Small animal emergency care

Quick reference guide

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For this English edition:
Small animal emergency care. Quick reference guide

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Preface

The purpose of *Small Animal Emergency Medicine. Quick Reference Guide* is to present the basic principles of diagnosis and treatment of the most common emergencies encountered in small animal medicine. This pocket guide is a quick reference for veterinarians to rapidly access information in a simple format. It is also quite useful for veterinary students and recent veterinary graduates starting out in emergency medicine. All diagnostic and therapeutic recommendations are based on the current literature and involve procedures that are likely available to most clinicians consulting this guide.

The book is presented in the logical, traditional order used to evaluate patients in the emergency department; that is, according to ABCD. It begins with an introductory chapter that describes the systematic evaluation of emergency patients (triage and initial assessment), followed by a brief discussion of common emergencies categorised by type: cardiovascular, respiratory, hematological, gastrointestinal, urological, neurological, metabolic, reproductive and, finally, environmental.* All the emergencies are discussed in the following order: definition of the pathology, most common causes in small animals, diagnostic protocol, and currently recommended management approaches.

I hope that this book will be useful for general practitioners who are starting out in this exciting specialty of small animal emergency medicine.

Carlos Torrente Artero

*The digital version of the book includes an extra chapter on toxicological emergencies.

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1

Triage and initial assessment

The term “triage” means “to choose” and describes prioritising patient care according to the severity of patient condition and whether emergency care is necessary.

Remote triage

Initial contact between the owner of a pet needing emergency care and the veterinarian often occurs by phone. The information obtained during the phone conversation can often help determine whether the patient needs urgent, immediate care and should thus be brought to the clinic promptly and whether the clinic staff should prepare for special procedures (e.g. emergency surgery).

The basic information should include:

- What happened.
- When it happened.
- What has been done since it happened.
- What concurrent problems the patient has.

In the initial phone evaluation, four major organ systems are indirectly evaluated in order to determine the patient’s stability. These are the respiratory, cardiovascular, central nervous (CNS), and urogenital systems. The most common emergent problems with these systems are as follows:

- Difficulty breathing.
- Trauma.
- Neurological abnormalities/severely depressed mentation.
- Severe vomiting/diarrhoea.
- Visible bleeding.
- Pale mucous membranes.

- Extreme weakness or inability to walk.
- Inability to urinate.
- Ingestion of toxins or foreign bodies.
- Severe pain.

In-person triage

Triage is an essential part of emergency patient evaluation and should include basic, concise communication with the owner. This type of triage should be performed within minutes of admission and require only a few minutes. Postpone taking a detailed history until the patient has been minimally stabilised. At this stage, only basic information is required, including:

- The cause of the lesion or problem.
- When the clinical signs started.
- What emergency treatments the patient has already received.

Once basic information has been collected, evaluate the patient's respiratory, cardiovascular, and neurological status by observing the efficacy and effort of the patient's breathing, signs of generalised hypoperfusion, mentation level, and ability to walk. Based on the above, cases should be prioritised as follows:

- **Level 1: patient in catastrophic or life-threatening condition.** These patients require immediate treatment, within a matter of seconds. Fortunately, such patients rarely present for emergency care. Examples include: patients with breathing difficulty due to thoracic trauma, those with cardiopulmonary arrest, and those with airway obstruction. Any unconscious patient a priori falls in this category.
- **Level 2: very severe or critical patient.** Treatment should be instituted within one hour of admission. Include in this category patients with multiple lesions and those who are in shock or bleeding but appear to have a patent airway and adequate lung function.

- **Level 3: severe patient.** Treatment should be instituted within a few hours of admission. Patients with open fractures, deep or penetrating wounds, or burns but lacking signs of shock or altered mentation fall into this category.
- **Level 4: less severe patient.** Treatment should be instituted within 24 hours of admission. Most veterinary trauma victims do not fall into this category, but some are brought in only later when the owner notes problems with walking, lameness, anorexia, vomiting, etc.

Consider emergency treatment of patients who do not present with altered vital signs but may have suffered:

- Poisoning by recent exposure to or ingestion of toxic substances.
- Recent seizures.
- Potentially severe trauma.
- Difficulty in urination or inability to urinate.
- Excessive bleeding.
- Organ prolapse.
- Hyperthermia.
- Open wounds.
- Fractures.
- Burns.
- Dystocia.

Primary assessment

The primary assessment should be rapid (few minutes) and evaluate the patient's ABCD (Box 1); it should be sequential and recorded in writing, and it should focus on identifying the optimal medical treatments for stabilisation.

The assessment should be performed in a strictly prescribed order, such that no body system is examined without having addressed the needs of the previous system. In this way, airway obstructions are detected and eliminated before proceeding to the following systems:

1. **Airway (A).** Securing a patent airway is the **main priority** for an emergency patient. The airway should be examined by auscultating breath sounds and checking for normal thoracic expansion, while also palpating and visually examining the oral cavity, trachea, and larynx, taking into account:
   - Potential airway obstructions or lesions: perform an orotracheal intubation if necessary.
   - Presence of blood, secretions, vomit, foreign bodies, or masses that may obstruct the airway: manual removal or suction may be necessary to clear the airway.
   - A comatose or moribund patient should always be intubated.
   - If necessary, cricothyroidotomy, transtracheal catheterisation, or emergency tracheostomy can quickly create a patent airway.

Box 1.

Patient ABCD.

- **A** : airway.
- **B** : breathing.
- **C** : circulation, cardiovascular system.
- **D** : disability, CNS deficits.

- Use whatever oxygen source is available to provide supplemental oxygen to the patient as needed. Oxygen will often alleviate dyspnoea and agitation, allowing the patient to breathe more effectively.

2. **Respiratory system (B).** Evaluation of the respiratory system is the second priority for the emergency patient. It should focus on examination, palpation, auscultation and percussion of organs involved in respiration, specifically: lung parenchyma, secondary bronchi, rib cage, and diaphragmatic and intercostal muscles. Evaluate:
   - Breathing: if the patient is not breathing, secure a patent airway, and intubate and ventilate immediately.
   - Respiratory rate, pattern, depth, effort, and breath sounds.
   - Presence of cyanosis.

3. **Cardiovascular system (C).** The cardiovascular exam should evaluate the following:
   - Heart rate and rhythm: absence of heart beat or pulse is an indication for the cardiopulmonary arrest protocol; extreme bradycardia or tachycardia may compromise cardiac output and tissue perfusion.
   - Pulse (quality, rate, and rhythm): asynchrony, irregular pulse, and absence of a detectable peripheral pulse in the distal limbs may be indicative of cardiovascular compromise.
   - Capillary refill time (CRT): excessively prolonged CRT may indicate peripheral vasoconstriction or hypoperfusion.
   - Mucous membrane colour: pale or cyanotic membranes are abnormalities that require immediate patient evaluation.
   - Body temperature: a difference of more than 4 °C between core and peripheral temperatures suggests insufficient peripheral tissue perfusion and tends to result from vasoconstriction. Temperatures <34 °C or >41 °C may be life-threatening.

- Cardiovascular abnormalities should be quickly corrected. It is critical to detect and control any haemorrhage:
  - External haemorrhages may be initially controlled using sterile dressings.
  - Arterial bleeds may be controlled by manually applying pressure to sponges around the wound, applying compressive bandages, or, if the bleed is identified, using haemostats and ligation.

4. **Central nervous system (D).** Evaluate:
   - Mentation: extreme alterations (stupor, coma, or seizures) require rapid determination of the underlying cause and immediate treatment. Metabolic causes (hypoglycaemia) and causes of increased intracranial pressure should also be considered.
   - The presence of obvious head or spinal cord lesions (asymmetry, displacements, etc.).

Secondary assessment

The secondary assessment takes place during patient recovery and stabilisation. It includes a more thorough physical examination, a more detailed history, and diagnostic testing: radiographs and ultrasound; complete, specific laboratory testing; as well as special procedures (invasive or noninvasive). The secondary assessment enables the clinician to make decisions regarding definitive therapy, prognosis, and options for patient management.

2

Cardiovascular emergencies

Hypovolaemic shock

Definition

The term shock refers to a syndrome of multifactorial aetiology characterised by insufficient cellular energy production, commonly associated with tissue hypoperfusion and usually caused by a decrease in or abnormal distribution of blood flow to tissues. It is often characterised by a mismatch between tissue oxygen supply and demand due to inadequate delivery or inefficient cellular use.

Causes

In most critical patients, shock results from a significant reduction in effective oxygen delivery to the tissues (DO_2). It is mainly due to loss of intravascular volume (hypovolaemic shock), poor distribution of vascular fluid (distributive shock) or failure of the heart to pump (cardiogenic shock) (Box 1).

Hypovolaemic shock can be caused by significant blood loss (internal or external haemorrhage), extracellular fluid loss that exceeds fluid and solute intake (repeated vomiting, diarrhoea, polyuria), or internal loss of plasma volume due to fluid exudation or transudation into the extravascular space.

Box 1.

Types and causes of shock.

Hypovolaemic, due to a loss of circulating blood volume:

- Haemorrhage (external or internal).
- Severe dehydration (polyuria/polydipsia, vomiting, diarrhoea, burns, etc.).
- Trauma.

Cardiogenic, due to decreased antegrade blood flow from the heart:

- Congestive heart failure.
- Arrhythmias.
- Cardiac tamponade.
- Drugs (anaesthetics, beta blockers, calcium channel blockers, etc.).

Distributive, due to a decrease in systemic vascular resistance:

- Sepsis.
- Obstruction (dirofilariasis, arterial thrombosis).
- Anaphylaxis.

Metabolic, due to compromise of cellular energy synthesis:

- Hypoglycaemia.
- Cyanide poisoning.
- Mitochondrial dysfunction.
- Cytopathic hypoxia (sepsis).

Hypoxaemic, due to decrease in arterial oxygen (CaO_2):

- Anaemia.
- Severe pulmonary disease.
- Carbon monoxide poisoning.
- Methaemoglobinaemia.

Diagnosis

Diagnosis and classification of this type of shock are based on the clinical history (which can suggest total or plasma losses and internal or external losses of intravascular volume), as well as the evaluation of physical and analytical perfusion parameters. Physical parameters include mentation, mucous membrane colour, capillary refill time, pulse pressure (amplitude and duration), arterial blood pressure, heart rate, cardiac auscultation and, finally, central temperature and the difference between central and peripheral temperatures.

In uncomplicated hypovolaemia, physical and analytical perfusion indicators (e.g. lactate) tend to change predictably, which lets us estimate the severity of the fluid loss and classify the type of shock in the patient:

- Compensated shock.
- Early decompensated shock.
- Late decompensated shock.

Table 1 shows the changes in physical indicators of perfusion according to the degree of hypovolaemia.

Various diagnostic procedures should be performed in every patient with signs of shock in order to determine the degree of organ compromise and the potential aetiology. These include blood count, complete serum biochemistry, coagulation profile, urinalysis, lactate levels, and blood gas (preferably arterial) analysis. Diagnostic imaging should be performed only after stabilising the patient.

Table 1. Changes in physical perfusion parameters according to degree of hypovolaemia.

Clinical signs	Hypovolaemic compensated shock	
Mentation	Alert or moderately depressed	
Temperature	Normothermic	
Heart rate (bpm)	130–150 (cats: possible bradycardia)	
Mucous membranes	Pink or congested	
CRT	<2 s	
Pulse amplitude	Increased	
Pulse duration	Slightly decreased	
Metatarsal pulse	Easily palpated	
Arterial blood pressure (mmHg)	Normal: • SBP >100 • MAP >80	
Lactate (mmol/l)	3–5	

CRT: capillary refill time; SBP: systolic blood pressure; MAP: mean arterial pressure.

Treatment

Early recognition and rapid initiation of treatment are critical to successful treatment of shock patients. While the details may differ, the foundation of hypovolaemic shock therapy is intravenous fluid support. Different fluids that expand the circulatory volume are available: isotonic crystalloids, hypertonic crystalloids, and synthetic colloids. Although these can be equally effective at treating shock at appropriate doses, recommendations vary depending on the patient's condition, degree of dehydration, and the presence of active bleeding or cerebral or pulmonary lesions (in such cases it is best to use limited volumes and boluses to effect).

	Early hypovolaemic decompensated shock	Late hypovolaemic decompensated shock
	Depressed	Significantly depressed
	Hypothermic	Severely hypothermic
	150–170 (cats: possible bradycardia)	170–220 (cats: possible bradycardia)
	Pink or pale	White or greyish
	2 s	>2 s
	Decreased	Very decreased
	Decreased	Very decreased
	Palpable	Not palpable
	Decreased: • SBP: 80–100 • MAP: 60–80	Very decreased: • SBP <80 • MAP <60
	5–8	>8

The choice of fluids and rate of administration depend on the patient's condition and the degree of haemodynamic compromise. In general, fluid therapy should be gradual and incremental, constantly reevaluating, quantifying, and determining the total volume of fluids the patient needs based on the patient's response to each administration. Therapeutic recommendations vary based on the degree of hypovolaemia and whether concurrent pathologies or conditions exist (Table 2).

Table 2. Treatment recommendations based on degree of hypovolaemia and concurrent clinical conditions.

Fluid type	Uncomplicated hypovolaemia*			
	Mild (loss of 10–20 % of blood volume)	**Moderate** (loss of 20–30 % of blood volume)	**Severe** (loss of >30–40 % of blood volume)	
Isotonic crystalloids	• Dog: 20–40 ml/kg. • Cat: 10–20 ml/kg.	• Dog: 40–60 ml/kg. • Cat: 20–40 ml/kg.	• Dog: 60–90 ml/kg. • Cat: 40–60 ml/kg.	
	Practical protocol with initial dose of isotonic crystalloids for all types of hypovolaemia: • Dog: 10 ml/kg (mild), 20 ml/kg (moderate) or 30 ml/kg (severe) over 10–15 minutes. • Cat: 10–20 ml/kg over 10–15 minutes, with simultaneous passive or active warming in case of hypothermia.			
Colloids	Variable indications (hypoproteinaemia): • Dog: 5 ml/kg bolus. • Cat: 2.5 ml/kg bolus.	• Dog: 5–10 ml/kg. • Cat: 5 ml/kg.	• Dog: 10–20 ml/kg. • Cat: 5–10 ml/kg.	
	Practical protocol with initial colloid dose for all types of hypovolaemia: • Dog: 5–10 ml/kg over 10–15 minutes. • Cat: 2.5–5 ml/kg over 10–15 minutes, with simultaneous passive or active warming in case of hypothermia.			
Hypertonic crystalloids (7.5 %)	Not indicated.	Variable indications; use low doses in large patients who are not dehydrated.	• Dog: 4–7 ml/kg over 5–10 minutes. • Cat: 2–4 ml/kg over 5–10 minutes.	

Adapted from Fragio et al., 2012.

*Due to controlled haemorrhage, dehydration, gastrointestinal or urinary losses, and third space losses (into body cavities).

	Hypovolaemia with cranioencephalic trauma	Hypovolaemia with pulmonary contusion	Hypovolaemia with abdominal haemorrhage
	• Dog: 40–90 ml/kg. • Cat: 20–60 ml/kg. Depends on the severity of hypovolaemia.	• Dog: 10–15 ml/kg. • Cat: 5–10 ml/kg.	• Dog: 10–20 ml/kg. • Cat: 5–15 ml/kg.
	During the infusion, evaluate the response and repeat if necessary to a maximum dose of 90 ml/kg in dogs and 60 ml/kg in cats.		
	• Dog: up to 20 ml/kg. • Cat: up to 15 ml/kg.	• Dog: 5 ml/kg bolus. • Cat: 2.5 ml/kg bolus.	2.5–5 ml/kg.
	During the infusion, evaluate the response and repeat if necessary to a maximum dose of 20 ml/kg in dogs and 15 ml/kg in cats.		
	• Dog: 4–7 ml/kg over 5–10 minutes. • Cat: 2–4 ml/kg over 5–10 minutes.	• Dog: 2–4 ml/kg over 5–10 minutes. • Cat: 1–2 ml/kg over 5–10 minutes.	• Dog: 2–4 ml/kg over 5–10 minutes. • Cat: 1–2 ml/kg over 5–10 minutes.

If blood loss is a priori significant or acute, or if the haematocrit after initial fluid therapy with crystalloids falls below 20 %, patients in hypovolaemic shock may require a blood transfusion. Given that the dose and rate of administration of blood products vary with the patient's haemodynamic status, transfusion of blood, packed red blood cells, or plasma should be considered in any patient with acute haemorrhage who does not respond adequately to traditional resuscitation techniques. The therapeutic target for blood products is to maintain the haematocrit above 25 % and normalise clotting times.

Whole blood can be given at rates of 20–25 ml/kg/h if necessary, and packed red blood cells and fresh frozen plasma can be given at rates of 10–20 ml/kg/h. However, administration over a longer duration (2–3 hours), while taking the aforementioned precautions, enables safer monitoring of the transfusion process, avoiding complications such as circulatory overload and transfusion reactions.

The combination of factors including the species, presenting complaint, examination findings, etc., can help the clinician determine what type of shock the patient has and carefully evaluate the most appropriate measures of care in each case. Table 3 shows the value for each parameter that clinicians should aim to reach in most of their shock patients.

Table 3. Acceptable values in patients recovering from hypovolaemic shock.

Parameter	Value
Mentation	Alert
Mucous membranes	Pink
Capillary refill time	<2 s
Heart rate	• Cats: 180–220 bpm • Small-breed dogs: 100–160 bpm • Large-breed dogs: 60-100 bpm
Respiratory rate	20–40 rpm
Systolic blood pressure (SBP)	>100 mmHg*
Mean arterial pressure (MAP)	>80–100 mmHg*
Central venous pressure	5–10 cmH_2O
Lactate	<2.5 mmol/l
Urine output	At least 1–2 ml/kg/h

*Active, noncompressible, or cavitary haemorrhages may be exceptions. In such situations, an MAP of 70 mmHg or SBP of 90 mmHg with an improvement in clinical signs during gradual administration of limited fluid volumes is considered acceptable until the haemorrhage can be definitively controlled.

Cardiopulmonary arrest

Definition

Cardiopulmonary arrest (CPA) is characterised by the abrupt cessation of breathing and the electromechanical activity of the heart.

Causes

Cardiopulmonary arrest (CPA) is characterised by the abrupt cessation of breathing and the electromechanical activity of the heart. CPA is caused by metabolic or organ failure linked to severe or persistent states of hypoxaemia, hypercapnia, acid-base imbalance, vasovagal responses (e.g. pain), hypothermia, electrolyte disturbances, hypotension, hypertension, or arrhythmias. The appearance and persistence of one or more of the aforementioned conditions in the presence of pathologies such as systemic inflammatory response syndrome (SIRS)/ sepsis, heart failure, pulmonary disease, systemic neoplasms, coagulopathies, toxicities, polytraumas, or anaesthetic procedures predispose the patient to experiencing CPA.

Diagnosis

CPA is characterised by the loss of consciousness in the patient and the absence of spontaneous ventilation, heart sounds audible on auscultation, and detectable pulse pressure.

Treatment

The cardiopulmonary resuscitation procedure is based on basic life support (BLS), advanced life support (ALS), and prolonged life support (PLS).

Basic life support (BLS)

Cardiac massage

1. Patient positioning: lateral or dorsal recumbency on a hard surface.

2. Compressions:
   - Location: 4th–6th intercostal space, at the costochondral junction in small dogs and cats. In the mid-thoracic region of large dogs.
   - Method: using the heel of the hand, arms extended.
   - Rate: 100–120 thoracic compressions per minute, with a 1:1 relaxation:compression ratio and 30–50 % displacement of the thoracic wall.
   - Cycles: 2 minutes per resuscitator without breaks (or less than 10 seconds).

3. If external massage is ineffective: internal cardiac massage. This is an invasive technique that can cause massive haemorrhage and pulmonary, vascular, and cardiac injury (Box 2). The procedure is as follows:
   - Quickly surgically prepare the left thoracic wall and thoracotomy over the 5th or 6th intercostal space, from the dorsal origin of the rib to the sternum.
   - In between two breaths, make an incision into the pleural space using the tips of curved Mayo scissors or haemostats.
   - Widen the opening with separators, avoiding the internal thoracic artery.
   - Perform a pericardectomy and clamp the descending aorta caudal to the heart.
   - Hold the heart with the base in the palm of the hand and the fingers encircling the apex. Perform direct cardiac compressions at a rate similar to that of external massage.

Box 2.

Indications for internal cardiac massage.

- Large dogs.
- Pneumothorax or haemothorax.
- Severe pulmonary contusions.
- Flail chest.
- Thoracic trauma with rib fractures.
- Cardiac tamponade.
- Diaphragmatic hernia.
- External massage is not effective (2–5 minutes without effective circulation).

Patent airway

1. If a complete obstruction is suspected, proceed as follows:
- Extend the patient's neck and check the oral cavity for blood, vomit, foreign bodies, etc.
- Aspirate, clean, and remove any airway obstruction.
- If the obstruction remains:
  - Introduce an 18 or higher gauge needle through the tracheal rings or cricopharyngeal membrane and administer 100 % oxygen (0.2–0.5 l/kg/min).
  - Perform a tracheostomy.

2. If there is no airway obstruction: intubate and ventilate.

Assisted ventilation

Positive-pressure ventilation should be performed with oxygen, preferably 100 % oxygen, using an Ambu bag or intermittent positive-pressure ventilation (IPPV):

- Inspiratory:expiratory ratio of 1:2, with an inspiratory time of 1 s. Monitoring of end-tidal carbon dioxide ($ETCO_2$). Increasing quantities of CO_2 recorded on capnography-

capnometry during resuscitation are considered indicative of adequate BLS and early return of spontaneous circulation.

- Rate: 12–15 bpm (small dogs and cats), 10–12 bpm (large dogs).
- Cycle: minute volume of 10 ml/kg or airway pressure (P_{aw}) <20 cmH_2O.

Advanced life support (ALS)

Establishing venous access is important in drug and fluid therapy for patients in CPA. While central venous access is preferred, peripheral intravenous (IV), intraosseous (IO), or even intratracheal (IT) access may substitute if catheterisation is impossible or available staff is a limiting factor.

Electrocardiographic evaluation (ECG)

ECG evaluation of drug treatment of arrhythmias and underlying conditions that can cause or aggravate them (acidosis, hypoglycaemia, etc.).

Asystole:

- Adrenaline: 0.01 mg/kg IV, IO or IT (×2), every 3–5 minutes up to three times. If the patient does not respond: increase the dose to 0.1 mg/kg.
- Atropine: 0.04 mg/kg IV, IO or IT (×2), every 3–5 minutes up to three times.

PEA (pulseless electrical activity):

Same as in asystole.

Sinus bradycardia:

- Rate <60 bpm in dogs and <140 bpm in cats.
- Atropine: 0.04 mg/kg IV, IO or IT (×2), every 3–5 minutes up to three times.

Ventricular tachycardia:

- Lidocaine: 2 mg/kg IV or IO.
- Magnesium sulphate: 30 mg/kg IV.
- If the ventricular tachyarrhythmia is refractory or high-grade (R-on-T, polymorphic, or pulseless), give amiodarone: 2.5–5 mg/kg IV or IO.

Refractory ventricular fibrillation or ventricular tachycardia:

External defibrillation alternating with BLS cycles (2 minutes). If ineffective at first, the second dose can be increased by 50 %, but subsequent doses may not be similarly increased. After each defibrillation cycle, immediately resume thoracic compressions for a full 2-minute cycle, after which reassessment of the rhythm by ECG will show if defibrillation must be repeated. The recommended starting doses are:

- 4–6 J/kg (monophasic defibrillators).
- 2–4 J/kg (biphasic defibrillators).

Increase in vagal tone:

If vagal tone increases (increase in cerebrospinal fluid pressure, gastrointestinal disease, respiratory disease) correct the hypothermia, administer atropine, stop treatment, and reverse the drugs:

- Naloxone (0.01 mg/kg IV or IO) for opioids.
- Flumazenil (0.01 mg/kg IV or IO) for benzodiazepines.
- Atipamazole (0.1 mg/kg IV or IO) or yohimbine (0.1 mg/kg IV or IO) for α_2 adrenergic agonists.

Sinus tachycardia:

- Rate >160 bpm for large dogs, >180 bpm for small dogs, and >240 bpm for cats.
- Start symptomatic treatment to improve oxygenation and acid-base, electrolyte, and fluid balance, and to control pain.

Fluid therapy

Fluid therapy varies greatly depending on whether the patient is in hypovolaemic or euvolaemic CPA.

Hypovolaemic patient:

- Isotonic replacement crystalloids at a maximum dose of 60–90 ml/kg (dogs) or 40 ml/kg (cats) for the first hour, with subsequent reevaluation. It is preferable to give 1/4–1/2 dose at a time.
- Use colloids if necessary: slow 5 ml/kg bolus in dogs or 2.5 ml/kg bolus in cats.
- Administer 7.5 % NaCl if necessary: 1–4 ml/kg for 5–10 minutes.

Euvolaemic patient:

Initial bolus of isotonic crystalloids at 10–20 ml/kg in dogs and 5–10 ml/kg in cats.

Blood glucose normalisation

Glucose (0.5–1.5 ml/kg IV) is given as a slow drip in a 1:1 ratio with normal saline.

Acid-base balance

If the pH is <7.1, bicarbonate <10 mmol/l or cardiopulmonary resuscitation (CPR) is extended: give 0.5 mmol/kg sodium bicarbonate (3 ml/kg in an isotonic solution of $NaHCO_3$).

Electrolytes

Correct serum potassium, calcium, and magnesium levels.

Prolonged life support (PLS)

In the period following CPA, vital signs, temperature, tissue perfusion, and oxygenation and respiratory capacity of the patient should be monitored. Considerations include:

- Controlled induced hypothermia (33–34 °C) can improve survival.
- The $PaCO_2$ should be 35–40 mmHg.
- The PaO_2 should be >80 mmHg.
- The SaO_2 should be >94–96 %.
- The MAP should be >80 mmHg.
- The central venous pressure should be between 5 and 12 cmH_2O.
- Acidaemia and hyperlactataemia should be corrected.

Mentation and diuresis should be restored after the arrest. To achieve this, intravenous fluid therapy with crystalloids and/or colloids is recommended, along with a constant rate infusion (CRI) of inotropes or vasopressors if necessary:

- Dopamine: 5–20 µg/kg/min as a vasopressor. If the blood pressure does not respond, administer noradrenaline: 0.05–0.2 µg/kg/min.
- Dobutamine: 2.5–10 µg/kg/min as an inotrope.

Congestive heart failure

Definition

Congestive heart failure (CHF) is a syndrome that results from the heart's inability to pump blood in sufficient volumes to satisfy the metabolic demand of tissues. In most patients, signs include a decrease in cardiac output, a reflexive increase in peripheral vascular resistance, and an increase in venous pressure due to circulatory overload.

Causes

CHF can be categorised by its cause (pericardial, myocardial, valvular, or vascular disease or conduction disorder) or by its predominant pathophysiological malfunction (systolic or diastolic disorder, left or right-sided disorder).

It is important to identify the cause and type of heart failure because treatment depends on a precise diagnosis. The most common diagnoses in dogs are atrioventricular valve degeneration, dilated cardiomyopathy, and pericardial effusion.

Diagnosis

Diagnosis is based on the clinical history and the presence of appropriate signs. Exercise intolerance, night-time cough or exercise-induced cough (dogs), dyspnoea, cyanosis, and syncope are typical signs. The crucial sign is dyspnoea without primary respiratory disease (in most cases, the heart rate will be elevated).

In dogs, signs of left heart failure can vary but commonly include dyspnoea, tachypnoea, cough, cyanosis, haemoptysis, murmurs, arrhythmias, cachexia, cardiogenic shock, pulmonary oedema, and hypothermia. Right heart failure signs include pleural effusion, weak heart sounds, jugular venous distension (sometimes with a visible venous pulse), pale mucous membranes, hepatosplenomegaly, ascites, and syncope.

The most common signs in cats are tachypnoea, dyspnoea, murmur or gallop rhythm, arrhythmias, and an increase in adventitious breath sounds (crackles) or, in the case of a cavitary effusion, attenuation of heart and lung sounds (common).

Besides routine tests (blood count, clinical biochemistry, urinalysis, blood gas analysis, etc.) that can provide supplementary information, the most relevant diagnostics are thoracic radiographs, abdominal ultrasound, and ECG evaluation.

Treatment

The specific treatment depends on the degree and type of CHF, but in general it is based on administering oxygen therapy, decreasing the circulatory overload or its manifestations in body cavities (pleural effusion or ascites) and in the lung parenchyma (cardiogenic pulmonary oedema), maximising cardiac output, and ensuring proper myocardial contractility and heart rate:

- **Oxygen therapy:** free fluid, mask (if tolerated), O_2 cage, goggles, or nasal cannula. A flow of 50–100 ml/kg/min ensures effective O_2 concentrations of 40–60 %.
- **Sedation:** it is crucial to minimise stress and handling in some of these patients. Butorphanol (0.05.1 mg/kg IM, IV) or morphine (0.1–0.2 mg/kg) is recommended.
- **Pleurocentesis or abdominocentesis:** drain the pleural effusion. Drain any abdominal effusion if present and impeding ventilation, though this is of lower priority.

- **Diuretics:**
  - Furosemide: 2–6 mg/kg IV every 1–2 hours in dogs and 1–2 mg/kg IV every 1–2 hours in cats until the breathing pattern improves and the respiratory rate decreases; then decrease the dose to 2 mg/kg every 8–12 hours in dogs and 0.5–1 mg/kg every 12–24 hours in cats. In patients with treated CHF who experience an exacerbation, give furosemide as a constant rate infusion at 0.5–2 mg/kg/h.
  - Intensive administration of diuretics can compromise the patient's fluid and electrolyte balance; thus, it is crucial to strictly monitor the patient's creatinine, sodium, chloride, and potassium levels, and hydration status.
- **Vasodilators:**
  - Nitroglycerine (venous vasodilator) patch: 0.2 mg/kg/h, constant release during the first 24–48 hours of treatment of the signs of congestion when pulmonary oedema is present.
  - Enalapril or benazepril: 0.5–1.5 mg/kg PO every 12–24 hours.
  - Hydralazine (arterial vasodilator): 1–2 mg/kg PO every 12 hours. Its effect is unpredictable; it can induce intense vasodilation, hypotension, and increased heart rate.
  - If the patient does not respond to these treatments, sodium nitroprusside (arterial and venous vasodilator) can be administered: 1–10 µg/kg/min with continuous monitoring of arterial blood pressure and provision of inotropes as needed.
- **Positive inotropes.** If the patient presents with dilated cardiomyopathy or decreased cardiac output:
  - Dobutamine (inotrope): 5–20 µg/kg/min in dogs and 2.5–10 µg/kg/min in cats (contraindicated in cats with hypertrophic cardiomyopathy).

- Dopamine (inotrope and vasopressor): 5–20 µg/kg/min. Can be used in hypotensive patients or if dobutamine is not available. If the patient develops tachycardia or hypertension, taper the dose or discontinue the medication.
- Pimobendan (inotrope and vasodilator): 0.5 mg/kg every 12 hours PO in dogs and cats (contraindicated in cats with hypertrophic cardiomyopathy).

- **Antiarrhythmics.** Consider administration of the following in patients with supraventricular tachyarrhythmias or haemodynamically significant atrial fibrillation:
  - Esmolol: 0.05–0.25 mg/kg IV, followed by a continuous rate infusion of 50–200 µg/kg/min.
  - Propranolol: 0.02 mg/kg IV bolus to a maximum of 0.1 mg/kg.
  - In case of refractory atrial fibrillation or simultaneous contractile dysfunction (canine dilated cardiomyopathy), digoxin can be added: 0.010–0.015 mg/kg/12 hours PO; the following day, decrease to 0.005 mg/kg/12 hours.
  - In cats with hypertrophic cardiomyopathy, begin treatment with calcium channel blockers (diltiazem) at 0.1 mg/kg IV as a slow drip and CRI of 1 µg/kg/min until clinical improvement occurs; may be continued at a dose of 7.5 mg/cat every 8 hours or change to atenolol at a dose of 12.5 mg/cat every 12 hours.
  - Another treatment option in dogs is procainamide: 6–8 mg/kg, followed by a CRI of 25–50 µg/kg/min.

  If the patient presents with ventricular tachyarrhythmias (ventricular tachycardia) of haemodynamic significance, consider administration of:
  - Lidocaine: 2 mg/kg IV followed by a CRI of 40–60 µg/kg/min. In cats, 0.25–0.5 mg/kg IV followed by a CRI of 10–20 µg/kg/min (caution for cats: suspend administration if seizure or vomiting occurs).
  - Procainamide: 2 mg/kg IV followed by a CRI of 25–40 µg/kg/min.
  - Amiodarone: 5–10 mg/kg in dogs as a slow IV bolus (20–30 minutes).

- Beta blockers or calcium channel blockers can alternatively be used in cats with restrictive or unclassified cardiomyopathy who present with significant ventricular arrhythmias (see doses above).

Arrhythmias

Definition

An arrhythmia is an alteration in the heart's rhythm. Based on whether the heart rate decreases or increases, they are called bradyarrhythmias or tachyarrhythmias, respectively. The origin (supraventricular or ventricular) and the haemodynamic significance are clinically relevant to identifying the optimal therapy.

The most significant arrhythmias are as follows:

- Bradyarrhythmias: sinus arrest, atrial standstill, escape beats, first-, second-, and third-degree atrioventricular (AV) block, and severe sinus bradycardia.
- Tachyarrhythmias: supraventricular (sinus tachycardia, atrial tachycardia, and atrial fibrillation) and ventricular (ventricular premature complexes and ventricular tachycardia).

Causes

Arrhythmias can be caused by systemic problems that impact perfusion and myocardial metabolism (hypoxia, hypoperfusion, acidosis, electrolyte alterations, pain, poisoning, etc.), or innate (primary cardiomyopathies) or degenerative myocardial diseases.

Diagnosis

Diagnosis is based on the clinical history, auscultation to detect anomalies in the heart rhythm or rate, as well as other signs of cardiac pathologies (murmur, gallop, asynchronous pulse, hypoperfusion, syncope, etc.), pulmonary diseases (dyspnoea, auscultation anomalies), or systemic diseases. An electrocardiogram is required to confirm the disease category and significance.

Treatment

Bradyarrhythmias

- **Sinus arrest:** pauses between R-waves that exceed two R-R intervals. Associated with increased vagal tone, common in brachycephalics and in sinus node disorders (SSS: sick sinus syndrome). Causes syncope or collapse. Requires pacemaker, but atropine (0.04 mg/kg) or propantheline bromide (7.5–30 mg/animal every 8–12 hours PO) is also an option.
- **Atrial standstill:** absence of P waves. Frequently associated with hyperkalaemia due to urinary obstruction, anuria, or Addisonian crisis. Treatment is based on resolving the primary cause.
- **Ventricular escape:** presence of ventricular complexes and bradycardia without sinus activity. No specific treatment is required aside from identifying and treating the cause of the arrhythmia.
- **First-degree AV block:** normal QRS complexes with a prolonged PQ segment. Arrhythmia secondary to noncardiac causes that resolves upon treating the primary cause. Linked to excessive vagal tone.
- **Second-degree AV block:** presence of P waves not followed by QRS complexes. The treatment consists of identifying and treating the underlying cause (cardiac or extracardiac), but in symptomatic cases (generally due to severe

second-degree blocks, such as Mobitz type 2 blocks), administration of atropine potentially followed by dopamine (1–3 µg/kg/min) may be necessary. In cases that are severe or secondary to severe cardiac disease, pacemaker implantation may be necessary.

- **Third-degree AV block:** presence of P waves without supraventricular QRS complexes, but with ventricular escape beats. Treatment is identical to that previously described. Palliative drug therapy may help minimally.

Tachyarrhythmias

Supraventricular tachyarrhythmias

- **Sinus tachycardia:** QRS morphology is normal, but the heart rate is over 140 bpm (large breeds), 180 bpm (small breeds) or 200 bpm (cats). It is usually secondary to fever, pain, fear, hypovolaemia, hypoxia, etc. No specific treatment is required. Does not respond to vagal manoeuvres.
- **Atrial tachycardia:** QRS morphology is preserved, though sometimes shortened, and P waves are absent; may occur in rapid bursts. May respond to vagal manoeuvres. Drug therapy:
  - Diltiazem: 0.25 mg/kg IV as a slow bolus, repeat 15 minutes later. Consider CRI of 2–6 µg/kg/min if recurrent or causing haemodynamic instability.
  - Esmolol: 0.05–0.14 mg/kg bolus every 5 minutes to a maximum of 0.5 mg/kg; CRI of 50–200 µg/kg/min.
  - Verapamil: 0.05 mg/kg IV as a slow bolus, repeat up to twice and administer to effect.
  - Digoxin: 0.01–0.02 mg/kg IV, followed by a quarter-dose every 30 minutes up to four times.
- **Atrial fibrillation:** supraventricular morphology is maintained, but the rhythm is irregular, with an absence of P waves and presence of f waves. The treatment is the same.

Ventricular tachyarrhythmias

- **Ventricular tachycardia:** wide and abnormal QRS complexes. Consider antiarrhythmic treatment if severe (>180 bpm, RonT, multifocal, or repetitive) or if haemodynamic effects occur:
  - Lidocaine: 2–4 mg/kg IV in dogs; repeat the dose until the arrhythmia responds or signs of toxicity appear (vomiting or seizures). Afterwards, continue with a CRI at 25–75 µg/kg/min.
  - Magnesium sulphate at a dose of 0.15–0.3 mmol/kg IV over 10 minutes can help to control ventricular arrhythmias.
  - Procainamide can be given in refractory cases: 6–12 mg/kg as an IV bolus, followed by a CRI of 20–50 µg/kg/min.
  - If the patient does not respond to the above treatments, give esmolol: 0.5 mg/kg as a slow IV bolus, followed by a CRI of 25–200 µg/kg/min.
- **Ventricular fibrillation:** fibrillation waves without identifiable complexes. The approach is as follows:
  - Electrical defibrillation: use interspersed with cardiac massage and in doses increasing by 2–5 J/kg for a total of three times. Check the pulse and ECG after each defibrillation and prior to delivering the next one.
  - Lidocaine is not recommended in cases of ventricular fibrillation when intending to use a defibrillator. In dogs, amiodarone is a drug alternative in these cases and is the preferred treatment of postdefibrillation ventricular arrhythmias: slow 5 mg/kg IV bolus over 10 minutes; a dose of 2.5 mg/kg may be repeated within 3–5 minutes.

3

Respiratory emergencies

Upper airway obstruction

Definition

Upper airway obstruction is common in small animals and often associated with diverse pathologies of the nasopharynx, larynx, and trachea. It can lead to severe pulmonary disease and complications such as noncardiogenic pulmonary oedema and aspiration pneumonia.

Causes

Illnesses causing upper airway obstruction are common in veterinary medicine and a frequent reason for emergency consultation. Although these illnesses are diverse and of varying severity, the most common ones are laryngeal paralysis; laryngitis; nasopharyngeal disorders (infections, polyps); oropharyngeal, laryngeal, or tracheal neoplasms; brachycephalic airway syndrome; and tracheal collapse.

Diagnosis

Clinical signs vary by species, aetiology, chronicity, comorbidities, and the severity of the obstruction. Patients may present with varying degrees of dyspnoea, which can lead to complete respiratory failure. In moderately affected patients, clinical signs generally include phonation changes, dysphagia, nausea, inspiratory stertor or stridor, and nonproductive cough. Stertor and rhonchi commonly occur in obstructive processes that affect the nasal passages or nasopharynx. Stridor, in contrast, is more common in extrathoracic (laryngeal or tracheal) airway obstruction. Normally in these patients, the intensity of respiratory sounds and associated clinical signs (cyanosis, orthopnoea, syncope, etc.) increases as the obstruction worsens. Open-mouthed breathing can relieve symptoms if the problem is limited to the nose or nasopharynx. Panting is common in dogs, but open-mouthed breathing is much less common in cats, so its occurrence in this species suggests possible respiratory pathology. Hyperthermia is another common sign of airway obstruction; left untreated, it can lead to heat stress or heat stroke.

Physical examination and auscultation are generally sufficient to find the source of the problem. Altered respiratory sounds of the affected area help to localise it on auscultation. Increased respiratory noise can be better localised via detailed auscultation of the larynx, trachea, and both hemithoraces. Inspiratory dyspnoea with stertor or stridor typically results from extrathoracic obstructions, whereas intrathoracic obstructions tend to be characterised by expiratory dyspnoea and adventitious sounds. Careful pulmonary auscultation permits identification of lung pathologies.

Treatment

Definitive treatment depends on the underlying aetiology, but emergency stabilisation should aim to decrease the patient's oxygen demand and improve the patient's ability to breathe and oxygenate tissues. Minimise stress when handling, given the patient's fragility and the elevated risk of decompensation. Treatment should be based on:

- **Anxiolytics or sedatives:** acepromazine 0.005–0.05 mg/kg IV, IM, or SC. Use with caution if secondary vasodilation or hypotension may pose a problem for the patient. It can be given in combination with butorphanol 0.1–0.6 mg/kg IM, IV, or SC to enhance the effect and minimise the required dose.
- **Oxygen therapy:** oxygen cage, continuous flow, or mask according to patient tolerance and degree of dyspnoea.
- **Corticosteroids:** ultra short-acting glucocorticoids such as dexamethasone sodium phosphate 0.2–0.4 mg/kg IV, IM, or SC as needed to reduce laryngeal or tracheal inflammation or oedema.
- **Temperature control:** provide passive or active cooling according to the degree of hyperthermia.
- **Artificial airway:** if the aforementioned do not improve the patient's condition and cyanosis or signs of respiratory distress appear (hypoxaemia refractory to oxygen supplementation or strong hypercapnia), intubate. If this is not possible, an emergency cricothyroidotomy under sedation can be performed until an emergency temporary tracheotomy can be performed.

Allergic bronchitis/ feline asthma

Definition

Allergic bronchitis is defined as an (often chronic) inflammation of the lower airways (bronchi and bronchioles) as well as alveoli.

Causes

The aetiopathogenesis of allergic bronchitis is complex, but it is generally defined as lower airway inflammation along with an exaggerated response to external agents (inhaled allergens) and intrinsic antigenic stimulation resulting from exposure to chemical, infectious or parasitic agents. Overproduction of antibodies (IgE), eosinophilic infiltration, massive mast cell degranulation, insufficient activation of pulmonary leucocytes, and massive release of inflammatory mediators (leukotrienes, histamine, bradykinin, etc.) play critical roles in the pathogenesis of this hypersensitivity response. Release of such mediators leads to oedema of the pulmonary mucosa, hypertrophy of bronchial and bronchiolar smooth muscle, accumulation of bronchial secretions, and constriction or collapse of terminal bronchioles.

Diagnosis

This type of pulmonary pathology is generally characterised by clinical signs including cough, expiratory effort with audible or auscultable wheezing, and a predictable improvement on glucocorticoids. Feline asthma attacks can present acutely

with superficial or open-mouthed breathing, severe coughing spells, and tracheal hypersensitivity.

Thoracic radiology is required to diagnose feline asthma. Although age of presentation (middle age), breed (higher incidence in Siamese), concurrent parasitosis, and peripheral eosinophilia in a symptomatic cat may suggest asthma, definitive diagnosis requires demonstration of eosinophilia in the bronchial epithelium and/or a generalised marked bronchial or bronchiointerstitial pattern. Lung hyperinflation, flattening of the diaphragm, and alveolar infiltration with consolidation of the right middle lobe may be observed. In any case, bronchoscopy and bronchoalveolar lavage (with predominance of neutrophils and eosinophils) permit confirmation of the diagnosis, characterisation of the process (e.g. pulmonary parasitosis), and identification of possible complications that may require treatment (e.g. pneumonia).

Treatment

Emergency treatment of acute allergic bronchitis or asthma attack is based on:

- **Minimising patient stress:** handle cautiously, avoid stress, and postpone further tests until the patient is stabilised. Sedate if necessary and give parenteral treatments (IM or SC) while the patient is oxygenated.
- **Oxygen therapy:** use the most effective (highest FiO_2—fraction of inspired oxygen—) and least stressful method the patient can tolerate. In an asthma attack, continuous flow oxygen or oxygen cages are recommended because they minimise patient handling.
- **Bronchodilators:**
  - Inhaled salbutamol by mask: 50 µg/kg every 30–60 minutes until dyspnoea improves.
    Do not use in severe acute cases if handling stresses the patient.

  - Terbutaline: 0.01 mg/kg SC, followed by 0.625–1.250 mg/cat PO every 12 hours.
  - If the above are unavailable, aminophylline can be given: 2–5 mg/kg as a slow IV drip.
  - If the patient does not respond and no cardiac disease exists, give adrenaline (0.2 ml/10 kg at 1/1,000 IM or 2 ml/10 kg at 1/10,000 IV) and atropine (0.04 mg/kg SC or IM, or 0.015 mg/kg IV, single dose).
- **Fast-acting glucocorticoids:**
  - Dexamethasone (sodium phosphate): 1–2 mg/kg IM or IV as a slow drip.
  - Prednisolone (sodium succinate): 50–100 mg/cat IV as a slow drip (15 minutes).
  - Methylprednisolone (sodium succinate): 10 mg/kg IV as a slow drip (15–30 minutes).

Pneumonia

Definition

Pneumonia is an inflammation of the pulmonary parenchyma of generally infectious aetiology that spreads by inhalation or haematogenous spread of bacteria, fungi, viruses, protozoa or parasites.

Causes

It may be a primary infectious process, but in small animals it often occurs concurrently with underlying predisposing pathologies that can be pulmonary as well as systemic.

Most bacterial pneumonias occur concomitantly with other respiratory infections. The infectious agents most commonly isolated from patients with bacterial pneumonia are as follows: *Pasteurella* spp. (gram-negative bacilli), *E. coli* (gram-negative bacillus), *Staphylococcus* spp. (gram-positive cocci), and *Streptococcus* spp. (gram-positive cocci). *Mycoplasma* spp. are frequently isolated in bacterial cultures of pneumonia patients as infectious agents or, frequently, as agents of coinfections (62 %). Anaerobic bacteria are isolated less often, but their presence can suggest a pulmonary abscess.

Bacterial, viral, fungal, protozoal, and parasitic pneumonias often occur in patients with predisposing conditions such as unconsciousness, immobility, primary or concurrent upper airway disease, immunosuppression, regurgitation, or oesophageal motility disorders, or as a result of anaesthetic procedures or invasive medical/surgical treatments of the respiratory tract.

Diagnosis

Diagnosis is based on the patient's clinical signs, history, and exam findings suggestive of the disease process; it is confirmed by diagnostic imaging (thoracic radiographs), blood tests, and, ideally, culture and identification of the responsible infectious agent.

Clinical signs of pneumonia are wide-ranging and variable, from patients who are virtually asymptomatic to those with lethargy, inappetence, dyspnoea, marked respiratory effort, nasal discharge and cough. The clinical history should aim to identify potential predisposing factors, and the physical examination should identify anomalies on pulmonary auscultation; most patients will present with increased breath sounds, crackles, or wheezes. Concomitant fever, productive cough, and purulent nasal discharge, though possibly intermittent, indicate potential infection of the lung tissue.

Diagnosis in patients with compatible signs and predisposing factors should be confirmed with diagnostic imaging and isolation from biological samples (transtracheal aspirate or bronchoalveolar lavage). The radiographic presence of alveolar opacities, bronchograms, consolidation, and an interstitial pattern with an asymmetric distribution on radiographs is suggestive of pneumonia. In such cases, while diagnostic tests such as a complete blood count (leukogram), serum biochemistry, and urinalysis can help to quantify the magnitude of the infection and its complications, they rarely contribute directly to the diagnosis.

Treatment

Patients without breathing difficulty who have mild/moderate pneumonia can be treated outpatient with long-term oral antibiotics and supportive therapy. However, patients with severe pneumonia, uncontrolled risk factors, or systemic complications of infection should be hospitalised and treated as follows:

- **Oxygen therapy:** patients with dyspnoea or hypoxaemic respiratory distress should receive supplemental O_2. The method (continuous flow, O_2 cage, nasal goggles, nasal cannula, etc.) depends on the degree of compromise and the patient's particular conformation and behaviour. Nasal catheters or cannulas are commonly used, and a flow rate of 50–100 ml/kg/min is generally recommended. Always try to use the lowest flow rate and percentage of supplemental O_2 that will effectively alleviate the patient's compromise. In long-term use (>24–48 h), do not exceed a concentration of 60 % in order to avoid toxic effects of oxygen on alveolar epithelium. If the breathing pattern or blood gas measurements deteriorate, or the patient's respiratory effort becomes excessive, intubation and mechanical ventilation may be indicated.
- **Antibiotic therapy:** empirical therapy is the usual first step in these patients, but culture, isolation, and susceptibility testing of all samples (saliva, aspirates, tracheal or bronchoalveolar lavages or other fluids such as blood, urine, etc.) are recommended before beginning therapy. For severe pneumonia, broad-spectrum antibiotic therapy with good pulmonary tissue penetration is generally recommended. If possible, the choice of antibiotic should consider the aetiopathogenesis of the process, and the selected drugs should be given parenterally for several weeks. Empirical therapy may start with amoxicillin-clavulanic acid, combinations of penicillins (amoxicillin, ampicillin or ticarcillin) with fluoroquinolones (enrofloxacin or marbofloxacin), or clindamycin with fluoroquinolones or macrolides (azithromycin).

- **Bronchodilators:** use is controversial but they can be helpful in cases of bronchospasm or accumulation of secretions due to alterations in ciliary activity. However, they can suppress cough, exacerbate the respiratory ventilation/perfusion mismatch, and facilitate the migration of secretions to adjacent alveoli. The following can be administered:
  - Adrenergic agonists: terbutaline 0.01 mg/kg IV or IM every 4–6 hours.
  - Methylxanthines: aminophylline or theophylline 5 mg/kg every 8 hours, as an IV infusion over at least 30 minutes, up to a dose of 10 mg/kg in dogs.
- **Mucolytics:** though frequently used in veterinary medicine, they lack clear proof of efficacy. N-acetylcysteine can be given orally or intravenously with previous dilution (use with caution).
- **Nebulisation:** nebulisation of normal saline (0.9 % NaCl) every 4–8 hours can hydrate the mucociliary system, liquefy secretions, and facilitate their migration and expulsion. Vaporisers and humidifiers do not work for this purpose, nebulisers must be used. For patients with nonproductive cough, combine nebulisation with thoracic percussion (coupage) to dislodge secretions.

Pleural space diseases

Definition

Pleural disease is caused by an abnormal accumulation of fluid or air or abnormal organ position that alters the pleural space and its normal function.

Causes

The most common pleural space diseases include pathological fluid accumulation (transudate, modified transudate, septic or aseptic exudate, chyle or blood), presence of air (pneumothorax) or presence of masses, tissues, or organs that occupy the pleural potential space (diaphragmatic hernia).

Diagnosis

Diagnosis of pleural disease is based on the patient's clinical history, physical examination, and diagnostic imaging. These patients often present with tachypnoea, orthopnoea with resistance to sternal recumbency, cyanosis, and open-mouthed, superficial, asynchronous breathing with a significant abdominal component. Auscultation reveals a lack of respiratory sounds (generalised, lateralised or ventral) in various lung fields, attenuated heart sounds, or shifting of sounds from the normal position (heart beat).

Thoracic radiographs are extremely useful to confirm the diagnosis, appreciate the severity of the disease, and identify other intrathoracic pathologies. Thoracic-focused assessment with sonography for trauma (T-FAST) can confirm diagnosis of mild pleural effusions and guide subsequent therapy (thoracentesis, thoracic surgery, etc.). Blind or ultrasound-guided thoracentesis can often stabilise patients with cavitary effusions and permit sampling for biochemical and cytological analysis (fluid density, gross appearance, biochemical characteristics, cellularity) (Table 1).

Table 1. Characteristics of pleural effusion fluid.

Effusion type	Colour	Proteins	Cell count	
Pure transudate	Clear or yellowish	<2.5 g/dl	<1,500 cells	
Modified transudate	Clear, yellowish, or pink	2.5–3.5 g/dl	1,500–3,000 cells	
Exudate	Variable	>3.5 g/dl	>3,000 cells	
Chyle	Milky	>2.5 g/dl	500–10,000 cells	
Blood	Red	Greater than or equal to peripheral blood	1,500–4,000 cells	

SIRS: systemic inflammatory response syndrome.

Treatment

Emergency treatment is based on supplemental oxygen, sedation or anaesthetic induction, and therapeutic thoracentesis when indicated. Specific treatment depends on the type of pleural pathology, the responsible aetiology, and the presence of related pathologies. For example, in cases of haemothorax, it is important to determine whether the cavitary bleed is localised (trauma, neoplasm) or instead due to a coagulopathy.

	Cell type	Differential diagnosis
	Mesothelial cells, neutrophils, and monocytes	• Hypoalbuminaemia • Circulatory overload
	• Mesothelial cells, neutrophils, and monocytes • Variable	• Congestive heart failure • Lung lobe torsion • Neoplasm • Vasculitis • Pulmonary thromboembolism • Diaphragmatic hernia • Idiopathic
	• Neutrophils, macrophages, and bacteria (septic) • Abnormal cellularity (neoplasm)	• Septic (pyothorax): foreign body, nocardiosis, etc. • Aseptic: neoplasm, inflammation, lung lobe torsion, filariasis, SIRS, etc.
	Lymphocytes, macrophages and neutrophils	• Congestive heart failure • Lung lobe torsion • Mediastinal mass • Dirofilariasis • Granuloma • Thoracic duct rupture
	Neutrophils with erythrophagocytosis and erythrocytes	• Trauma • Coagulopathy • Neoplasm

Such cases may require transfusion of blood products (packed red blood cells or plasma) and preventive thoracentesis if breathing is compromised. Autotransfusion may be considered.

In patients with pyothorax, conservative treatment includes continuous thoracic drainage. Long-term broad-spectrum antibiotic therapy guided by culture (aerobic and anaerobic) of pleural exudate is strongly indicated. Periodic pleural lavages with normal saline are commonly performed. Refractory cases may require a surgical approach.

In pneumothorax, thoracentesis may be critical to not only diagnosis but also therapy. If the pneumothorax is recurrent, high-grade or tension type, placement of an emergency pleural drain may be indicated. In these cases, perform intermittent or continuous postoperative aspiration if the severity or patient progression requires it. Refractory cases may require an exploratory thoracotomy.

Surgery is the definitive treatment for diaphragmatic hernia. However, stabilisation by conservative measures (oxygen therapy, sedation, fluid therapy, thoracentesis and draining of effusions, etc.) is always indicated before surgery. In case of stomach entrapment, emergency surgery is required after thoraco-gastrocentesis, since dyspnoea may be severe and the risk of recurrence high.

4

Haematological emergencies

Anaemia

Definition

Anaemia is defined as a decreased capacity to transport oxygen in the blood due to decreased haemoglobin and circulating erythrocytes.

Causes

Anaemia is a common laboratory abnormality in small animals but is not in itself a diagnosis; relevant clinical investigations must be performed to identify the underlying cause in each patient. The three main pathophysiological mechanisms of anaemia are blood loss, haemolysis, and ineffective erythropoiesis (Box 1).

Box 1.

Main causes of anaemia.

1. Blood loss

- **Normal haemostasis:** trauma, surgery, parasitosis, NSAIDs, steroids, neoplasms, etc.
- **Abnormal haemostasis:** thrombocytopaenia, thrombocytopathy (von Willebrand disease), congenital or acquired coagulopathies (disseminated intravascular coagulation—DIC—, hepatopathy, rodenticide toxicity, etc.).

2. Haemolysis

Congenital, infectious, immune, chemical (zinc, copper, sulphonamides), hypophosphataemia-induced, mechanical (DIC), neoplastic (haemangiosarcoma, histiocytosis).

3. Ineffective erythropoiesis

- **Refractory anaemias** (respond only to transfusions of blood and blood products): caused by chemicals, chronic renal failure, chronic illnesses (organ dysfunction, infection, cancer, idiopathic—immune?—), etc.
- **Nonrefractory anaemias:** aplastic anaemia; pancytopaenia; drug- or chemical-induced, infectious, immune, neoplastic, radiation-induced, or congenital anaemia; anaemia due to malabsorption or cobalamin deficiency; idiopathic anaemia, etc.

Diagnosis

The diagnosis is made in the laboratory, but the clinical signs vary based on how acute the anaemia is, what type it is, and the cause. Regardless of the cause, it is common to observe pale mucosae, lethargy, weakness, and anorexia. In case of haemorrhagic losses, signs of external, internal, or occult bleeding may be found.

Patients with hyperacute haemorrhage may show signs of circulatory collapse from acute hypovolaemia and hypoxaemia, with lethargy, pallor, tachycardia, and tachypnoea. In case of internal haemorrhage (e.g. retroperitoneal, in muscle fascia) or gastrointestinal bleeding, signs may be less obvious and harder to interpret; thus, localising the bleed may be more difficult. The haemorrhage may appear as a local bleed (trauma, surgery). If it is in multiple locations, on the surface, and accompanied by petechiae, ecchymoses, haematomas, or cavitary bleeding, it may be indicative of an underlying alteration of haemostasis (primary or secondary).

Haemolytic anaemia may be accompanied by icterus, haemoglobinaemia or pigmenturia. Signs of anaemia due to ineffective erythropoiesis may be more subtle depending on the chronicity and the patient's ability to adapt. In these cases, patients tend to present with signs of the underlying pathology.

Although signs may suggest anaemia, laboratory testing is required to determine the severity and cause of the alterations. In emergency cases, the most useful items for the initial diagnosis and determination of the aetiology include microhaematocrit and total protein, blood smear, platelet count, and basic coagulation parameters (prothrombin time and activated partial thromboplastin time). Along with the total protein levels, the microhaematocrit can suggest a potential active bleed (generally protein <6 g/dl); the leucocyte-platelet layer (buffy coat) can reveal leucocyte levels; and examination of the supernatant can show haemoglobinaemia or bilirubinaemia. The blood smear can offer information on erythrocyte morphology, count,

and colour and reveal agglutination; the reticulocyte count shows the degree to which the anaemia is regenerative. A decrease or alteration in other cell lines (leucocytes or platelets) can point to the underlying pathogenesis (central or medullary versus peripheral).

Treatment

It depends on the cause, severity, and progression of the anaemia. Patients with hyperacute bleeds require intensive fluid therapy in hopes of maintaining perfusion and nearly always before considering transfusion of blood or blood products. Transfusion of fresh whole blood or blood products is indicated in many clinical situations, especially in emergency and intensive care medicine (Table 1). The main objective of haemotherapy is to maintain oxygen delivery in the face of a blood volume loss that can cause severe hypoperfusion or even death.

Table 1. Haemotherapy.

Blood product	Indication
Packed red blood cells (pRBCs)	• Hypovolaemic anaemia (acute haemorrhage, shock), along with fluids (saline crystalloids with or without colloids) or plasma. • Normovolaemic anaemia (haemolytic, aplastic, iron deficiency, due to chronic renal failure or myelodysplastic syndrome).
Fresh whole blood	• Transfusions in cats. • Also in hypovolaemic anaemia.

When transfusing **fresh whole blood**, the volume to administer depends on the degree of anaemia and animal's clinical condition and size.

Generally, administration of 2 ml/kg of whole blood leads to a 1 % increase in the recipient's haematocrit, assuming no haemorrhage or haemolysis.

The transfusion is slow at first, 10–25 ml over 15 minutes, monitoring for reactions. If no reaction occurs, a rate of 10–20 ml/kg/h is appropriate for normovolaemic animals and 20–60 ml/kg/h is appropriate for hypovolaemic animals. Do not exceed 2–4 ml/kg/h in animals with heart failure. The transfusion should take no longer than 4 hours in order to protect the functional components and avoid infection. If the transfusion volume is a limiting factor, do not give whole blood.

Administration of **pRBCs** should be restricted to cases of necessity such as severe anaemia. There is no specific haematocrit value that indicates whether pRBCs are required since each case progresses differently. In dogs, a rule of thumb is haematocrit values below 20 % can induce myocardial hypoxia, and below 12 %, oxygen reserves are depleted, so transfusion is almost always required. These are generally gravely ill animals with circulatory, respiratory and metabolic complications that exacerbate the signs of anaemia. Even if the haematocrit is not in the critical range (<10–12 %), the acute presentation of anaemia and related clinical signs (weakness, tachycardia, tachypnoea, or persistent hyperlactataemia) can justify giving a transfusion.

Coagulopathies

Definition

Coagulopathy is defined as the clinical syndrome resulting from a congenital or acquired deficiency in one or more of the clotting factors that comprise the secondary haemostasis system.

Causes

Box 2 shows the main causes of blood dyscrasias associated with acquired or congenital coagulopathies.

Diagnosis

Diagnosis should be based on the clinical history, clinical signs, and complete laboratory testing, as described previously. Defects in secondary haemostasis are often associated with single or multiple haematomas and bleeding into subcutaneous tissues, body cavities, muscles, and joints. The most useful diagnostic tests for alterations of secondary haemostasis are as follows:

- **Activated clotting time** (ACT): evaluates the intrinsic and common pathways, i.e. factors XII, XI, IX, VIII, X, V, II, and fibrinogen.
- **Prothrombin time** (PT): is a more sensitive test of these same factors.
- **Activated partial thromboplastin time** (APTT): evaluates the extrinsic and common pathways, i.e. factors III, VIII, X, V, II, and fibrinogen.

Box 2.

Main causes of acquired and congenital coagulopathies.

Acquired:

- Deficiency or antagonism of vitamin K (rodenticide toxicity).
- Hepatopathy.
- DIC.
- Anticoagulants (administration of heparin, etc.).

Congenital:

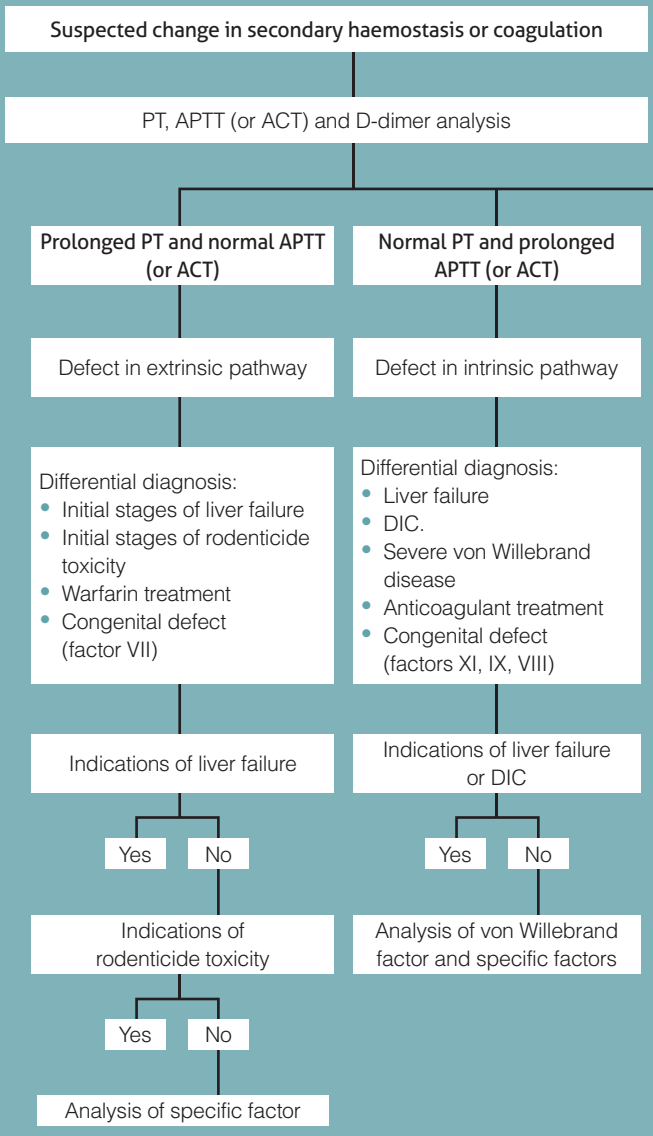
- Factor I: hypofibrinogenaemia and dysfibrinogenaemia (Bernese Mountain Dog, Borzoi, Lhasa Apso).
- Factor II: hypothrombinaemia (English Cocker Spaniel, Boxer).
- Factor VII: hypoproconvertinaemia (Beagle, Alaskan Malamute, Boxer, Bulldog).
- Factor VIII: haemophilia A (German Shepherd and other breeds).
- Factor IX: haemophilia B (various breeds).
- Factor X: Stuart-Prower deficiency (American Cocker Spaniel, Jack Russell Terrier, and other breeds).
- Factor XI: plasma thromboplastin antecedent deficiency (Springer Spaniel, Kerry Blue Terrier, Great Pyrenees).
- Factor XII: Hageman factor deficiency (Standard and Miniature Poodle, Shar Pei).

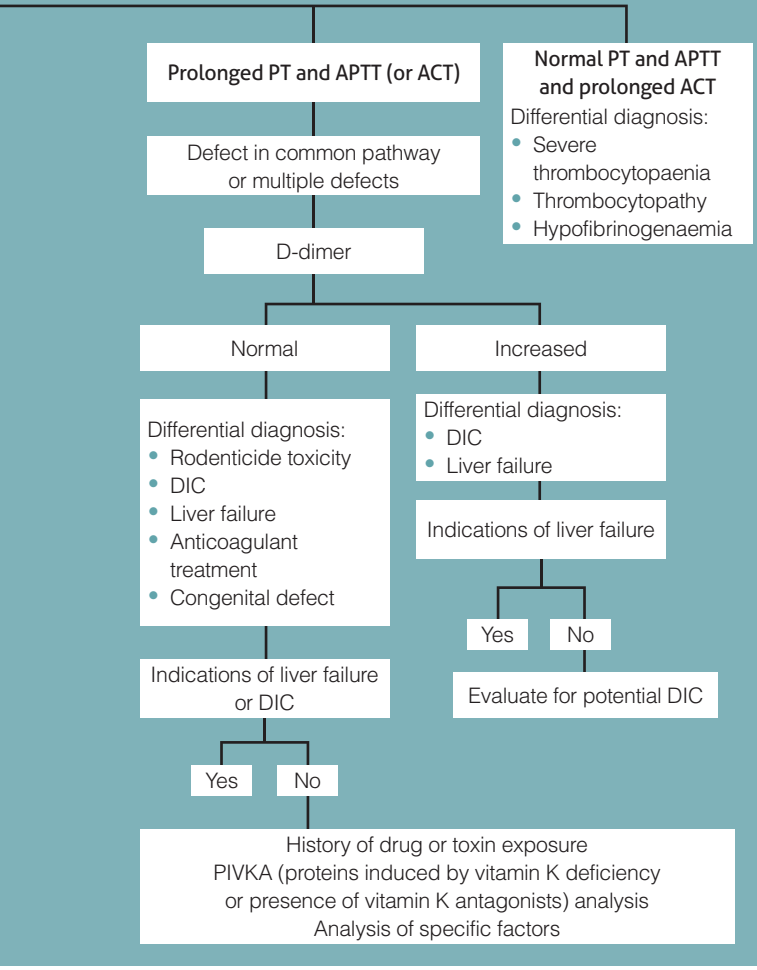
And to evaluate fibrinolysis:

- **Fibrin degradation products** (FDPs).
- **D-dimer**.

Figure 1 provides a useful flow chart for diagnosing secondary haemostasis disorders.

Figure 1. Flow chart for diagnosing alterations in secondary haemostasis.




Prolonged PT and APTT (or ACT)
Defect in common pathway or multiple defects
D-dimer
Normal
Differential diagnosis:
• Rodenticide toxicity
• DIC
• Liver failure
• Anticoagulant treatment
• Congenital defect
Indications of liver failure or DIC
Yes
No
History of drug or toxin exposure
PIVKA (proteins induced by vitamin K deficiency or presence of vitamin K antagonists) analysis
Analysis of specific factors
Increased
Differential diagnosis:
• DIC
• Liver failure
Indications of liver failure
Yes
No
Evaluate for potential DIC
Normal PT and APTT and prolonged ACT
Differential diagnosis:
• Severe thrombocytopaenia
• Thrombocytopathy
• Hypofibrinogenaemia

Treatment

Treatment for rodenticide toxicity (coagulopathy due to inactivation of vitamin K-dependent clotting factors) is administration of the antidote at a dose and duration appropriate to the specific toxin. Vitamin K_1 (phytonadione) 2–5 mg/kg/day PO or SC for several weeks, with follow-up check of clotting times (PT/APTT). In case of intense bleeding with hypoperfusion, severe anaemia, or bleeding in vital organs (lung tissue, CNS), simultaneous administration of fresh frozen plasma (FFP) 6–20 ml/kg every 8–24 hours is recommended.

In case of severe liver disease, administration of vitamin K_1 and FFP may be useful while the organ dysfunction persists. In cases of DIC, treatment of the primary cause is critical, but administration of FFP, heparin and pRBCs or whole blood as needed may improve the patient's condition.

FFP is the product of choice for congenital coagulopathies. For deficiencies in factors VIII and I (fibrinogen), the cryoprecipitate is the ideal treatment, but for deficiencies in factor II, VII, IX, X, or XI, the supernatant is optimal. If these products are unavailable, administer FFP.

5

Gastrointestinal emergencies

Gastric dilatation-volvulus

Definition

Gastric dilatation-volvulus (GDV) is a medical and surgical emergency caused by stomach dilatation, displacement and/or rotation, and the cardiovascular, respiratory, and gastrointestinal effects that result.

Causes

Generally affects large- or giant-breed middle-aged deep-chested dogs (e.g. Great Danes, Doberman Pinschers, German Shepherds, etc.). Overeating, exercise or excitation after meals, lack of division of meals, or ingestion of food that is difficult to digest may be risk factors for GDV.

Diagnosis

Diagnosis is based on the clinical history, physical examination findings, and radiographic confirmation. In patients with a conformational predisposition, nonproductive vomiting, drooling, a tympanitic, distended abdomen, and generalised perfusion abnormalities suggest this possibility. Abnormal pyloric displacement (anterodorsally on a lateral abdominal radiograph) gives rise to the “double bubble” or “Popeye’s arm” sign that is diagnostic for this syndrome.

Treatment

The most important element is prompt correction of circulatory collapse. Following initial stabilisation with fluids, treatment should decompress the stomach, differentiate between dilatation and dilatation-volvulus, reposition and fixate the stomach, and finally address intra- and postoperative complications. If the patient has only gastric dilatation, the clinician should treat medically and determine whether a surgical preventive gastropexy is necessary.

Correction of circulatory collapse

- Administer supplemental O_2 (continuous flow or mask).
- Place one or two large-bore (14–16 gauge) peripheral catheters in the cephalic or external jugular veins.
- Take blood samples for basic testing: haematocrit (Hct), total protein, glucose, electrolytes, urea, creatinine, lactate, acid-base balance and coagulation.
- Monitor SpO_2, arterial blood pressure, and ECG.
- Administer an IV bolus of isotonic crystalloids (10–30 ml/kg) simultaneously with colloids (10–20 ml/kg) to effect. In large dogs or those with severe circulatory compromise, crystalloids may be replaced with hypertonic saline (4 ml/kg over 10–15 minutes).
- In cases of severe dilatation and haemodynamic compromise, it may be helpful to preventively trocarise the stomach during haemodynamic stabilisation to facilitate orogastric tube placement.
- Start antibiotic therapy with cefazolin (25 mg/kg every 8 hours IV) or ampicillin (25 mg/kg every 8 hours IV) and cytoprotective drugs (omeprazole: 0.7 mg/kg/day IV).

Decompression and gastric lavage

For critical patients, orogastric intubation using physical restraint and palliative gastrocentesis may be considered, but the patient should be sedated or anaesthetised, intubated, and undergo stomach decompression along with gastric lavage.

Methadone (0.2–0.4 mg/kg IV) or fentanyl (2.5–5 µg/kg) and benzodiazepines such as diazepam or midazolam (0.2–0.5 mg/kg) can be given as a slow bolus to facilitate orotracheal intubation and orogastric tube placement using a flexible, lubricated tube of sufficient length. If orogastric tube placement fails, trocarise the most superficial, tympanitic area of the stomach to facilitate the procedure. Once the tube is placed, perform several gastric lavages with warm water (37 °C). During haemodynamic stabilisation, tube placement, and decompression, monitor the patient's physical and haemodynamic parameters and recheck basic laboratory values.

Gastropexy

The goals of the surgery are decompression, return of the stomach to its normal position, evaluation of the viability of the stomach and spleen, removal of nonviable or compromised tissue, and fixation of the stomach to avoid recurrence. In case of dilatation-volvulus, early surgical treatment (within 2 hours of admission) is advised. This carries a lower risk of morbidity and mortality and avoids subsequent complications such as arrhythmias, infarctions, thrombotic events, gastric perforation, and peritonitis.

Postoperative management

Postoperative management should aim to restore fluid balance as well as prevent and treat gastrointestinal and systemic complications resulting from the emergent problem (Box 1). Postoperative treatment should be proportional to the severity of the case and its complications. The following is recommended:

- A balanced fluid therapy regimen proportional to the patient's perfusion-hydration status.
- Treatment with blood products (plasma or packed red blood cells) if required (coagulopathy or anaemia, respectively).

- Multimodal analgesia: opioids and lidocaine CRI at 20–30 µg/kg/min.
- Broad-spectrum antibiotic therapy: cefazolin, ampicillin or combinations with aminoglycosides or fluoroquinolones.
- Cytoprotective drugs: omeprazole (1 mg/kg IV or PO, every 12–24 h) and sucralfate (250–500 mg/dog every 8 hours).
- Antiemetics and prokinetics: metoclopramide CRI at 1–2 mg/kg/day.
- In case of signs or increased risk of thrombosis, heparin can be added to the regimen (enoxaparin: 0.8 mg/kg SC every 6 hours).

The following should be carefully monitored postoperatively in high-risk patients: ECG, blood pressure, SpO_2, urine production, lactate, electroytes, ALT, AST, alkaline phosphatase, urea, creatinine, Hct, total protein, blood sugar, coagulation and acid-base balance.

Box 1.

Possible postoperative complications.

- Cardiac arrhythmias (normally ventricular).
- Fluid overload.
- Paralytic ileus.
- Repeated vomiting.
- Pancreatitis.
- Dehiscence of sutures.
- Gastric ulceration.
- Ischaemic necrosis of stomach or spleen.
- Acute renal failure.
- Sepsis, DIC.

Acute pancreatitis

Definition

Inflammatory disease of the pancreas characterised by intrapancreatic activation of digestive enzymes that autodigest the tissue. This condition is relatively common in both dogs and cats. While the severity is variable, patients who are severely affected may undergo extensive pancreatic necrosis, systemic inflammatory response syndrome, multiple organ dysfunction, and death.

Causes

Although pancreatitis is often idiopathic, several risk factors increase the morbidity and mortality of this disease: advanced age, obesity, gastrointestinal disease, endocrinopathies (e.g. diabetes mellitus, hyperadrenocorticism or hypothyroidism) and hepatic lipidosis or other concurrent hepatobiliary pathologies in cats.

Some factors associated with pancreatitis in dogs and cats are: recent abdominal trauma; hypercalcaemia; biliary obstruction; duodenogastric reflux; hypertriglyceridaemia (Schnauzers); recent overeating or ingestion of rich or fatty food; and, finally, medical, environmental, or surgical conditions leading to splanchnic hypoperfusion or severe hypothermia.

Diagnosis

Clinical signs of acute pancreatitis are variable and nonspecific, particularly in cats. Affected patients often present with anorexia, vomiting, weakness, depression and sometimes diarrhoea. The most severe cases present with fever, dehydration, abdominal pain, and icterus.

Diagnosis requires integration of the clinical history, physical examination, laboratory findings, and results of diagnostic imaging. Blood count and biochemistry results are variable and nonspecific and usually reflect abnormalities caused by the patient's concurrent illness or complications of the systemic inflammatory response. Neutrophilic leukocytosis, thrombocytopaenia, hyperbilirubinaemia, transaminase elevation, azotaemia, hypoalbuminaemia, hyper- or hypocalcaemia, and hyper- or hypoglycaemia are common findings. Laboratory determination of pancreas-specific lipase by immunoreactivity (cPLI and fPLI for dogs and cats, respectively) is currently the main tool for diagnostic confirmation, since it is both specific and sensitive. Repeated measurement of this parameter enables monitoring of disease progression.

With respect to diagnostic imaging, abdominal ultrasound can confirm the clinical suspicion and is particularly useful for monitoring patient progress.

Treatment

It is based on adequately managing the patient's underlying condition, instituting supportive and symptomatic treatment, and preventing complications associated with inflammation.

Fluid therapy

If the patient presents with signs of generalised hypoperfusion, a bolus of isotonic replacement crystalloids (10–30 ml/kg IV) should be given until the patient is haemodynamically stable. Given the inflammatory aetiology of the process and the significant loss of fluids into third spaces (intestinal lumen, peritoneal cavity) and interstitial spaces (linked to vasodilation and increased permeability), a simultaneous colloid bolus is recommended (2.5–10 ml/kg IV), particularly if protein or albumin levels are low. Consider use of vasopressors (dopamine or noradrenaline) if the patient is refractory to fluid resuscitation.

Once the patient is haemodynamically stable, establish a hydration regimen that accounts for both maintenance requirements and replacement of potential losses (vomit, diarrhoea or third-spacing) via comprehensive monitoring and daily weight checks.

If necessary, supplement the fluids (with glucose, potassium, magnesium). In case of a related coagulopathy, use of fresh frozen plasma is an option (10–15 ml/kg every 8, 12 or 24 hours IV).

Analgesia

Pain treatment is indicated in all patients with pancreatitis. It is based on using opioids and ketamine (0.2–2 mg/kg/h) and/or a lidocaine (20–30 µg/kg/min) CRI in severe cases. Some patients may benefit from epidural or intraperitoneal analgesia. Use of NSAIDs is generally contraindicated, except in cases of patients with refractory pain who are haemodynamically stable and not dehydrated or azotaemic.

Nutrition

For mild or moderate pancreatitis, fasting 24–48 hours is recommended, followed by progressive reintroduction of liquids and small quantities of a hyperdigestible low-fat diet. When pancreatitis is severe or there is a clinical history of prolonged anorexia, enteral feeding is recommended because it helps to structurally maintain the gastrointestinal mucosa and decrease bacterial translocation, which in turn minimises the release of inflammatory mediators and propagation of the systemic inflammatory response. It is easy to implement naso-oesophageal or nasogastric tube feeding or feeding via oesophagostomy, so these are quite useful tools for managing patients. Patients with vomiting or intolerance can receive temporary parenteral nutrition.

Antibiotics

Routine prophylactic antibiotic use is controversial. In patients who do not respond to the regular treatment or those with a potentially higher risk of septic complications, samples should be taken for culture (e.g. abdominal fluid) and broad-spectrum antibiotic therapy administered.

Other therapies

Antiemetics, prokinetics, cytoprotective medications, heating pads, etc., can improve the patient's symptoms and enhance recovery.

Surgical treatment

Surgical treatment for pancreatitis is controversial and generally not indicated. However, it may be considered if the patient's condition deteriorates despite intensive medical treatment or if there is evidence of infected necrotic or inflamed pancreatic tissue (abscesses).

6

Urological emergencies

Acute renal failure

Definition

Acute renal failure (ARF) is defined as a sudden decline in renal filtration and excretion function, leading to retention of uraemic toxins and loss of fluid, electrolyte, and acid-base balance. Although oliguria and anuria appear in many ARF patients, others are not oliguric and may even be polyuric, which carries a better prognosis.

Causes

Causes of acute renal failure (ARF) include:

- **Prerenal:** decrease in blood flow, hypoperfusion, or excessive renal vasoconstriction.
- **Renal:** prolonged haemodynamic or ischaemic conditions, infectious diseases, exposure to toxins, or renal manifestations of systemic illnesses.
- **Postrenal:** obstruction or diversion of urine that may originate from the ureters, bladder, or urethra.

Diagnosis

The clinical history is often suggestive of the aetiology of renal failure, but the clinical signs and examination findings are often nonspecific. Patients generally have a history of weakness, vomiting, diarrhoea, anorexia, and oliguria/anuria or polyuria.

The examination may show signs of dehydration, uraemic halitosis, oral ulcers, tachycardia or bradycardia, and hypothermia, as well as enlarged kidneys that are painful on palpation.

The diagnosis of ARF and confirmation of its aetiology require laboratory testing, diagnostic imaging, and other specific diagnostic modalities.

Blood tests often show these patients to be haemoconcentrated (increased haematocrit and plasma proteins) due to dehydration, but in severe gastritis or uraemic enteritis, the haematocrit may decrease along with the platelet count. The degree of azotaemia depends on the severity and duration of the disease, but elevated urea, creatinine and phosphorus levels are typical. In case of oliguria/anuria, hyperkalaemia may be noted; and certain toxicities (ethylene glycol) typically exhibit hypocalcaemia with an increased anion gap.

Urinalysis is very useful not only for identifying the origin of the ARF (prerenal, renal or postrenal), but also confirming the diagnosis and sometimes the primary aetiology. ARF of renal origin commonly manifests with isosthenuria (urinary specific gravity: 1.007-1.015) and, depending on the aetiology, alterations in urinary pH, proteinuria, haematuria, pyuria, and active and varied urinary sediment (casts, crystals, bacteria, etc.). Urine culture is recommended when bacteria are found in the sediment.

Radiographs may show nephromegaly, renal calculi (nephroliths) or ureteral calculi. Depending on the pathology, ultrasound can confirm nephromegaly with either unchanged or altered corticomedullary architecture, pyelectasia, etc.

Supplemental diagnostic tests are sometimes indicated, such as cytology of ultrasound-guided aspirates (for lymphosarcoma) and ultrasound-guided biopsy through laparoscopy or traditional laparotomy. Serology or PCR for infectious diseases (primarily leptospirosis, leishmaniosis and ehrlichiosis) may be indicated depending on the clinical suspicion.

Treatment

Treatment of ARF includes control of azotaemia and extrarenal signs of the disease process, supportive therapy, and, in some cases, specific treatment of the underlying process. In other words, treatment is based on correcting the patient's hydration status and electrolyte and acid-base alterations, as well as establishing or maintaining urine production, and treating the original cause of the ARF.

Fluid therapy

In case of perfusion deficits (shock), a balanced isotonic crystalloid (e.g. Lactated Ringer's) should be given at 20–30 ml/kg (dogs) or 10–20 ml/kg (cats), with a maximum dose of 80–90 ml/kg/h and 40–60 ml/kg/h, respectively, until haemodynamic parameters (pulse pressure, heart rate, mentation, etc.) recover.

If the patient is dehydrated but presents without perfusion deficits, give an isotonic crystalloid appropriate to both electrolyte levels (primarily sodium and potassium) and acid-base status. For severe acute hyponatraemia or hyperkalaemia, 0.9 % NaCl may be a better option. The volume should be calculated as a function of the estimated percent dehydration and the patient's maintenance requirements, adjusting the infusion rate over the period needed to rehydrate (4–24 hours).

The ARF patient's fluid therapy plan should be reviewed several times daily to tailor the infusion rate and fluid composition to the circulatory volume, hydration status, ongoing losses that accumulate during hospitalisation (vomiting, diarrhoea, etc.) and, above all, urine production. In euvolaemic or hypervolaemic patients who are normo- or hypertensive and well-hydrated, if urine production is below 1 ml/kg/h, consider the use of diuretics. Such cases require catheterisation and uninterrupted monitoring of urine output (closed collection system). If the patient responds well and diuresis is restored, fluid and electrolyte balance must also be controlled because losses in

urine may be significant (polyuria). Stable patients and those leading up to hospital discharge should be gradually weaned from intravenous fluid therapy.

Recovery of electrolyte and acid-base balance

Although alterations in electrolyte and acid-base balance are variable and common in ARF, the most significant ones relate to potassium (hyperkalaemia) and metabolic acidosis:

- **Hyperkalaemic patients** ($[K^+]$ >8 mEq/l): if signs of cardiotoxicity appear (bradyarrhythmia, absence of P waves, peaked T waves, prolonged QRS), the therapeutic approach should be based on temporary protection of the myocardium, potassium transport into cells, and maximisation of excretion (diuresis). Use of 10 % calcium gluconate (0.5–1 ml/kg as a slow IV drip) can temporarily (15–20 minutes) counter the cardiotoxicity associated with hyperkalaemia, at which point insulin (0.2–0.4 IU/kg of regular insulin with 1–2 g of glucose/IU of insulin given) should be administered to transport potassium intracellularly. Blood sugar should be checked and fluids supplemented as appropriate. Alternatively, bicarbonate can be used for intracellular potassium transport (2 mEq/kg IV).
- **For patients with metabolic acidosis** (pH <7.2; $[HCO_3^-]$ <12 mEq/l): give sodium bicarbonate ($NaHCO_3$).

$$NaHCO_3\ (mEq/l) = 0.3 \times \text{body weight (kg)} \times (24 - [HCO_3^-] \text{ of the patient})$$

One-third of the dose should be given as a slow bolus and the rest over 4–6 hours; the patient's acid-base status and sodium levels should be carefully monitored. Do not use in hypernatraemic, hyperosmolar, hypoventilating or hypercapnic patients.

Diuretics

Although there exists no strong evidence that their use leads to an improved prognosis for veterinary patients, diuretics present a therapeutic option for complementary longer-term therapy in patients with anuria/oliguria, particularly if more effective options (haemodialysis or peritoneal dialysis) are not available. Despite their limitations, some options used alone or in combination are as follows:

- **Furosemide:** 2–4 mg/kg bolus (dogs) and 2 mg/kg (cats). Diuresis should begin within 30 minutes. If the first dose is ineffective, a second bolus can be given. If the second dose is ineffective, furosemide should be discontinued. If effective, continue with a 0.2–1 mg/kg/h CRI.
- **Mannitol:** 0.2–0.5 g/kg as a slow IV drip (5–10 minutes). This can be repeated 30 minutes later (up to a maximum of 1.5 g/kg/day) to continue the diuretic effect if the patient responds to the initial bolus. If the patient does not respond to the initial bolus, do not repeat administration. The use of mannitol is contraindicated in patients with signs of overhydration, dehydration, hyperosmolarity, active bleeding, congestive heart failure, or vasculitis.
- **40% dextrose:** 0.5–1 g/kg over 15-20 minutes. Treatment can be repeated every 8–12 hours if effective. If diuresis is not restored, do not continue the treatment.
- **Diltiazem:** ARF secondary to leptospirosis. Give an initial 0.3–0.5 mg/kg bolus over 10 minutes followed by a 3–5 µg/kg/min CRI. The infusion should be continued until creatinine normalises (48–72 hours). It can be given with furosemide until diuresis is restored. Monitor heart rate and blood pressure; suspend administration if the patient develops significant hypotension (systolic blood pressure <80 mmHg).
- **Dopamine:** low doses (1–3 µg/kg/min) are not currently recommended because its efficacy has not been demonstrated. Consider administration (5–10 µg/kg/min) in oliguric/anuric and hypotensive patients.

Supplemental treatment

ARF patients often present with nausea and vomiting, for which antiemetics (metoclopramide, maropitant, chlorpromazine, ondansetron) and gastric cytoprotective drugs (ranitidine, famotidine, omeprazole, and sucralfate) may be indicated. Their efficacy varies, and the choice depends on the clinician's preference and drug availability.

Nutritional support should start as early as possible while taking into account the patient's condition. If vomiting can be controlled with drugs, enteral feeding is preferred (assisted or unassisted with nasoesophageal or nasogastric tube). Parenteral feeding can be considered if vomiting is not controlled.

Specific therapies

As previously explained, the primary aetiology of ARF is often unknown; thus, therapy is based on control of uraemia and its manifestations. Specific treatment can be administered to patients with pyelonephritis, leptospirosis or ethylene glycol toxicity.

Feline urethral obstruction

Definition

Urethral obstruction in cats results from a sudden or intermittent blockage of urine passage through the urethra. It is classified by degree (partial or total) and aetiology. These clinical signs are often observed: haematuria, dysuria, periuria, pollakiuria, and stranguria.

Causes

The aetiology of urinary tract obstructions in cats is variable, but it is most common in certain breeds (Persians) and in middle-aged, male, neutered, obese, sedentary indoor cats who eat dry food. Urinary obstructions in cats are often associated with bladder or urethral pathologies: feline idiopathic cystitis, urolithiasis, infection, anatomic defects, neoplasms, or urethral plugs.

Diagnosis

Diagnosis of feline urinary tract obstructions is based on the patient's clinical history and clinical signs. Generally, affected cats present with difficulty urinating, painful urination (accompanied by vocalisation), increased urinary frequency, blood in the urine, or urination outside the litter box. Some cats only present with behaviour changes, and stop using the litter box or exhibit aggression.

In case of an inability to urinate due to complete urinary obstruction, the clinical history often reflects other systemic signs such as lethargy, anorexia, and vomiting. The examination reveals a firm, distended bladder that is difficult or impossible to express manually. In cases of postrenal azotaemia or

electrolyte alterations (hyperkalaemia), dehydration and global perfusion deficiencies are common: hypothermia, bradycardia, altered mentation, weak pulse, etc.

The diagnostic protocol for obstructed patients should involve a complete blood count and biochemistry, including assessment of electrolytes and acid-base balance, and urinalysis with culture, as well as a plain radiograph (or with contrast if indicated) and full urinary tract evaluation on abdominal ultrasound. An electrocardiogram is recommended in cases of hyperkalaemia or severe perfusion abnormalities.

Treatment

Treatment depends on the patient's condition and the most likely underlying cause. Nonetheless, the priority is to restore fluid, electrolyte, and acid-base balance, as well as reestablish urinary flow.

Fluid therapy

If the patient develops signs of hypoperfusion, start intravenous isotonic fluid therapy without potassium (10–30 ml/kg bolus of 0.9 % NaCl over 15 minutes) and provide heat to restore the body temperature. In these cases, oxygen therapy (continuous flow) is recommended. If hyperkalaemic cardiotoxicity ($[K^+]$ >8 mEq/l) occurs, administer emergency treatment (see previous section).

If the patient does not show signs of hypoperfusion, start an intravenous infusion of isotonic solution based on estimated dehydration and maintenance fluid requirements (without potassium if $[K^+]$ >6.5 mEq/l).

During monitoring, it is important to review the fluid therapy plan because postobstructive polyuria is common. In these cases, adequate fluid support and potassium supplementation are required.

Urethral catheterisation

In very depressed patients, catheterisation may be performed with only sedation (opioids with or without benzodiazepines), but in most patients, analgesia and potentially general anaesthesia may be indicated during the procedure. For analgesia, butorphanol (0.2–0.4 mg/kg) or buprenorphine (10–20 µg/kg) can be used. Anaesthetic induction can be performed with diazepam (0.5 mg/kg) and ketamine (2.5–5 mg/kg) slow IV infusion, followed by anaesthetic maintenance with isoflurane and oxygen.

Catheterisation should be performed aseptically and atraumatically. The exteriorised penis should be massaged gently as small clots or plugs often emerge at the tip. If catheterisation is difficult, partial emptying of the bladder via ultrasound-guided cystocentesis may facilitate the procedure. If catheterisation is impossible, repeated palliative cystocentesis or placement of a cystotomy tube is indicated until the patient is stabilised and surgical removal of the obstruction (urethrostomy) can be performed.

If catheterisation is possible, once urinalysis and culture samples are obtained, lavage the bladder with warm saline (37 °C) and connect the catheter to a sterile collection system. Urethral catheters should be left in place until azotaemia resolves and the patient's symptoms improve.

Supplemental treatment

Analgesia (buprenorphine: 20 µg/kg SC every 8 hours) is necessary. Addition of antibiotics is controversial if the obstruction has a noninfectious cause. Antibiotic therapy is recommended in cases of confirmed UTI (urinary tract infection), according to urine culture and antibiotic susceptibility results.

Depending on the case and the clinical approach, consider the following during hospitalisation:

- Prazosin: reduces urethral spasm at doses of 0.25–1 mg/cat PO every 8–12 hours.
- Diazepam: relaxes skeletal muscle of the urethral sphincter at doses of 1–2.5 mg/cat PO every 12 hours.
- Meloxicam: analgesic and anti-inflammatory, give 0.05–0.1 mg/kg PO every 24 hours for up to 4–5 days.

Start a wet diet (prescription diet in cases of urolithiasis) as promptly as possible, promote fluid intake, minimise environmental stress, and perform a behavioural intervention.

7

Neurological emergencies

Seizures

Definition

Seizures are a transient sign caused by paroxysmal changes in the electrical activity of cerebral cortical neurons; they may appear as partial or generalised tonic-clonic seizures and occur along with behavioural changes including loss of consciousness, involuntary urination and defecation, vocalisation, or facial tremors. Veterinary medicine includes two categories of neurological emergencies linked to seizure activity:

- **Status epilepticus** is defined as seizures lasting longer than 5 minutes or multiple seizures without recovery of mentation between episodes.
- **Cluster seizures** are defined as two or more seizures within 24 hours, but with recovery of mentation between episodes.

Causes

Seizure aetiology is quite variable and may be due to both intracranial and extracranial causes. Age of onset, breed, type of episode, and progression can help narrow down the differential diagnosis in many patients. Box 1 shows the most common causes of seizure episodes by patient age.

Box 1.

Most common causes of seizure by patient age.

- **Patients <1 year old:** congenital anomaly (e.g. hydrocephalus), cranioencephalic trauma, hypoglycaemia, toxicity, infectious process (e.g. distemper), and portosystemic shunt.
- **Patients 1–5 years old:** idiopathic epilepsy, immune-mediated encephalitis, neoplasms, and other causes mentioned above.
- **Patients >5 years old:** neoplasm, hepatic encephalopathy, hypoglycaemia (insulinoma), acquired metabolic disease (e.g. uraemia), infectious processes, and other causes of encephalitis.

Diagnosis

The clinical history, physical examination, and full neurological examination are critical elements of the diagnostic protocol for seizure patients. Complete the protocol with laboratory testing (complete blood count, biochemistry, and urinalysis) as well as specific tests based on the clinical suspicion and differentials (e.g. pre- and postprandial bile acids, ammonia, insulin levels, and tests for toxicity or infection, etc.). In case of a possible underlying systemic process or intracranial disease, basic imaging (thoracic radiographs and abdominal ultrasound) can help distinguish an infection from a neoplasm. If primary intracranial disease is suspected, more advanced diagnostic imaging (MRI, CT scan), CSF analysis, collection of samples for histopathological analysis (cytology or biopsy) or blood testing for specific infectious diseases should be performed.

Treatment

The main goal of seizure treatment is to stop it as quickly as possible. General treatment for epileptic patients, and particularly those who present in status epilepticus or with cluster seizures, should focus on emergency evaluation and stabilisation (ABC, temperature, blood glucose, and blood pressure), drug treatment for seizures, and prevention and treatment of possible complications (e.g. cerebral oedema associated with continuous seizure activity).

Acute drug treatment usually employs benzodiazepines (diazepam), barbiturates, (phenobarbital and pentobarbital), and propofol. Table 1 shows the protocol for managing a patient in status epilepticus or with cluster seizures.

Veterinary surgeons are now using new anticonvulsant drugs adapted from human medicine. One of these, levetiracetam, seems to be effective when given as a 60 mg/kg bolus followed by 20 mg/kg every 8 hours. This drug is often given in combination with the aforementioned drugs for acute seizure control.

Diazepam and phenobarbital are the drugs of choice for acute seizures. Chronic therapy is often based on phenobarbital alone (dogs and cats) or in combination with potassium bromide (dogs only) if necessary for adequate seizure control.

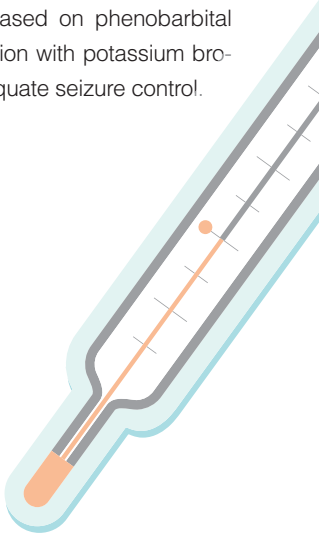


Table 1. Acute drug treatment for seizures or status epilepticus in dogs and cats.

Drug	Dose	Notes
Diazepam	• IV bolus: 0.5–1 mg/kg, may be repeated 2–3 times. • CRI: 0.5–1 mg/kg/h. • Rectal: 0.5–1 mg/kg (2 mg/kg if already receiving phenobarbital).	If a CRI is necessary due to refractory seizures in spite of giving IV boluses, use of a central venous line is preferable.
Phenobarbital	2–4 mg/kg IV every 20–30 minutes to a maximum of 18–20 mg/kg.	• Administer with diazepam to prevent recurrence of seizures once brain levels of diazepam begin to decrease (30 minutes). • The recommended dose can be repeated every 20–30 minutes up to a cumulative maximum of 20 mg/kg. • Once the seizures are controlled, follow with a maintenance dose (3–5 mg/kg IV or IM every 12 hours for 24–48 hours). • Oral anticonvulsive therapy should be given every 12 hours as soon as the patient is stable and able to swallow.
Propofol	2–8 mg/kg slow IV bolus, administering 25 % of the dose every 30 seconds to effect.	• Administration as a CRI (0.1–0.4 mg/kg/min) may be considered if the patient does not respond to the above drugs or the response to boluses is only partial or temporary. • Carefully monitor physiological parameters (propofol can stop tonic-clonic activity but not cerebral seizure activity).

Cranioencephalic trauma (CET)

Definition

Cranioencephalic trauma (CET) is defined as an alteration in neurological function or other evidence of cerebral pathology caused by external trauma that induces primary or secondary physical damage to the brain.

Causes

Trauma-induced brain injuries are a common reason for presentation at the emergency clinic. Causes are myriad: accidents involving vehicles, falls, crush injuries, penetrating wounds, fight injuries, etc.

Diagnosis

Primary brain injuries are often a direct result of trauma and occur at the moment of trauma. These traumas include skull fractures, vascular injuries leading to haemorrhage, oedema and lesions of brain tissue (brain concussion, contusion and laceration).

Secondary brain lesions may occur as a result of physiological changes that are caused by the primary lesion and ultimately result in increased intracranial pressure (ICP). These changes occur hours to days after the initial trauma and are induced by the abundant release of excitatory neurotransmitters (especially glutamate) and inflammatory mediators (cytokines); the infiltration, accumulation, and activation of inflammatory cells; as well as the release of nitric oxide, which leads to excessive vasodilation and a loss of autoregulatory function due to pressure from the injured brain tissue.

Evaluation of CET patients should include physical and neurological examinations, with special attention to the level of consciousness, posture, pupil size, and pupillary light reflex. The modified Glasgow scale is a quantitative measure of these parameters that has proven to be a useful 48-hour prognostic indicator in dogs with CET. The minimum, most relevant testing should include haematocrit and total protein, glucose, lactate, and blood gas and acid-base analysis. Patients with severe neurological signs or who deteriorate despite intensive systemic and intracranial treatment should undergo diagnostic imaging to identify lesions that potentially require surgical treatment. In these cases, CT and MRI are the methods of choice.

Treatment

The therapeutic approach to CET requires a global assessment with a special emphasis on both the intra- and extracranial aspects that need to be treated first. Patients with traumatic brain injury often present with other currently or potentially life-threatening problems: penetrating wounds in body cavities (thorax, abdomen) caused by the same traumatic event, airway obstructions, respiratory compromise, circulatory collapse, etc. Such situations should be evaluated during the initial exam and prioritised for appropriate treatment. Once the extracranial problems have been addressed and treated appropriately, the clinician should focus on intracranial problems: maintaining cerebral perfusion pressure and oxygenation and avoiding elevations in ICP.

Maintaining perfusion pressure and cerebral oxygenation

The main goal of treatment is to maintain the O_2 supply to brain tissue, which requires sufficient circulating blood volume, adequate systemic blood pressure, and good O_2 transport capacity:

- **Oxygen therapy:** administer via continuous flow, mask, or a nasal cannula. Maintain a PO_2 greater than or equal to 90 mmHg in dogs and 100 mmHg in cats, or an SaO_2 >95 %. Saturations below 89 % are equivalent to a PO_2 <60 mmHg and thus pose a risk of increased cerebral bloodflow and ICP.
- **Ventilation:** maintain a $PaCO_2$ and an $ETCO_2$ between 30 and 35 mmHg. Persistently low oxygen levels induce vasoconstriction and neuronal ischaemia, while hypercapnic states favour an increased ICP due to cerebral vasodilation.
- **Fluid therapy:** patients with cranial trauma often present in hypovolaemic shock; thus, the goal in restoring blood volume is to maintain a minimum MAP of 80 mmHg (see Ch. 2).
- **Neurological examination:** once fluid volume and oxygenation are restored, the neurological examination may be performed, along with additional testing to evaluate possible lesions of the nervous system or in the rest of the body. Repeat the neurological examination every 30–60 minutes to detect signs of deterioration and monitor treatment efficacy.

Avoiding/treating elevations in intracranial pressure (ICP)

Elevation of the head (25–30 degrees without jugular compression) and, most importantly, administration of hyperosmotic agents are the most practical measures for reducing intracranial hypertension in severe CET. The protocol is as follows:

- **Mannitol:** 0.5–1.5 g/kg over 15 minutes. It exerts an immediate rheological effect by decreasing blood viscosity and a rapid osmotic effect (after 15–20 minutes) that reduces cerebral oedema and induces diuresis. Prolonged effects occur over 2–6 hours. Do not give more than three boluses or administer as a CRI. Replace fluid volume a posteriori with crystalloids or colloids to maintain intravascular volume.
- **Hypertonic saline (7.5 %):** 3–5 ml/kg over 15 minutes. This is an option for use in hypovolaemic patients with severe cranial hypertension. Its use is contraindicated in cases of hypernatraemia.
- **Hyperventilation:** if the patient does not respond to the aforementioned therapies, hyperventilation may be used in a targeted manner. The goal is to maintain CO_2 pressure between 25 and 30 mmHg temporarily and to decrease ICP via hypocapnia-induced cerebral vasoconstriction.
- **Induced hypothermia-barbiturate coma:** may be used if the hypertension does not respond to the above measures. This requires intense monitoring and special equipment.
- **Other supplemental measures:** designed to minimise secondary brain damage, these include strict control of post-traumatic seizures, maintenance of euglycaemia (to prevent hypo- and hyperglycaemia), analgesia with pure opioid agonists that are reversible (with naloxone), and prompt nutritional support. Decompressive craniotomy is indicated in patients who do not respond to extra- and intracranial therapy and those who present with extra-axial haematomas, depressed fractures, or foreign bodies on diagnostic imaging.

8

Metabolic emergencies

Addisonian crisis

Definition

Addisonian crisis (hypoadrenocorticism) is an endocrine emergency resulting from insufficient adrenal glucocorticoids, mineralocorticoids, or both.

Causes

Hypoadrenocorticism (HA) is a disease that is more prevalent in dogs and predominantly affects young and middle-aged female dogs of particular breeds. Small animal clinical endocrine emergencies usually involve adrenal insufficiency (primary HA), which is generally immune-mediated and characterised by the destruction of glandular tissue as well as variable deficiencies in the synthesis of glucocorticoids (cortisol), mineralocorticoids (aldosterone), or both.

Diagnosis

HA can cause diverse signs that are generally nonspecific (weakness, vomiting, diarrhoea, anorexia, etc.), so it is often confused with other more common diseases.

The diagnosis of adrenal crisis should be based on the patient's clinical history, the presence of compatible signs, and laboratory diagnostic tests. The most common clinical signs are weakness, anorexia, vomiting, diarrhoea, abdominal pain, weight loss, polyuria/polydipsia, and signs of generalised

hypoperfusion (pale mucous membranes, prolonged CRT, weak pulse, tachypnoea, hypothermia) in the presence of bradycardia.

The most common clinicopathological signs are a decreased Na:K ratio (<27:1), azotaemia with iso- or hyposthenuria, anaemia and leukograms that are incompatible with the severity of the patient's disease, hypoglycaemia, hypercalcaemia, hypocholesterolaemia, hypoalbuminaemia, and metabolic acidosis. Patients with hypovolaemia and hyperkalaemia are often bradycardic and exhibit the following ECG anomalies: lack of P waves, prolonged QRS, and peaked T waves. Severe cases may involve ventricular fibrillation or asystole.

Definitive diagnosis is based on low resting cortisol (≤0.02 µg/dl) and cortisol levels below the reference range following ACTH stimulation.

Treatment

Treatment is based on fluid therapy, correction of electrolyte alterations, and hormone replacement.

Fluid therapy

If the patient is hypovolaemic, a 10–30 ml/kg bolus of 0.9 % NaCl every 15 minutes is advisable, taking into account the severity of the hypoperfusion and hyponatraemia, until the patient is haemodynamically stabilised. Monitor electrolytes (sodium and potassium) and repeat the neurological exam, especially in cases of severe hyponatraemia (Na <130 mEq/l), regularly (every 1–4 hours). In case of hypoglycaemia (glucose <50 mg/dl), a bolus of 40 % glucose diluted in normal saline (1:1) should be given as a slow infusion (0.5–1 ml/kg IV).

Correction of electrolyte alterations

If symptomatic hyperkalaemia occurs ([K^+] ≥7–8 mEq/l):

- Administer 10 % calcium gluconate (0.5–1 ml/kg) as a slow IV bolus with ECG monitoring. This protects the myocardium for approximately 20 minutes but does not resolve the hyperkalaemia.
- Administer regular insulin (0.1–0.2 IU/kg IV) followed by a glucose bolus (1–2 g/IU of insulin administered) and supplement the fluid regimen with 2.5–5 % glucose.
- Monitor glucose and electrolyte levels every 1–4 hours until they normalise.

Repeat the procedure as necessary until potassium levels decrease below the cardiotoxicity threshold (<7 mEq/l), normalise, or are controlled by restoring diuresis.

Hormone replacement

Once resting cortisol is determined and the ACTH stimulation test is performed, hormone replacement therapy may be initiated:

- **Mineralocorticoid:** injectable desoxycorticosterone pivalate at a starting dose of 2.2 mg/kg IM or SC every 25 days. Alternatively, patients who can tolerate oral dosing should receive fludrocortisone at a starting dose of 0.02 mg/kg/day.
- **Glucocorticoid:** hydrocortisone, initially 1.25 mg/kg IV and then 0.5–1 mg/kg IV every 6 hours, in decreasing doses until the patient is stabilised, and followed with oral prednisone. Another injectable option for adrenal crisis is prednisolone at a starting dose of 4 mg/kg IV, followed by 2–4 mg/kg every 8 hours, then given in decreasing doses. Finally, dexamethasone may also be given at a starting dose of 0.5 mg/kg IV, followed by doses decreasing by 0.05 to 0.1 mg/kg every 12 hours. Dexamethasone can be used as emergency hormone therapy because it will not interfere with the results of the ACTH stimulation test.

Supplemental treatment

In these patients, supplemental treatment usually includes fluid therapy according to their needs (dehydration, maintenance, etc.), monitoring and correction of hypoglycaemia, administration of gastroprotectants and antiemetics, and analgesia (opioids) if necessary. In most patients with severe adrenal crisis, oral replacement therapy can begin within 24–48 hours and be followed up on an outpatient basis. Medium- and long-term follow-up requires adapting the oral treatment (usually fludrocortisone and prednisone) to the patient's clinical progress and laboratory results during the days, weeks, and months following the adrenal crisis.

Diabetic ketoacidosis

Definition

Serious decompensation of diabetes mellitus that requires emergency treatment and is characterised by overproduction of ketone bodies in blood, resulting in severe acid-base and electrolyte imbalances.

Causes

Diabetic ketoacidosis is a common, severe complication of diabetes that occurs in both dogs and cats of middle or advanced age, and it is nearly always associated with a concurrent illness. In fact, 70 % of dogs and 90 % of cats with diabetic ketoacidosis suffer from a concurrent disease.

- In dogs, the most common associated causes are pancreatitis, urinary tract infections, and other endocrinopathies (hyperadrenocorticism). The presence of other conditions (obesity, gestation, diestrus) or chronic diseases (heart or

renal failure, etc.) in diabetic patients may also predispose them to this complication.

- In cats, acute pancreatitis, hepatic lipidosis, renal failure, viral infections, urinary tract infections, and neoplasms are the most common predisposing conditions.

Diagnosis

The clinical signs may generally be categorised as those related to uncontrolled diabetes (polyuria/polydipsia, polyphagia, weight loss, cataracts, etc.), those resulting from the concurrent pathology, and acute signs linked to the ketoacidotic crisis itself. The most common ones in dogs are polyuria/polydipsia, lethargy, anorexia, vomiting, dehydration, and weight loss. Cats often present with lethargy, poor body condition, icterus, hepatomegaly and dehydration.

When the clinical history and signs are compatible, the presence of hyperglycaemia with ketosis and severe metabolic acidosis with an increased anion gap is diagnostic. These patients should receive a complete blood count, serum biochemistry with electrolytes, acid-base analysis, urinalysis and culture, along with other hormonal or diagnostic testing based on the patient's particular condition (pancreatic lipase, diagnostic imaging, etc.).

Treatment

Treatment of ketoacidotic patients is based on fluid therapy, supplementation of required nutrients, administration of insulin, and monitoring of potential complications.

Fluid therapy

If the patient is hypovolaemic, a 10–30 ml/kg bolus of isotonic crystalloids every 15 minutes is advisable, taking into account the severity of the hypoperfusion and hyponatraemia, until the patient is haemodynamically stabilised. If the patient

is dehydrated but not hypovolaemic, isotonic crystalloids should be administered according to the degree of dehydration, maintenance requirements, and ongoing losses, while monitoring hydration, mentation, and electrolyte/acid-base balance. If the patient initially presents with hyponatraemia or hypochloraemia, administer 0.9 % NaCl, but in case of severe metabolic acidosis (pH <7.2; [HCO_3^-] <12 mmol/l), hypokalaemia or hyperosmolarity, balanced isotonic crystalloids (lactated Ringer's) are recommended. The fluid plan should be reevaluated every 6 hours for the first 24–36 hours and then every 12–24 hours based on patient progress.

Supplementation

Supplementation should be based on electrolyte and metabolic alterations that the patient presents. Potassium, phosphorus and magnesium are nearly always required, especially once insulin administration has started:

- **Potassium:** potassium (as potassium chloride) should be provided based on the changes in blood potassium levels (Table 1). In case of severe hypokalaemia (<2.0–2.5 mEq/l), administer a CRI to a maximum dose of 0.5 mEq/kg/h. Depending on severity, serum potassium levels should be checked every 1, 4 or 8 hours.
- **Phosphorus:** phosphorus (as potassium phosphate) should be provided based on the changes in blood phosphate levels. In case of severe hypophosphataemia (2.0 mg/dl) a CRI at a rate of 0.01–0.03 mmol/kg/h should be provided. Levels should be checked every 1, 4 or 8 hours depending on severity.
- **Magnesium:** magnesium (as magnesium sulphate) should be provided based on the changes in blood levels; ionised magnesium (Mg^{2+}) is preferred. Hypomagnesaemia should be suspected in cases of refractory hypokalaemia. In cases of refractory hypomagnesaemia, a CRI of 0.5–1 mEq/kg every 24 hours is recommended. Depending on the severity of the hypomagnesaemia, magnesium levels should be checked every 12 or 24 hours.

Table 1. Potassium supplementation in hypokalaemic patients.

Serum potassium concentration	Potassium added to 500 ml of fluids
1.6–2.0 mmol/l	80 mEq
2.1–2.5 mmol/l	60 mEq
2.6–3.0 mmol/l	40 mEq
3.1–3.5 mmol/l	20 mEq

- **Bicarbonate:** its use is recommended only in extreme acidaemia (pH <7,0; [HCO_3^-] <10 mmol/l), given that the acidosis in these patients can generally be corrected with fluids and insulin. In addition, inadequate bicarbonate supplementation can exacerbate the hypokalaemia that ensues from insulin treatment as well as favour ketone formation in the liver and induce a paradoxical cerebral acidosis. Despite these factors, if it is still considered necessary, give one-half or one-third of the calculated quantity (0.1 × weight in kg × base deficit) over 20–30 minutes, reevaluating the acid-base status every hour and repeating the dose until pH >7.0. The acid-base status should be rechecked every 8, 12 or 24 hours according to the severity of the metabolic acidosis.

Administration of insulin

Insulin therapy should be started once the perfusion deficiencies are corrected and rehydration is underway. In most patients, it can be started after 1 to 6 hours of fluid therapy. Ketoacidotic patients should be treated with a regular insulin CRI (Table 2) and receive blood glucose checks every 2 hours. Prepare the solution by adding 2.2 IU/kg (dogs) or 1.1 IU/kg (cats) of regular insulin to 250 ml of 0.9 % NaCl. This solution should be given IV at a rate of 0.05–0.1 IU/kg/h, considering a blood glucose drop of 50 mg/dl/h to be appropriate.

Table 2. Administration of regular insulin in patients with diabetic ketoacidosis.

Blood glucose (mg/dl)	Isotonic crystalloid composition	Rate of administration of insulin CRI
>250	0.9 % NaCl or lactated Ringer's	10 ml/h
200–250	0.45 % NaCl + 2.5 % glucose	7 ml/h
150–200	0.45 % NaCl + 2.5 % glucose	5 ml/h
100–150	0.45 % NaCl + 5 % glucose	5 ml/h
<100	0.45 % NaCl + 5 % glucose	Stop administration

This intravenous protocol should be continued until the patient begins to ingest and tolerate food, the ketonaemia resolves, the acid-base status normalises, and the biochemical parameters demonstrate clear improvement. The clinician should consider switching to a subcutaneous slow-acting insulin only once the patient is stable and well-hydrated.

Supplemental treatment

Antibiotics, antiemetics, heparin, gastroprotectants, etc., may be used depending on the patient's clinical situation and the particular complications that are causing or associated with the ketoacidosis.

9

Reproductive emergencies

Dystocia

Definition

The term “dystocia” applies when labour or delivery is proceeding abnormally or with difficulty, regardless of whether the cause is maternal or foetal.

Causes

Dystocia can be the result of insufficient or uncoordinated uterine contractions, abnormal foetal position, relative or absolute cephalopelvic disproportion, foetal death, or abnormalities that affect the birth canal (hereditary or acquired).

Diagnosis

Evaluation of a patient with dystocia should include a thorough physical and vaginal examination, a complete clinical history of the pregnancy, and a full reproductive history, including previous reproductive events, males used for breeding, time when labour began, frequency and intensity of contractions, and time interval between deliveries.

Presence of one or more of the following signs suggests the need for clinical assistance during labour:

1. Foetal membrane rupture without contractions.
2. Foetal membranes in the vulva for more than 15 minutes.
3. Absence of signs of labour within 24–36 hours of the mother's temperature decreasing below 37.5 °C.
4. Abnormal vaginal discharge: haemorrhagic, purulent secretion.
5. Delay of more than one week past the due date.
6. Presence of contractions without delivery for more than 2–4 hours.
7. More than 2 hours between deliveries or prolongation of labour (>24 hours).
8. Repeated contractions (>30 minutes) without delivery.

Diagnostic evaluation of patients with signs of dystocia should include a complete blood count and biochemistry with calcium and glucose levels. Radiographs and ultrasound can help to identify the cause of dystocia and evaluate the level of foetal distress.

Treatment

Medical treatment

- Foetus lodged in the birth canal: take radiographs to determine the position and presentation. Sterile gloves and sterile lubricant should be used during manipulation of the birth canal and extraction of the foetus. If possible, pull from the extremities ventrally, following the natural orientation of the birth canal.

- Non-obstructive dystocia: in case of normal foetal presentation, with no obstruction of the canal, with weak or nonexistent contractions, cervical dilation, and no foetal distress (heart rate >160 bpm), the following can be administered:
  - Oxytocin: 0.1–2 IU/kg IM or SC (dogs) and 0.5–1 IU/kg IM or SC (cats) every 30 minutes as long as delivery is progressing. Maximum: 20 IU total IM (dogs) and 4 IU total IM (cats).
  - 10 % calcium gluconate if oxytocin is ineffective, contractions are ineffective, or hypocalcaemia is detected: 3–5 ml/kg (dogs) and 3–5 ml (cats), as a slow intravenous drip (15 minutes).
  - 40 % dextrose if hypoglycaemia is detected or the above measures are ineffective: 0.5–1 ml/kg as a slow intravenous drip (15 minutes).

Surgical treatment

The indications for performing a Caesarean section are as follows:

- Partial primary or secondary uterine inertia that does not respond to medical treatment.
- Excessive foetal size relative to the birth canal.
- Pelvic canal malformations.
- Abnormal foetal presentation and inability to manipulate it.
- Foetal death.
- Systemic illness of the mother.
- Suspected uterine torsion, rupture, prolapse or hernia.
- Evidence of foetal distress.

Pyometra

Definition

Pyometra is an accumulation of purulent material in the uterus. This condition generally occurs in intact females of middle or advanced age, predominantly in dogs.

Causes

The primary pathology is cystic endometrial hyperplasia resulting from prolonged and repeated exposure to progesterone during diestrus (progesteronaemia) over the female's reproductive lifetime.

Opportunistic bacterial colonisation of the uterus by ascent from the vagina during oestrus and later progesterone-induced closure of the cervix in diestrus can favour development of metritis-pyometra.

Diagnosis

Signs and clinical detection vary based on whether the cervix is open (visible purulent vaginal discharge) or closed (absence of discharge, presence of signs of endotoxaemia and sepsis). Most cases involve recent oestrus (1–2 months) and one or more of these signs: anorexia, depression, lethargy, polyuria/polydipsia, vomiting/diarrhoea, vaginal discharge (open cervix), abdominal pain and distension, fever, and signs of hypovolaemic or distributive (septic) shock in severe cases.

Diagnostic tests often reflect the systemic effects of this pathology and are generally proportional to the severity of the complications secondary to the infection. Leukocytosis or leukopaenia, thrombocytopaenia, anaemia, azotaemia, hyper- or

hypoproteinaemia, hyper- or hypoglycaemia, increased liver enzymes, and alterations in coagulation (PT, APTT, fibrinogen) are all commonly observed. Although abdominal radiography can assist with diagnosis, the test of choice is ultrasound. The presence of multiple cavities containing anechoic material is diagnostic of pyometra in patients with a compatible clinical history and signs.

Treatment

The treatment of choice is surgery (ovariohysterectomy). However, depending on the patient's condition, prior medical treatment tailored to the specific situation may be required.

Initial stabilisation

- **Fluid therapy:** this is performed in order to reestablish patient perfusion and hydration, in addition to correcting possible electrolyte imbalances, using appropriate isotonic crystalloids (lactated Ringer's, normal saline, etc.). In case of hypoproteinaemia (TP <6 g/dl) or hypoalbuminaemia (Alb <2.0 g/dl), consider colloids (10–20 ml/kg/day).
- **Broad-spectrum antibiotic therapy:** enrofloxacin (5–10 mg/kg/day IV). If the patient exhibits clear signs of sepsis, broader coverage with ampicillin (22 mg/kg IV every 8 hours) or amoxicillin-clavulanic acid (12–25 mg/kg every 8–12 hours IV) is recommended. In cases of peritonitis with severe sepsis or septic shock, choose carbapenems while awaiting the peritoneal fluid culture and sensitivity results.
- **Analgesia:** pure opioids such as methadone at doses of 0.2–0.4 mg/kg IV or SC, or fentanyl as a bolus of 5 µg/kg every 15 minutes or a CRI of 5–10 µg/kg/h, can be given for intense pain.
- **Fresh frozen plasma:** in case of prolonged clotting times and thrombocytopaenia, administer 6–10 ml/kg plasma every 8 hours as a slow intravenous drip (2–3 hours) until clotting times normalise.

- **Low-molecular weight heparin:** in cases of hyperfibrinogenaemia, elevated D-dimers, shortened clotting times or signs of thrombosis, consider adding low-molecular weight heparin (dalteparin or enoxaparin) to the fluid regimen.
- **Supplemental treatment:** oxygen therapy, transfusion of blood products (albumin, packed red blood cells), etc.

Medical treatment

This is a temporary solution that is indicated only if the endometrium is healthy and the patient's life is not in danger. The following can be administered:

- **Prostaglandin $F_{2\alpha}$:** the veterinary literature contains various protocols with different recovery, relapse, and fertility rates after treatment. Antibiotics should always be provided simultaneously. Side effects can be pronounced: anxiety, tachypnoea, ptyalism, tachycardia, abdominal pain, fever, vomiting, and diarrhoea.
- **Aglepristone (antiprogestogen):** 10 mg/kg SC on days 1, 2, and 8 in dogs and days 1, 2, and 7 in cats. A follow-up ultrasound examination should be performed on day 14 to determine if the pyometra has resolved. If it has not resolved, repeat the treatment and ultrasound examination at 21 days. Complete the treatment with cloprostenol (1 µg/kg for 5 days, from days 3 to 7 of treatment).

Surgical treatment

This is the treatment of choice. Ovariohysterectomy patients should remain hospitalised for at least 48 hours to monitor their progress.

10

Environmental emergencies

Heat stroke

Definition

Hyperthermia designates the condition in which the patient's body temperature increases above the normal range for the species (39.5 °C). Hyperthermia linked to excessive exercise or exposure to humid, hot environments is referred to as "non-pyrogenic hyperthermia" and is characterised by a sharp increase in body temperature above the range set by the thermoregulatory centre of the hypothalamus (41 °C). The patient may present with varying degrees of hyperthermia; in order of increasing severity, they are:

1. **Heat stress:** thirst, muscle cramps, weakness.
2. **Heat exhaustion:** intense thirst, fatigue, weakness, muscle tremors, vomiting, diarrhoea.
3. **Heat stroke:** the above signs plus severe CNS dysfunction and clinical or laboratory evidence of multiorgan dysfunction.

Causes

Mere exposure to a warm environment or exercise in a warm environment does not in itself cause heat stroke; rather, the increase in body temperature is the cause of what is referred to as a "heat-related illness". Under such conditions, the patient is likely unable to shed sufficient heat by evaporation, radiation, conduction or convection, which may lead to life-threatening hyperthermia.

Box 1 lists conditions that can predispose patients to heat stroke.

Box 1.

Predisposing factors for heat stroke.

- Poor climate control in the immediate environment.
- Confinement in poorly ventilated areas.
- High relative humidity.
- Lack of access to water.
- Decreased respiratory function (brachycephaly, obesity, laryngeal paralysis, tracheal collapse, etc.).
- Presence of cardiac disease.
- Paediatric or geriatric patients.

Diagnosis

Diagnosis is based on clinical history, elevated body temperature, and the presence of related clinical signs. While the body temperature rapidly increases, three protective mechanisms are activated: thermoregulation, release of mediators linked to the acute phase inflammatory response, and synthesis of protective intracelullar heat-shock proteins. When these three mechanisms fail, the patient can develop heat stroke leading to organ dysfunction. The respiratory, cardiovascular, nervous, gastrointestinal, renal and haemostatic systems are most commonly affected. The systems affected and to what degree depend on the severity and duration of the hyperthermia.

Hyperthermia is generally obvious at admission, but the patient's temperature may be high, normal or low depending on whether the owner has already attempted cooling measures and whether the patient's perfusion has been compromised.

The most common clinical signs are tachypnoea, dyspnoea, auscultatory abnormalities (lung crackles, etc.), tachycardia, ventricular arrhythmias, hyperaemia, vasodilation (CRT <1 s), icterus (haemolysis), pulse pressure that varies with the degree of hypovolaemia, blindness, disorientation, ataxia, stupor, coma, seizures, vomiting, diarrhoea, haematemesis, melaena, haematochezia, oliguria/anuria, petechiae, ecchymoses and excessive bleeding from venipuncture sites.

Depending on the degree of hyperthermia and the patient's condition, laboratory values may vary proportionally to the hyperthermia. Patients may exhibit haemoconcentration; hypoglycaemia; electrolyte alterations; severe metabolic acidosis; azotaemia; glucosuria; elevated transaminases, muscle enzymes, and bilirubin; and often thrombocytopaenia and coagulopathies (DIC).

Treatment

Treatment consists of cooling appropriate to the patient's condition and medical treatment to protect the cardiovascular system and prevent complications.

Cooling of the patient is based on measures that maximise heat loss via evaporation, conduction, radiation or convection. These techniques should be used until the core temperature reaches the high normal range (39–39.5 °C) and, ideally, declines at a rate of 0.1–0.2 °C/min.

Cooling techniques

External cooling (passive and active)

- Immerse or bathe in cold water. Do not bathe the patient in ice water because this may cause excessive vasoconstriction and result in an inability to dissipate body heat.
- Dampen the patient with cold, wet towels and place fans nearby to encourage heat loss through evaporation.
- Apply ice packs, not directly in contact with the skin, to anatomical regions with major vascular plexuses (cervical, axillary and inguinal regions) to encourage heat loss through conduction.

Internal active cooling

- Administer room temperature or cool intravenous fluids (immerse infusion bag in an ice water bath).
- Perform a gastric lavage or an enema with cold water in extreme or refractory cases. Consider peritoneal lavage under aseptic conditions in an appropriately equipped hospital.

Cooling should be stopped when the body temperature reaches 39.5 °C. This will help prevent rebound hypothermia.

General medical treatment

- Evaluate and ensure the patient's ABC; this can be achieved by administering continuous flow oxygen and watching for respiratory compromise. Begin fluid therapy with cold or room temperature solutions:
  - Patients in shock: 0.9 % NaCl at a dose of 50–90 ml/kg (dogs) and 40–60 ml/kg (cats); give a quarter dose every 15 minutes until perfusion parameters normalise. Synthetic colloids may be added if the patient exhibits hypoproteinaemia (TP <6 g/dl) or hypoalbuminaemia (Alb <1.5 g/dl). The dose is a 5–10 ml/kg bolus (dogs) or a 2.5–5 ml/kg slow bolus over 10–15 minutes (cats). If given in combination with crystalloids, reduce the crystalloid dose by 50 %.
  - Patients with refractory hypotension: administer dopamine (5–15 µg/kg/min) or norepinephrine (0.05–0.3 µg/kg/min).
  - Hypoglycaemic patients: give a slow bolus of 40 % dextrose (0.5–1 ml/kg).
- Evaluation of a possible neurological lesion: perform a neurological exam with special attention to mentation and cranial nerve functions. Evaluate and correct possible deficiencies due to hypoperfusion and abnormal haematocrit, total protein, and glucose values. If these anomalies persist or the patient deteriorates, neuroprotective measures should be implemented: elevate the patient's head 15–30° and consider giving mannitol (0.5–1 g/kg IV over 20–30 minutes).
- Evaluation of renal function: place an indwelling urinary catheter to measure urine output. Perform a urinalysis to look for signs of acute kidney injury (casts, glucosuria, isosthenuria, etc.). Ensure euvolaemia and maintenance of mean arterial pressure (MAP) (>80 mmHg) and urine output (>2 ml/kg/h).
- Evaluation of clotting function: administer fresh frozen plasma (6–10 ml/kg IV every 8 hours) if there are signs of DIC or low-molecular weight heparin if a hypercoagulable state or thrombosis is suspected.

- Evaluation of the gastrointestinal tract: if signs of gastric ulceration or hypoperfusion appear, give omeprazole (1 mg/kg IV every 24 hours) and sucralfate.
- In case of multiorgan dysfunction, severe hypoperfusion, or damage to mucosa, give broad-spectrum antibiotics, e.g. ampicillin (22 mg/kg IV every 8 hours) along with enrofloxacin (5 mg/kg IV or SC every 24 hours).
- Correct any acid-base alterations that the patient may have and start nutritional support (enteral or parenteral) as promptly as possible.

The prognosis of these patients depends on the particular predisposing factors, the number of organ systems affected, and the degree of neurological dysfunction. Most patients who survive the first 48 hours will recuperate if they receive appropriate care.

Hypothermia

Definition

Hypothermia designates the condition in which the patient's body temperature decreases below the normal range for the species. The varying degrees of severity are as follows: mild (36.7–37.7 °C), moderate (35.5–36.7 °C), severe (33–35.5 °C), and critical (<33 °C).

Causes

Hypothermia can result from any condition or state that induces an increased loss of body heat, a decrease in heat production, or dysfunction of the thermoregulatory centre of the hypothalamus.

The categories are primary (or accidental) and secondary. The primary type results from prolonged exposure to low ambient temperatures, while the secondary type is much more common and results from the patient's disease, trauma, surgical intervention, or use of drugs that affect thermoregulation.

This is a relatively common condition in veterinary emergencies and may induce cardiovascular, respiratory, neurological, and metabolic effects. Early intensive treatment can decrease morbidity and mortality of emergency and critical patients.

Diagnosis

The diagnosis is based on body temperature measurement with a rectal or oesophageal thermometer.

Physiological and laboratory parameters may vary with the rate of temperature decline. Potential variations are haemoconcentration, leukopaenia, thrombocytopaenia, coagulopathies, hyperkalaemia, hyperglycaemia, glucosuria, lactic acidosis, bradycardia, and arrhythmias.

Treatment

Treatment consists of warming tailored to the patient's particular situation, medical treatment to protect the cardiovascular system, and medical treatment to prevent complications related to both the patient's primary condition and the hypothermia itself.

Management of this type of patient is based on core warming, which includes correction of core temperature and intensive administration of warm fluids (40–45 °C).

Warming techniques

Passive external warming

- This includes the use of blankets to prevent body heat loss and encourage the animal's own thermogenesis mechanisms.
- It is indicated in healthy patients with mild primary or secondary hypothermia.
- It preserves peripheral vasoconstriction and allows for uniform warming.

Active external warming

- This consists of applying heat sources: hot water bags, hot air circulators, incubators, hot air dryers, etc. These heat sources should be placed near the largest parts of the body (thorax or abdomen). The treatment should focus on restoration of the core body temperature and not that of the extremities. In any case, avoid direct contact between radiant heat sources and the skin, since improper use can cause severe burns. The simultaneous use of passive external warming measures can enable prompt return to a normal body temperature.
- This is indicated in patients with moderate-severe hypothermia and in weak patients with mild hypothermia or those who do not respond to passive external warming.
- It decreases peripheral vasoconstriction, allowing blood recirculation and thus favouring progressive warming.

Active internal warming

- This consists of administration of warm fluids (isotonic crystalloids) at 40–45 °C, at a dose of 10–20 ml/kg via an intraperitoneal or intrapleural route (using aseptic technique) or via gastric or colonic lavage.

- It is indicated in patients with severe to critical hypothermia who have suffered cardiopulmonary arrest, as well as those refractory to the aforementioned therapies.
- These techniques minimise the likelihood of refractory hypothermia, but they can be difficult to manage and should therefore be performed only by experienced personnel using advanced monitoring equipment.
- The administration of warm, humidified air or oxygen to intubated patients may help by increasing the temperature by 1–2 °C/h.