phenobarbital (2–4 mg/kg IV or orally) may be used for longer-term seizure control. Severe hypoglycemia and prolonged seizure activity can result in cerebral edema. Treatment for cerebral edema involves oxygen administration, elevation of the head 15–30 degrees above the horizontal plane, and administration of hypertonic agents (Chapter 70) [85]. Hyperosmotic agents such as mannitol and hypertonic saline do not cross an intact blood brain barrier and thus reduce cerebral edema by osmotically drawing water out of the brain. Common doses are mannitol 0.5–1 g/kg IV or 7.0–7.5% NaCl 3–5 ml/kg IV over 15–20 minutes.

Hypoglycemia caused by insulinoma can be very challenging to treat. Dextrose administration in these patients can trigger tumor secretion of insulin and rebound hypoglycemia. Therefore, several other medications are available in the treatment of insulinoma with the effect of supporting blood glucose by increasing endogenous glucose production. Dexamethasone (0.5 mg/kg IV every 12–24 hours) is given to increase hepatic gluconeogenesis and glycogenolysis as well as inhibit peripheral glucose uptake [86]. A CRI of the hormone glucagon with concurrent dextrose infusion is an additional treatment for insulinoma-associated hypoglycemia [87, 88]. Glucagon should be reconstituted with the provided diluent and added to 1 l of 0.9% NaCl resulting in a solution with a glucagon concentration of 1 μg/ml. This medication is usually started at a dose of 5 μg/kg/minute and the infusion dose may be increased as needed up to 20 μg/kg/minute to maintain a concentration greater than 73 mg/dl [86, 87]. A syringe pump should be used to allow for accurate dosing of glucagon. This medication cannot be given orally. Finally, the drug diazoxide, which inhibits insulin release and stimulates hepatic release of glucose via gluconeogenesis, is also potentially useful for treatment of hypoglycemia in dogs with insulinoma. Diazoxide also causes adrenomedullary release of epinephrine, which increases insulin resistance. The dose ranges from 10 to 40 mg/kg/day orally and is generally titrated as indicated by the blood glucose concentration [86]. The most common adverse effect seen with use of this drug is gastrointestinal upset, including diarrhea, vomiting, and anorexia, which may be minimized by administering this medication with food [88].

Blood glucose should be monitored closely, at least every two hours, or more frequently, in all patients following a hypoglycemic episode until it can be established that the primary cause has been resolved or effectively controlled. Any patient that presents with symptomatic hypoglycemia should be hospitalized until the patient is eating and drinking normally.

Conclusion

Blood glucose monitoring is an important aspect in the management of acutely and critically ill companion animals. The prompt recognition and treatment of both hyperglycemia and hypoglycemia are essential for the veterinary technician and clinician. There are a number of ways to measure blood glucose concentrations in veterinary patients; however, portable blood glucose meters and POC analyzers are most commonly used to quickly measure blood glucose cage-side in the hospital. Continuous glucose monitoring devices allow for non-invasive minute-to-minute monitoring of interstitial glucose concentration, potentially allowing for quicker detection and treatment of aberrations in blood glucose. Glycemic control in critically ill patients is achieved in most patients with the use of insulin and/or dextrose. It is extremely important for both veterinary clinicians and technicians to understand the clinical indications for their use as well how to administer these medications safely and effectively to our patients.

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Critical Nursing Care of the Neonate

Autumn Davidson and Janice Cain

The neonatal period, for purposes of this chapter, is the first two weeks of life, although some authors refer to the immediate (moments to hours) postpartum period as the newborn period. Neonatal mortality rates are greatest during the first week of life, especially the first 48 hours; reportedly the average can be as high as 18–30% [1–3]. Veterinary intervention in the prenatal, parturient, and postpartum periods can increase neonatal survival (and hence reduce the need for admission to the critical or intensive care unit) by controlling or eliminating factors contributing to puppy and kitten morbidity and mortality.

Poor prepartum condition of the dam, dystocia, congenital malformations, genetic defects, low birth weight, postnatal injury, environmental extremes, malnutrition, parasitism, and infectious disease all contribute to neonatal morbidity and mortality. Veterinary intervention improves neonatal survival by managing pregnancy (obstetrics) including ovulation timing or gestational aging, labor and delivery (vaginal or surgical) to reduce stillbirths and fading neonates, optimizing nutrition of the dam and neonates, controlling parasitism, and reducing infectious disease. Promoting proper genetic screening for selection of breeding stock minimizes inherited congenital defects. Recognition and appropriate medical and/or surgical management of dystocia are critical to neonatal survival. Medical management should be based on objective criteria (tocodynamometry as well as Doppler or ultrasound fetal heart-rate evaluation), and response to therapy (calcium gluconate, oxytocin); surgical management should be timely, whether elective or emergent (Figure 74.1).

Care of the small-animal neonate in the clinical setting is challenging. Small-animal neonates are very small and fragile. Veterinary neonatal technology has not achieved the advances available to human neonatologists (e.g. prepartum diagnostics/intervention, ventilator therapy, extracorporeal membrane oxygenation, hypothermic therapy) or even in some cases available for other veterinary species (e.g. no canine surfactant is available, no studies exist on bovine or synthetic substitution). Evidence and consensus-based guidelines in the veterinary literature are limited. Another factor that must be considered is the cost for substantial medical intervention in the neonatal period. This choice is usually made by the breeder, as the neonate is not yet adopted into a family for which a strong human/animal bond will exist. It is this bond that is the usual driving factor for the decision by most owners to pursue significant intensive medical care for their pets. While veterinarians and staff can be dismayed by the reality of this situation, it is important to consider the whole picture from the breeder's viewpoint, including emotions, time, energy, and other costs involved in raising a litter. The discussion between the healthcare team and owner about costs and expected outcomes should be approached in a nonjudgmental, informative manner.

Small-Animal Neonatal Physiology

The neonatal cardiovascular system is characterized by low pressure, low volume, and low peripheral resistance requiring a high heart rate, central venous pressure, cardiac output, and plasma volume for homeostasis. The cardiac sympathetic nervous system and baroreceptor development are immature. Myocardial hypoxemia results in bradycardia and anoxia results in hypotension. Chest wall compliance is stiff and carotid body chemoreceptors immature, predisposing to hypoventilation and hypoxemia, which can then promote bacterial translocation [4]. Premature neonates (less than around 62 days of gestation counting from the luteinizing hormone surge) can lack surfactant [5]. Neonates have red blood cell macrocytosis and polychromasia, are

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Figure 74.1 (a) Tocodynamometry unit. (b) Monitoring uterine activity in a Chihuahua bitch in stage II labor. (c) Tocodynamometry results showing normal myometrial contraction (x-axis strength of contraction in mm Hg, Y-axis time in minutes). (d, e) Fetal heart-rate monitoring with a handheld Doppler.

hemoconcentrated at birth, then develop an anticipated pediatric anemia. Mucous membrane color in the neonate tends to be red and not representative of pallor or shock. Decreased renal and hepatic metabolic and excretory capacity persist until four to six months of age; both glucosuria and proteinuria are normally present. Blood pressure dictates renal blood flow. Blood urea nitrogen, creatinine, and urine specific gravity are low. Sterile at birth, intestinal flora are acquired during and following birth [4]. Human neonates born by cesarean section fail to acquire typical/normal intestinal flora immediately as they would have from vaginal delivery; this can have repercussions later in life [6]. Intestinal motility is dependent upon a pressure gradient rather than myoelectrical activity; ileus, bacterial overgrowth and intussusception can result. Neonates exhibit essential reflexes (rooting, righting, heat seeking, suckling); electroencephalography is representative of sleep, and neurotransmitters are immature. Neonates have a lower nociceptive threshold than adults [7]. Their body weight to surface area makes chilling more likely. Absorption of oral drugs is altered by high gastric pH and increased mucous content, decreased gastrointestinal motility, and decreased bile production. Drug distribution is impacted by

neonatal increased body water content and decreased fat content. Lower plasma proteins decrease drug binding. The blood brain barrier is incomplete [4].

Cesarean Section: Emergency (Surgical Management of Dystocia) or Elective

Most commonly, neonates are admitted to critical or intensive care after unsuccessful management of dystocia resulting in an emergency cesarean section, less commonly following an elective section (recommended for brachycephalic and giant breeds with high incidence of dystocia, and singleton litters), and sometimes after medically managed dystocia. Once the decision for cesarean section has been made, time is of the essence. A well-organized team will have the operating room prepared during the time of medical evaluation and management of dystocia; the team can then proceed quickly. Factors to consider for positive outcome for both the dam and neonates include adequate staffing and veterinary expertise, optimal anesthesia, surgical efficiency, and rehearsed neonatal resuscitation. Intraoperative intravenous (IV) balanced crystalloids are advised.

There are numerous reports of anesthetic protocols for cesarean section; the authors prefer the following: Preanesthetic glycopyrrolate as an anticholinergic (anticipate vagal stimulation from manipulation of a gravid uterus), owing to its limited placental transfer, combined with an opioid sedative (75% of usual dose/nonpregnant body weight) to lessen anxiety and provide analgesia for the dam [8, 9]. Preoxygenation for three to five minutes is advised if the dam is not distressed by its administration; this is facilitated by preanesthetic sedation. As much surgical prepping as possible should be performed before anesthetic induction. Anesthetic induction is achieved quickly with IV propofol (4–7 mg/kg IV over 60–90 seconds) to permit endotracheal intubation and ventilation. Repeat administration of propofol as mini-boluses (1.7–3 mg/kg over 60 seconds) as needed to maintain a light surgical plane of anesthesia. Alfaxalone (1.5–4.5 mg/kg/60 seconds)/maintenance (1.1–2.2 mg/kg/60 seconds) is an alternative induction agent. A local anesthetic block in the region of the intended ventral midline incision using bupivacaine 0.25% (2.5 mg/ml) or 0.5% (5 mg/ml) at 1–2 mg/kg, lidocaine 2% (20 mg/ml) at 1–5 mg/kg, or a combination (lidocaine 2%, equal volume 0.5% bupivacaine) is advised, reducing the amount of general anesthetic needed during surgery by providing analgesia [10].

The surgical technique employed during the cesarean section depends in part on the surgeon's preference, litter size, condition of the uterus, and status of the dam and neonates; the authors' follows. Laparotomy is performed



Figure 74.2 Bulldog clamp on umbilicus intraoperatively.

expeditiously and efficiently. Exposing only part of the uterus as needed to deliver fetuses while maintaining a sterile field, is helpful, especially in a large dog with a large litter. A hysterotomy incision is made on either side of the uterine bifurcation. Fetuses are delivered quickly and passed to the recovery team. The umbilicus can be clamped immediately, or if preferred, the placenta can be delivered attached to the fetus and managed by the recovery team (see Neonatal Resuscitation, below) (Figure 74.2). Similarly, the surgeon may tear fetal membranes, or may defer to the team (Figure 74.3). Usually, all fetuses can be delivered from a single hysterotomy incision, but a second incision may become necessary for delivery of fetuses cranial in the opposite horn.

In most cases, all fetuses can be delivered within 15 minutes from the time of anesthetic induction. This promotes prompt recovery of the neonates; movement, breathing, and vocalization are observed nearly immediately after delivery. After all fetuses are delivered, inhalant anesthesia (isoflurane or sevoflurane) can be added to the regimen and surgery completed in a routine manner. The authors' opinion is not to perform ovariohysterectomy at the time of cesarean section unless indicated by the condition of the uterus (devitalized/necrotic) because of the higher risk of maternal mortality from postoperative hemorrhage and potential prolongation of operative/anesthetic/recovery time. A single dose of perioperative antibiotic appropriate for fetuses/neonates (e.g. a cephalosporin) is advised.

Neonatal Resuscitation

Resuscitation is indicated for most neonates delivered by cesarean section because of maternal (and fetal) anesthesia. Neonatal vigor can also be negatively impacted by dystocia, even with an eventual vaginal delivery, and may require intervention; if the dam's actions fail to stimulate respiration, vocalization, and movement within one



Figure 74.3 (a) Amniotic membrane over a neonate's face. (b) Tearing the amniotic membrane intraoperatively.

minute of birth intervention is advised. Maintaining a neonatal resuscitation kit is optimal (Protocol 74.1). Neonatal resuscitation techniques are based on the same principles (airway, breathing, circulation) as any cardiopulmonary resuscitation with one modification; ventilation takes precedence over chest compressions (Protocol 74.1).

Neonates should be received into a warm towel, their amniotic membranes should be immediately removed from the muzzle, and the proximal airway promptly cleared by gentle suction with a bulb syringe or DeLee aspirator, while the neonate's head is lowered to enhance drainage (Figure 74.4). Neonates should not be swung to clear airways because of the potential for concussion and resultant cerebral hemorrhage [11]. Prolonged airway suction can be harmful [12].

After confirming a heartbeat (palpation, auscultation, Doppler), dry and stimulate the neonate to promote respiration; brisk rubbing of the muzzle and thorax stimulates sensory respiratory receptors. Movement of the neonate and vocalization are positive signs, as spontaneous breathing and vocalization at birth are positively associated with survival through seven days of age. Reversal of any narcotic used in the dam's anesthetic protocol should be considered (naloxone 0.0001 mg/g intramuscularly or subcutaneously) [10]. Ventilatory support should be initiated if hypopnea is evident, beginning with constant flow-by O_2 (40–60%) delivered by face mask. If this is ineffective after one to two minutes, positive pressure ventilation (around 30–60 breaths/minute) with a snugly fitting mask, or endotracheal intubation and rebreathing bag (using a 2-mm endotracheal tube or a 12- to 16-gauge IV catheter), is advised (Figure 74.5). Anecdotal success with Jen Chung acupuncture point stimulation (GV26) has been claimed when a 27-gauge or actual acupuncture needle is inserted into the nasal philtrum at the base of the nares and rotated when cartilage is contacted (Figure 74.6). GV26 reportedly increases blood pressure and the brain inspiratory centers [13].

A detectable improvement in heart rate (normal around 180–200 beats/minute) should follow successful ventilatory support; myocardial hypoxemia is the most common cause of neonatal bradycardia (< 150 beats/minute) or asystole. The use of doxapram (sublingually or intramuscularly) as a respiratory stimulant is unlikely to improve hypoxemia associated with hypopnea and resultant hypoventilation and is not currently recommended [10]. Atropine is also not advised in neonatal resuscitation [10]. The mechanism of neonatal bradycardia is hypoxemia-induced myocardial depression; it is not vagally mediated, and anticholinergic-induced tachycardia can exacerbate myocardial oxygen deficits. Persistent hypopnea warrants reevaluation of effective proximal airway clearance (no fluid present) and positive pressure ventilation (thoracic excursions). If marked bradycardia (< 30 beats/minute) or asystole is present, direct transthoracic cardiac compressions (around 90–120 beats/minute) are advised as the first step (two-finger lateral thoracic compressions unless it is a chondrodystrophic breed, then sternal); positive pressure ventilation should also continue. Cardiac compressions should be coupled with ventilation (3 : 1), due to neonatal myocardial oxygen requirements. Epinephrine 0.00001–0.0001 mg/g body weight (e.g. a 500-g neonate gets 0.05–0.5 ml of 1 : 10 000 [0.1 mg/ml] epinephrine solution) IV or intraosseously (IO) can be considered, as well as bicarbonate (guided by blood gas analysis ideally, sampling can be challenging) at 0.001–0.003 mEq/g body weight [10, 14]. Intracardiac injections are problematic as

Protocol 74.1 Neonatal Resuscitation Protocol

Equipment

- Syringes (TB)
- 20–25 gauge needles, 18–24 gauge over-the-needle catheters, EZ-IO® catheters
- Drugs:
  - Epinephrine (1 mg/ml) to be freshly diluted 1 : 9 with sterile water or saline to 0.1 mg/ml solution
  - 50% dextrose to be freshly diluted with sterile water to 2.5–5%
  - Ceftiofur
  - Lidocaine freshly diluted with dextrose 5% in water, 0.9% sodium chloride, or lactated Ringer's solution to 1%
- Oxygen sources
- Suction (pediatric bulb syringes, DeLee mucus traps)
- Small face masks
- Towels (small)
- Heat sources (Baer Hugger, warm water blanket, infrared lamp, hair dryer, warm IV fluid bags)
- Puppy box (insulated) with heat support
- Multiple clean mosquito forceps and small scissors
- 3-0 monofilament suture for umbilical cord ligation
- Tincture of iodine 2%
- Shallow stainless-steel bowls for warm water baths
- Pediatric/neonatal stethoscope
- Doppler
- Neonatal gram scale

Ceftiofur Protocol

- Reconstitute vial with 20 ml sterile water to 50 mg/ml solution.
- Stable for 12 hours at room temperature (once reconstituted).
- Stable for 7 days if refrigerated (once reconstituted).
- Stable for 8 weeks if frozen (once reconstituted).
- Freeze in 1 ml increments in labeled RTT.
- Neonatal dose is 2.5 mg/kg (0.0025 mg/g) SC twice daily for 3–5 days.

Resuscitation Protocol

Airway, Breathing, and Circulation (ABC)

1) Clear muzzle of amniotic membranes and airway of fluids by suction (DeLee aspirator, bulb syringe). Do not swing. Place with head below thorax to improve drainage.
2) Gentle brisk towel drying to stimulate respiration; provide flow-by oxygen as necessary.
3) If not breathing, start positive pressure ventilation using snug face mask and oxygen.
4) If heart rate is slow, improve airway clearance/ventilation/oxygenation.

Is Resuscitation Effective?

1) Is the puppy vocalizing?
2) Is the mucous membrane color improving?
3) Is the puppy moving?
4) Keep in mind that even a nonviable puppy can have red color in the mucous membranes from the maternal circulation and fetal hemoglobin.
5) Acupressure (GV26) if hypopneic: 27-gauge or acupuncture needle into the nasal philtrum, insert and turn.
6) External cardiac compression (lateral unless chondrodystrophic breed, then sternal) if persistent bradycardia (< 60 beats/minute despite interventions) at 3 : 1 compression/ventilation ratio (90 : 30).

Medications if ABCs Fail

- Epinephrine 0.00001–0.0001 mg/g body weight IO/IV
- ± bicarbonate 0.001–0.003 mEq/g body weight IO/IV
- Atropine not advised
- Doxapram not advised

Prolonged Problematic Case

1) Hypothermic? Warm water bath at 95–98°F
2) Hypoglycemic? Dextrose 2.5–5.0% IV, IO

Reasons to Stop

1) No pulse after 10 minutes (check with Doppler or pediatric stethoscope).
2) Agonal breathing for more than 20 minutes.
3) Severe congenital defect (client consent).

they are markedly traumatic. Venous access in the neonate is challenging; the single umbilical vein is one possibility if not already thrombosed; the jugular vein another. The proximal humerus, proximal femur, and proximomedial tibia are potential intraosseous sites for effective drug administration (Box 74.1; Figure 74.7).

Continued hypopnea and hypoxia can indicate that the neonate is premature; prematurity is complicated by a lack of surfactant and contributes to irreversible respiratory distress. Replacement is not available for the canine or feline neonate and premedication of the dam with corticosteroids does not induce its production [15].

Hypothermia and hypoglycemia can complicate resuscitation efforts. Neonates cannot thermoregulate (shiver). Chilled neonates will respond suboptimally to resuscitation. Loss of body temperature occurs rapidly when a neonate is damp post-delivery. Keeping the neonate warm is important both during resuscitation and in the immediate

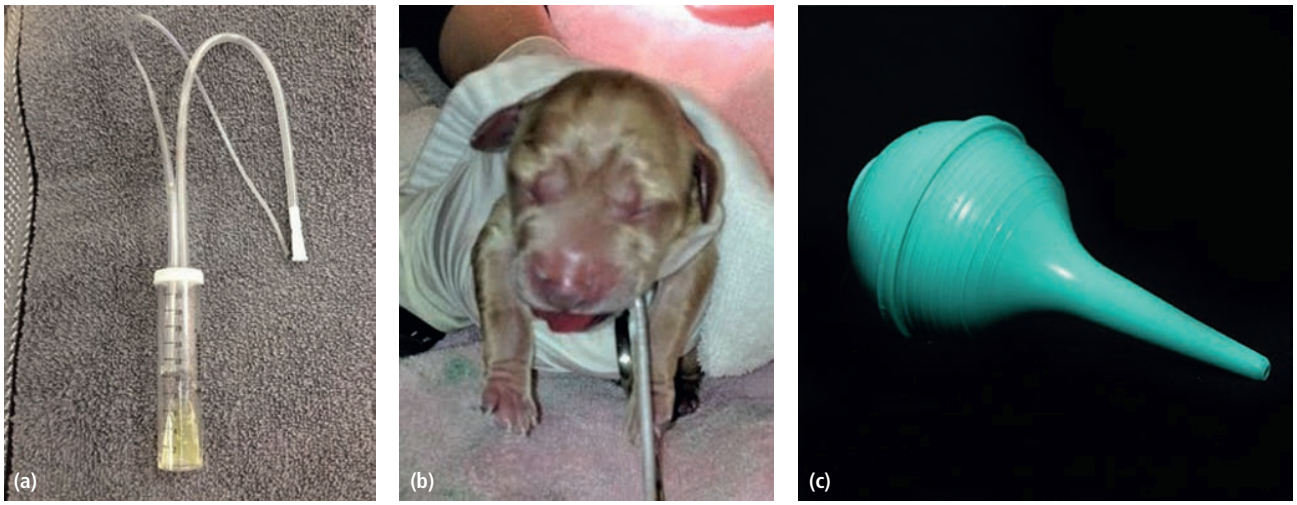


Figure 74.4 (a) DeLee aspirator containing neonatal airway fluid. (b) DeLee in use in proximal airway of neonate. (c) Human preemie bulb syringe.

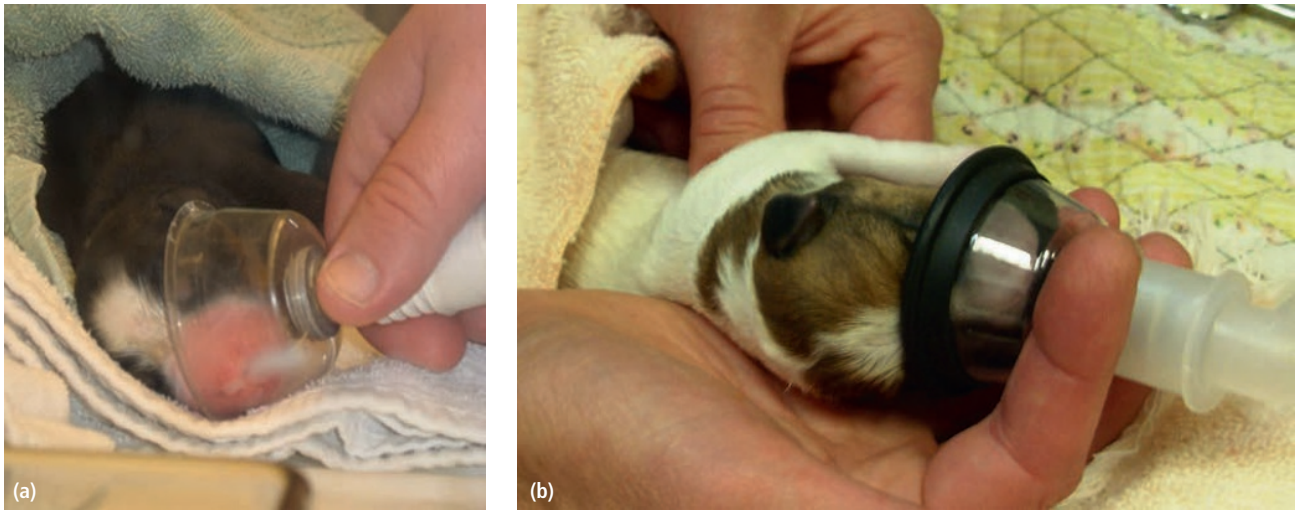


Figure 74.5 (a) Flow-by oxygen delivered by face mask. (b) Positive pressure ventilation delivered with snugly fitting face mask.



Figure 74.6 (a) GV26 acupuncture site, cadaver. (b) GV26 acupuncture, neonate.

Box 74.1 Intraosseous Catheterization

Sites

- Trochanteric fossa of femur
- Wing of ilium
- Proximal humerus
- Lateral tibia

Technique

1) Select site, clip/aseptic preparation.
2) Perform lidocaine 1% block 0.001 mg/g SC ± stab incision.
3) Insert needle with twisting motion through 1 cortex.
4) 18–22 gauge spinal or hypodermic needle.
5) Evaluate placement with 0.2–0.5 ml non-heparinized 0.9% saline infusion.
6) Apply aseptic bandage.

Complications

- Bilateral cortical perforation.
- Growth plate perforation.
- Trauma/fracture (unlikely due to immature calcification).
- Short catheter lifespan (~24 hours); easily dislodged (replace with IV catheter in 24 hours if continued IV access necessary).
- Bacterial contamination, bone necrosis, osteomyelitis.
- Slow infusion rate.

Advantages

- Quick access.
- Rapid distribution, equal efficacy as IV route.
- Appropriate for any isotonic solution given IV.



Figure 74.7 (a) Proximal humerus intraosseous catheter in place, cadaver. (b) Proximal femur trochanteric fossa, cadaver. (c) Cephalic catheter in septic neonate.

postpartum period. Working under a heat lamp or within a Bair Hugger® (3M, Maplewood, MN) warming device is helpful. During a prolonged resuscitation, placing a chilled neonate's body into a warm water bath (95–99°F; 35–37°C) for a few minutes can improve response (Figure 74.8). Prompt drying with thermal support should follow. After successful resuscitation, neonates should be placed in an insulated box (an insulated box with ventilation is ideal if an incubator is not available) with warmed but not excessively heated bedding until they can be placed with their dam for nursing (Figure 74.9).

Neonates lack glucose reserves and have minimal capacity for gluconeogenesis. Providing energy during prolonged resuscitation efforts becomes critical. Clinical hypoglycemia involves blood glucose levels less than 80 mg/dl and is best treated with IV or IO dextrose at a dose of 0.1–0.2 ml of a 2.5–5.0% (25–50 mg/ml) dextrose solution. Single administration of parenteral glucose is adequate if the neonate improves and can then be fed or nurses. Because of the potential for phlebitis if administered intravenously, 50% dextrose solution should only be applied to mucous membranes; however, circulation must be adequate for any absorption from the mucosa. Neonates repeatedly administered dextrose should be monitored for hyperglycemia because of immature metabolic regulatory mechanisms. If a neonate is too weak to nurse, a mixture of a warmed, balanced crystalloid (half-strength saline) and 2.5% dextrose may be administered by gavage (0.25–1 ml) until the neonate can be fed or nurses (Protocol 74.2). Note that 5% dextrose in lactated Ringer's or Normosol® (ICU Medical, Lake Forest, IL) solution is hypertonic and contraindicated unless volume expansion is desired. Subcutaneous

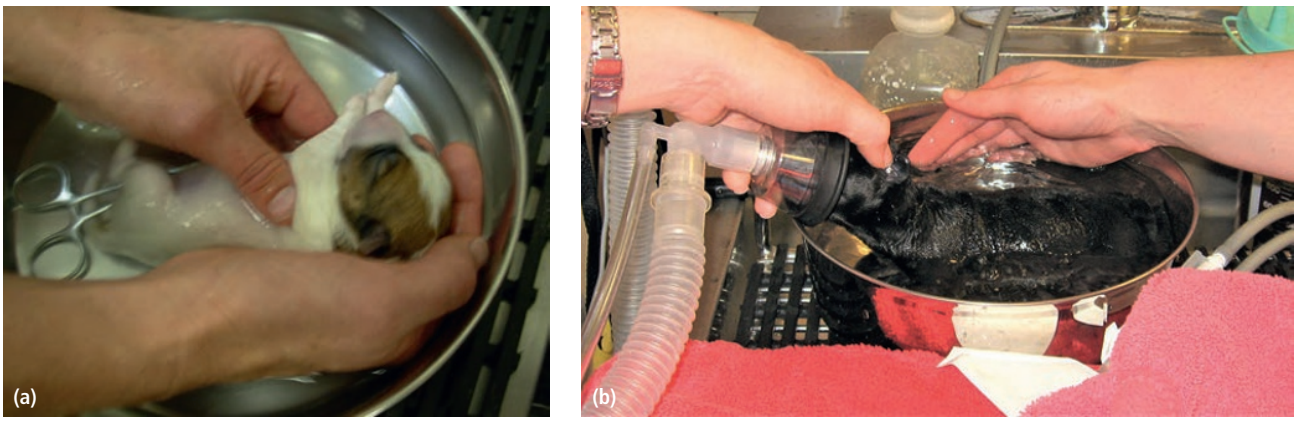


Figure 74.8 (a) Neonate receiving chest compressions while in warm water (around 98.6°F; 37°C) bath. (b) Neonate in warm water bath receiving flow-by oxygen.

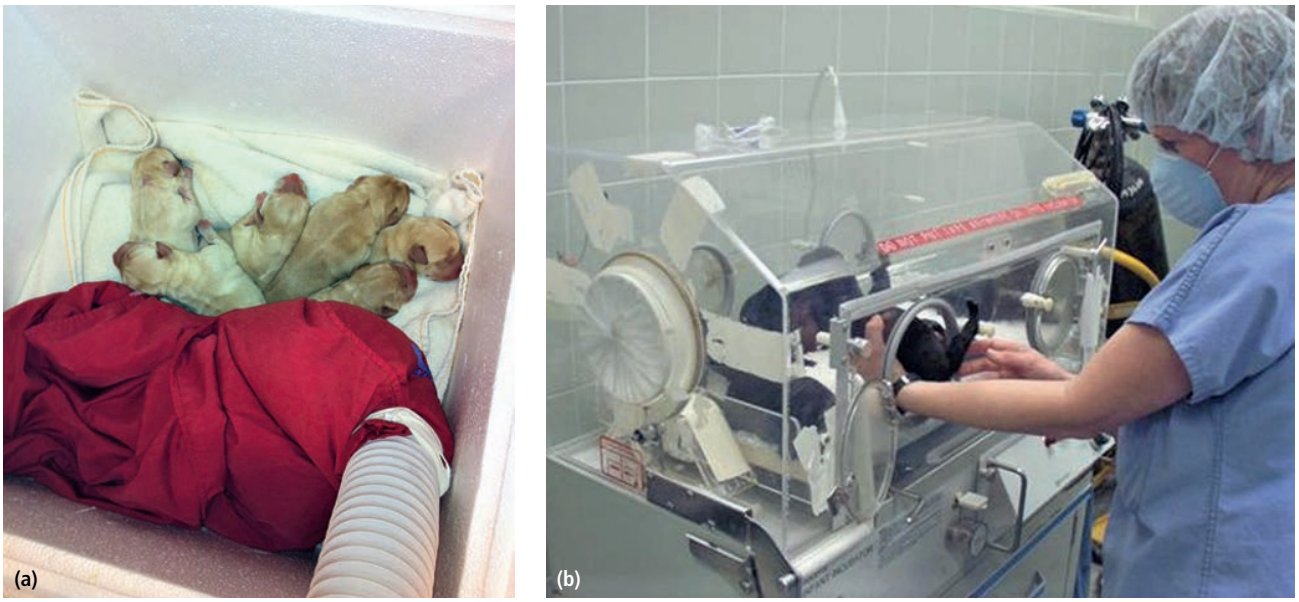


Figure 74.9 (a) Styrofoam (insulating) neonatal receiving box with warm air bag post-resuscitation. (b) Human neonatal incubator.

Protocol 74.2 Placement of a Stomach Tube in Neonates

Equipment

- Syringes (12 cc for mild, 3 cc for tube placement confirmation)
- Soft red rubber or silicone feeding tube (5–10 Fr), closed end with side ports; use the largest size that can be easily placed through the esophagus to lessen the chance of inadvertent airway introduction
- Sharpie pen or adhesive tape (mark depth of tube for insertion)
- Water-soluble lubricant
- Bowl
- Dam's/conspecific colostrum, milk, or commercial formula

Technique

1) Place neonate in dorsal recumbency on a flat, padded surface. The cranial portion of the neonate can be moderately elevated (similar to the normal nursing position). Measure the distance from the rostral muzzle to just beyond the last rib with the feeding tube and mark the tube. This identifies the length to reach the distal esophagus/stomach.
2) Lightly lubricate the distal portion of the tube without occluding the openings.
3) Gently introduce the tube into the oropharyngeal area, which should stimulate a suckle or swallowing reflex. The tube can be gently advanced to the mark.
4) Test tube placement (esophageal/gastric vs. tracheal):
   a) Place the exposed end of the tube in a shallow bowl of water. If bubbling results, the tube is likely in the airway. Remove and reinsert.
   b) Infuse a small amount of 0.9% saline or water (1–2 ml) through the tube and monitor for struggling/coughing. Remove and reinsert if occurs.
5) Slowly inject colostrum, milk, or formula (warmed to approximately body temperature; 95–99°F; 35–37°C); monitoring for any distress. Volume fed should be around half stomach capacity, which is 4–5 ml/100 g neonate weight; smaller, more frequent feedings are best.
6) Follow by stimulation to urinate, defecate if less than 14 days of age.



Figure 74.10 Subcutaneous abscess secondary to dextrose administration.

administration of glucose solutions can result in abscessation and is not advised (Figure 74.10). Colostrum acquired from the dam can be administered orally by gavage (0.25–1 ml) every 15–30 minutes until the neonate is capable of suckling. Colostrum supplemented neonates have a lower incidence of necrotizing enterocolitis [16].

APGAR Scoring

APGAR scoring in human neonatal resuscitation is routinely employed at one and five minutes after birth to measure neonatal viability in the first minutes of life objectively. It has been described in small-animal neonatal resuscitation. The authors recommend the term *APVAR* as small-animal neonates do not grimace (Box 74.2). One study evaluated heart rate, respiratory effort, reflex irritability, mobility, and mucus membrane color five minutes after birth and found that lower scores correlated with higher mortality two hours after birth [17]. Another study evaluated heart rate, respiratory effort, reflex irritability, mobility, and mucus membrane color as well as umbilical vein lactate level. The study found that the presence of lactate was predictive of neonatal mortality within 48 hours [18]. Dystocia or anesthesia-associated hypoxia, followed by anaerobic glycogenolysis and metabolic acidosis, has been found to be a major contributor to intrapartum mortality in many species; adaptive mechanisms (lower metabolic rate, low body temperature) may contribute to neonatal hypoxia tolerance [19]. A later study evaluated evaluating general vitality (mucous membrane color, heart rate, respiratory rate), irritability reflex and mobility evaluation, rectal temperature, glucose, β-hydroxybutyrate, lactate, and hydration status (urine specific gravity) between 10 minutes and eight hours after birth, finding

Box 74.2 Veterinary Small Animal APVAR (Appearance, Pulse, Vocalization, Activity, Respiration)

Scoring

A Appearance (mucous membrane color): 2 = pink, 1 = blue or pale, 0 = very blue/pale
P Pulse/heart rate: 2 = normal (~200 bpm), 1 = slow; 0 = none
V Vocal/crying: 2 = immediate, 1 = minimal, 0 = none
A Activity: 2 = moving vigorously, 1 = weakly, 0 = limp
R Respiration: 2 = normal, 1 = intermittent, agonal, 0 = none

Additional Notes

- A: normal physical exam
- B: meconium significant
- C: physical anomaly
- Vital neonate score 8–10
- Fair neonate score 6–8
- Poor neonate score <6



Figure 74.11 Cyanotic neonate and normal littermate. *Source:* Courtesy of Dr. P. Olson.

low vitality, relative hypoglycemia, and low birth weight to be predictive of increased neonatal mortality (Figure 74.11) [20].

Management of the Umbilicus And Placenta

Debate exists concerning the management of the umbilicus and placenta in the immediate postpartum/post-cesarean section period. Bitches tend to ingest the placenta and nip the umbilical cord immediately after delivery, followed by removal of the fetal membranes by licking. The surgeon's preference may be to clamp and ligate the umbilicus prior to removing the neonate from the surgical field, or to remove the fetus and placenta as a single unit delivered to the recovery technician/nurse. Removal of the fetal membranes by the surgeon is also variable. Some advocate retaining the placental blood flow/umbilical patency to avoid asphyxiation before respiration is initiated [21]. In any case, resuscitation should always take precedence over placental/umbilical care unless hemorrhage from the umbilicus exists. Following successful resuscitation, the umbilicus of neonates should be double clamped, trimmed to 1 cm from the body wall, ligated and then saturated with 2% tincture of iodine to reduce contamination and prevent ascent of bacteria into the peritoneal cavity (omphalitis, peritonitis); the alcohol-based tincture of iodine promotes faster desiccation of the umbilicus (<24 hours) than water-based betadine (Figure 74.12).

Postnatal Support

Thermal support during this period is important and can be accomplished with a warm air blanket, hairdryer on a low setting, or overhead heat lamps most effectively (Figure 74.13). Post-resuscitation nursing should be encouraged for immediate acquisition of colostrum. This can be accomplished even while the dam is recovering from anesthesia with appropriate direct supervision from a knowledgeable technician/nurse or breeder (Figure 74.14). Removal of any residual antiseptic from the dam's mammary skin avoids contact dermatitis in the neonates (Figure 74.15). Neonates should be stimulated by rubbing, and placed on a nipple from which a small amount of colostrum has been expressed, and should be encouraged to suckle. The neonate's mouth can be opened gently and placed on a nipple; the taste of colostrum can improve suckling effort. Gently stoking the dorsum of a neonate against the fur simulates the dam's grooming and results in improved effort to latch on and suckle. Weighing neonates with a gram scale before and after this first nursing confirms effective suckling. They should then be placed in the above-mentioned warm box while the dam continues to recover. Following anesthesia, most dams are uncoordinated and lack normal maternal instincts for 12–24 hours; they should always be under supervision when with their neonates.

Neonatal Examination

Immediately post-resuscitation, a quick, thorough physical examination of the neonate should be performed. This will determine whether the neonate is vital and normal and can be reunited with the dam for supervised nursing with the goal of eventual discharge to the owner's care, or whether further critical intervention is indicated. Vital signs should be obtained (Table 74.1). The head, oral cavity, nares, hair

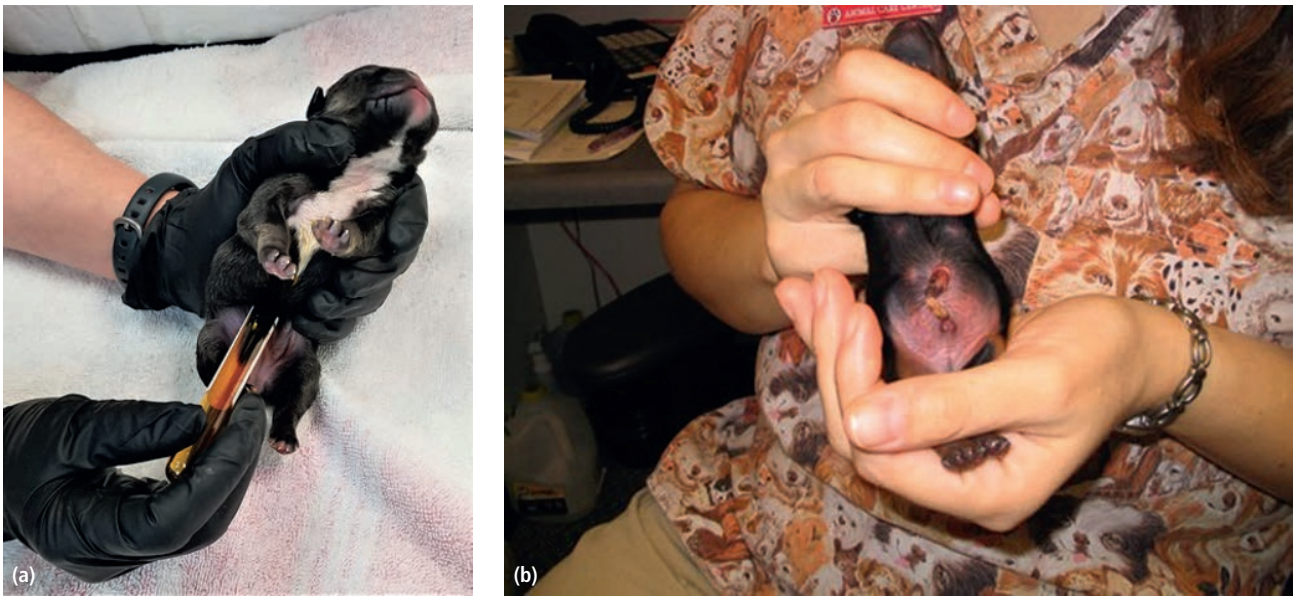


Figure 74.12 (a) Application of 2% tincture of iodine ("dunk") to ligated umbilicus. (b) Normal appearance of umbilicus after ligation and dunking.



Figure 74.13 Judicious use of overhead warm air for thermal support and flow-by oxygen during resuscitation/umbilical care.

coat, limbs, digits, umbilicus, anus, and urogenital structures should be visually inspected. In the normal neonate, the mucous membranes are reddish pink and moist, a suckle reflex is present (stronger is better), the coat full and clean, and the urethra and anus patent (Figure 74.16). The eyelids and ear canals are closed. External genitalia should not be ambiguous or malformed (Figure 74.17). Canine testes descend after six weeks of age, feline testes are intrascrotal at birth but very small. Urination can be stimulated by gently dabbing the vulva or prepuce, and defecation (meconium or first stool) by dabbing the anus with a fresh, moistened cotton ball. The thorax should be auscultated; vesicular breath sounds and a lack of murmur are normal; subtle flow murmurs, if present, should resolve within 24–48 hours.

The abdomen should be pliant and not painful, and the umbilicus non-patent. Muscle tone should be strong.



Figure 74.14 (a) Assisted first nursing. (b) Continued nursing during the dam's recovery.



Figure 74.15 Contact dermatitis; neonates' dorsal nasal planum resulting from exposure to residual preoperative antiseptic while nursing.

Table 74.1 Normal values of body temperature, heart and respiratory rates in the newborn.

Vital sign	Puppies	Kittens
Body temperature	95–98°F (35.0–37.2°C)	95–98°F (35.0–37.2°C)
Heart rate	200 beats/minute	200–250 beats/minute
Respiratory rate	10–18 breaths/minute	10–18 breaths/minute



Figure 74.16 Evaluating the neonatal suckle reflex.

A normal neonate will squirm and vocalize when examined, will attempt to right itself if placed on its side or dorsum, and will crawl and root to find the dam. Seeking and "piling up" with littermates conserves heat. Successful nursing is followed by sleeping quietly; occasional twitching is normal. Neonates awaken and seek to nurse frequently (every 10–20 minutes). Continuous crying, limp muscle tone, cold extremities, and a poor suckle reflex indicate problems (Figure 74.18).

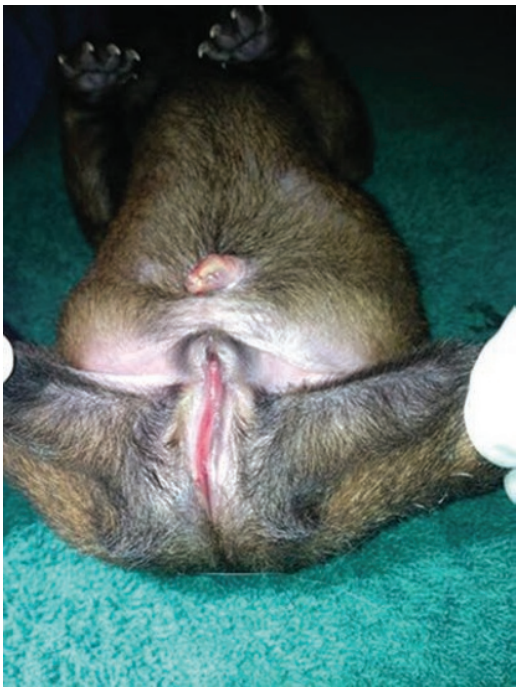


Figure 74.17 Anogenital malformation.



Figure 74.18 Fading neonate; hypothermic, hypovolemic, lethargic.

Neonatal Diagnostics

Only the minimum amount of blood to accomplish the pertinent tests required in the critically sick neonate is drawn (Box 74.3). Typically, packed cell volume/total solids, blood glucose, blood urea nitrogen and a blood smear provide a minimal data base. A complete blood count, chemistry panel, urinalysis, and coagulation testing are reserved for cases requiring more diagnostics. Normal neonatal laboratory values are not the same as for adults; appropriate reference ranges have been published (Tables 74.2–74.8) [37].

Box 74.3 Neonatal Sampling

- Blood sampling must not exceed 10% of total blood volume/day (0.075 ml/g body weight; e.g. 3.75 ml from a 500-g neonate/day):
  - Minimum volume required for testing advised.
  - Use appropriately small collection tubes containing anticoagulants (EDTA or heparin for whole-blood testing to avoid dilutional errors).
- Jugular vein or (larger neonates) cephalic vein sampling optimal; 25-gsauge needle; minimize alcohol due to cooling effect; butterfly catheter or tuberculin syringe preferable.
- Needle stick of foot pad acceptable if unable to acquire IV sample.

Necropsy of a neonate dying without obvious cause is always warranted to provide proper veterinary care of the littermates, and clients should be advised (tactfully, ahead of time) to refrigerate (not freeze) deceased neonates and present them promptly for evaluation. Postmortem evaluation at commercial laboratories can be prohibitively expensive; however, clinicians can more economically perform the necropsy and submit pertinent tissues for histopathology (with or without culture, serology, or polymerase chain reaction, PCR). If an obvious cause for death is not seen grossly (i.e. crushed or suffocated neonate, obvious anatomic defect) tissues should always be evaluated microscopically to avoid false conclusions. For example, the gross lesions of bacterial septicemia mimic canine herpes and can be misleading. Ideally samples of lung, thymus, umbilicus, kidney, and liver should be submitted, in addition to any other suspicious tissues.

Early Neonatal Problems

Neonatal mortality occurs most commonly in the first seven days of life. The most common and significant acquired neonatal problems are hypoxia (addressed previously during resuscitation), hypothermia and hypoglycemia. Neonatal dogs and cats lack thermoregulatory mechanisms until four weeks of age, so the ambient temperature must be warm enough to facilitate maintenance of a body temperature of at least 97°F (36°C), this is most critical with orphans lacking maternal companionship or small litter sizes (Box 74.4). Hypothermia negatively impacts immunity, nursing, heart rate, and digestion.

Post-resuscitation exogenous heat should be supplied best in the form of an adjustable overhead heat lamp. Heating pads and warm air blankets run the risk of burning or overheating neonates which are incapable of moving away from excessively hot surfaces. The heat source should be focused on one section of the nest box, enabling the dam to move away if too warm (Figure 74.19). Unlike resuscitation warming, chilled older neonates must be rewarmed slowly (30–60 minutes) to avoid peripheral

Table 74.2 Puppy biochemical parameters from birth to approximately eight weeks of age.

	Weeks 1–2		Weeks 4–5	Weeks 7–8
Biochemical parameter	**Days 1–3**	**Days 8–10**	**Days 28–33**	**Days 50–58**
Albumin (g/dl)	1.76–2.75	1.71–2.5	2.17–2.97	2.38–3.22
Alkaline phosphatase (iu/l)	452–6358	195–768	153–490	153–527
Alanine transaminase (iu/l)	9.1–42.2	4.1–21.4	4.3–17.4	10.3–24.3
Bilirubin (mg/dl)	0.04–0.38	0.01–0.18	0.02–0.15	0.01–0.11
Blood urea nitrogen (mg/dl)	29.5–118	29.1–66.7	13.1–46.2	16.8–61.4
Calcium (mg/dl)	10.4–13.6	11.2–13.2	10.4–13.2	10.8–12.8
Cholesterol (mg/dl)	90–234	158–340	177–392	149–347
Creatinine (mg/dl)	0.37–1.06	0.28–0.42	0.25–0.83	0.26–0.66
Gamma-glutamyltransferase (iu/l)	163–3558	–	–	–
Glutamate dehydrogenase (iu/l)	1.8–17.0	0.2–17.7	1.2–9.0	1.6–7.3
Glucose (mg/dl)	76–155	101–161	121–158	122–159
Total protein (g/dl)	3.7–5.77	3.26–4.37	3.71–4.81	4.04–5.33
Triglycerides (mg/dl)	45–248	52–220	36–149	39–120
Phosphorus (mg/dl)	5.26–10.83	8.35–11.14	8.66–11.45	8.35–11.14

Source: Adapted from Center et al. [22]; Kuhl et al. [23]; Harper et al. [24].

Table 74.3 Puppy biochemical parameters up to 12 months of age.

Biochemical parameter	2–3 months	4–6 months	7–12 months
Albumin (g/dl)[a]	2.6–3.7	2.6–3.7	2.6–3.7
Alkaline phosphatase (iu/l)	88–532	126–438	4–252
Alanine transaminase (iu/l)	≤29	≤32	5–45
Amylase (iu/l)[a]	≤1683	≤1683	≤1683
Aspartate aminotransferase (iu/l)	7–19	3–23	2–26
Bilirubin (mg/dl)	0.01–0.13	0.01–0.13	≤0.3
Blood urea nitrogen (mg/dl)[a]	9.8–37.3	9.8–37.3	9.8–37.3
Calcium (mg/dl)	10.4–13.6	10–13.2	10.4–12
Chloride (mEq/l)[a]	99–120	99–120	99–120
Cholesterol (mg/dl)	99.6–499.6	99.6–499.6	135–278
Creatine kinase (iu/l)	31–255	40–192	≤134
Creatinine (mg/dl)	0.39–0.49	0.27–0.88	0.21–0.89
Gamma-glutamyl transferase (iu/l)	≤6.2	≤4.3	≤3.2
Globulins (g/dl)	1.9–2.5	2.2–3.5	2.2–4.5
Glucose (mg/dl)	97.1–166.2	97.1–166.2	76–119
Glutamate dehydrogenase (iu/l)	1.6–9.6	1.9–8.7	1.2–8.0
Lactate dehydrogenase (iu/l)	68–290	≤442	9–269
Lipase (iu/l)	≤241	≤139	≤154
Magnesium (mEq/l)[a]	1.4–5.2	1.4–5.2	1.4–5.2
Phosphorus (mg/dl)	6.4–11.3	5.6–9.6	3.5–7.8
Potassium (mEq/l)	4.5–6.3	3.9–6.1	4.2–5.6
Sodium (mEq/l)	140–156	139–159	138–158
Total protein (g/dl)	4.3–5.8	4.5–7.3	4.9–6.7
Triglycerides (mg/dl)	19.1–205.5	19.1–205.5	40–169
Trypsin-like immunoreactivity (µg/l)	5–35	–	–

[a] Parameters for which significant age variation was not found in puppies.
Source: Adapted from the following sources: Harper et al. [24]; Kley et al. [25]; Kraft et al. [26]; Kraft et al. [27]; Laroute et al. [28]; Vajdovich et al. [29].

Table 74.4 Kitten biochemical parameters from birth to eight weeks of age.

	Week 1			Week 4	Week 8
Biochemical parameter	Day 0	Day 1	Day 7	Day 28	Day 56
Albumin (g/dl)	2.5–3.0	1.9–2.7	2.0–2.5	2.4–4.9	2.4–3.0
Alkaline phosphatase (iu/l)	184–538	1348–3715	126–363	97–274	60–161
Alanine transaminase (iu/l)	7–42	29–77	11–76	14–55	12–56
Amylase (iu/l)	310–837	310–659	187–438	275–677	407–856
Aspartate aminotransferase (iu/l)	21–126	75–263	15–45	15–31	14–40
Bilirubin (mg/dl)	0.1–1.1	0.1–1.6	0.0–0.6	0.0–0.3	0.0–0.1
Blood urea nitrogen (mg/dl)	26–45	34–94	16–36	10–22	16–33
Calcium (mg/dl)	9.4–13.9	9.6–12.2	10.0–13.7	10.0–12.2	9.8–11.7
Cholesterol (mg/dl)	65–141	48–212	119–213	173–253	124–221
Creatine kinase (iu/l)	91–2300	519–2654	107–445	125–592	102–1512

Table 74.4 (Continued)

	Week 1			Week 4	Week 8
Biochemical parameter	**Day 0**	**Day 1**	**Day 7**	**Day 28**	**Day 56**
Creatinine (mg/dl)	1.2–3.1	0.6–1.2	0.3–0.7	0.4–0.7	0.6–1.2
Gamma-glutamyl transferase (iu/l)	0–2	0–9	0–5	0–1	0–2
Glucose (mg/dl)	55–290	65–149	105–145	117–152	94–143
Lactate dehydrogenase (iu/l)	176–1525	302–1309	117–513	98–410	62–862
Lipase (iu/l)	12–43	21–131	8–46	4–86	6–70
Phosphorus (mg/dl)	5.9–11.2	4.9–8.9	6.7–11.0	6.7–9.0	7.6–11.7
Total protein (g/dl)	3.8–5.2	3.9–5.8	3.5–4.8	4.5–5.6	4.8–6.5
Total solids (g/dl)	3.1–4.4	3.2–5.2	3.0–4.6	4.0–6.0	4.1–6.2
Triglycerides (mg/dl)	23–132	30–644	129–963	43–721	16–170

Source: Adapted from Levy et al. [30].

Table 74.5 Kitten biochemical parameters up to 12 months of age.

Biochemical parameter	**< 3 Months**	**4–6 Months**	**7–12 Months**
Alkaline phosphatase (iu/l)	≤564	37–333	21–197
Alanine transaminase (iu/l)	10–50	≤77	≤85
Amylase (iu/l)[f]	≤1800	1800	≤1800 (≤2200 Oriental breeds)
Aspartate aminotransferase (iu/l)	≤20	≤30	≤30 (≤40 Oriental breeds)
Bilirubin (mg/dl)[a]	≤4	≤4	≤4
Blood urea nitrogen (mg/dl)[b]	17–35	17–35	17–35
Calcium (mg/dl)[f]	9.2–12.0	9.2–12.0	9.2–12.0
Chloride (mEq/l)	97–125	102–122	104–124
Creatinine (mg/dl)	0.16–1.26	0.33–1.21	–[c]
Creatine kinase (iu/l)	≤188	≤160	≤128
Gamma-glutamyl transferase (iu/l)[f]	≤4	≤4	≤4
Glutamate dehydrogenase (iu/l)[f]	≤7	≤7	≤7 (≤16 Oriental breeds)
Glucose (mg/dl)[b]	70–150	70–150	70–150
Lactate dehydrogenase (iu/l)	68–280	≤442	9–269
Lipase (iu/l)	≤280	≤280	≤280
Magnesium (mEq/l)*	1.2–5.2	1.2–5.2	1.2–5.2
Potassium (mEq/l)	3.7–6.1	4.2–5.8	3.7–5.3
Phosphorus (mg/dl)	6.5–10.1	6–10.4	4.5–8.5
Sodium (mEq/l)*	143–160	143–160	143–160
Total protein (g/dl)[d]	–	3.3–7.5	3.3–7.5
TLI (µg/l)	17–49[e]		

Source: Adapted from Kraft et al. [26, 27, 31].
[a] Adult values reached after 1 week of age.
[b] Adult values reached after 8 weeks of age.
[c] Reference ranges have not been reported for kittens over 8 months of age; 0.8–2.3 mg/dl (adult).
[d] Adult levels are reached between 8 months and 8 year of age.
[e] Data from Steiner [32].
[f] Parameters for which significant age variation has not been found in kittens.

Table 74.6 Hematologic values for growing, healthy Beagle dogs.

Hematologic parameter	Age (in weeks)															
	Birth[b]	1[b]	2[b]	3[b]	4[b]	6[b]	8	12[a]	16[a]	20[a]	24[a]	28[a]	40[a]	44[a]	52[a]
RBC ($\times 10^6$/μl)	4.7–5.6 (5.1)	3.6–5.9 (4.6)	3.4–4.4 (3.9)	3.5–4.3 (3.8)	3.6–4.9 (4.1)	4.3–5.1 (4.7)	4.5–5.9 (4.9)	(6.34)	(6.38)	(6.93)	(7.41)	(8.45)	(8.69)	(8.47)	(7.68)
Hemoglobin (g/dl)	14.0–17.0 (15.2)	10.4–17.5 (12.9)	9.0–11.0 (10.0)	8.6–11.6 (9.7)	8.5–10.3 (9.5)	8.5–11.3 (10.2)	10.3–12.5 (11.2)	(14.3)	(15.0)	(16.0)	(16.7)	(17.7)	(18.2)	(18.8)	(18.1)
PCV (%)	45.0–52.5 (47.5)	33.0–52.0 (40.5)	29.0–34.0 (31.8)	27.0–37.0 (31.7)	27.0–33.5 (29.9)	26.5–35.5 (32.5)	31.0–39.0 (34.8)	(40.9)	(43.0)	(44.9)	(47.6)	(48.8)	(50.8)	(50.2)	(49.3)
MCV (fl)	(93.0)	(89.0)	(81.5)	(83.0)	(73.0)	(69.0)	(72.0)	(64.6)	(67.4)	(64.8)	(64.2)	(57.8)	(58.4)	(59.3)	(63.5)
MCH (pg)	(30.0)	(28.0)	(25.5)	(25.0)	(23.0)	(22.0)	(22.5)	(22.8)	(23.5)	(23.0)	(22.5)	(20.5)	(20.9)	(22.1)	(23.6)
MCHC (%)	(32.0)	(32.0)	(31.5)	(31.0)	(32.0)	(31.5)	(32.0)	(35.3)	(34.8)	(35.6)	(35.1)	(36.1)	(35.9)	(37.3)	(37.1)
nRBC/100 WBC	0–13 (2.3)	0–11 (4.0)	0–6 (2.0)	0–9 (1.6)	0–4 (1.2)	0	0–1 (0.2)								
Reticulocytes (%)	4.5–9.2 (6.5)	3.8–15.2 (6.9)	4.0–8.4 (6.7)	5.0–9.0 (6.9)	4.6–6.6 (5.8)	2.6–6.2 (4.5)	1.0–6.0 (3.6)								
Total WBC ($\times 10^3$/μl)	6.8–18.4 (12.0)	9.0–23.0 (14.1)	8.1–15.1 (11.7)	6.7–15.1 (11.2)	8.5–16.4 (12.9)	12.6–26.7 (16.3)	12.7–17.3 (15.0)	(17.1)	(16.3)	(14.6)	(15.6)	(15.5)	(14.4)	(13.9)	(14.0)
Segmented neutrophils	4.4–15.8 (8.6)	3.8–15.2 (7.4)	3.2–10.4 (5.2)	1.4–9.4 (5.1)	3.7–12.8 (7.2)	4.2–17.6 (9.0)	6.2–11.8 (8.5)	(9.8)	(9.0)	(8.9)	(9.1)	(9.1)	(9.9)	(8.7)	(8.1)
Band neutrophils	0–1.5 (0.23)	0–4.8 (0.50)	0–1.2 (0.21)	0–0.5 (0.09)	0–0.3 (0.06)	0–0.3 (0.05)	0–0.3 (0.08)	(0.08)	(0.1)	(0.02)	(0.02)	(0.08)	(0.02)	(0.02)	(0.04)
Lymphocytes	0.5–4.2 (1.9)	1.3–9.4 (4.3)	1.5–7.4 (3.8)	2.1–10.1 (5.0)	1.0–8.4 (4.5)	2.8–16.6 (5.7)	3.1–6.9 (5.0)	(5.7)	(5.9)	(4.5)	(5.3)	(4.8)	(3.4)	(4.0)	(4.7)
Monocytes	0.2–2.2 (0.9)	0.3–2.5 (1.1)	0.2–1.4 (0.7)	0.1–1.4 (0.7)	0.3–1.5 (0.8)	0.5–2.7 (1.1)	0.4–1.7 (1.0)	(0.9)	(0.9)	(0.8)	(0.7)	(0.7)	(0.5)	(0.6)	(0.5)
Eosinophils	0–1.3 (0.4)	0.2–2.8 (0.8)	0.08–1.8 (0.6)	0.07–0.9 (0.3)	0–0.7 (0.25)	0.1–1.9 (0.5)	0–1.2 (0.4)	(0.4)	(0.4)	(0.3)	(0.5)	(0.8)	(0.6)	(0.5)	(0.5)
Basophils		0–0.2 (0.01)			0–0.15 (0.01)										
Platelets ($\times 10^3$/μl)	178–465 (302)	282–560 (352)	210–352 (290)	203–370 (272)	130–360 (287)	275–570 (371)	240–435 (324)								

MCH, mean corpuscular hemoglobin; MCV, mean corpuscular volume; MCHC, mean corpuscular hemoglobin concentration; nRBC/100 WBC, number of nucleated red blood cells per 100 white blood cells; PCV, packed cell volume; RBC, Red blood cells; total WBC, total white blood cell count. Values in parentheses are mean values.

[a] Mean values from Anderson and Gee [33].

[b] Normal ranges and/or mean values from Earl et al. [34].

Table 74.7 Hematologic values for growing, healthy cats.

	Age (in weeks)										
Hematologic parameter	**0–2[b]**	**2–4[b]**	**4–6[b]**	**6–8[b]**	**8–9[b]**	**12–13[b]**	**16–17[b]**	**20[a]**	**30[a]**	**44[a]**	**52[a]**
RBC ($\times 10^6$/μl)	5.29 ± 0.24	4.67 ± 0.10	5.89 ± 0.23	6.57 ± 0.26	6.95 ± 0.09	7.43 ± 0.23	8.14 ± 0.27	7.4 ± 0.7	8.0 ± 0.5	7.9 ± 0.8	7.7 ± 0.8
Hemoglobin (g/dl)	12.1 ± 0.6	8.7 ± 0.2	8.6 ± 0.3	9.1 ± 0.3	9.8 ± 0.2	10.1 ± 0.3	11.0 ± 0.4	10.7 ± 1.2	12.1 ± 1.8	13.0 ± 2.1	13.3 ± 1.8
PCV (%)	35.3 ± 1.7	26.5 ± 0.8	27.1 ± 0.8	29.8 ± 1.3	33.3 ± 0.7	33.1 ± 1.6	34.9 ± 1.1	33.4 ± 3.3	37.1 ± 3.4	37.3 ± 3.5	36.6 ± 3.6
MCV (fl)	67.4 ± 1.9	53.9 ± 1.2	45.6 ± 1.3	45.6 ± 1.0	47.8 ± 0.9	44.5 ± 1.8	43.1 ± 1.5	45 ± 5.2	46 ± 3.5	47 ± 3.4	47 ± 3.9
MCH (pg)	23.0 ± 0.6	18.8 ± 0.8	14.8 ± 0.6	13.9 ± 0.3	14.1 ± 0.2	13.7 ± 0.4	13.5 ± 0.4				
MCHC (%)	34.5 ± 0.8	33.0 ± 0.5	31.9 ± 0.6	30.9 ± 0.5	29.5 ± 0.4	31.3 ± 0.9	31.6 ± 0.8	32 ± 2.0	33 ± 3.3	34 ± 3.0	36 ± 3.1
Total WBC ($\times 10^3$/μl)	9.67 ± 0.57	15.31 ± 1.21	17.45 ± 1.37	18.07 ± 1.94	23.68 ± 1.89	23.20 ± 3.36	19.70 ± 1.12	15.9 ± 6.0	21.9 ± 6.8	18.3 ± 7.8	24.0 ± 12.5
Segmented neutrophils	5.96 ± 0.68	6.92 ± 0.77	9.57 ± 1.65	6.75 ± 1.03	11.00 ± 1.41	11.00 ± 1.77	9.74 ± 0.92				
Band neutrophils	0.06 ± 0.02	0.11 ± 0.04	0.20 ± 0.06	0.22 ± 0.08	0.12 ± 0.09	0.15 ± 0.07	0.16 ± 0.07				
Lymphocytes	3.73 ± 0.52	6.56 ± 0.59	6.41 ± 0.77	9.59 ± 1.57	10.17 ± 1.71	10.46 ± 2.61	8.78 ± 1.06	6.2 ± 2.1	5.3 ± 1.2	6.1 ± 2.0	5.5 ± 2.7
Monocytes	0.01 ± 0.01	0.02 ± 0.02	0	0.01 ± 0.01	0.11 ± 0.06	0	0.02 ± 0.02				
Eosinophils	0.96 ± 0.43	1.40 ± 0.16	1.47 ± 0.25	1.08 ± 0.20	2.28 ± 0.31	1.55 ± 0.35	1.00 ± 0.19				
Basophils	0.02 ± 0.01	0	0	0.02 ± 0.02	0	0.03 ± 0.03	0				

RBC, Red blood cells; PCV, packed cell volume; MCV, mean corpuscular volume; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; nRBC/100 WBC, number of nucleated red blood cells per 100 white blood cells; total WBC, total white blood cell count. Values in parentheses are mean values.

[a] Normal ranges from Anderson et al. [35].

[b] Normal ranges ± one standard deviation from Meyers-Wallen et al. [36].

Table 74.8 Regenerative response in puppies and kittens.

	Puppies		Kittens	
PCV (%)	**Polychromasia**	**Reticulocytes[a]**	**Polychromasia**	**Reticulocytes[a]**
>25	1–2+	>80 000/µl	1–2+	>60 000/µl
15–25	2–3+		2–3+	
<15	4+		4+	

[a] These values represent the minimum absolute numbers of reticulocytes required for an interpretation of a regenerative response. If the PCV is lower, the absolute number of reticulocytes should increase proportionately.

Box 74.4 Normal Rectal Temperature of Neonates, First Four Weeks of Life, and Associated Ambient Temperature Range

- Neonatal normal body temperature (rectal):
  - Week 1: 95–99°F (35–37°C)
  - Weeks 2–3: 97–100°F (36–37°C)
  - At weaning 99–101°F (37–38°C)
- Environmental warmth required (ambient air temperature in room):
  - Week 1: 84 – 89°F (28 – 31°C)
  - Weeks 2 – 3: 80°F (26°C)
  - Week 4: 69 – 75°F (20 – 23°C)
  - Week 5: 69°F (20°C)

Note: Presence of dam and littermates improves thermoregulation in the litter. Overheating can be problematic for both the dam and litter. Heating elements in or above the nest box, which should only cover a portion of the box, will reduce the need for ambient (room) heat. Euthermic puppies may pile up, may spread out, and sleep/nurse/sleep. Chilled neonates will pile up, may vocalize, and may eventually become lethargic. Overheated littermates will spread out, will be restless and cry, and will have very pink/red mucus membranes, and the dam may pant excessively or even leave the box.

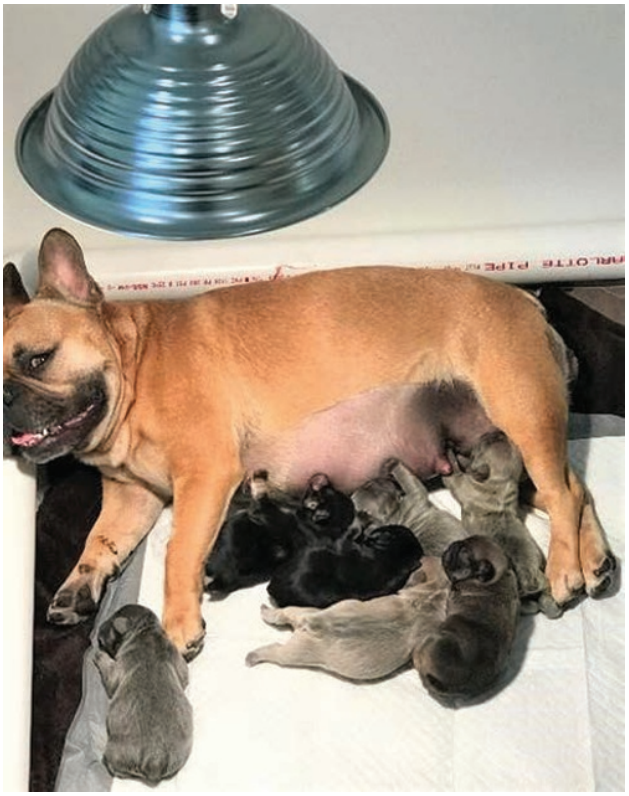


Figure 74.19 Early sign of overheating of a whelping box using an overhead heat lamp; the bitch is panting and appears anxious.

vasodilation and dehydration. Tube feeding neonates should be delayed until the neonate is euthermic; hypothermia induces ileus, and regurgitation and aspiration can result. Appropriate, cautious thermal support should continue through four weeks of life. Overheating is problematic as neonates cannot pant and have poor peripheral vasodilation capabilities, of concern when transported in a small box.

Neonates have minimal body fat reserves and limited metabolic capacity to generate glucose from precursors. Glycogen stores are depleted shortly after birth, making adequate nourishment from nursing vital. Even minimal fasting can result in hypoglycemia. Hypoglycemia can also result from endotoxemia, septicemia, portosystemic shunts, and glycogen storage abnormalities. Rapid blood glucose assessment with a drop of blood obtained by a digital pad prick is indicated if hypoglycemia is suspected; IV or IO glucose therapy should follow (Figure 74.20; Protocol 74.3). Oral fluid and glucose replacement may be preferable to parenteral if the neonate has an adequate swallowing reflex and is not clinically compromised. In one study, median glucose concentration at day 1 in puppies subsequently dying was 88 mg/dl (56–128 mg/dl) compared with 120 mg/dl (96–149 mg/dl) in puppies still alive at day 21 [20].

The neonatal caloric requirement is 0.133 calories/g/day during the first week of life, 0.155 cal/g/day for the



Figure 74.20 A quick blood glucose measurement can be acquired using a glucometer and one drop of blood acquired from a pad prick.

Protocol 74.3 Hypoglycemia Therapy

1) Warm fluids to neonatal body temperature.
2) Acute hypoglycemia (blood glucose < 80 mg/dl):
   a) Optimal: IV or IO bolus 2.5–5.0% (25–50 mg/ml) dextrose solution at 0.001 ml/g neonate weight (e.g. 500 g neonate gets 0.5 ml).
   b) Alternative: balanced crystalloid (half-strength saline) and 2.5% dextrose may be administered by gavage (0.25–1 ml).
   c) Suboptimal: 1 drop 50% dextrose solution applied to mucous membranes.
3) Recheck blood glucose approximately hourly, and repeat as necessary.
4) Encourage nursing; consider stomach tubing with colostrum/artificial milk.
5) Avoid hyperglycemia/osmotic diuresis and rebound hypoglycemia can occur.

second, 0.175–0.198 calories/g/day for the third, and 0.220 calories/g/day for the fourth [16]. Bottle or intermittent orogastric tube feeding is indicated for neonates who fail to gain weight or nurse effectively until weaning (Figure 74.21). Premature low flow human baby bottles are superior to commercial veterinary bottles in puppies; syringes modified with a nipple or commercial feline neonatal bottles are preferred in kittens. The neonate should be in a normal feeding position (ventral recumbency with slight elevation of the head) while suckling a bottle or receiving gavage (Figure 74.22).

Normal neonatal weight gain is an increase of 5–10% body weight/day. Commercially manufactured milk replacement formulas are usually superior to homemade versions (high lactose), but none is equivalent to the dam's milk, which contains high levels of lipids [38]. The use of milk obtained from the dam or another lactating bitch should be considered and is superior if available. Most replacement formulas lack appropriate calcium, amino acids, and essential fatty acids. An osmotic diarrhea (usually yellow, curdled fecal appearance) can result from overfeeding formula; constipation can also occur necessitating diluting the product 25–50% with water or a balanced crystalloid solution. Neonates should gain weight steadily from the first day after birth (a transient mild loss from birth weight is acceptable on day 1), with puppies gaining 1–3 g/day/2.2 kg of anticipated adult weight and kittens 50–100 g/week. Neonatal weights should be recorded at least daily for the first two weeks, then every one to three days until one month of age. The limited size of neonatal stomach (reportedly 4–5 ml/100 g), and propensity for regurgitation/aspiration if overdistension from overfeeding occurs, necessitates multiple small feedings around the clock [16]. Most commercial milk replacers deliver approximately 1.0 kcal/ml; smaller more frequent volumes fed are preferable (50–80% stomach capacity). One study reported superior thermoregulation and weight gain in neonates supplemented arbitrarily with a maltodextrin enriched milk replacer during the first 48 hours of life [39]. Kittens and puppies under three weeks of age lack voluntary elimination and must have the micturition and defecation reflexes stimulated multiple times daily using a cotton ball with lubrication on the anogenital area if the dam fails to groom the neonate sufficiently. Constipation can result in vomiting, hypoglycemia, and dehydration.

Hypovolemia and dehydration are potential life-threatening conditions in neonates, most commonly resulting from decreased fluid intake, vomiting, and diarrhea. The immature autonomic nervous system results in an altered response to shock; tachycardia is already present and neonatal mean arterial pressure low. Skin turgor and urine specific gravity in neonates are not indicative of hydration status due to their increased body water content/plasma volume and immature/limited glomerular filtration rate. Fluid (warmed to body temperature) replacement via IV or IO administration is preferred (Box 74.5). Intraperitoneal (IP) administration of fluids is an alternative but can be painful and slowly



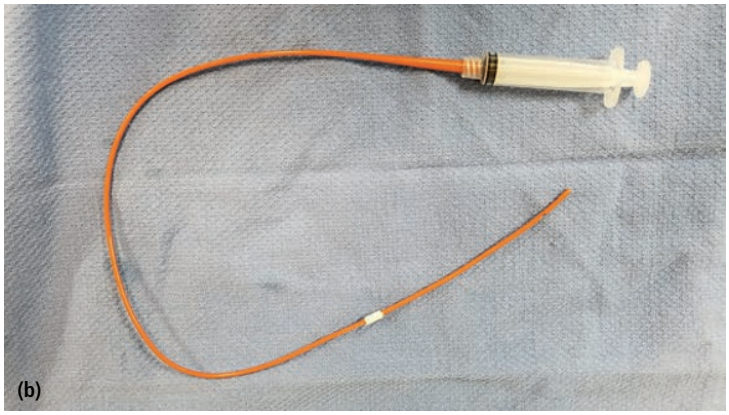




Figure 74.21 (a) Measuring the distance from the mouth to the stomach, just caudal to the last rib. (b) Marked large-diameter orogastric tube. (c) Orogastric feeding with a smaller-diameter tube with which inadvertent placement in the airway can occur more easily.

absorbed; the subcutaneous (SC) route is appropriate only with mild dehydration.

Neonatal Immunodeficiency

Canine and feline neonates are immunodeficient at birth. Incompletely developed immune systems during the first 10 days of life make neonates vulnerable to systemic infection (most commonly bacterial and viral). Adequate ingestion of colostrum must occur promptly postpartum for neonates to acquire passive immunity, as transplacental transfer is less than 5%. The intestinal absorption of immunoglobulin G generally ceases by 24 hours after parturition. Colostrum-deprived canine neonates should be given 10–20 ml/kg (0.01–0.02 ml/g) of serum from an immunocompetent adult dog to achieve adequate immunoglobulin levels. Kittens should receive 15 ml/kg (0.015 ml/g).

Blood typing is important for cats. The serum can be given orally if within the first 24 hours of life, otherwise it must be given parenterally, preferably subcutaneously, divided into reasonably small volumes with both routes [40, 41].

Fading Neonates

Neonatal bacterial septicemia can cause rapid deterioration resulting in death if not recognized and treated promptly. Factors that reportedly predispose a neonate to septicemia include endometritis in the dam, prolonged delivery/dystocia, feeding of replacement formulas (likely a comorbidity), use of ampicillin, stress, low birth weight (< 350 g for a medium-size breed of puppy, < 100 g for a kitten), and chilling (body temperature < 96° F; Figure 74.23).

The organisms most frequently associated with septicemia are *Escherichia coli,* streptococci, staphylococci, and *Klebsiella* spp. Umbilical contamination is the most likely route of entry. Omphalitis leads to peritonitis, bacteremia, and pneumonia. Abscessation can occur later at other sites (e.g. neonatal ophthalmia; Figure 74.24). Premortem diagnosis can be challenging; clinical signs may not be noted owing to sudden death. Commonly, a decrease in weight gain, failure to suckle, hematuria, persistent diarrhea, unusual vocalization, abdominal distention, and pain, and sloughing of the extremities indicate septicemia may be present. Prompt therapy with broad-spectrum bactericidal antibiotics, improved nutrition via supported nursing, tube or bottle feeding, maintenance of







Figure 74.22 Bottle feeding. (a) Canine neonate. (b) Feline neonate. (c) Proper position for feeding via gavage. *Source:* Courtesy of Dr. P. Dedrick.

Box 74.5 Intraosseous/intravenous Fluid Guidelines

- Warm fluids to neonatal body temperature.
- Ideal: IV or IO administration balanced crystalloid shock dose (puppy 0.02–0.04 ml/g body weight; kitten 0.02–0.03 ml/g body weight) bolus (e.g. 500 g neonate receives 10–20 ml). IO requires very slow infusion rate.
- Suboptimal: IP administration balanced crystalloid (avoid hypertonic solutions).
- Less optimal: SC administration balanced crystalloid (avoid hypertonic solutions).
- Maintenance fluid rate 1.2–1.8 ml/g neonate weight/day as multiple boluses or continuous infusion.

body temperature, and appropriate fluid replacement are indicated. The third-generation cephalosporin antibiotic ceftiofur sodium is an appropriate choice for neonatal septicemia (Box 74.1). Ceftiofur minimally alters normal intestinal flora and is usually effective against the causative organisms. Ceftiofur sodium should be administered at a dose of 0.0025 mg/g SC every 12 hours for no longer than five days. Because neonates less than 48 hours old have reduced thrombin levels, presumptive therapy with vitamin K_1 may be used (0.01–1 mg/neonate SC). The prognosis is guarded but not hopeless.

Incompatible blood types of the parental cats can cause feline neonatal mortality. This incompatibility arises when queens with type B blood give birth to kittens who inherited the sire's blood type A. Type B is rare in mixed breed and Siamese cats but is much more common in certain



Figure 74.23 Low-birthweight puppy with littermates.



Figure 74.24 Neonatal ophthalmia secondary to bacteremia.

purebreds such as British Shorthair and Devon Rex cats. Type B cats have naturally occurring anti-A antibodies. When the kittens nurse and absorb these antibodies, the kittens' own red blood cells are hemolyzed, leading to anemia and organ failure. The clinical course is determined by the severity of the hemolytic reaction. In all cases, the kittens are born healthy and nurse vigorously. Some kittens may die suddenly in the first day, while others linger longer and fade during the first week of life. Clinical neonatal isoerythrolysis is suggested by pale mucous membranes and dark red brown urine and is confirmed by documenting hemolytic anemia and the blood types of the parents. If neonatal isoerythrolysis is suspected, the kittens are removed from the queen and fostered on a type A queen or bottlefed for the first two days of life. Neonatal isoerythrolysis can be avoided in catteries by testing the blood types of breeding animals and avoiding matings of type B queens with type A toms.

Canine herpes virus (CHV) is a commonly blamed cause for fading puppy syndrome resulting in neonatal death. Premortem diagnosis of CHV infection in neonates can be challenging. Postmortem diagnostics include appropriate histopathology, virus isolation, or PCR. Pathognomonic changes occurring in the kidneys include multifocal petechial hemorrhages, although this can be seen with bacterial septicemia and associated thromboembolic disorders as well (Figure 74.25). Intranuclear inclusion bodies can be difficult to find. Diagnosis by virus isolation or CHV-specific PCR is confirmatory and desirable, especially before litter mortality reaches 100%.

Until recently, treatment of CHV infection in neonates has been reported to be unrewarding and rare, with recovery suspected to be associated with residual cardiac and neurologic damage. Treatment with immune serum from affected dams is reported to be ineffective in infected puppies. Successful treatment with the antiviral agent acyclovir has been reported [42]. Acyclovir is an antiviral agent with activity against a variety of viruses including herpes simplex. Acyclovir is preferentially taken up by susceptible viruses and converted into the active triphosphate form, inhibiting viral DNA replication. It is poorly absorbed after oral administration and is primarily hepatically metabolized, and can



Figure 74.25 Pinpoint hemorrhagic renal capsular foci in a confirmed canine perinatal herpes infection.

increase the toxicity of nephrotoxic drugs. The half-life in humans is approximately three hours. Its use in veterinary medicine is not well established, and it should be used with caution and only in situations where indicated. The safety and effectiveness in humans younger than two weeks of age is not established. The dose was extrapolated from that for humans (20 mg/kg orally every six hours for seven days).

Congenital and Acquired Abnormalities

Cardiovascular

Innocent (flow) murmurs are transient and are not pathologic. The ductus arteriosus normally closes by five days after birth. Continuous murmurs and murmurs that persist beyond two to three days of life are abnormal. The most common congenital heart diseases are subaortic stenosis and patent ductus arteriosus in dogs, and tricuspid valve dysplasia and ventricular septal defect in cats [43]. Cardiac defects can arise from environmental stress, infection, or intoxication of the dam, but the heritability of many has been documented. Clinical signs are often not apparent until after weaning, although affected neonates may not be as vigorous as normal littermates. Accurate cardiac auscultation of subtle, persistent murmurs is best after four to six weeks of age; echocardiography is the preferred mode of evaluation once adequate pediatric size is attained, usually at three to four months of age.

Respiratory

Primary ciliary dyskinesia, the rare immotile cilia syndrome, should be suspected in neonates exhibiting persistent or recurrent mucopurulent nasal discharge, coughing, and abnormal breath sounds without other demonstrable cause. The presence of a cleft palate, persistent right aortic arch, and congenital megaesophagus should be ruled out (Figure 74.26). Abnormal mucociliary transport and neutrophil function result in chronic rhinitis, tracheobronchitis, and bronchopneumonia. Situs inversus can also be present. The long-term prognosis, even with supportive therapy, is poor. Neonatal dyspnea with evidence of gastrointestinal disorders can occur secondary to congenital peritoneopericardial or pleuroperitoneal diaphragmatic hernias. Thoracic wall abnormalities, such as pectus excavatum, a sternal intrusion into the thorax, can cause dyspnea and is variably associated with poor growth. Surgical correction of thoracic wall abnormalities is sometimes possible once adequate body size is attained. Reports of a neonatal respiratory distress syndrome secondary to a deficiency of surfactant exist, and affected neonates died within five days of age. Surfactant deficiency secondary to prematurity is predictably problematic. Proper timing of elective cesarean sections is vital to avoid premature delivery of surfactant deficient neonates. When ovulation timing is incomplete, determination of term pregnancy can be attempted by examination of fetal dental mineralization on radiographs (Figure 74.27) and ultrasound presence of gastrointestinal motility [44].



Figure 74.26 Lateral positive contrast image illustrating congenital megaesophagus. Non-ionic, water-soluble iodine contrast media is advised in neonates.



Figure 74.27 Abdominal radiograph of term pregnancy illustrating mineralized fetal dentition (arrowhead).

Gastrointestinal

Examination of the neonatal oronasal/pharyngeal cavity should always be carefully performed. Congenital palate defects occur in dogs with an incidence of up to 25%, less commonly in kittens A secondary cleft palate is a congenital oronasal fistula resulting in incomplete closure of the

hard and soft palates (Figure 74.28). Secondary cleft palate occurs alone or in combination with primary cleft palate, which involves the lip and premaxilla (Figure 74.29). Cleft palate results from incomplete fusion of the palatine shelves, most critical at 25–28 days of gestation, attributed to genetic (recessive or incompletely dominant polygenic inheritance), teratogenic (drugs, supplements), nutritional (folic acid deficiency), or infectious (viral) factors.

Affected neonates are diagnosed by visual inspection of the face and oral cavity. Ineffective nursing/suckling results; these neonates fail to thrive, developing aspiration pneumonia and rhinitis. Feeding by orogastric tube is indicated until the neonate reaches a size permitting oral surgery, traditionally advised at 8–12 weeks of age. Palatoplasty in such young puppies is difficult due to patient size and anticipated postoperative orofacial growth, which often requires multiple surgeries. Esophagostomy or gastrostomy tube placement can facilitate feeding over time but requires significant client commitment and can still result in aspiration (Figure 74.30). Palatal prostheses are problematic in dogs. A successful method to manage nutrition in mild to moderately affected dogs until approximate adult size is attained, facilitating a single surgical correction, has been reported. Feeding the dam's colostrum for 24 hours followed by either the dam's milk or artificial milk replacer by intermittent (every two to six hours) orogastric tube is instituted. At four to five weeks of age, transition to a dry (unsoaked) commercial pediatric kibble for small breeds is made. Water is made available at this time through an overhead ballpoint tube cap system (Figure 74.31). Surgery can be delayed until the



Figure 74.28 Secondary cleft palate.



Figure 74.29 Primary cleft palate with unilateral cleft lip.



Figure 74.30 Placement of an esophagoscopy tube in a neonate.



Figure 74.31 Effective drinking-water delivery to a puppy with a secondary cleft palate.

pet has achieved adequate size, or may never become necessary, as some degree of closure of the cleft occurs with time (Figure 74.32) [45].

A developmental anomaly resulting in extrusion of a portion of the gastrointestinal tract outside of the body wall, occurring within the umbilical canal (omphalocele) or lateral to the umbilical canal (gastroschisis) occurs in both dogs and cats (Figure 74.33). The condition is usually hopeless in small pediatric veterinary patients presented to the veterinarian hours after birth; however, a 30–70%

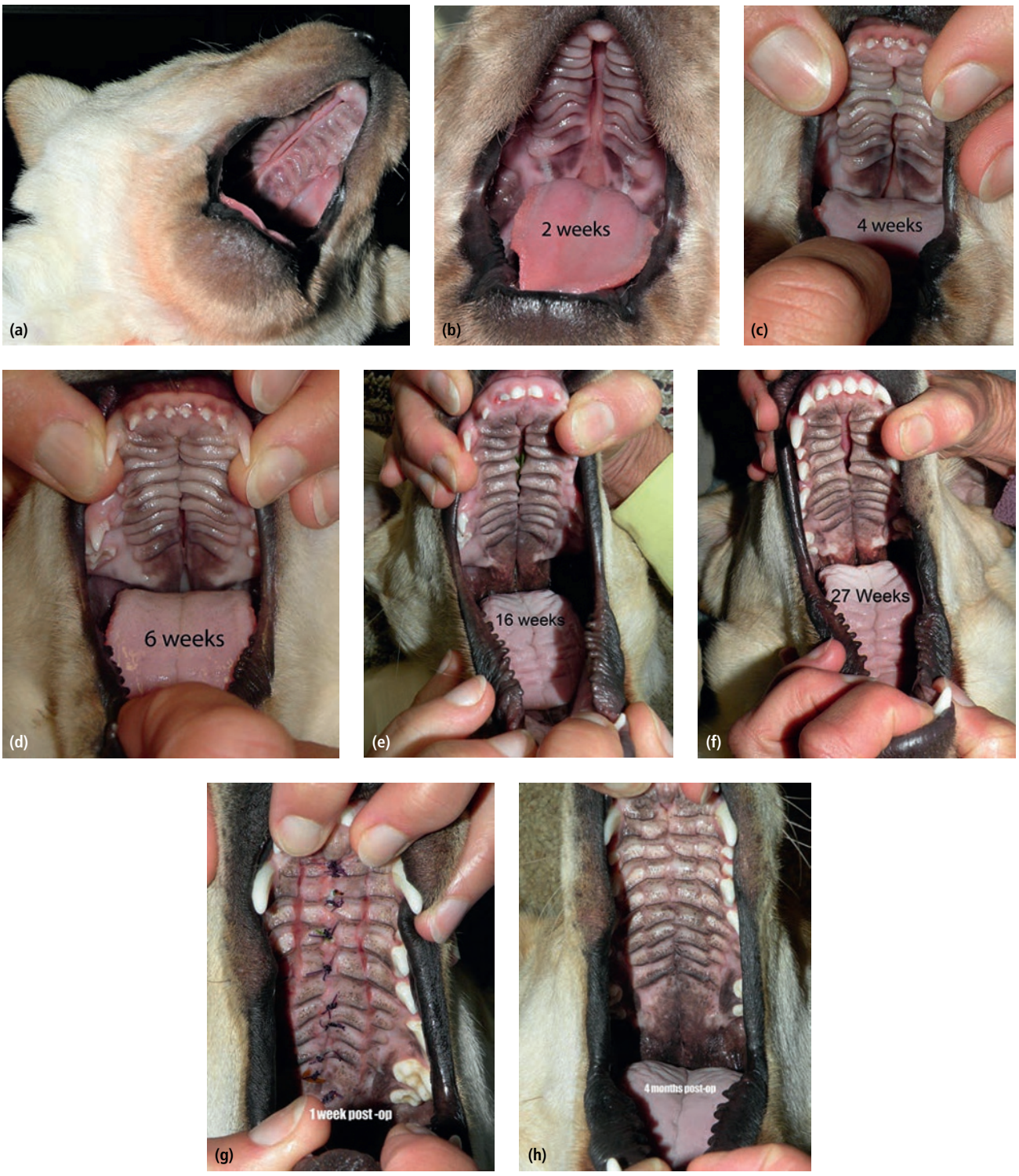


Figure 74.32 Progression of spontaneous closure of a congenital secondary cleft palate: (a) neonate; (b) 2 weeks; (c) 4 weeks; (d) 6 weeks; (e) 16 weeks; (f) 27 weeks; (g) 1-week postoperatively at 16 months; (h) 4 months postoperatively.



Figure 74.33 (a) Stillborn siblings. Omphalocele present in the smaller neonate. (b) Omphalocele.

survival rate is reported in humans with immediate postpartum surgical intervention; the diagnosis is made prepartum with abdominal ultrasound, based on the recognition of fetal gastric wall (rugal) structures or intestinal contents in an abnormal location. Earlier surgical intervention before inevitable septic contamination occurs may improve the prognosis in veterinary patients in whom the diagnosis is made in the immediate postpartum period.

Enteric duplication or agenesis can be confirmed ultrasonographically in pediatric patients. Duplication is rare, can occur anywhere in the intestinal tract and the clinical signs may be nonspecific (abdominal distension, discomfort). A fluid-filled juxtaintestinal formation with variable peristalsis and contents can be seen with ultrasound. Enteric agenesis usually results in severe, life-threatening clinical signs in the neonatal period with failure to defecate. Ultrasonographic findings usually include marked fluid and gas distention of bowel proximal to the defect.

Umbilical hernias can be significant if large enough to permit bowel evisceration and strangulation. More commonly, omentum becomes trapped in a small umbilical hernia in pediatric dogs and cats; closure of the hernia over time results in a benign omental mass misinterpreted as a hernia. Evaluation of a painful or enlarging umbilical mass with ultrasound permits differentiation of omentum from entrapped bowel, which has a typical enteric appearance. Early repair is indicated if bowel entrapment is evident. Omphalitis resulting from bacterial umbilical contamination in the postpartum period places the neonate at risk for bacterial septicemia. The umbilicus should be closed and dry 24 hours postpartum; erythema or drainage indicates antibiotics should be instituted because of the potential for peritonitis. Excessive umbilical attention by the dam can result in exposure of the subcutaneous tissues with risk of peritonitis (Figure 74.34).

Musculoskeletal

Both canine and feline neonates can be born with pelvic limb genu varum of unknown etiology; because their joints are lax, simple bandaging in a flexed position for two to

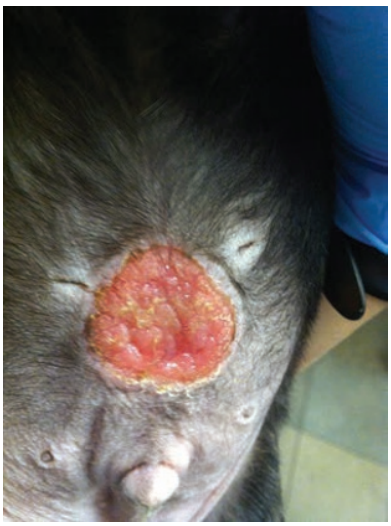


Figure 74.34 Umbilical dehiscence resulting from excessive maternal grooming.

three days combined with gentle physical therapy can resolve the condition (Figure 74.35). Puppies with noticeable flattening of the sternum at two to four weeks of age are called "swimmer puppies" by breeders. Swimmer puppies fail to develop normal ambulation at 14–21 days of life, moving instead by paddling their limbs laterally and caudally. Compression and deformation of the sternum and thorax occurs concurrently (Figure 74.36). Obese puppies from small litters raised on slippery surfaces are predisposed. Treatment should be instituted immediately upon diagnosis, consisting of caloric restriction, physical therapy, and improved traction on the floor the nest box. Placement of loose hobbles helps to control limb movements and promotes normal ambulation within days (Figure 74.36). The prognosis for swimmer puppies treated before four weeks of age is good.

Neurologic

Neonates sleep most of the time when not nursing. They respond to odors, touch and pain, all adaptive mechanisms for survival. Invasive procedures such as dewclaw removal and tail docking should be accompanied by pain management or avoided altogether. An open fontanelle is commonly present at birth in normal puppies and should close within two to five days. Congenital hydrocephalus is most commonly a breed related characteristic in brachycephalic puppies, and is a result of intrauterine exposure to toxins or infectious causes in kittens. It can be associated with a persistent open fontanelle, and eventually behavioral problems, blindness, and seizures. The diagnosis can be made with ultrasound through the open fontanelle (Figure 74.37). The prognosis is guarded; surgical



Figure 74.35 (a, b) Neonatal pelvic limb genu varum. (c) Correctional bandage in place.



Figure 74.36 (a) "Swimmer" Border Terrier puppy. (b–d) Hobbles in place in a similarly affected Labrador Retriever.

placement of a shunt and medical therapy (omeprazole, furosemide, prednisone, and acetazolamide) can allow partial remission.

Urogenital Disorders

Dysuria, urinary incontinence, or hematuria/pyuria can indicate neonatal urogenital disorders, many of which are not apparent until three to four weeks of age. The presence of a persistent patent urachus causes micturition through the umbilicus and can be diagnosed within the first week of life. Eventual surgical ligation is indicated. Cystic urachal diverticula can predispose the bladder to recurrent infection because of abnormal urine flow in the region, surgical repair is indicated usually after six to eight weeks of age; the diagnosis is confirmed with ultrasound. Renal dysplasia is a heritable problem in several canine breeds, ultrasound can identify the typical marked morphologic abnormalities in puppies at six to eight weeks of age in breeds at risk. Congenital renal polycystic disease of brachycephalic cats can similarly be identified with ultrasound in 8–12-week-old kittens. Neonatal urolithiasis, with or without associated urinary tract infection, can cause outflow obstruction and signs of acute abdominal pain. Lower urinary tract infection in neonates has the potential for ascending and causing pyelonephritis if not





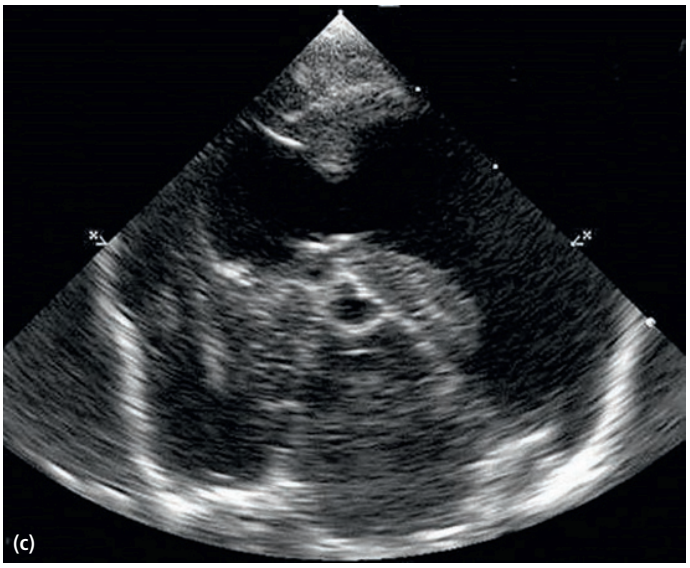


Figure 74.37 (a) Hydrocephalic puppy (left) with sibling. (b, c) Intracranial ultrasound (transfontanelle) in a hydrocephalic puppy, showing an increased amount of cerebrospinal fluid present in the ventricles. *Source:* Images courtesy of T.W. Baker.

detected and controlled. Ectopic ureters can cause incontinence during the postpartum period; clinical signs are most evident after weaning when the dam is no longer cleaning the puppy, but astute breeders may note that a puppy is leaking and may seek evaluation.

Ophthalmologic

Ocular anomalies account for 15% of all congenital defects in puppies and 9% in kittens. Eyelid agenesis occurs most commonly in kittens and is apparent at birth. The prognosis is guarded for preservation of corneal integrity and correction of the defect and blink reflex. Eyelids separate at 10–14 days of life, before this, neonatal ophthalmia can occur causing an accumulation of fluid below the lids. Bacterial infection is most likely in puppies, viral (herpes, chlamydia) in kittens. Treatment includes gentle manual separation of the lids and topical triple antibiotic ointment (erythromycin in kittens). Microphthalmos, enophthalmos, strabismus, distichiasis, and entropion become apparent after the eyelids separate.

Dermatologic

Hypotrichosis is usually regional and cosmetic (Figure 74.38). Sibling suckling secondary to the lack of adequate nursing/hunger can cause cutaneous hematomas; separation or rotation of nursing neonates may be necessary until solid food is introduced (Figure 74.39). Most infectious, parasitic, and inherited dermatopathies become apparent in older pediatric patients (e.g. demodicosis, cheyletiellosis, nutritional imbalances, ichthyosis, epidermal dysplasia, acrodermatitis, dermatomyositis).



Figure 74.38 Congenital hypotrichosis (right) with normal littermate.

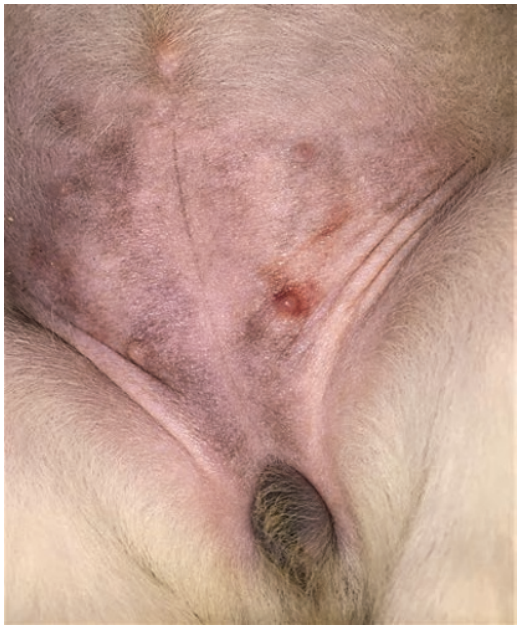


Figure 74.39 Cutaneous hematoma resulting from suckling by a littermate.



Figure 74.40 Anasarca; cesarean section was necessitated by the resultant obstructive dystocia.

Miscellaneous

Anasarca, a lethal congenital edema, can occur with or without concurrent cardiovascular abnormalities. Generalized subcutaneous edema, with intrathoracic and IP fluid accumulation, are present at birth; prenatal diagnosis with ultrasound is possible (Figure 74.40). Anasarca puppies commonly cause an obstructive dystocia. Therapy is unrewarding and euthanasia likely indicated. A breed tendency suggests it is a heritable condition (Bulldogs, Labrador Retrievers). Congenital hereditary lymphedema (French Bulldogs) results in edema of the extremities and sometimes head and is associated with morphologic lymphatic abnormalities. Mild cases can resolve with time.

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75

Safe Handling and Care of Patients Exposed to Radioactive and Anti-Neoplastic Agents

Michael S. Kent and Kristen Sears

The safe handling and care of patients exposed to either radioactive or chemotherapeutic agents is essential. These patients present a particular challenge in that they may need extensive and intensive nursing care and may have the potential to endanger the very people providing that care if proper precautions are not taken. How to best protect yourself is not always straightforward. Multiple studies have shown that improper handling of the agents and patients treated with these agents results in exposure of the nursing staff. This can carry a serious health risk for caregivers. To decrease this risk to a minimum, procedures and protocols should be established at each practice using or caring for patients exposed to these agents. It is important that each facility has knowledge of federal, state, and local regulations when developing such a plan. Exposure to these agents can occur during chemotherapy drug or radiopharmaceutical preparation or administration, or from handling an exposed or treated patient or waste from that patient. Limiting contamination of areas where drugs are stored and prepared, and the areas where patients are treated and cared for is essential to maintain a safe work environment. General radiation safety is also extremely important when using radiation to diagnose or treat patients; this includes the use of diagnostic x-ray machines. This chapter covers radiation and chemotherapy safety.

Radiation

Definitions and Terms

Radiation is energy that travels as waves or in the form of high-energy particles. Radiation is only damaging to cells if it is ionizing, meaning that it has the ability to break bonds or liberate bound electrons from molecules within cells. X-rays, beta particles, gamma rays, electrons, and photons are all different types of ionizing radiation used in veterinary medicine for diagnostic and therapeutic purposes.

The unit of absorbed dose for radiation is the Gray (Gy), which is defined as a joule/kg or the amount of energy deposited in a mass. This unit does not take into account the biological damage potential, which varies with different types of radiation. For example, 10 Gy of alpha particles or neutrons will cause more severe damage to cells than will 10 Gy of electromagnetic radiation. To help account for this variability, the term rem or Sievert is used. These units of absorbed dose take into account a factor assigned to different types of radiation based on the severity of their biological effects on humans.

The curie (Ci) and the becquerel (Bq) are units of activity for a radioactive substance. A becquerel is the SI unit for radioactivity and is defined as one disintegration (dis)/second. The curie is defined as the approximate activity of 1 g of the isotope ^{226}Ra (radium) and is equal to 3.7×10^{10} dis/second. Generally, millicurie (mCi) doses of a radiopharmaceutical are used for imaging or therapeutic purposes.

Dose Limits

The Nuclear Regulatory Commission (NRC) is the body charged with regulating nuclear materials used for medical reasons within the United States [1]. By special agreement with contract states, licensing a facility to use radioactive substances may also be delegated to the state government. The NRC has set maximum limits of radiation exposure for humans. These limits are different for the public, radiation workers, fetuses, and other groups (Table 75.1) [2]. These differences are based on the relative risk of each group. For example, fetuses are more sensitive to radiation damage than adults, particularly during the first three months of gestation, so they have a relatively low legal limit for

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Table 75.1 Annual dose limits for whole body exposure.

Personnel	Dose limit
Occupation dose limit (adult)	5 rems (0.05 Sv)
Member of the public (adult)	0.1 rem (1 mSv)
Minor (< 18 years of age; occupational dose limit)	0.5 rems (0.005 Sv)
Embryo/fetus (for mother who is a radiation worker)	0.5 rem (5 mSv) over term of pregnancy

Source: Adapted from NRC Regulations [2].

exposure. The general public is also to be protected from increased exposure as compared with radiation workers.

A stochastic effect is an adverse effect that can occur at any dose exposure, but whose risk increases as the dose increases. The risk of developing cancer after radiation exposure is one example of this. Compared with someone who was not exposed, exposure to even low doses of radiation increases the risk of developing cancer later in life. However, the increased risk of developing cancer with low level exposure when receiving, treating, and handling radioactive patients when proper precautions are taken is difficult to quantify but is probably very low.

Radiopharmaceuticals

Veterinary Uses

A radiopharmaceutical is a radioactive drug that is either used to diagnose or treat disease. The radiopharmaceuticals most commonly used in veterinary medicine are ^{18}F-fluorodeoxyglucose (^{18}FDG), technetium-99 (^{99m}TC) and iodine-131 (^{131}I), although others such as samarium-153 (^{153}Sm) and phosphorus-32 (^{32}P) have also been used [3–7]. ^{18}FDG is used in diagnostic positron emission tomography (PET) or PET/computed tomography (CT) and has a half-life of just under 110 minutes. PET, while relatively uncommonly used in veterinary medicine, is becoming more readily available either on site or in some cases at a human facility. While PET is most commonly used for staging and monitoring response to treatment or disease progression, in oncology patients it is also being used to help diagnose other diseases such as certain musculoskeletal disorders and infectious diseases. ^{99m}TC is used in diagnostic radiology and has also has a relatively short half-life. When bound to pertechnetate it is most commonly used for the diagnosis of portosystemic shunts and thyroid disorders. When bound to methylene diphosphonate, it is taken up by osteoblasts and can be used to identify active areas of bone remodeling. It is therefore commonly used to help to identify affected areas in patients with osteosarcoma or other cancers that have metastasized to bone [8–10]. ^{131}I is most commonly used to treat hyperthyroidism in cats and occasionally to treat thyroid carcinomas in either dogs or cats [11–13]. A list of the most commonly used radiopharmaceuticals and their half-lives is given in Table 75.2.

Table 75.2 Commonly used radiopharmaceuticals used in veterinary medicine and their corresponding half-lives.

Radionucleotide	Approximate physical half-life
^{18}F-fluorodeoxyglucose (^{18}FDG)	110 minutes
Iodine 125 (^{125}I)	59 days
Iodine 131 (^{131}I)	8 days
Samarium 153 (^{153}Sm)	1.93 days
Technesium-99 M (^{99m}Tc)	6 hours

Concept of Half-Life

How long a particular radiopharmaceutical is actively emitting radiation is based on the physical half-life ($t^{1/2}$) of the particular isotope. The physical half-life is defined as the amount of time required for the radioactivity to decrease by half and is constant for each isotope. In general, after 10 half-lives an element is considered no longer radioactive. This is the reason that when a patient dies directly after receiving a radiopharmaceutical, their body must be stored in a shielded freezer designated for this purpose and is not released for 10 half-lives. In the case of ^{131}I this would be approximately 80 days.

The biological half-life is defined as the amount of time for a patient to eliminate one half of the compound from the body. Most agents are excreted in the urine and/or feces. When a radiopharmaceutical is given to a patient, the length of time they are actively giving off radiation is based on both the physical half-life of the element and the biological half-life. Combining these terms gives you the effective half-life, which can be variable between patients due to variation in the biological half-life between individuals.

Radiation Protection

As with patients who are exposed to x-rays for diagnostic imaging (radiographs or CT), patients receiving external beam radiation therapy are not radioactive and do not present a risk of exposure to caregivers. This is in contrast to patients who receive a radiopharmaceutical agent resulting in residual radioactive material being present in their body. This leads to risk for anyone who comes in contact with

them, their blood or other body fluids such as urine, or with their feces. These patients also have the potential to contaminate the environment, which could also place their owners or caregivers at risk for exposure to radiation.

The most basic concept of radiation protection is to limit exposure as much as possible. This concept should be applied to anyone potentially coming into contact with any form of radiation. The term ALARA, or as low as reasonably achievable, is a mainstay of radiation protection and should be closely adhered to. To limit exposure when working with radiation or with a radioactive patient, the three main factors to keep in mind are time, distance, and shielding. Limiting the amount of contact time with a patient will decrease the amount of dose that the caregiver absorbs. Since radiation dose falls off as the distance squared, increasing the distance from a patient substantially decreases the dose received. For example, If you are 2 meters away from a patient who is radioactive you will receive one quarter of the dose you would have received if you were 1 meter away. Shielding the radioactive source or patient will also greatly reduce potential exposure. For these reasons, patients treated with a radiopharmaceutical are housed separately from other patients and kept out of commonly entered areas. This can be difficult if the patient is ill, particularly in the first few days after being treated.

External Beam Radiation Protection

One potential source of exposure to radiation occurs while taking radiographs. While it is a well-established fact that you should never place any part of your body in the primary beam if you are holding a patient for radiographs, you may still receive exposure from scatter radiation. Whenever possible, everyone but the patient should leave the room when a radiograph is being taken. Chemical sedation and restraint devices such as sand bags can be used to limit the need to have someone in the room. In some states, it is not permissible to be in the room while a radiograph is being taken. Regardless of the law, every attempt should be made to limit exposure if someone is holding an animal during this procedure. With critically ill patients, it may at times be impractical to sedate or restrain the patient appropriately for radiographs without having one or more people holding the animal. If allowed by law in your state, precautions should be taken if holding an animal while a radiograph is being taken to minimize radiation exposure.

To decrease the time of exposure, careful technique should be used to decrease the number of repeat radiographs that are needed. Wearing lead gloves will not prevent exposure of your hands if they are in the primary beam and the field should be properly collimated so that no one has any body part within the primary beam. Additionally, lead gowns, gloves, and thyroid shields will largely protect you from scatter radiation and should be worn. No one person should be designated to hold animals for radiographs and this task should be shared among trained individuals, thus limiting the exposure to any one person. Lastly, anyone taking radiographs should have received training in the proper use of equipment and the steps that can be taken to reduce the risk of exposure. Anyone taking radiographs should be properly monitored using a radiation detection device (dosimeter) to ensure that they are not receiving too large of a cumulative dose. Radiation detection badges are usually checked monthly or quarterly and reports are to be made available to the individual so they are aware of what exposure they may have received.

Radionucleotide Safety

Any facility using a radionucleotide will have a designated radiation safety officer and a radiation safety plan as part of their licensing requirements. This plan will detail how long a patient needs to be held after exposure and safety procedures. If a treated patient is brought into a practice for care other than where it was initially treated, the facility should be contacted and notified. Each treating facility will have a plan in place for handling radioactive patients and should be familiar with local rules and regulations. The length of time after exposure that there is a concern for people working with the animal or its blood depends on the particular radionucleotide. For ^{18}FDG with its short half-life, there is little risk of exposure 18 hours after injection. With ^{99m}TC there is little concern of exposure after 48 hours, while patients treated with I^{131} can present a hazard for one month or more. It should be remembered that the animal's blood, urine and feces are also radioactive. These patients should be isolated, their cages labeled as radioactive, and contact minimized until it is determined whether they still present a hazard. Minimal precautions to be taken include wearing gloves, gowns, plastic shoe covers, and a face shield when coming into contact with these patients. Cages should not be hosed to limit the risk of aerosolizing and spreading radioactive material present. The cages can also be covered with plastic lined absorbent material to help contain any excreta such as urine. Geiger–Müller counters can be used to measure electron or photon radiation directly from the surface of a patient or from areas that may have become contaminated. These detectors generally give readouts in counts/minute or milliroentgen/hour (mR/hour).

In a patient treated with I^{131} a Geiger counter can be placed over their thyroid area and over any waste or bodily fluids to check for radioactivity. To check whether the work environment is contaminated, wipe tests should be done. Wipe tests are done by taking swabs of the cage, counters,

floors, and other areas where an irradiated patient was present and placing the wipes in a scintillation counter. Wipe tests are generally part of any radiation safety plan for a facility using radionucleotides and they should be able to run the plan. Remember that once an area is contaminated, it may take up to 10 physical half-lives for the area to be safe unless decontamination procedures are followed and the contamination is removable. All waste from animals should be kept separately, labeled and checked for radioactivity. It should be stored properly and shielded until it is no longer radioactive before disposal. As the local legal requirements vary for radioactive waste disposal the treating facility should be consulted regarding waste disposal or withholding times before any animal waste is disposed of in municipal trash.

Although generally not required as part of routine employee monitoring, if there is concern that an exposure might have taken place with radioactive iodine a bioassay test can be performed to see if they have absorbed any radionucleotide. This procedure does require specialized equipment. It is a noninvasive test where a gamma counter is placed near the neck area to see if a person is emitting gamma rays from absorbed radionucleotide that may have accumulated in the thyroid.

Chemotherapy

According to American Society of Health-System Pharmacists, a drug should be considered hazardous if it is genotoxic, carcinogenic, teratogenic, and/or could lead to decreased fertility or cause serious organ or other toxicities at low doses in experimental animals [14]. Drugs that are classified as chemotherapeutic or antineoplastic agents fall into at least one of these categories and should be handled with care. Drugs for which there are no data regarding these potential toxicities, such as new or investigational drugs, should also be handled as hazardous materials. Chemotherapeutic drugs are most commonly used to treat dogs and cats with cancer, although some of these same agents have also been used to treat inflammatory and autoimmune diseases such as granulomatous meningoencephalitis and immune-mediated hemolytic anemia.

As there is little information available as to a safe level of exposure to any one particular drug or chemical, it is always safest to limit exposure to ALARA. In a sense, it is the same concept as presented for radiological hazards earlier in this chapter. Unlike with radiation, however, there is no chemotherapy "dosimeter" to wear or other way to quantify exposure that is commercially available.

The risk of exposure to a hazardous agent is based on the properties of the drug itself and the amount exposed to. Exposure can occur by drugs being inhaled (if they are aerosolized), absorbed through the skin, or ingested. Sources of exposure to hazardous chemicals for the nursing staff can occur during transport and handling of chemotherapy drug vials, drug preparation, administration, handling of animal waste from patients who have received a hazardous drug, and from working in an environment that has become contaminated from any of the above [15].

There are multiple studies of healthcare workers that show residual drug in their urine [16, 17]. In addition, chromosomal changes consistent with exposure to mutagens have been found in people who regularly work with chemotherapy [18, 19]. As with radiation, the additional risk of developing cancer after exposure to low levels of chemotherapy is poorly understood.

Limiting Exposure

The Occupational Health and Safety Administration states that sound practices require that a hazardous safety and drug plan be developed at institutions where hazardous drugs are used to minimize the risk to employees [20]. Additional recommendations from the Board of Pharmacy will vary from state to state.

All chemotherapeutic drugs should be mixed in a biological safety hood. Minimally a class II laminar flow biological safety cabinet should be used [16, 20]. A pharmacy isolator may be preferable as it has been shown that the area directly outside of laminar flow hoods can become routinely contaminated [21, 22]. The use of a closed drug delivery system such as the PhaSeal™ (Becton Dickinson, Franklin Lakes, NJ) system while administering chemotherapy can also decrease the risk of exposure and avoid contamination of the work environment [16, 23]. Proper training in the use of a closed system is important to help avoid accidents leading to contamination [24].

For larger spills or those taking place outside the hood, a spill kit should be used. These kits are available commercially prepackaged, or you can assemble your own (Protocol 75.1.). See Protocol 75.1 for steps to follow to contain a spill.

One of the most important ways to protect yourself from exposure to patient or environmental chemotherapy hazards is by wearing personal protective equipment (PPE) [15]. Minimum PPE to be worn for hazardous drug safety would be a gown, gloves, and goggles. Additional PPE, such as a respirator, may be indicated for certain agents with a high aerosol potential (e.g. nitrogen mustard). Gloves should meet the American Society for Testing and Materials Standard D6798. Specially manufactured chemotherapy gloves should be worn whenever mixing, administering or handling excreta from patients who have received chemotherapy. Chemotherapy gloves can be made

Protocol 75.1 Containing a Spill of Chemotherapy Drug

Minimum Contents Needed for a Spill Kit

- PPE (mask, gloves, gown, goggles)
- Absorbent materials (pads, towels)
- Plastic bags
- Incident report form
- Cautionary signage

Procedure

1) Put on gloves, mask, and goggles.
2) If there is a large spill, restrict access to the spill area and place cautionary signage, if applicable.
3) Wait for any aerosols to settle.
4) Gently place absorbent pads on spill.
5) Place absorbent pads in plastic bags marked as "hazardous waste."
6) Rinse spill with equal amount of neutralizing agent (such as Peridox™, Contec, Spartanburg, SC) by pour-over method (do not spray).
7) Use more absorbent pads to pick up the rinse, and thoroughly dry the area, moving from the outside of the spill inward.
8) All contaminated materials, including PPE, should then be put in a second plastic bag and disposed of in proper containers.
9) Fill out an incident report form, if applicable.

from different materials but are always powder free. Double gloves should be worn; the first glove goes underneath the gown, then the second pair extends over the cuff of the gown at the wrist, so no skin is exposed. These gloves must undergo testing by the manufacturer to ensure that they are not permeable to chemotherapy agents and will be clearly labeled as such. Examination gloves in general are not sufficient to protect from exposure.

Gowns should be single use, close in the back, and be rated for their impermeability to hazardous drugs. They should have closed cuffs to facilitate gloving. Surgical scrubs, lab coats, isolation gowns, and other permeable or absorbent materials do not provide enough protection and may increase exposure by absorbing and holding contamination against the skin.

The disposal of contaminated materials and waste from chemotherapy preparation, administration and from patients exposed to chemotherapy is regulated by federal (Environment Protection Agency), state, and local regulation, and an institutional plan should be developed to make sure any facility using these hazardous drugs is in compliance. In general, all needles and other sharps should be disposed of in a separate appropriately labeled chemotherapy designated sharps container. Needles should not be removed, recapped or broken off from syringes that come in contact with hazardous chemicals and blood or bodily fluids from patients exposed to chemotherapy agents. Syringes should never be reused.

Most of the data we have on drug excretion have been extrapolated from studies on human patients, but it is safe to assume that the routes of excretion are the same. It has been recommended that bedding contaminated with blood, urine, feces, or other bodily fluids from patients treated with chemotherapy are treated as potentially hazardous for at least 48 hours after treatment. Any waste from a treated patient should be considered a potential exposure hazard. PPE should be worn when handling waste and it should be disposed of as chemotherapy waste. Patients that remain in the hospital after receiving chemotherapy should have their cages clearly marked with cautionary signage, and the staff should be trained in safe handling of waste products. Kennels that have housed chemotherapy patients should not be washed with a high-pressure hose to avoid aerosolization of the waste. Soiled bedding should be stored separately from other laundry and prewashed, and then washed for a second time with regular laundry. The laundry bag should either be disposed of as chemotherapy waste or washed with the bedding if it is reusable.

Checking for environmental contamination with hazardous drugs is not easy, as commercial wipe tests are not readily available. This means that to minimize exposure several basic steps should be taken. Work areas where hazardous drugs are prepared or used should be clearly labeled with appropriate signage and should be separate from other drug preparation areas. No eating, drinking, smoking, applying of cosmetics, or other activities of this type should be done in an area where hazardous drugs are used or where patients treated with these drugs are housed.

Personnel that are pregnant, believe they might be pregnant, are actively trying to conceive, or are breastfeeding should avoid any chemotherapy agents and the area in which they are administered. As this may require reassignment of duties or changes in job descriptions, labor laws should be followed, but minimally the employee should be made aware of potential risks to their fetus if they are exposed to chemotherapeutic agents. Proper handling of all hazardous drugs for preparation, transport, and administration should be carried out as outlined elsewhere in this chapter. The work areas and patient handling areas should be regularly cleaned.

Chemotherapy Elimination

How long the drug is present in a patient after it is dosed depends on many factors including properties of the drug itself, its metabolism, and individual patient and species characteristics. For most cytotoxic drugs, full pharmacokinetic information is not available in veterinary patients. One study showed that vincristine, vinblastine, cyclophosphamide, and doxorubicin could all be detected in dog urine after infusion [25]. Another study looked at residual drug in dog urine after dogs were treated with several commonly used chemotherapy drugs including cyclophosphamide, doxorubicin, vinblastine, and vincristine. After oral cyclophosphamide dosing, there was no detectable drug in the urine after one day. With vincristine and vinblastine, low levels of drug were detectable for up to seven days post-treatment. Doxorubicin still had low but detectable levels of drug at three weeks after administration [26]. In a separate study, these same investigators found that most serum samples contained little to no detectable drug by seven days after treatment [27]. These results indicate that care should be taken when handling chemotherapy patients for up to several weeks after treatment.

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Handling the Suspected Cruelty Case

Alison Liu

Veterinary professionals, especially those working in emergency medicine, may become involved with the evaluation and treatment of animals that are part of criminal animal cruelty cases. Animal cruelty cases are medicolegal cases, which means that they have both medical and legal aspects. Veterinary forensic medicine is veterinary medicine practiced within a legal context.

While the term "animal cruelty" is used commonly in the veterinary community and in society, animal cruelty is a legal determination. All 50 of the United States have animal cruelty laws, but how they are defined and what conduct they prohibit may differ. While veterinary professionals are not expected to be legal experts, they should have a general understanding of their local animal-related laws, including but not limited to veterinary reporting of suspected animal cruelty.

Response to animal cruelty cases varies by community. In many cities and counties, local humane societies or animal welfare organizations have deputized humane officers that respond to and investigate animal cruelty complaints. In other communities, law enforcement agencies (e.g. police departments, sheriffs' offices, animal control) enforce animal cruelty laws. Veterinary professionals should be familiar with which agency responds to animal cruelty complaints in their community so that when they are presented with a suspicious case, they know to whom to make their good faith report. The National Link Coalition maintains a database with respective reporting agencies by state and community [1].

Veterinary Roles

Veterinary professionals can have several roles in suspected animal cruelty cases, which can vary based on how the case is initiated (i.e. veterinary or law enforcement). One veterinarian may be responsible for providing both the initial evaluation and continuing medical care. That veterinarian may also have been the individual who made the good faith report of suspected animal cruelty to law enforcement. In many instances, all three roles, which are elaborated below, are carried out by the same veterinarian. Alternatively, responsibilities may be handled by multiple veterinarians each serving more specialized roles.

Recognizing and Reporting

Veterinarians frequently play an essential role in animal cruelty cases by recognizing suspected cruelty and reporting to law enforcement. While animal cruelty cases may be thought of as originating in animal shelters and welfare organizations, any veterinarian may be presented with an animal that has been the victim of non-accidental injury and/or neglect. Veterinarians working in emergency medicine and general practice are not uncommonly presented with animals with suspicious injuries. Reporting suspected animal cruelty can be a difficult and uncomfortable task, especially if the veterinarian has a long-standing relationship with the client. The veterinarian does not have the burden of investigating and determining whether a crime has been committed or who is guilty of a crime (it may not be the owner); those are the responsibilities of law enforcement, prosecutors, and the courts.

Veterinarians should be familiar with their state's laws regarding reporting suspected animal cruelty. As of October 2021, 36 states had laws regarding veterinarians reporting suspected animal cruelty; 19 states had mandatory reporting laws, and most states provide civil immunity to veterinarians who report in good faith [2]. The referenced AVMA website [2] contains updated information.

The reporting of suspected animal cruelty aligns with the veterinarian's broader role of protecting public health.

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"The Link" is the well-established connection between animal cruelty and other forms of family and interpersonal violence. In a survey of battered women entering shelters, 71% of pet-owning women reported that their intimate partners injured, killed, or threatened a family pet, and 57% reported that actual harm or killing had occurred [3]. Another similar survey revealed that of battered women reporting violence toward their pets, 88% of the reported cases occurred in the presence of women and 76% occurred in the presence of children [4]. Concern for safety of their pets can be a reason that survivors delay or do not leave their abusive situation. Reporting animal cruelty may provide an individual or family an opportunity to leave an unsafe situation.

Recognizing Non-accidental Injury

Non-accidental injury is physical trauma purposely inflicted by a person on an animal. One of the most challenging aspects is acknowledging it as a differential. While there is no single, consistent indicator, there may be various aspects of either the reported history or clinical presentation of the patient that increase the veterinarian's index of suspicion (Box 76.1).

Forensic Evaluation

Forensic veterinary medicine is veterinary medicine practiced within a legal context. An animal that is part of an alleged animal cruelty case is not only a patient; it can also be evidence in that criminal case. The findings from the examination and medical treatment of the patient may be used in a court of law. Documentation of the medical findings should be accurate, objective, and thorough.

In most cases, the veterinarians providing medical care will also fulfill the forensic evaluation role by communicating the animal's medical status to law enforcement and prosecutors. They may also be asked to provide expert witness reports or testimony in a hearing or at trial. In some jurisdictions, local law enforcement may establish relationships with designated veterinarians, who serve as forensic veterinarians in all animal cruelty related investigations. However, the involvement of a designated forensic veterinarian does not preclude any other veterinary professionals involved in the care of the animal from being called to testify on behalf of either the prosecution or defense.

Medical Care

After the animal is assessed, a treatment plan should be developed to address the presenting medical concerns. Owner consent must be obtained for any treatments and procedures. When animals are seized by law enforcement and the owner has not provided consent, medical care should not extend beyond what is considered "medically necessary." Spays and neuters are common examples of "elective" procedures that should not be performed except in instances where they are considered a medical necessity. When an animal's medical needs exceed the capabilities of the hospital or shelter, the animal should ideally be transferred to another veterinary facility that is able to provide appropriate care.

Documentation of the Suspected Cruelty Victim

History

As with any animal that presents to a hospital for evaluation of a medical concern, it is important to obtain a thorough history. Client service representatives, veterinary assistants, and veterinary technicians typically have initial contact with owners and serve as essential communicators.

Box 76.1 Features That May Increase a Veterinary Health Care Worker's Index of Suspicion for Non-Accidental Injury

History

- History is inconsistent with the injuries
- History is discrepant (e.g. changes from person to person, owner changes their story)
- No history is provided
- Another animal in the household has a history of injuries or death
- Absence of an accidental scenario
- Animal is kept primarily or only indoors
- Known or suspected violence in the home
- Behavior of the owner arouses suspicion

Clinical Findings

- Repetitive injuries (the animal has presented previously with injuries or has injuries in various stages of healing)
- Injuries cannot be explained by an accident
- Fractures feature prominently
- Multiple fractures
- Fractures involving multiple areas of the body
- Transverse fractures
- Fractures in later stages of healing
- Multiple fractures at different stages of healing

Source: Adapted from references [5–10].

A comprehensive set of questions should be asked of the client and attention to details they provide may increase the index of suspicion for reporting suspected cruelty (Box 76.1). The history should be thoroughly documented in the patient's medical record.

When law enforcement brings animals for evaluation, veterinary staff should inquire whether scene photos and/or video as well as any reports were created and if so, request them for review. Photographs and video of where the animal was found can provide the veterinarian better context when evaluating the animal's medical findings as they relate to their living environment. Scene documentation can also help the veterinarian assess the suitability of living conditions, a frequent question that arises in neglect cases.

Physical Examination

A thorough physical examination should be performed at presentation. While full physical examination would ideally occur prior to treatment that could alter the patient's presenting state, thorough examination should never preclude medical stabilization. As with any other emergency patient being triaged, pain medication should be provided as clinically indicated.

The initial physical examination should include assigning a body condition score, which is especially crucial in underweight animals. The Purina Body Condition Scale is a widely used tool that had been scientifically validated [11–13]. When evaluating an underweight animal, the veterinarian does not want to under or overestimate how much weight the animal has lost; hence, the value of the objective scoring system. Final assessment of the animal's ideal body condition after weight gain may provide further guidance in assessing whether the estimate of their initial body condition was accurate.

A pain assessment should also be performed. A standardized pain scale, such as the Glasgow Composite Measure Pain Scale or the Colorado State University's Acute Canine (or Feline) Pain Scale can be used. At a minimum, documentation of abnormal behavior interpreted as pain during examination should be noted in the medical record. Reevaluation after analgesics have been administered can serve as additional documentation of an animal's pain or discomfort during initial intake.

Scanning the animal for an identifying microchip may be helpful in cases involving stray or abandoned animals. This information can provide law enforcement a potential lead in a case where the ownership status is unknown. In some cases, claims made by an apparent "good Samaritan" of recently finding an animal may be dispelled after it is determined that the microchip is registered to them.

Photography

Every animal should be documented by a taking a standard series of whole-body photographs during initial examination. In a survey where prosecutors were asked what type of animal evidence influenced their decision to prosecute a case, approximately two thirds cited photographs as a significant factor [14].

Photographs should be taken as close to the time of initial examination as possible because the animal's appearance will change with time and after receiving treatment. Photographs taken after treatment may affect a veterinarian's ability to draw conclusions about a particular injury. For example, gunshot wounds will appear remarkably different after they are clipped, cleaned, and surgically addressed. Certain characteristics that help determine which wound was an entrance and which was an exit wound will be lost. A wound that is left to heal by second intention may appear different several days later. While photographs should be taken prior to non-urgent medical intervention, photography should not delay any life-saving medical care.

A photograph of the animal's identification (e.g. an identifying case board or label) should be taken at the commencement of the photo series and should include the date, case number, and animal identification number or name. Photographic views should include all sides of the animal's entire body (Figure 76.1). Photographs of dentition will document the appearance of the teeth, which can be used to estimate the age of the animal. Areas of interest, such as a wound or other lesion, should also be photographed. When photographing areas of interest, overview, mid-range, and close views should be taken to help orient the viewer (Figure 76.2). A scale placed in the visual field, adjacent to the area of interest, of at least one photograph can help provide relative size information. The American Board of Forensic Odontology number 2 photo scale is an inexpensive scale that is widely accessible online. If a scale is not available, an object that has a standardized size such as a coin can be placed in the visual field.

Because photographs are potential evidence in a criminal case, they should not be deleted or altered. Deleting a photograph in a series will cause a gap in the digital photo numbering system. Photographs should be saved in a secured location, such as a secured drive or cloud-based storage system. If photographs that are used in a written report are altered (e.g., cropped, enlarged, edited), ensure that the original unaltered photograph has been saved.

Videography

Video can be another tool in documenting an animal's presenting condition. Video can be especially impactful in cases in which the animal has a neurologic condition or



Figure 76.1 Photographic series of a dog at initial intake. (a) Photo label with date, case, and animal identifying information. (b) Left lateral. (c) Right lateral. (d) Cranial. (e) Ventral. (f) Dorsal. (g) Caudal. (h) Oral (right side of the mouth).

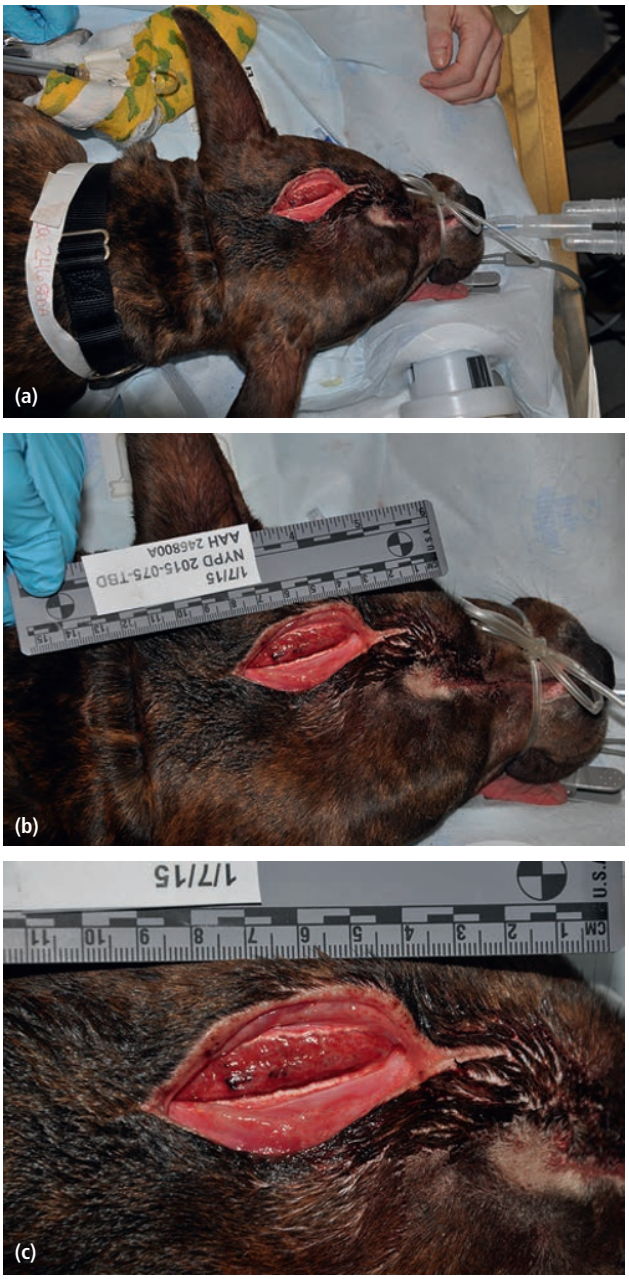


Figure 76.2 Photographic series of an area of interest (sharp force injury). (a) Overview. (b) Mid-range and (c) close, both with standardized measurement device in frame.

lameness. Video may also better visually communicate an animal's pain and discomfort than words and photographs alone.

Laboratory Tests

Every animal should have survey laboratory tests performed at initial intake regardless of their physical examination findings. An ideal minimum database includes a complete blood count, comprehensive serum biochemistry, and urinalysis. Dogs should be tested for heartworm disease and cats should be tested for feline leukemia and feline immunodeficiency viruses. The biochemistry panel should include creatine phosphokinase (also known as creatine kinase, CK) and aspartate aminotransferase (AST), which are often excluded from basic biochemistry panels; both can be beneficial in assessing muscle injury.

Animals with significant laboratory work abnormalities should have their bloodwork rechecked at appropriate intervals. The normalization of certain values shortly after initial presentation may help the veterinarian form conclusions about injuries. For example, a high alanine aminotransferase (ALT) level at initial presentation that normalizes shortly after presentation without treatment may indicate acute hepatic injury.

A fecal ova and parasite test should also be performed. Ruling in or out intestinal parasites is especially important in underweight animals. Intestinal parasites are often reported by owners as the reason their pet is underweight. In large, multi-animal cases (e.g. hoarding or other neglect case involving multiple animals) a high prevalence of intestinal parasites may serve as further evidence of the lack of appropriate care and poor environmental conditions of the population.

Diagnostic Imaging

Radiographs should be taken as part of the initial minimum database. Orthogonal thoracic and abdominal radiographs should be considered the minimum survey views. Additional views should be taken based on physical examination findings. A dog with a strangulating wound to one of its limbs should have radiographs taken of the affected limb and of the contralateral limb for comparison. Animals that have blunt force trauma injuries or are alleged to have experienced trauma should have full body radiographs, including skull, thorax, abdomen, pelvis, and limbs. It is not uncommon to diagnose additional injuries not identified on physical examination (Figure 76.3). This is a common diagnostic tool used to identify injuries at different stages of healing. Healing or healed rib fractures are examples of older injuries that are typically not palpated during physical examination.

Because animals that are part of alleged cruelty investigations frequently present to veterinarians with a limited or ambiguous medical history, performing survey radiographs allows for the most comprehensive evaluation of that animal. Not only can radiographs help identify additional injuries not seen on physical examination, they can also diagnose unrelated medical conditions that the veterinarian may need to address.

Ultrasound can be contributory as part of the initial evaluation. Thoracic and abdominal point of care ultrasound

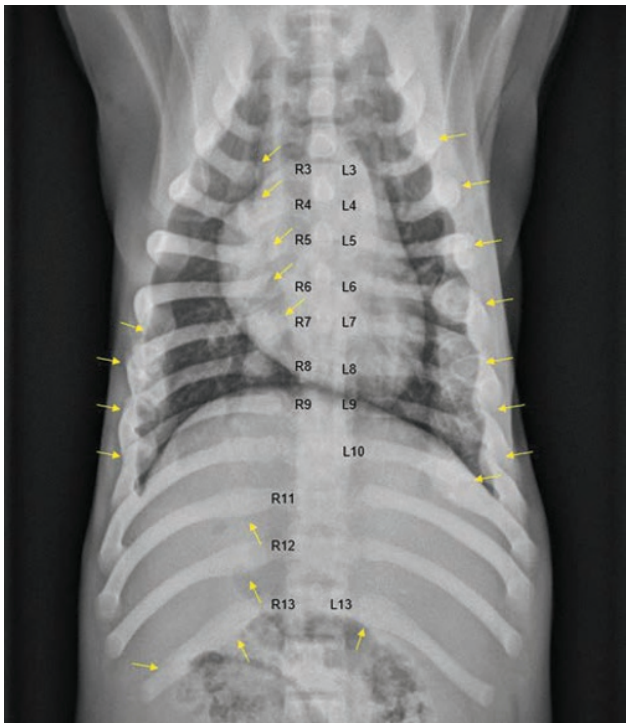


Figure 76.3 Ventral-dorsal thoracic radiograph of four-month-old dog that was presented for reportedly being malnourished. In addition to being underweight, the dog was found to have many fractures at different stages of healing, including 22 rib fractures (shown with arrows).

should be considered in cases of known or suspected trauma to the abdominal and thoracic cavities (Chapters 27 and 39). Animals that show evidence of liver injury on bloodwork may benefit from a complete abdominal ultrasound. For underweight animals, ultrasound may help to rule out certain disease processes that account for weight loss. As with the diagnostic workup of any non-criminal case, ultrasound may be indicated for animals with a variety of other medical conditions (e.g. pyometra, intestinal disease).

Computed tomography (CT) can be a valuable tool for evaluating animals with traumatic injuries. CT provides superior detail compared with radiography and can reveal injuries that were not apparent radiographically. In a prospective study of dogs that presented to a hospital with a history of motor vehicle accident injuries, CT was determined to be more sensitive for detecting thoracic trauma than radiography [15]. Magnetic resonance imaging may be indicated for certain neurologic conditions. When considering advanced imaging, the animal's overall health status must be considered. Critically ill animals may be medically unstable for sedation, and advanced diagnostics such as these should not be done in patients in which it is medically contraindicated.

Injuries

Animal cruelty cases are often classified into two categories: physical abuse and neglect. Physical abuse may also be referred to as intentional abuse or non-accidental injury. Neglect cases involve failing to provide proper care or acts of omission. Below is an overview of some of the types of injuries that may be seen in animal cruelty cases. Additional resources are provided for reference at the end of this chapter.

Physical Abuse

Blunt Force Trauma

Blunt force trauma is the most common type of physical trauma that veterinarians encounter. Blunt force trauma is trauma caused by impact of a blunt object against a body, impact of the animal against a blunt surface, or a combination of both. Blunt force injuries include abrasions, contusions, lacerations, and fractures. Abrasions are superficial injuries that result in loss of the top layer of skin from friction with a blunt object. Contusions are bruises caused when a blunt impact ruptures blood vessels, resulting in blood leaking into surrounding tissue. Lacerations are tears in soft tissue and typically have irregular, jagged margins. Bony fractures can occur with significant blunt force injury. Skin injuries may not always be easily detected on physical examination, especially those that are small and superficial. Careful examination of the skin by parting the hair coat can help detect subtle injuries. In areas where skin injury is identified or suspected, clipping the hair coat will provide better visualization (Figure 76.4). It is not



Figure 76.4 The skin contusions on the dorsal aspect of this dog's head are best appreciated after fur is clipped.

uncommon for animals to have no recognizable soft tissue injury on physical examination. The absence of injury does not rule out blunt force trauma.

Thorough evaluation of the animal's injuries may allow the veterinarian to conclude whether the injuries are more consistent with an accidental scenario or non-accidental injury. In a retrospective study of dogs and cats that sustained non-accidental blunt force trauma compared to those that were injured from a motor vehicle accident, there was a difference in the pattern of injuries. Box 76.2 summarizes the injuries that were found to be significantly associated with the respective groups. Note that head injuries (scleral hemorrhage, dental fractures, skull fractures) were found to be associated with non-accidental injury, whereas a more caudal pattern of skeletal injuries (pelvic/sacral fractures, sacroiliac luxations) was found to be more commonly associated with motor vehicle accidents.

Penetrating Trauma

Penetrating trauma is trauma that involves injury from penetration of an object into the body and includes sharp force trauma and gunshot wounds. Some bite wound injuries can also be classified as penetrating trauma.

Sharp force trauma is injury resulting from a sharp object, such as a knife, scissors, glass, or other sharp object. Stab wounds are sharp force wounds that are deeper than they are wide, whereas incisional wounds are sharp force wounds that are longer than they are deep (e.g. surgical incision). Sharp force injuries typically cause a significant amount of hemorrhage and a minimal amount of bruising in contrast to blunt force injuries. The edges of the wound are straight and well defined; however, these physical features may be more challenging to appreciate the older the wound is. The measurements of sharp force wounds do not always equate to the size of the instrument used to cause them. For example, a stab wound may be deeper than the length of the blade if the knife is pressed firmly into the animal, depressing the skin and underlying soft tissues.

Gunshot injuries are caused by penetration of a projectile from a firearm. Gunshot wounds may be difficult to distinguish from other injuries, especially if the wounds are older. Radiographs can confirm the presence of a projectile and assess the extent of the injuries; however, projectiles that pass through and exit the body (referred to as through-and-through injuries) will not be detected radiographically.

While low velocity firearms like air-powered pellet guns and BB guns have less speed than high velocity firearms such as handguns and rifles, low velocity firearms can cause devastating injuries. Law enforcement should be notified in cases where a projectile is recovered so that if indicated, they can take possession of it for evidentiary purposes. Projectiles should not be handled with metal forceps to prevent damaging them. While the veterinarian's basic assessment of the projectile (e.g., being consistent with a certain type of weapon such as a pellet) can be contributory to the initial investigation, further analysis should be reserved for law enforcement ballistic experts.

The veterinarian should attempt to determine the entrance and exit wound, if present. Entrance wounds are usually smaller than exit wounds. They often have a rounder shape in contrast to exit wounds that typically have a more irregular shape. Entrance wounds typically have an inward beveled appearance and there may be hair that has been pushed into the wound from the projectile entering the tissue (Figure 76.5).

Bite wounds can be classified as penetrating injury if the tooth penetrates through the tissue. Bite wounds frequently also involve some degree of blunt trauma in the form of crushing injury from the animal's jaws.

Burns

Burns can either be intentional or accidental. While there may be characteristics that are more supportive of one versus the other, the veterinarian may not be able to form a definitive conclusion about the nature of the burns. Burns that are suspected to be accidental may constitute making a good faith report if it appears there has been a significant delay in seeking treatment.

Box 76.2 Injuries Found to be Significantly Associated with Non-Accidental Blunt Force Trauma Compared with Motor Vehicle Trauma

Non-accidental

- Scleral hemorrhage
- Teeth fractures
- Skull fractures
- Rib fractures
- Vertebral fractures
- Damage to claws
- Older fractures

Motor vehicle accident

- Pneumothorax
- Pulmonary contusions
- Sacroiliac luxations
- Pelvic/sacral fractures
- Abrasions
- Degloving injuries

Source: Intarapanich et al. (2016) [6]

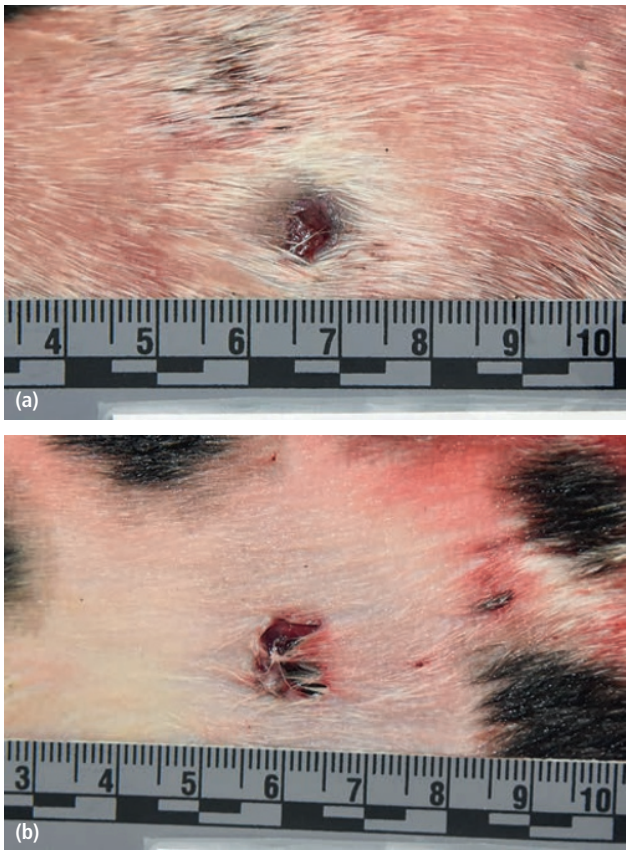


Figure 76.5 Gunshot wounds in a Dalmatian dog. (a) Entrance wound; note the round shape, smaller size, and hair being pulled into the entry wound. (b) Exit wound; note the more irregular shape and larger size of the exit wound.

Taking a biopsy of the suspected burn may be contributory as it can provide confirmation that the wound is a burn as well as establish an estimated age of the injury. Histopathology cannot definitively differentiate between chemical and thermal burns.

Neglect

Chronic Inadequate Feeding

Chronic or prolonged inadequate feeding is inadequate feeding over a long period of time (generally at least several weeks) and is frequently referred to as starvation. Because "starvation" often evokes thoughts of complete withholding of food, it is the author's opinion that chronic or prolonged inadequate feeding are usually preferable terms. It is not uncommon for non-medically trained individuals to assume that an emaciated animal has been withheld food. Only after ruling out medical conditions that would account for weight loss and monitoring the animal's weight for a period of time after initial presentation will a veterinarian be able to determine the cause of the presenting emaciation. It generally takes one to two months for an emaciated animal to reach ideal body condition and for a veterinarian to form their final conclusions.

Animals that show evidence of malnourishment should initially be fed a refeeding diet to minimize the risk of refeeding syndrome, a potentially fatal complication that can occur when reintroducing food to an animal that has experienced prolonged malnutrition. The shift from fat to carbohydrate metabolism can result in dangerous electrolyte abnormalities. A refeeding protocol should consist of a restricted caloric diet during the first days. After these first days, if the animal does not show signs of refeeding syndrome, the caloric intake can be gradually increased until feeding for resting energy requirements is reached (Chapter 42).

The animal should be weighed and their body condition monitored regularly. Most underweight animals can be weighed every one to two weeks. After an animal reaches an ideal body condition, the weight gained can be compared as a percent of initial intake weight. Most animals that were emaciated at initial presentation should be expected to gain approximately one third or more of their presenting body weight.

Animals should only be dewormed if parasites are detected in their fecal examination. The presence of intestinal parasites in an otherwise asymptomatic animal is typically an incidental finding and not a significant contributing factor to weight loss. Animals that are immunosuppressed and/or exposed to poor environmental conditions are most susceptible to infections, including parasitism.

Hair Coat Matting

Hair coat matting resulting from inadequate grooming over a long period of time can lead to meaningful medical issues including dermatitis, wounds, myiasis (infestation by fly larvae), impaired mobility, and pain. Dogs with severe hair matting may need to be anesthetized to be adequately groomed. Mats should be examined and photographed. The weight of the removed hair mats should be obtained and compared to the intake weight of the animal. Because matting can prevent full examination of the skin, dogs should be examined before and after grooming.

Hair matting along the limbs can cause strangulation wounds from ischemic necrosis (Figure 76.6). This can result in soft tissue infection, necrosis, osteomyelitis, and injury to ligaments and tendons. In more severe cases, complete separation of the paw from the limb can occur. While many strangulation wounds can heal with regular wound management over several weeks, some injuries are so severe that the limb must be amputated. A biopsy of the wound can be contributory by confirming the diagnosis and establishing an estimated age of the wound.



Figure 76.6 Left thoracic limb paw, dorsal surface. This extensive wound is a strangulation injury caused by severe hair matting that resulted in severe tissue infection, necrosis, and sloughing.

Embedded Collar

Embedded collar wounds are injuries to the neck caused when a collar, chain, leash, or other neck lead becomes embedded in the skin, resulting in injury to the skin and possibly underlying tissues. This injury is most commonly seen in young, growing animals. Collars should be cut away, measured, and that measurement compared to the circumference of the animal's neck. In the event the collar has been removed and does not accompany the animal to the veterinarian, conclusions about the cause of these wounds can often still be made based on these wounds' characteristic appearance of being fully or partially circumferential and located in the cervical region; collar embedment wounds frequently have granulation tissue. Whenever possible, a skin biopsy should be considered and may help establish an estimated age of the wound. A wedge biopsy perpendicular to the wound should be taken and should include haired skin through scar tissue to healthy tissue.

Medical Neglect

Medical neglect can include virtually any injury or illness where it is alleged that treatment was not sought or not sought in a reasonable timeframe. These cases are often also referred to as "failure to treat." Establishing an estimated timeframe is a crucial component of medical neglect cases. This may be done by reviewing previous medical records, taking biopsies of wounds, or assessing degree of healing seen on radiography of fractures. When considering reporting a suspected medical neglect case, a review of previous medical records may be beneficial in demonstrating previous attempts to seek medical care. However, it is important to note that this information may not be available at the time the veterinarian initially sees the animal and may only be discoverable when law enforcement performs their investigation.

Exposure to Environmental Extremes

Heat stroke is the most serious form of heat-induced injury and can become rapidly fatal. Thermal tissue injury can occur when the body is no longer able to dissipate heat adequately to maintain temperature below a safe point.

While animals that are housed both outdoors as well as indoors can become victims of heat-related injury, animals that are confined are less able to exert control over their environment and are at greater risk for experiencing heat stroke. For example, consider how being restricted by a short tether in direct sunlight on an outdoor balcony without water when the ambient temperature is 90°F increases a dog's risk for heat injury in contrast to that same dog being loose inside a backyard with multiple areas of shade and access to water.

Animals confined inside vehicles not being operated can become victims of heat-induced injury very quickly. Even when the ambient temperature outside is relatively mild, the temperature inside the vehicle can rapidly rise, creating dangerous conditions. A study by McLaren, et al. found that in ambient temperatures of 72–96°F, the temperature inside a car increased on average 40°F in a one-hour period with 80% of the temperature rise occurring during the first 30 minutes. In this same study, cracking the windows 1.5 inches (3.8 cm) did not make a significant difference [16].

Animals exposed to frigid temperatures should be evaluated for signs of hypothermia. Signs of hypothermia, especially if mild, such as shivering and heat seeking behavior, frequently resolve quickly once the animal is removed from the environment. Animals that have experienced extreme cold are at risk of developing frostbite, which is most often seen along the extremities.

With any suspected case of exposure to extreme temperatures, it is important to take the animal's temperature as soon as possible while keeping in mind that their temperature frequently changes drastically during transportation from the environment to the hospital. Because of this, animals may have a normal body temperature at time of initial examination. It is not uncommon for animals exposed to environmental extremes to show no appreciable abnormalities on physical examination or diagnostics.

Special Considerations for Deceased Animals

Veterinary hospitals may be presented with deceased animals that veterinary staff become suspicious about after hearing the owner or caregiver's reported history. Examination of a deceased animal should not be performed until legal consent is obtained. In contrast to live animals that may have been seized by law enforcement, examination of deceased animals is not medically necessary. Because a necropsy results in the collection of evidence, either consent from the owner or a search warrant obtained by law enforcement is necessary prior to being performed.

Like the examination of live animals, the external examination of deceased animals should include photographs of all sides of the entire body. Photographs should also be taken of any external packaging around the body as that packaging is being opened (e.g. bags, boxes, carriers).

Radiographs can aid in diagnosing conditions and can provide direction on areas of the body where post-mortem examination should be better focused. A complete necropsy should then be performed and photographs taken. The body should ideally be sent to a veterinary diagnostic laboratory for a pathologist to perform the necropsy. If the necropsy is performed by a veterinarian in-house, tissue samples should be collected and submitted for histopathology. While some diagnoses can be made based on gross dissection (e.g. traumatic injuries), histopathology is often necessary for a definitive diagnosis. Histopathology is also contributory in ruling out other conditions and providing an estimated duration of injuries or illness.

Summary

Veterinary professionals working in emergency medicine may encounter animals that they believe may have been victims of animal cruelty. Careful evaluation of the patient's reported history and assessment of their injuries may increase a veterinarian's index of suspicion and support making a good faith report to law enforcement. Animal cruelty can be classified as either physical abuse or neglect. Because animals in criminal animal cruelty cases can be evidence, their condition should be thoroughly documented, while simultaneously providing necessary medical care.

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Section Eleven

Wellness for the Veterinary Health Care Team

77

Self-Compassion: The Cornerstone of Wellbeing

Deborah A. Stone

Introduction

Veterinary professionals consistently work in emotionally charged and physically demanding environments. Continuously serving clients, patients, and other practice stakeholders is a familiar expectation within every work shift. Veterinary professionals are known to give of themselves regularly to an exhaustive level to patients, clients, and the practice team, thus often realizing that there is not much left for themselves. Too often, they may feel stuck in an endless cycle of going to work, giving their all, going home, and doing it all over again the next day.

Practice team members regularly experience the joys of positive outcomes as well as the stresses associated with negative outcomes and a constantly changing profession. The wellbeing of veterinary professionals is a growing concern within the veterinary industry, and as a result, there are increased endeavors to raise awareness and develop actionable resources.

This chapter introduces the powerful yet challenging construct of self-compassion. It explores why the construct is so challenging and provides resources to help us to understand the importance of giving ourselves a break when things do not go as we had hoped or planned.

In addition, the constructs of compassion fatigue and burnout are discussed. These terms may already be familiar from a significant number of available resources. The nuances between compassion fatigue, burnout, and self-compassion are also discussed.

Compassion Fatigue

An often-mentioned construct in the wellbeing space is compassion fatigue. This term is associated with individuals working across all caregiving professions, including human and animal health. Compassion fatigue, in the health arena, is the result of individuals caring for and giving to their patients to the point where there is often little or no energy to care for themselves.

Literature consistently advises caregivers to be mindful of prioritizing self-care over patient care, so the best outcomes are realized. This often resembles the oxygen and mask analogy, where the flight attendant announces, in the case of an emergency, first place the mask on yourself before placing on a child or someone in need. According to one of the leading compassion fatigue researchers, Charles R. Figley [1], "There is a cost to caring. Professionals who listen to clients' stories of fear, pain, and suffering may feel similar fear, pain, and suffering because they care. Sometimes we feel we are losing our sense of self to the clients we serve." This likely resonates with many veterinary professionals as they listen to and empathize with the clients who bring in their pets for end-of-life care and share the fear, pain, and suffering of losing their best friend or family member.

Figley, together with co-researcher, Robert G. Roop, specifically addresses compassion fatigue in the veterinary profession. As noted in their book, *Compassion Fatigue in the Animal-Care Community* [2], "For years, compassion fatigue was an unspoken occupational hazard of humane work. It caused diminished productivity, high attrition rates among shelter workers, and, worst of all, despair." This text advises the importance of self-care and of developing specific plans that can mitigate the symptoms of compassion fatigue (Box 77.1).

How to Address Compassion Fatigue

The veterinary profession continues to learn about compassion fatigue and the impact it has on practice team members. There are many resources, including assessments, available

Advanced Monitoring and Procedures for Small Animal Emergency and Critical Care, Second Edition. Edited by Jamie M. Burkitt Creedon and Harold Davis.
© 2023 John Wiley & Sons, Inc. Published 2023 by John Wiley & Sons, Inc.
Companion website: www.wiley.com/go/burkitt/monitoring

Box 77.1 Common Personal Symptoms of Compassion Fatigue

- Bottled-up emotions
- Sadness and empathy
- Inability to get pleasure from activities that previously were enjoyable
- Isolation
- Difficulty concentrating
- Feeling mentally and physically tired
- Chronic physical ailments
- Voicing excessive complaints about your job, your manager(s) and/co-workers
- Lack of self-care, including poor hygiene and a drop-off in your appearance
- Recurring nightmares of flashbacks
- Substance abuse or other compulsive behaviors such as over-eating or gambling

Source: American Veterinary Medical Association. Work and compassion fatigue.

Box 77.2 Personal Approaches to Combat Compassion Fatigue

- Focus on building your resilience
- Take time to be alone with yourself
- Engage in meditation and/or mindfulness-based stress reduction
- Engage with co-workers
- Connect with other colleagues
- Practice expressive writing
- Practice your spiritual beliefs
- Complete basic hygiene tasks
- Wash up before you leave work

Source: American Veterinary Medical Association. Work and compassion fatigue.

to help veterinary professionals reduce the impact of compassion fatigue (Box 77.2) so they may continue doing more of what they love to do, both personally and professionally.

Burnout

Another often referred to construct that presents a significant number of resources in the veterinary space is burnout. According to Hayes et al. [3]:

> Burnout has been defined as a psychological state typified by emotional exhaustion, depersonalization, or cynicism towards patients and colleagues, and a reduced sense of personal accomplishment. Burnout syndrome has been documented in a wide range of health care professions, and job stress is recognized as the principal cause of burnout.

Stressors associated with burnout are often linked to the workplace in areas such as level of exhaustion and the many nuances associated with practice culture. Examples of these stressors may be seen when practice team members are scheduled to work long intervals of overtime, the practice leadership is perceived by team members as not being approachable to ask questions, or may be consistently overwhelmed by a constantly overscheduled patient caseload.

To reduce the symptoms of compassion fatigue and burnout (Box 77.3), experts advise the necessity of self-care. Examples of self-care in regard to compassion fatigue might include taking care of yourself physically and mentally and connecting with others to discuss successes and challenges. Through self-care and reflection, you may remember what brings you joy in your life Asking yourself: are you experiencing that joy and if not, why not? And if so, how can you do more of that joy?

Examples of self-care in regard to burnout (Box 77.4) are very similar to those in compassion fatigue. They may also include moments of reflection to determine whether your current work environment is still working for you. How much of the increased cases and stress is associated with a temporary change event or is this truly the culture? Do you have a voice that is heard by the practice leadership? The reflective self-care may help to determine whether you stay or go.

The examples for both compassion fatigue and burnout both indicate reflection, which may present uncomfortable

Box 77.3 Common Symptoms of Burnout

- Excessive stress
- Fatigue
- Insomnia
- Sadness, anger, or irritability
- Alcohol or substance misuse
- Vulnerability to illnesses

Source: Mayo Clinic. Job burnout: How to spot it and take action.

Box 77.4 Handling Burnout

- Evaluate your options
- Seek support
- Try a relaxing activity
- Get some exercise
- Get some sleep
- Mindfulness

Source: Mayo Clinic. Job burnout: How to spot it and take action.

processes. Making significant life changes is very rarely easy. Doing the work and reflecting on where you are now and where you would like to go will provide increased chances for positive change and making goals a reality.

Self-Care and Wellbeing

As we saw with the compassion fatigue and burnout discussions, self-care is critical to reduce many potentially debilitating symptoms. Self-care intersects these two constructs, is included in our roadmaps to wellbeing, and comes in many different forms.

This chapter refers to wellbeing and constructs related to wellbeing. According to the American Association of Veterinary Medical Colleges (AAVMC) [4], "Health is a state of being, often represented by numbers in a medical history. In contrast, wellbeing is a state of living that speaks to our overall quality of life". The AAVMC reference to health being "represented by numbers" refers to the medical diagnostic results included in our personal medical history. The definition of wellbeing used by the National Wellness Institute, "Well(being) is an active process through which people become aware of, and make choices toward, a more successful existence".

According to the American Veterinary Medical Association (AVMA) [5]:

> "Your mental and physical wellbeing depends largely on your ability to care for yourself in addition to your patients. You do not have to do it alone, but you have to do it. You're the one who has to prioritize your own care as well as that of your patients and clients. Why? It is simple: If you are not taking care of yourself, you'll be less able to care for others. Your own wellbeing affects your ability to care for your patients and your loved ones."

Self-care may be a personal challenge for many veterinary professionals to weave into their life routines due to many variables, which may include, "I don't have time" or "I don't know where to start." Concerning where to start, the AVMA lists nine dimensions of wellbeing [5] that allows opportunities to reflect and target areas that may need your attention. When reflecting on these nine dimensions (Box 77.5), you are trying to identify gaps in your wellbeing. If you identify a gap or a deficit, consider referring to the resources provided at the end of this chapter. Assessing the nine dimensions of wellbeing may be a first step toward identifying where to start to develop self-care plans and help establish goals, improve wellbeing, and do more of what we love to do.

We have discussed terms and concepts that may already be familiar to many veterinary professionals. Even if this serves as a refresher, the reminder for the common denominator, self-care, may prove helpful to keep on all our radars and assess our progress.

Box 77.5 The Nine Dimensions of Wellbeing

1) Occupational – Being engaged in work that gives you personal satisfaction, and aligns with your values, goals, and lifestyle
2) Intellectual – Learning new things; Participating in activities that foster critical thinking and expand your worldviews
3) Spiritual – Having a sense of inner harmony and balance
4) Social – Surrounding yourself with a network of support built on mutual trust, respect, and compassion
5) Emotional – Being able to identify and manage your full range of emotions, and seeking help when necessary
6) Physical – Taking care of your body e.g. getting enough sleep, eating a well-balanced diet, exercising regularly, etc.
7) Financial – Being aware of your personal finances and adhering to a budget that enables you to meet your financial goals
8) Creative – Participating in diverse cultural and artistic experiences
9) Environmental – Taking an active role in preserving, protecting, and improving the environment

Source: American Veterinary Medical Association. The nine dimensions of wellbeing.

Introducing Self-Compassion

The introduction of the construct self-compassion allows for yet another dimension of self-care. What happens when things do not go as planned, whether in our personal or professional lives? What do we tell ourselves? Are we as kind to ourselves as we are to our colleagues and friends? There is a significant amount of research, literature, and empirical data associated with the self-compassion construct. In the context of this chapter, insight and steps to help veterinary professionals understand and practice self-compassion are provided.

In spite of all the things veterinary professionals do every day in practice, such as providing patient care, communicating with clients, and working with team members, we also often hold onto events we believe did not go as well as planned. We may ruminate about how "I could have done something differently," or "Why didn't I?" or "If I only," and castigate ourselves for things that may have been out of their control.

What happens if a mistake was actually made? Do we continue to ruminate about the "woulda, coulda, shoulda"? Do we get stuck and incessantly beat ourselves up? We may hold onto those events to the detriment of our own

wellbeing, which leads us to the important self-care construct, self-compassion.

Self-compassion dives into how truly giving ourselves a break and treating ourselves as we would our own best friend, if in need of support, is OK. Caring for and demonstrating empathy toward others is nothing most veterinary professionals need to learn. Caring for ourselves and giving ourselves a break with as much compassion is. Exploring the components of self-compassion will provide additional insight into how we may strengthen our heart, mind, soul and, body to, as mentioned previously, do more of what we love to do.

The self-compassion construct is supported by more than 4000 studies and significant empirical data are available. Much of the research associated with self-compassion included in this chapter comes from the studies conducted by one of the leading self-compassion researchers, Dr. Kristin Neff. In addition to the discussion, available resources are listed for additional information.

Self-Compassion: What is It?

According to Dr. Kristin Neff [6], "Having compassion for oneself is really no different than having compassion for others." Neff provides an example [6]:

> Think about what the experience of compassion feels like. First, to have compassion for others you must notice that they are suffering. If you ignore that homeless person on the street, you cannot feel compassion for how difficult his or her experience is. Second, compassion involves feeling moved by others' suffering so that your heart responds to their pain (the word compassion literally means to "suffer with"). When this occurs, you feel warmth, caring, and the desire to help the suffering person in some way. Having compassion also means that you offer understanding and kindness to others when they fail or make mistakes, rather than judging them harshly. Finally, when you feel compassion for another (rather than mere pity), it means that you realize that suffering, failure, and imperfection is part of the shared human experience.

Veterinary professionals have the compassion for others down perfectly. Now, to address the compassion for ourselves.

Neff's research continues by acknowledging how the compassion we express for others may lead us to look inward and show that same compassion for ourselves [6]:

> Self-compassion involves acting the same way toward yourself when you are having a difficult time, fail, or notice something you don't like about yourself. Instead of just ignoring your pain with a 'stiff upper lip' mentality, you stop to tell yourself 'This is really difficult right now,' how can I comfort and care for myself in this moment?

Neff's body of work with self-compassion encompasses propositions that are relevant to the veterinary profession, as can be seen with this consideration, "Instead of mercilessly judging and criticizing yourself for various inadequacies or shortcomings, self-compassion means you are kind and understanding when confronted with personal failings – after all, who ever said you were supposed to be perfect?" [6].

Self-Compassion: Three Core Elements

According to Neff's self-compassion model, there are three core elements: self-kindness, common humanity, and mindfulness.

Kindness

The motivational core of self-compassion is kindness. According to Neff [7]:

> It's a warm, friendly, and supportive attitude toward ourselves as we wade through the mud of life. Too often when we struggle, we are more likely to beat ourselves up than put a supportive arm around our own shoulder. Even people who are unfailingly kind to others often treat themselves like crap. Self-kindness reverses this tendency so that we are genuinely good to ourselves.

Veterinary professionals may have learned about the impostor syndrome construct that addresses perfectionism. In relation, according to Neff, "We can't be perfect. Our lives will inevitably involve struggle." "Self-kindness provides the resources to cope with hardship and makes it more bearable. It's a rewarding and fulfilling emotion, the sweetness that counters the bitterness of life."

Common Humanity

According to Neff's research, "Also central to self-compassion is recognition of our own humanity. In fact, this is what differentiates self-compassion from self-pity." Neff's studies revealed that, "Compassion is predicted on the idea that all conscious beings are intrinsically worthy of humane treatment". This includes you. It is to be hoped that this resonates with all veterinary professionals who read this chapter.

Mindfulness

According to Neff, "The foundation of self-compassion is the ability to turn mindfully toward our discomfort and acknowledge it". Neff continues, "Mindfulness allows us to see clearly when we've made a mistake or failed." Turning toward zones of discomfort and acknowledging it is often difficult, but Neff's research reveals that, "Mindfulness is essential to self-compassion so we can know when we're suffering and respond with kindness."

Self-Compassion: What it is Not

Self-Compassion Is Not Self-Pity

According to Neff's research [6]:

> When individuals feel self-pity, they become immersed in their own problems and forget that others have similar problems. They ignore their interconnections with others, and instead feel that they are the only ones in the world who are suffering. Self-pity tends to emphasize egocentric feelings of separation from others and exaggerate the extent of personal suffering. Self-compassion, on the other hand, allows one to see the related experiences of self and other without these feelings of isolation and disconnection.

In addition, the self-pity card may play a role in "I am the victim," thus justifying negative behavior and justification for stoking self-pity.

Self-Compassion Is Not Self-Indulgence

According to Neff's research [6]:

> Self-compassion is also very different from self-indulgence. Many people say they are reluctant to be self-compassionate because they're afraid they would let themselves get away with anything. 'I'm stressed out today so to be kind to myself I'll just watch TV all day and eat a quart of ice cream.' This, however, is self-indulgence rather than self-compassion. Remember that being compassionate to oneself means that you want to be happy and healthy in the long term.

Neff's research reveals more about the impact of indulgence [6]:

> In many cases, just giving oneself pleasure may harm wellbeing (such as taking drugs, over-eating, being a couch potato), while giving yourself health and lasting happiness often involves a certain amount of displeasure (such as quitting smoking, losing weight, exercising). People are often very hard on themselves when they notice something they want to change because they think they can shame themselves into action – the self-flagellation approach. However, this approach often backfires if you cannot face difficult truths about yourself because you are so afraid of hating yourself if you do. Thus, weaknesses may remain unacknowledged in an unconscious attempt to avoid self-censure. In contrast, the care intrinsic to compassion provides a powerful motivating force for growth and change, while also providing the safety needed to see the self clearly without fear of self-condemnation.

Self-Compassion Is Not Self-Esteem

Neff's research contrasts the two constructs [6]:

> Although self-compassion may seem similar to self-esteem, they are different in many ways. Self-esteem refers to our sense of self-worth, perceived value, or how much we like ourselves. While there is little doubt that low self-esteem is problematic and often leads to depression and lack of motivation, trying to have higher self-esteem can also be problematic. In contrast to self-esteem, self-compassion is not based on self-evaluations. People feel compassion for themselves because all human beings deserve compassion and understanding, not because they possess some particular set of traits (pretty, smart, talented, and so on). This means that with self-compassion, you do not have to feel better than others to feel good about yourself.

Self-Compassion Assessments

According to Neff and Germer [8], "The path to self-compassion often begins with an objective assessment of how self-compassionate or not we are". Researchers of self-compassion frequently use the self-compassion scale to measure the link between self-compassion and wellbeing. Neff and Germer have adapted a shorter version (Protocol 77.1) of the scale. This gives an idea of the reflective questions that are included in the assessments. Details of the electronic version of this assessment, as well as the full version, are available in the resources section at the end this chapter.

As a reminder, "The path to self-compassion often begins with an objective assessment of how self-compassionate or not we are" [8]. Scores are intended to give insight into the level of self-compassionate you may or not have toward yourself. It reflects a snapshot in time and may act as a starting point to learn more about yourself.

Protocol 77.1 Self-Compassion Scale Short Form

How I Typically Act Towards Myself in Difficult Times

Please read each statement carefully before answering. Indicate how often you behave in the stated manner, using the following scale:

Almost never-1, Occasioanally-2, About half of the time-3,
Fairly often-4, Almost always-5

1) When I fail at something important to me I become consumed by feelings of inadequacy.
2) I try to be understanding and patient towards those aspects of my personality I don't like.
3) When something painful happens I try to take a balanced view of the situation.
4) When I'm feeling down, I tend to feel like most other people are probably happier than I am.
5) I try to see my failings as part of the human condition.
6) When I'm going through a very hard time, I give myself the caring and tenderness I need.
7) When something upsets me I try to keep my emotions in balance.
8) When I fail at something that's important to me, I tend to feel alone in my failure.
9) When I'm feeling down I tend to obsess and fixate on everything that's wrong.
10) When I feel inadequate in some way, I try to remind myself that feelings of inadequacy are shared by most people.
11) I'm disapproving and judgmental about my own flaws and inadequacies.
12) I'm intolerant and impatient towards those aspects of my personality I don't like.

Scoring Key

Self-Kindness Items:	2, 6
Self-Judgment Items (Reverse Scored):	11, 12
Common Humanity Items:	5, 10
Isolation Items (Reverse Scored):	4, 8
Mindfulness Items:	3, 7
Over-identification Items (Reverse Scored):	1, 9
To reverse score items	(1=5, 2=4, 3=3, 4=2, 5=1).

To Compute a Total Self-Compassion Score

- First reverse score the negative subscale items: self-judgment, isolation, and over-identification
- Then take the mean of each subscale
- Next compute a total mean (the average of the six subscale mean)

Norms and Score Significance

There are no clinical norms or scores which indicate that an individual is high or low in self-compassion. Rather, scores are mainly used in a comparative manner to examine outcomes for people scoring higher or lower in self-compassion.

You can consider these scores (as an ad hoc rubric):

LOW: 1.0–2.49
MODERATE: 2.5–3.5
HIGH: 3.51–5.0

(When trying to determine whether self-compassion levels are high or low relevant to a particular sample, some researchers use a median split.)

Source: Neff K. [9] Dr. Kristin Neff has granted permission to share this assessment.

Protocol 77.2 How Would You Treat a Friend?

Please take out a sheet of paper and answer the following questions:

1) First, think about times when a close friend feels really bad about themselves or is really struggling in some way. How would you respond to your friend in this situation (especially when you are at your best)? Please write down what you typically do, what you say, and note the tone in which you typically talk to your friends.
2) Now think about times when you feel bad about yourself or are struggling. How do you typically respond to yourself in these situations? Please write down what you typically do, what you say, and note the tone in which you talk to yourself.
3) Did you notice a difference? If so, ask yourself why. What factors or fears come into play that lead you to treat yourself and others so differently?
4) Please write down how you think things might change if you responded to yourself in the same way you typically respond to a close friend when you are suffering.

Why not try treating yourself like a good friend and see what happens?

Growing self-compassion starts with assessing, reflecting, listening, and learning more about how we can care as much, if not more, about ourselves as we do others. Taking time out to check in regularly with ourselves and engaging in reflective exercises is helpful toward not only our self-compassion but also our wellbeing. Neff [6] provides an example of a reflective exercise (Protocol 77.2). There are more exercises and information on how to grow your self-compassion in the resources section at the end of this chapter.

Conclusion

Veterinary professionals do many tremendously good things for those they serve in every work day. They provide medical care to patients, communicate with clients to help them understand what is best for their pet, and engage with their practice team on many levels. They demonstrate empathy and kindness to practice stakeholders even when they may be on fumes from exhaustion or constant stress.

Learning about wellbeing and self-compassion is about being mindful that veterinary professionals are also worthy of compassion and kindness, especially from ourselves.

Since much of this chapter highlights the work of self-compassion expert, Dr. Kristin Neff, I end this chapter with a few words from her most recent book, *Fierce* [7]:

> I've been practicing self-compassion daily for almost twenty-five years now. Although I'm definitely stronger, calmer, and happier because of it, my inner bulldog barks less often than it used to, I still struggle. I'm as imperfect as ever, and this is how it should be. Being human is not about getting it right, it is about opening up your heart-whether you get it wrong or right. I've learned to do this over time, just by moving through all my mistakes and difficult experiences.

I trust this may resonate with all my veterinary colleagues and friends.

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Companion website: www.wiley.com/go/burkitt/monitoring

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