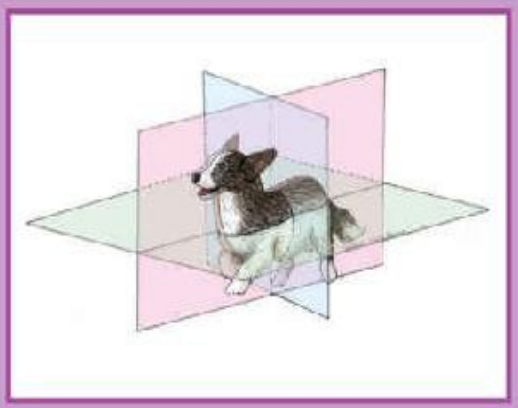
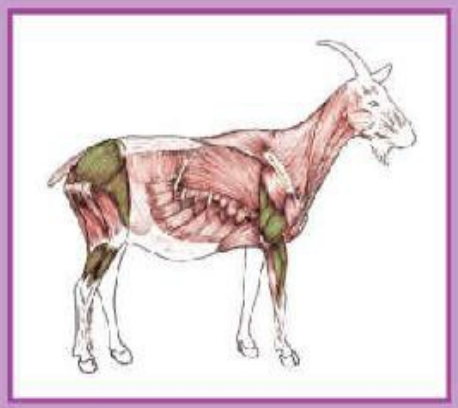
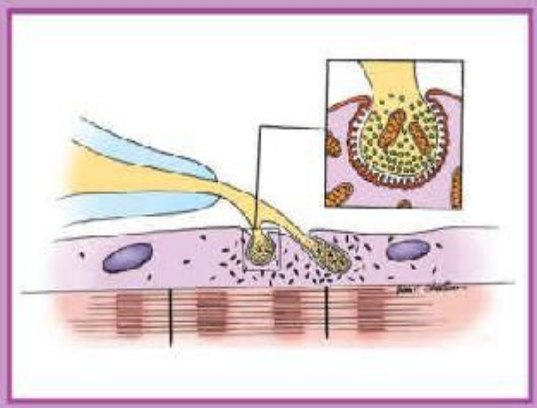
Third Edition

VETERINARY MEDICAL TERMINOLOGY







Dawn E. Christenson

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Veterinary Medical Terminology

THIRD EDITION

Dawn E. Christenson, LVT, MDiv

Veterinary Technician Educator, Retired, College of Veterinary Medicine, Michigan State University, East Lansing, Michigan

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Senior Content Strategist: Brandi Graham
Content Development Specialist: Laura Klein
Publishing Services Manager: Catherine Jackson
Senior Project Manager: Sharon Corell
Design Direction: Ryan Cook

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Preface

This text is designed to be used for self-study, as part of a medical terminology course or to enhance other veterinary-related coursework. It has been created to assist veterinary technology, preveterinary, and veterinary students alike. The text is intended to provide students with a basic foundation in the language of veterinary medicine—veterinary medical terminology. In my experience as an educator, I learned that if students are to be successful, they must immediately apply what they have learned. That is why anatomy, physiology, disease concepts, and clinical case information are included at an introductory level. The scientific information provides immediate application of the terminology presented in each chapter. Clinical case studies provide students with an opportunity to test their knowledge at the end of each chapter. It is my hope that the scientific information brings this new language to life and gives students an insatiable desire to learn even more. If you've learned a foreign language, you know that simply memorizing words does not make you fluent in the language. Context is everything. Likewise, medical terms must be read, spoken, and used in context. Repetition and practice in all aspects of the language makes you fluent. In my experience, facts memorized without reinforcement or application are soon forgotten. And if we forget medical terminology, we do our patients a disservice. So, students, I encourage you to lay a strong foundation for your career, by learning medical terminology well. Veterinary medicine and its language are ever-evolving. A commitment to lifelong learning is essential to the success of every veterinary professional and the patients they serve.

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Learning Resources on Evolve

Students, your professional success is important—so important that we have gone to great lengths to create multiple tools to help you in your quest to become fluent with veterinary medical terminology. Many students find pronouncing medical terms very difficult. I'll admit—some of them can be challenging. No student wants to sound foolish by mispronouncing or misusing medical terms. Well, the cavalry has arrived! We've created an Evolve website to accompany this edition. The website features outstanding student learning resources, including an audio glossary and interactive games, to reinforce the terms and concepts introduced in this text.

Audio Glossary

On the accompanying Evolve site for this text, you'll find more than 4500 veterinary medical terms that are pronounced, spelled out, and defined. You'll be able to see the term spelled correctly, hear the term pronounced accurately, and see correct usage of each term in a sample sentence. This adds a whole new sensory dimension to the learning process to help you achieve competency with both the spoken and written language.

Interactive Games

To further help you immerse yourself in the language (without drowning), we have created interactive games for the Evolve site: Hangman, Part Puzzler, Listen and Spell, and Word Shop. Hangman is a fun way to interact with the terms. Believe it or not, the more fun you have with a subject, the better you'll remember it. Part Puzzler will give you practice dividing terms appropriately. Listen and Spell provides the audio pronunciation for a word. You listen to the word and then type the word as you think it should appear. That is a fabulous way to hone your spelling skills. Finally, Word Shop provides a definition and then permits you to drag and drop word parts in the order necessary to create the correct term that matches the definition. Immediate feedback with each of the

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games helps you rapidly hone your skills.

All of these Evolve features will enhance your experience and hopefully make learning medical terminology fun and easy for you. If nothing else, these features provide a convenient and effective means for you to assess your progress as you learn veterinary medical terminology.

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Flash Cards

For years, I watched students labor over creating "flash cards" of word parts and terms that they could use to study medical terminology anytime and anywhere. That time spent creating the flash cards could have been devoted to actually studying and learning various terms and word parts. To help you manage your study time wisely, we have also created *Saunders Veterinary Terminology Flash Cards*. That's right—printed, illustrated, color-coded, well-organized flash cards have been created with you in mind. These flash cards give you a convenient, effective tool that you can use anywhere to help you master prefixes, suffixes, root words, and abbreviations. Be sure to order a set, to enhance your learning!

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Teaching Recommendations

Instructors and students, it is absolutely essential that Chapter 1 be completed first. All other chapters build on and use the information contained in Chapter 1, especially the directional terminology. Chapters 2 through 12 may be completed in any order, permitting easy integration in any curriculum. Most chapters focus on either an individual body system or closely related systems. And each chapter has a detailed table of contents, permitting both instructors and students to find and cross-reference informational content.

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Open Letter to Students

Dear Student,

I remember, when I was a student, how I struggled with learning medical terminology. It truly was like a foreign language to me. And with the intensity and complexity of my professional education (that was quite overwhelming at times), this "foreign language" complicated matters even more for me. It inhibited my learning. I've been confused, frustrated, overwhelmed, and put to sleep by medical terminology. As a student, I found lists of words, for the sake of lists, completely useless. Memorizing was hard, if not impossible, for me. (It's just not a good learning strategy for me, even today.) If I couldn't fully understand and apply to patients what I was learning, I couldn't remember it. That's why I have more than just medical terms in this book. The added information is intended to help you immediately apply terms to relevant information that will be important to you as a veterinary professional. The more connections you make, the better your understanding and retention of the information. That's certainly been my experience, as it has for many of my colleagues and students.

Through my experiences as a student, practicing credentialed veterinary technician, and teacher, I have found that medical terminology does not have to be an ominous "monster" or a "millstone." Learning medical terminology can actually be a fun adventure! All you need is a little curiosity. I still have that curiosity. Even after 40 years as a veterinary professional, I have an insatiable curiosity for learning new terms. Whenever I run across an unfamiliar term, I look it up. Call me a geek, but I love learning the origins of new terms. And I am surprised at times how some of those dusty, crusty old Greek and Latin roots can be downright amusing. That makes learning them both fun and memorable.

I know firsthand that medical terminology initially feels awkward and unwieldy. What language doesn't? If you're like me, you'll probably wonder how you'll ever remember all of the terms, what they mean, and (OMG) how to spell them. But you will. Once

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you start using them on a regular basis, they'll become second nature. You'll begin to take them for granted. Hopefully, along the way, you'll have some fun. Who knows, you might even feel adventurous and make up some bogus terms, so that you and your friends can talk "code." I've done that. Just for fun, I've created terms like stomatomegaly [*stoma* a mouth + *-megaly* enlarged, big; i.e., a big mouth], ornithencephaly [*ornith(o)-* bird + *encephal(o)-* brain + *-y* condition of; i.e., condition of being a bird brain], aerocephalon [*aer(o)-* air + *cephal(o)-* head + *-on* an; i.e., an air head], and condylocephalus [*condyl(o)-* knuckle + *cephal(o)-* head + *-us* a; i.e., a knucklehead]. It's loads of fun! You should try it!

To be serious for a moment, veterinary medicine *is* serious business. Life is serious business. Still, you need to enjoy the journey. Otherwise, what's the point? Your success and the successful outcomes of the patients you eventually serve are important to me. That's why I've done my very best to set you up for success with the design and content of this text. I want you to read it (yes, actually read it!) and use it. So, unlike most scientific resources, I've written it in a very conversational style. I've tried to make it fun where possible. And I've included information from actual clinical cases to make it relevant. How well you learn this information will impact your patients. So, I ask you to do your very best.

Someday, you'll be in casual conversation with family or (nonveterinary) friends and someone will stop you asking: "What did you say? Why do you always have to use such big words?" That's cool. And you'll feel like you've really arrived when you fully engage in conversation with seasoned veterinary professionals and you understand and use all of the medical lingo. That is SO gratifying. You'll get there. Trust me. Just give it time. It'll probably sneak up on you and at some point you'll simply recognize, "I'm there! I made it!" Until then, be persistent and consistent. Remember, your patients and their humans will depend on you. So, I want you to take your education seriously. I also hope that this text and its resources will make the learning process a little easier for you. And I hope to make the learning enjoyable, full of surprises, and maybe even make you laugh a little. In the end, if I

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can contribute in a small way to your successes educationally and professionally, I will have done my job.

Warmest regards,

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Acknowledgments

First, I would like to express my deepest gratitude to Bea for her thorough and critical review of this text. Bea, your "eagle-eye," while frustrating at times, helped me make this text the best it can be. Thank you. And at a personal level, thank you for walking with me on life's journey. You are truly the greatest blessing to me.

To all of my colleagues and graduates, thank you for years of encouragement and contributions to my personal and professional growth. Each of you helped hone me as an educator.

To all of the reviewers of the previous edition, thank you for your critiques and suggestions. Your comments were very valuable in guiding both the content and organization of this edition.

To my editorial and production teams, thank you for all of your flexibility, hard work, patience, and understanding. You're the best!

Finally, to Ellie, the best Cardigan Welsh Corgi ever: Thank you for sharing more than 16 years of life with me and providing 11 years as the best teaching assistant anyone could hope for. Rest in peace, little Miss Diddle-butt, until we meet again.

Consultant

Beatrix VanKampen, BS, LVT, Veterinary Sonographer, Retired
Veterinary Medical Center, Michigan State University East Lansing, Michigan

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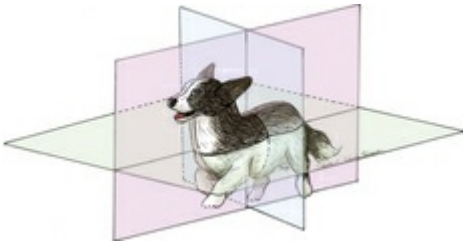

Fundamentals and Applied Directional Terminology

Basics of Word Structure,
- Root Word,
- Prefix,
- Suffix,
- Combining Vowel,
- Compound Word,
- General Rules,

Applied Directional and Imaging Terminology,
- Body Planes,
- Directional Terms Related to Body Surfaces,
- Applied Radiographic Terms,
- Applied Sonographic Terms,
- Case Study,
- Case Study Questions,

GOALS AND OBJECTIVES

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By the conclusion of this chapter, the student will be able to:

1. Recognize common root words, prefixes, and suffixes.
2. Understand the function of the root words, prefixes, suffixes, combining vowels, and combining forms.
3. Divide simple and compound medical terms into their respective parts.
4. Recognize, correctly pronounce, and appropriately use common directional terms.
5. Recognize the planes of the body.
6. Demonstrate a basic understanding of directional terminology, as it relates to the body and body planes.
7. Demonstrate a basic understanding of medical and directional terms, as they relate to veterinary diagnostic imaging.

"IMPORTANT NOTE: Information from this chapter will be applied in all subsequent chapters."

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Basics of Word Structure

Before we embark on the contents of this chapter, allow me to explain a few things. Refer to the Chapter 1 introductory table in Appendix C, for a moment. It probably looks daunting to you, but don't panic. I don't expect you to memorize or to fully understand all of the terminology listed in that table right now. My job is to guide your learning. I hope to do that in a logical manner, using numerous examples and repetition. I will present each of the terms listed in the introductory table in context to aid your understanding. Let's start slowly using a few directional terms to explain the basic structure of medical terms in general. Yes, some of the terms listed are big and foreign. Yet they are just words … some are really big words. Each one can be broken down into smaller digestible parts. Recognition of the parts makes defining, understanding, and using the terms much easier. Words, including medical terms, can be likened to trains. They are each built of important parts. Without each of the necessary parts, the train can't deliver precious cargo and words can't deliver appropriate meanings.

Do not, I repeat—do not—skip over the following information. What follows is essential to your understanding of medical terminology. You must grasp the function of each type of word part (i.e., root word, prefix, suffix, and combining vowel) before we can discuss the directional and imaging terminology presented in this chapter.

Root Word

The ***root word*** is the foundation of any word. It gives substance and meaning to a word, much like each boxcar and tank car in a train give it mass and value. Like a train's cars, root words come in a variety of sizes, "shapes," and "colors," each holding a special cargo (i.e., meaning) within.

It is from the ***root word*** that most of the meaning of a given word is derived. In general, words may contain one or multiple root words. For example, the word *football* contains two root words (root

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1 = foot, root 2 = ball). Similarly, medical terms may contain one, two, or many root words. But be not ye anxious! Don't be alarmed by the length and complexity of some medical terms. Once you begin to recognize various root words (just like brand names and logos on a train's cars), you will begin to understand and "unpack" the overall meanings of medical terms.

There are many examples of root words throughout this chapter and book. In Appendix C, the root words and their corresponding meaning are shown in bold. Let's play with a couple of those root word examples. In the directional term ***cranial*** [*crani(o)-* head + *-al* pertaining to], the root word is *crani(o)-* meaning "head." It refers to the body part that we call the head. As you begin to learn the directional terms in this chapter, you will find that many of them reference a particular aspect of the body or body part. Even if you know very little about anatomy yet, you'll understand many directional terms because of their commonly referenced root words. For instance, everyone knows a head [*crani(o)-*] from a tail [*caud(o)-*]. So, it is easy to figure out, directionally, where to look for *cranial* versus ***caudal*** [*caud(o)-* tail + *-al* referring to] injuries on an animal or on a radiograph (or x-ray).

Prefix

The ***prefix*** can be likened to the train's engine with its whistle. The engine goes before the boxcars, and its whistle announces the approaching train. Likewise, a prefix goes before a root word and announces that a wealth of meaning is coming. (Note: in Appendix C, prefixes and their corresponding meaning are shown in italics.)

Do prefixes have much meaning? Yes, they do, but they cannot stand alone. Once again, let's think about this in terms of a train. Is an engine *the* train? No. It's only part of the train. True, the engine does have power (power of the *prefix:* active, altering meaning). What purpose would it serve to have an engine merrily chugging along the tracks all by itself? If, however, the engine pulls numerous boxcars and tank cars with their precious cargo, it serves a tremendous purpose. The same can be said of prefixes. Once a prefix is connected to one or more root words, it has purpose. And that purpose is to actively alter or modify the overall meaning of

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the root words that follow.

Let's look at a prefix found later in this chapter. In the medical term *contralateral* [*contra-* + *later(o)-* side + *-al* pertaining to], *contra-* is the prefix. It means "opposite." What is it opposite to? We can't know until we look at the rest of the term. The root word *later(o)-*, which follows the prefix in this example, means "side." So, the whole term doesn't refer to just the side or any side. Collectively, the whole term refers to the opposite side. Do you see how the prefix actively alters the meaning?

One final note: notice that the prefix *contra-*, when written alone, is immediately followed by a hyphen. This is standard practice whenever prefixes stand alone. By the way, did you notice that even the word *prefix* has a prefix within it? *Pre-* is the prefix of the word, meaning "before" or "in front of." That should help you remember where prefixes are found in medical terms: they go *before* a root word, in the "front" of a medical term.

Suffix

Let's return to our train analogy. In earlier days of the railroad industry, the very last car on a train was the caboose. It was almost always painted red, with blinking red lights aft. The function of the caboose was to provide storage and living space for railroad employees. Sadly, today's trains no longer have cabooses. The caboose has been replaced by a blinking red light attached to the rear of the very last car of the train. What do suffixes have in common with cabooses? The ***suffix*** is always *last*. Yet that doesn't mean it's unimportant. (Note in Appendix C that suffixes and their corresponding meanings are shown in italics.)

Like a prefix, a *suffix* is not a word in its own right, just as a caboose is not the whole train. So suffixes cannot stand alone. If you do see one written alone in this book or a medical dictionary, you'll recognize it as a suffix by the hyphen that precedes it (*-al*, for example). And even though suffixes are last, they do contribute important meaning to the root words and other word parts that precede them. In fact, suffixes are so important that the meaning of a medical term is interpreted **first** with the meaning of the *suffix*. Suffixes not only modify the word but they often indicate how the

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word should be used grammatically.

Okay, before you panic about grammar, let me explain. Let's look again at the directional term *cranial* [*crani(o)-* head + *-al* referring to]. The suffix (*-al*) makes this word an adjective. It must be used to describe something else, like the word *red* would be used to describe the caboose. So, to use this directional term correctly in context, we might say: The brain is located in the *cranial* vault. *Cranial* tells us specifically which vault or the type of vault we are referring to. Can we take the same root word in this example and add a *suffix* that would create a noun (person, place, or thing)? Yes. Yes we can. Let's use the suffix (*-um),* to indicate a "thing." The word ***cranium*** [*crani(o)-* head + *-um a,* the] would be defined as *the head*. To use both terms in context, we might say: The *cranium* [*crani(o)-* + *-um*] houses the brain in the *cranial* vault. Do you see the difference? The *cranium* is the head (a thing) and the *cranial vault* is a chamber of the head. One is a thing (*cranium*) and the other is an adjective, or a word used to describe (*cranial*) an attribute of that thing.

Combining Vowel

Going back to our train analogy, all the boxcars are joined together by couplers. In medical terminology, we join root words together with combining vowels. The letter "o" tends to be the default vowel of choice. However, any vowel can serve in this capacity. Depending on the subsequent word part to be joined to a root word, a ***combining vowel*** may or may not be needed. When a root word is joined to another root word that begins with a consonant, a combining vowel will always be used. And a combining vowel may be used even if the second root word begins with a vowel. When a root word is connected to a suffix that begins with a vowel, a combining vowel will not be used.

By the way, did you notice in previous examples of root words that they are shown with an "o" in parentheses [e.g., *crani(o)-*]? That is what is known as the *combining form* of the root word, with a recommended or default *combining vowel*. Please note: a ***combining form*** is not a word unto itself. It must be connected to another word part to create a whole word.

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Compound Word

Any word constructed of two or more root words is called a ***compound word***. As an example, let's combine two root words that you are already familiar with: *crani(o)-* and *caud(o)-*. The new *compound word*, ***craniocaudal*** [*crani(o)-* head + *caud(o)-* tail + *-al* pertaining to] is a directional term that would indicate head to tail. Clear as mud, right? Okay, picture this: imagine a dog wearing a miner's headlamp and looking over her shoulder and back so that the light is shining on her own tail. Got it? Well, we could say that the beam of light is pointed in a *craniocaudal* (head to tail) direction. We'll clarify this more later in the chapter when we apply it to diagnostic imaging.

General Rules

First, whenever you try to read the basic meaning of a new medical term, begin with the *suffix*. If you look at Medical Terms Introduced in Chapter 1 in Appendix C, you'll notice that the suffix for each word is shown last in the "division" column. However, the meaning of each suffix is written first, in the "basic definition" column. As you look at the table, notice how many of those suffixes mean the same thing. We are going to accumulate a bunch of suffixes that mean "pertaining/referring to" as we make our way through the book. Redundancies like this should be helpful to you.

Second, most of the time, when a *root word* is combined with a *suffix* that begins with a vowel, the *combining vowel* will be dropped. However, most of the time when two root words are connected (even if the second root begins with a vowel), a combining vowel *will* be used to connect the roots. Notice that in both of these scenarios I said "most of the time." Unfortunately there are always (thank goodness they're rare) exceptions to the rules. Sorry. I'm just the messenger.

Finally, try to have fun. A strong sense of humor, deep curiosity, and a medical dictionary will be your greatest allies throughout your educational and professional life. Life is short; so enjoy the journey. That is why I suggest having some fun by creating your own medical terms. "How?" you ask. That's simple. Transform everyday words, phrases, and experiences into medical lingo. Let

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me give you just a few silly examples.

We have all probably jokingly been called silly names by close friends or family. Think about common terms of endearment that we may use among those closest to us. For example, my brother, whom I love dearly, called me a knucklehead when we were growing up. Had he known medical terminology, he might have told me that I was *chondylocephalic* [*chondyl(o)-* knuckle + *cephal(o)-* head + *-ic* pertaining to]. There are worse things than being a knucklehead. Sometimes we poke fun at ourselves. I freely admit that I have, from time to time, suffered from *aerocephaly* [*aer(o)-* air + *cephal(o)-* head + *-y* condition of]. Yes, you read that correctly. I have sometimes been an airhead. Have you ever known someone afflicted by *stomatomegaly* [*stomat(o)-* mouth + *-megaly* a large condition; enlargement of]? I have. In fact, I've probably been accused of having a big mouth myself. I can be rather outspoken at times. Finally, I have also known some *ornithencephalic* [*ornith(o)-* bird + *encephal(o)-* brain + *-ic* referring to] folks. What about you? By the way, calling someone a birdbrain could be understood as a compliment. Some birds are extremely intelligent! Even the common crow has tremendous problem-solving ability.

You will never find terms like those I've just mentioned in a medical dictionary. However, you and your classmates could have some fun creating unique words. You may even create a secret "code" language just for you. Of course, some folks may scoff at such a silly idea. However, word games like this can serve to reinforce the medical terminology you're learning. And your laughter over the silly terms you create will probably help the information "stick" a bit better. If it does that, the game will be well worth it.

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Applied Directional and Imaging Terminology

Finally, we can begin to learn and apply some very useful medical terms. I will be spending a great deal of time and effort on directional terminology. This is one subset of medical terms that is vital for every veterinary professional to know and know well.

Body Planes

Directional terms help us to orient ourselves to our patients, their bodies, and locations of disease or injury affecting their bodies. Unfortunately the body doesn't come with a built-in GPS (Global Positioning System). We need directional terms to help us "map" locations on and views of our patients' bodies. As I said earlier, many of the directional terms that we use to help us in "mapping," or referencing particular body parts and attributes. But before we can consider such body part–specific directional terms, we need to consider the orientation of the body as a whole. That's where invisible body planes come into play.

Our patients are not two-dimensional creatures, like cartoons found in comic strips. Our patients' bodies are three-dimensional (3D). So, we orient the body in relation to three principal (3D) body planes. Look at the 3D plywood structure in Fig. 1.1. The three primary body planes are configured in a similar manner, with the intersection of each being perpendicular to the others. Notice too how the plywood boards all meet at a central point. Ah, but we could tip and turn the plywood structure in many different ways, perhaps eventually losing sight of the structure's original position. All of the boards look the same. If we superimpose such a structure on the body, we can begin to tell each board (plane) from the others.

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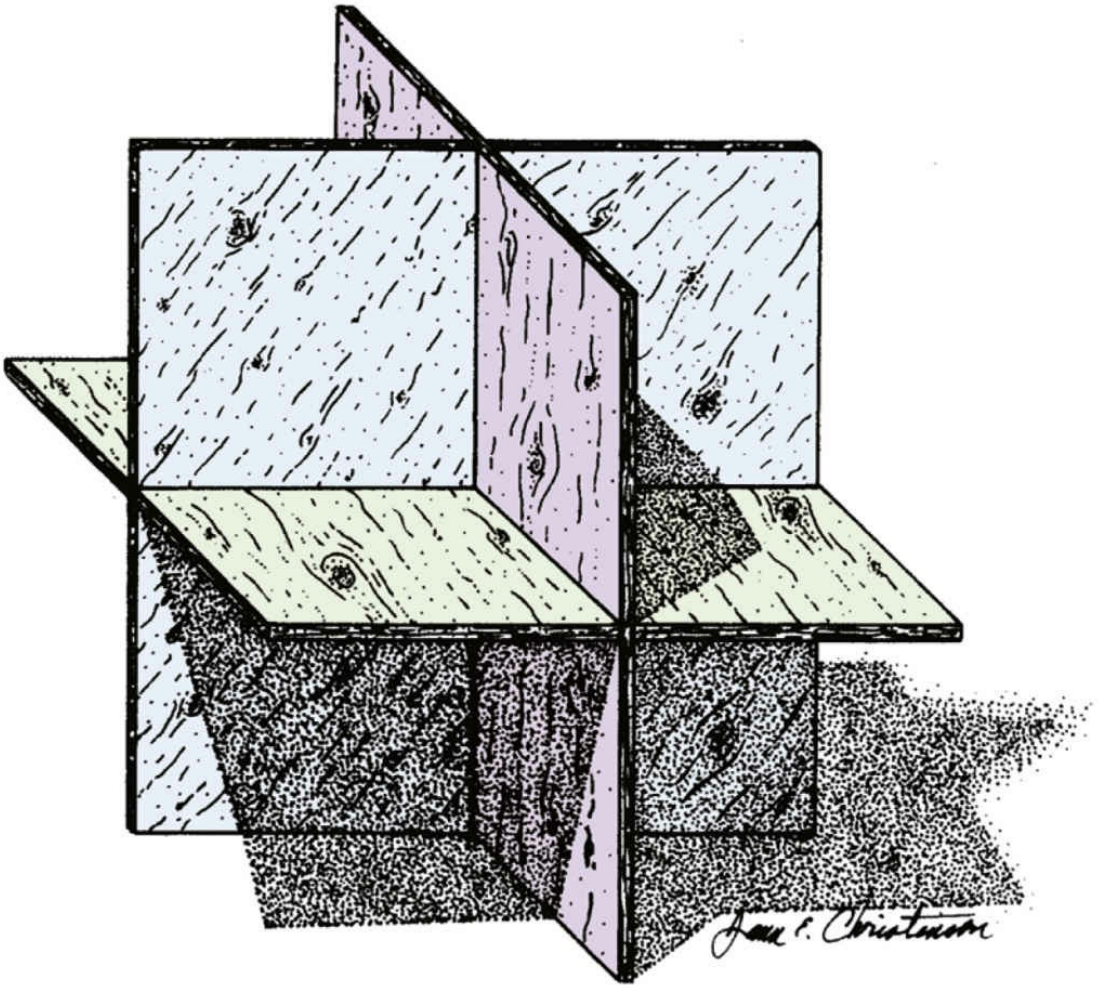

FIG. 1.1 A plywood structure simulating the body planes.

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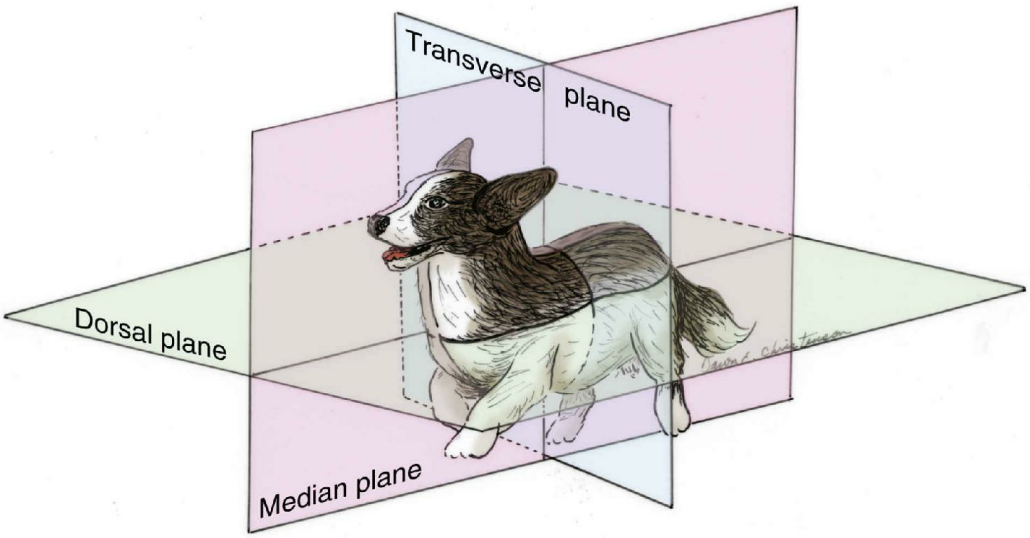

FIG. 1.2 The body planes.

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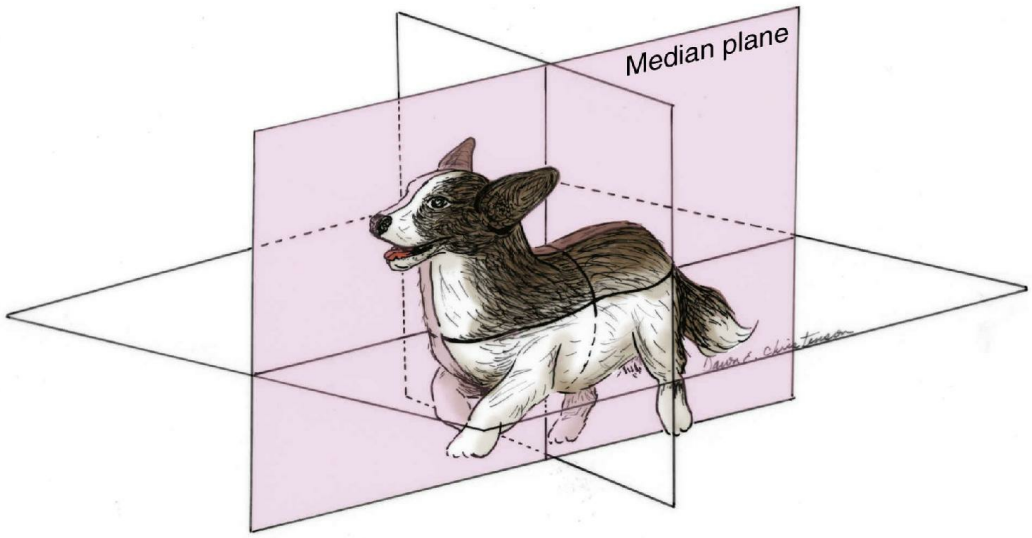

FIG. 1.3 Median plane.

In Fig. 1.2, we have replaced the plywood with plexiglass and superimposed the transparent structure on this Cardigan Welsh Corgi's body. Now that we have a body, we can begin to distinguish one sheet of plexiglass from the others. And if we name each sheet, as shown, we can begin to map the relationship of points on the body to the plexiglass sheets or *body planes*.

The ***median*** [*medi(o)-* middle + *-an* referring to] ***plane*** is the vertical plane that divides the body into equal right and left halves. It is shown in pink in Figs. 1.2 and 1.3. Yet its location is probably easier to visualize in the ***dorsal*** [*dors(o)-* the back + *-al* referring to] view of the horse (Fig. 1.4). The median plane is sometimes also called the ***midsagittal*** [*mid-* middle + *sagitt(o)-* straight arrow + *-al* referring to] ***plane.*** Why? It is distinguished from other ***sagittal*** [*sagitt(o)-* straight arrow + *-al* pertaining to] planes because the midsagittal plane is found exactly in the middle of the body. Other *sagittal* [*sagitt(o)-* straight arrow + *-al* referring to] planes lie parallel, to the right or left, of the midsagittal or median plane. An example of a sagittal plane is shown in Fig. 1.5.

Sagittal planes, whether to the right or to the left of midline, are all "straight like an arrow," running through the entire length of the body. (That's how they came up with the name, sagittal.) In fact, if you look closely at the horse in Fig. 1.5, you'll notice that this particular sagittal plane is shown actually subdividing each of the

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right limbs. How is this useful? By subdividing each limb, as shown, we can make relational reference, in part, to the median plane. This is how: the "inner" surface of the right limb, nearest the median plane, is directionally referred to as the ***medial*** [*medi(o)-* middle + *-al* pertaining to] surface. The side of the right limb, away from the median plane, is directionally referred to as the ***lateral*** [*later(o)-* side + *-al* pertaining to] surface.

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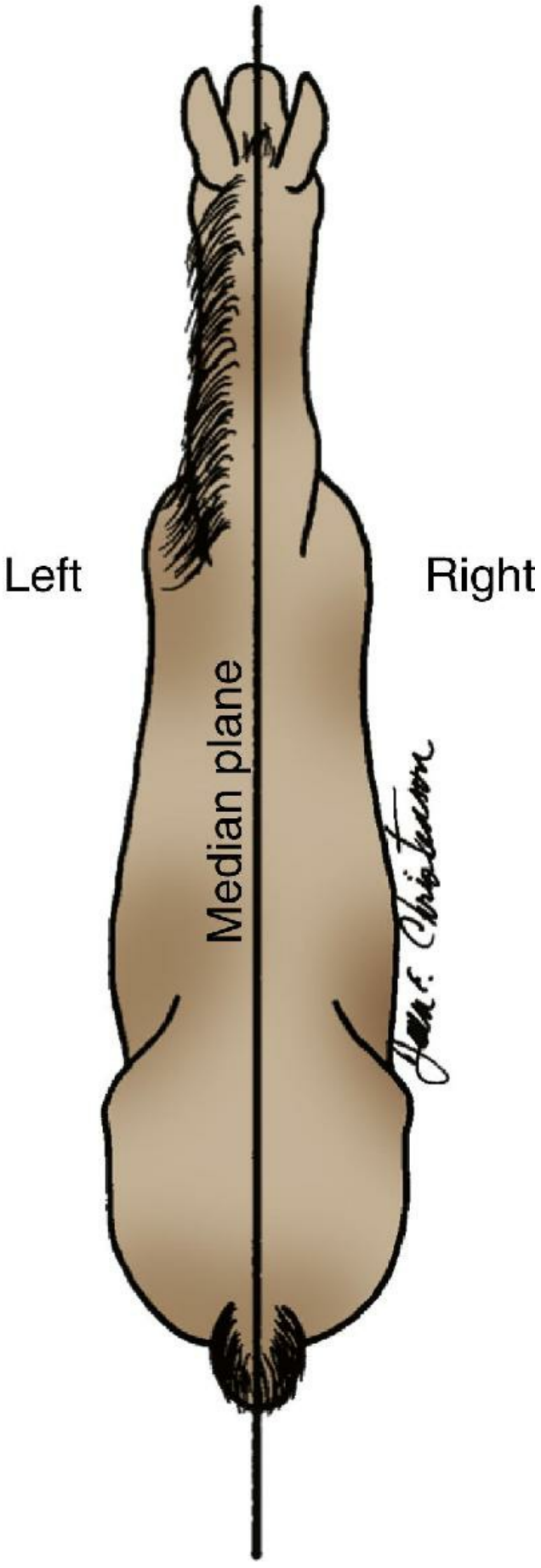

FIG. 1.4 Dorsal view of the median plane.

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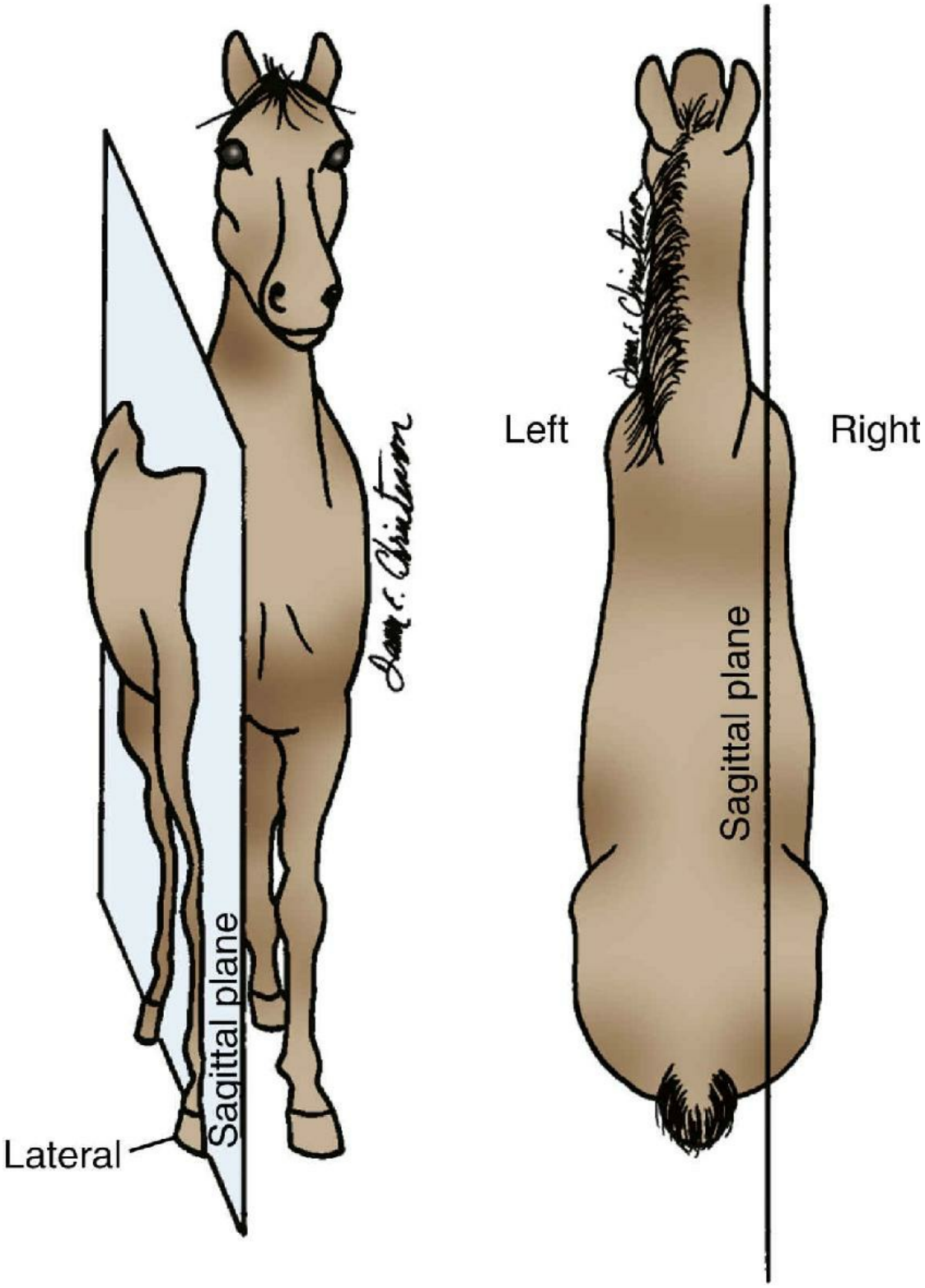

FIG. 1.5 Sagittal plane.

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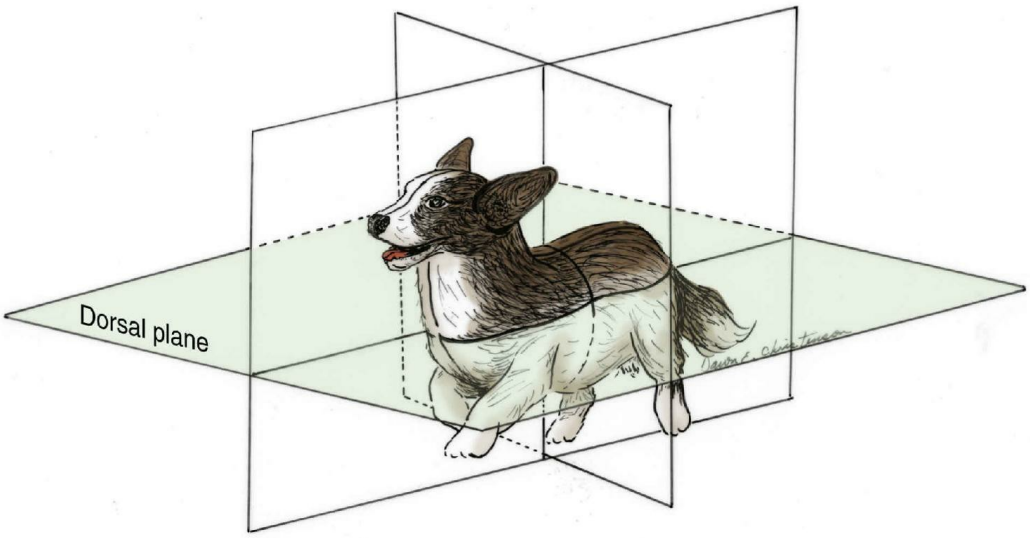

FIG. 1.6 Dorsal plane.

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FIG. 1.7 A dog in dorsal recumbency.

Let's look at another body plane. This time let's focus on the ***dorsal*** [*dors(o)- the back* + *-al* pertaining to] ***plane***. In Figs. 1.2 and 1.6 the dorsal plane is shown in green. It divides our Corgi's body into *dorsal* and ***ventral*** [*ventr(o)-* belly + *-al* referring to] portions. Why must we use the term *dorsal* and *ventral*? Why can't we simply refer to upper and lower portions? Good question. Consider this: what if our Corgi rolls over on her back, as in Fig. 1.7? After all, that position seems to be the preferred sleeping position for the breed. Now what was up is down and vice versa. So *up* and *down* are poor directional references. It's much more precise to reference the physical attributes of the Corgi's back and her belly. Now we can say, in directional terms, that she is lying in ***dorsal recumbency***. Without even seeing this dog, anyone hearing that would know that her ***ventrum*** [*ventr(o)-* belly + *-um* the] is facing up for all to see. Tuck this positioning information into a safe place in your mind. It will come in very handy when we discuss ***radiographic*** [*radi(o)-* rays, radiation + *graph(o)-* to record + *-ic* pertaining to] positioning

and views a little later in this chapter.

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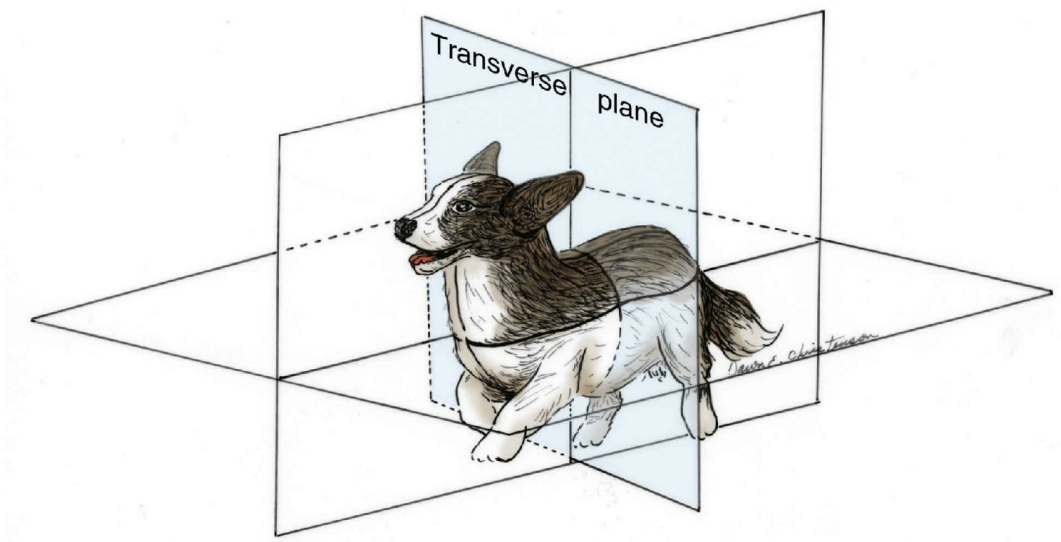

FIG. 1.8 Transverse plane.

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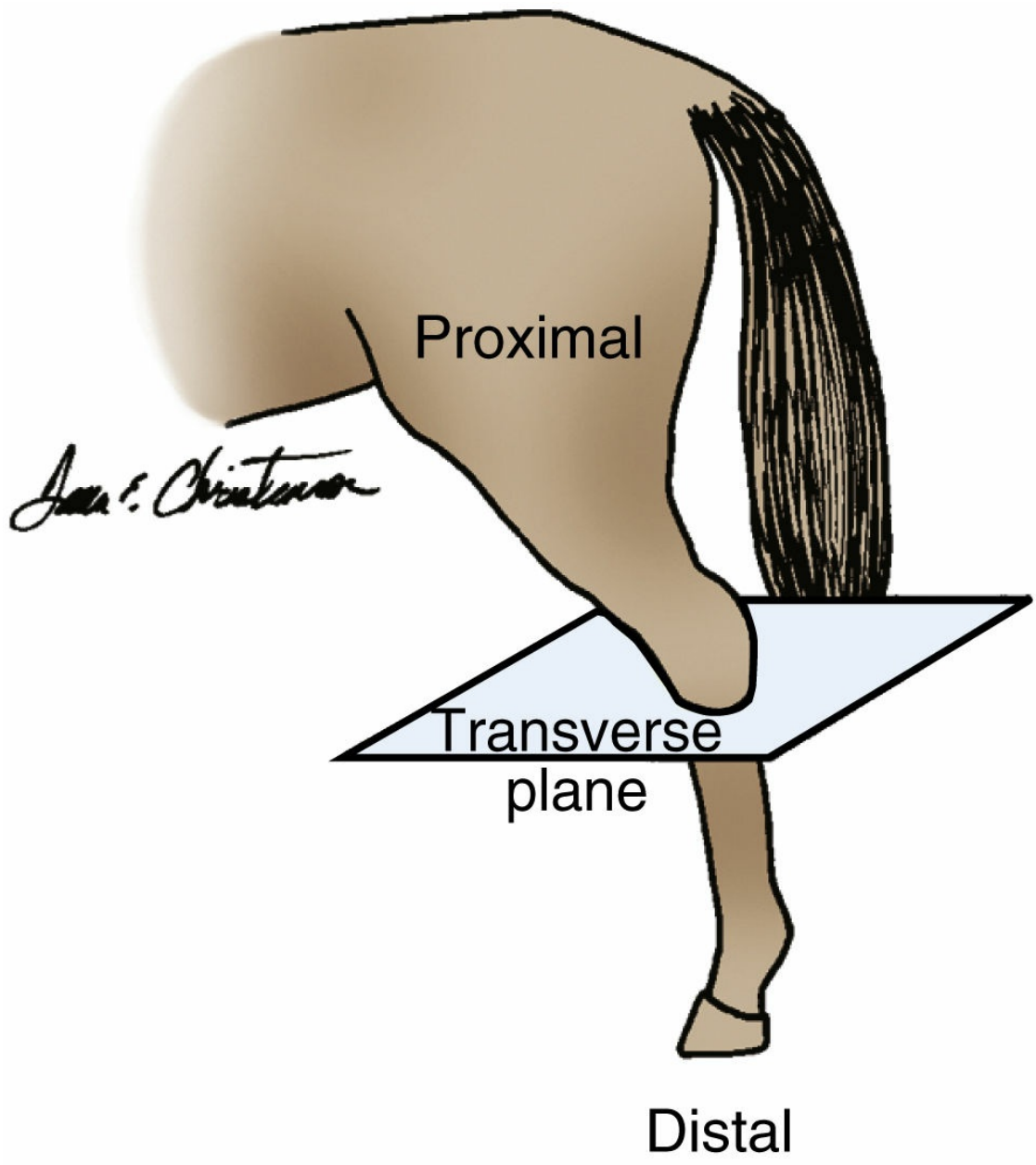

FIG. 1.9 Transverse plane of a limb.

The last body plane we need to discuss is the ***transverse plane*** [L. *transversus* crosswise; *trans-* across]. In Figs. 1.2 and 1.8, the transverse plane is shown in blue. In these figures, we find that the body is actually divided into ***cranial*** [*crani(o)-* head + *-al* referring to] and ***caudal*** [*caud(o)-* tail + *-al* referring to] portions. Since most veterinary patients have heads and tails, these directional references are very valuable, even for animals such as primates who may walk in an upright position. Of course directional terms such as *cranial* and *caudal* are best applied to the neck and trunk of the body. This is because the transverse plane cuts across the axis of the

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body, and that axis is represented by the spinal column. Ah, but we have other axes (ak'sēz) on the body besides the spinal column.

Each appendage that extends from the trunk itself, like the legs and the tail, is considered to have its own axis. Because of this, an alternative transverse plane can ***transect*** [*trans-* across + *-sect* cut] each appendage, as shown in Fig. 1.9. Now we cannot use the directional terms of *cranial* and *caudal* to distinguish one end of this horse's limb from the other. Likewise, if we were to transect the tail with a transverse plane, we couldn't use the term *caudal* at all. We're already at the tail. So, just how does a transverse plane subdivide a limb or a tail? It divides each of them into ***proximal*** [*proxim(o)-* near + *-al* referring to] and ***distal*** [*dist(o)-* distant + *-al* referring to] portions (i.e., the portion nearest the body and the portion farthest from the body). Let me clarify with a couple of examples to help you understand the use of terms like *proximal* and *distal*. For instance, we could say that the horse's foot is the most *distal* part of its limb. We could also say that its hip is the most *proximal* joint of its rear leg. We will work more with these terms more, later on.

Finally, when we are looking at a feature of the head, we cannot use the term *cranial*. We're already at the *cranium*. We can actually *transect* the head with an alternate *transverse plane* to divide it into front and rear portions. And to describe those portions we will reference specific body parts to avoid confusion. What parts are always available? The nose and the tail are quite permanent and prominent features. So, if we want to reference features to the rear of the head, we will talk about *caudal* aspects of it. Conversely, if we want to discuss features of the "front" of the head, we will talk about ***rostral*** [*rostr(o)-* nose, beak + *-al* referring to] aspects. Again, you don't need to know very many details about anatomy, to use and understand terms like *rostral* and *caudal*. Everyone knows the locations of noses versus tails.

Directional Terms Related to Body Surfaces

Now that we are familiar with the body planes and some of the basic directional terms used in relation to those planes, we can begin to build on that knowledge. Each major body part (e.g., the

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head, neck, chest, abdomen, leg, and tail) has an exterior surface. To help our visualization of those surfaces, let's imagine a Trojan horse, like the one used in Greek legend. It was built of wood. Having done a bit of woodworking myself, I know that the easiest and fastest method of construction would involve using planks and posts. Thus each part would be rather square, as shown in Fig. 1.10. Yes, this is a very rudimentary-looking creature. When you've finished laughing at the image, we'll move on. Ready? There is a method to my madness. This very square beast provides clear delineations of each surface. It is very important that you have a clear understanding of those surfaces. Without a sound, functional understanding of the body surfaces and their related terms, you will have great difficulty understanding directional terms used in ***radiography*** [*radi(o)-* rays, radiation + *-graphy* process of recording] and other areas of veterinary medicine.

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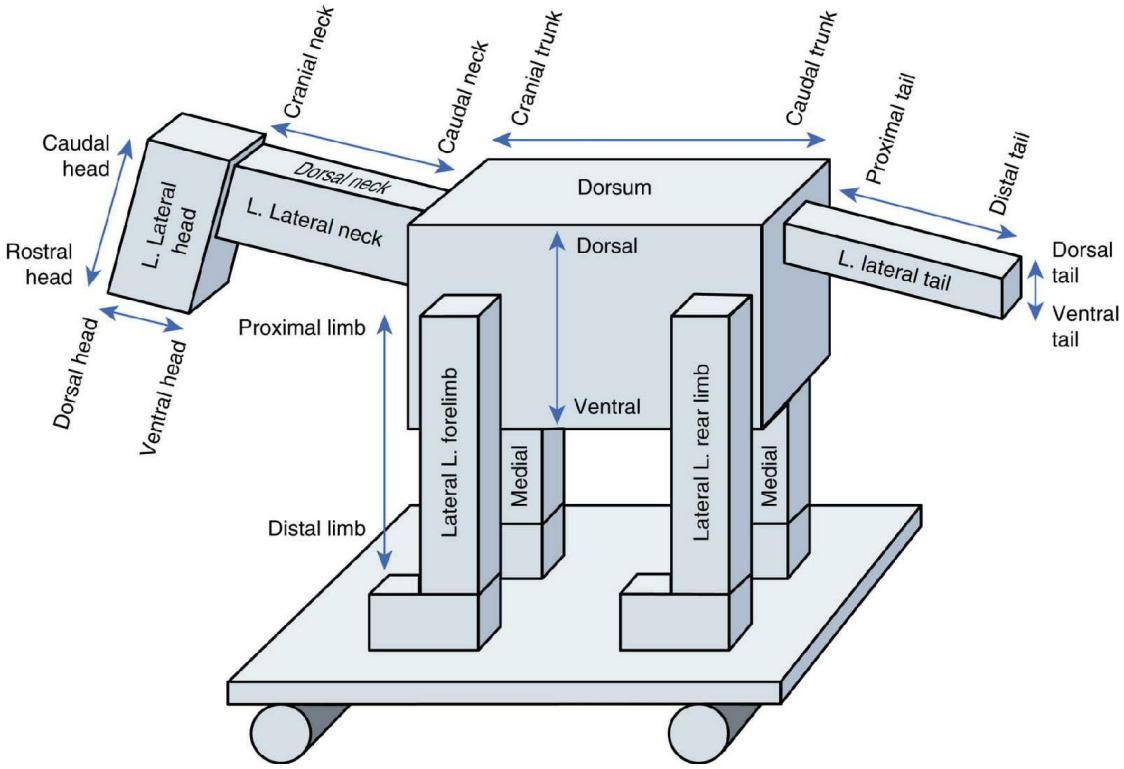

FIG. 1.10 Trojan horse schematic with directional terms related to body surfaces.

As you can see in Fig. 1.10, the limbs and the tail have *proximal* (near) and *distal* (far) points. On the head, neck, trunk, and tail, we can reference the *dorsal* and *ventral* surfaces. Again, we cannot use *up* and *down* or *top* and *bottom* because our patients may be positioned on their sides or their backs. So, we need to reference the surfaces consistently regardless of the position of the body. You will also notice, from the *left lateral view* of our Trojan horse, that we can label the side facing us as *left lateral*. The *contralateral* [*contra-* opposite + *later(o)-* side + *-al* referring to] aspect of this wooden beast would be labeled *right lateral*. Aha, but notice the aspect of the limbs that lie nearest the *median plane*. Those surfaces are labeled *medial*. It is important to note that *medial surface* can be used only in reference to the limbs because they are positioned in *sagittal planes*.

What about the ***anterior*** [*anter(o)-* the front + *i* + *-or referring to*] and ***posterior*** [*poster(o)-* the rear + *i* + *-or* referring to] surfaces of the limbs? First of all, the directional terms *anterior* and *posterior* are generally reserved for use with humans and perhaps primates. Second, the limb structure of any ***quadruped*** [*quadri-* four + *ped(o)-* foot] is so dramatically different from that of humans that we subdivide the proximal and distal portions a bit differently. In so

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doing, we directionally reference the part of the foot that contacts the ground (i.e., the sole or pads). That reference is different for *front* versus *rear feet*. As shown on the *distal* left front limb of a dog (Fig. 1.11), we see the ***palmar*** [*palm(o)-* palm + *-ar* pertaining to] surface. If it helps, think of the palm of your hand to remember that a *palmar surface* is found only in the front limbs of quadrupeds. Notice, however, that the *palmar* surface (i.e., everything shown in Fig. 1.11) of the limb includes more than merely the pads of the paw. It actually extends from the paw pads *proximally* to include the ***carpus*** [Gr. *karpos* wrist].

The same is true for the distal left the front limb of the horse shown in Fig. 1.12. The *palmar* surface includes the sole of the horse's hoof and extends proximally to include the carpus.

In the rear limb, the surface in contact with the ground is the ***plantar*** [*plant(o)-* sole + *-ar* pertaining to] surface (Fig. 1.13A and B). To help you remember, simply think about where you get *plantar* warts—on the soles of your feet. Correlate that with our *quadrupeds*. This means that the plantar surface is found only in the rear limbs. Like the palmar surface in the front limbs, the plantar surface of the rear limbs includes more than the pads and soles of the feet. It extends proximally to include the ***tarsus*** [Gr. *tarsos* broad flat surface]. The *tarsus* is commonly called the *hock* in domestic animals.

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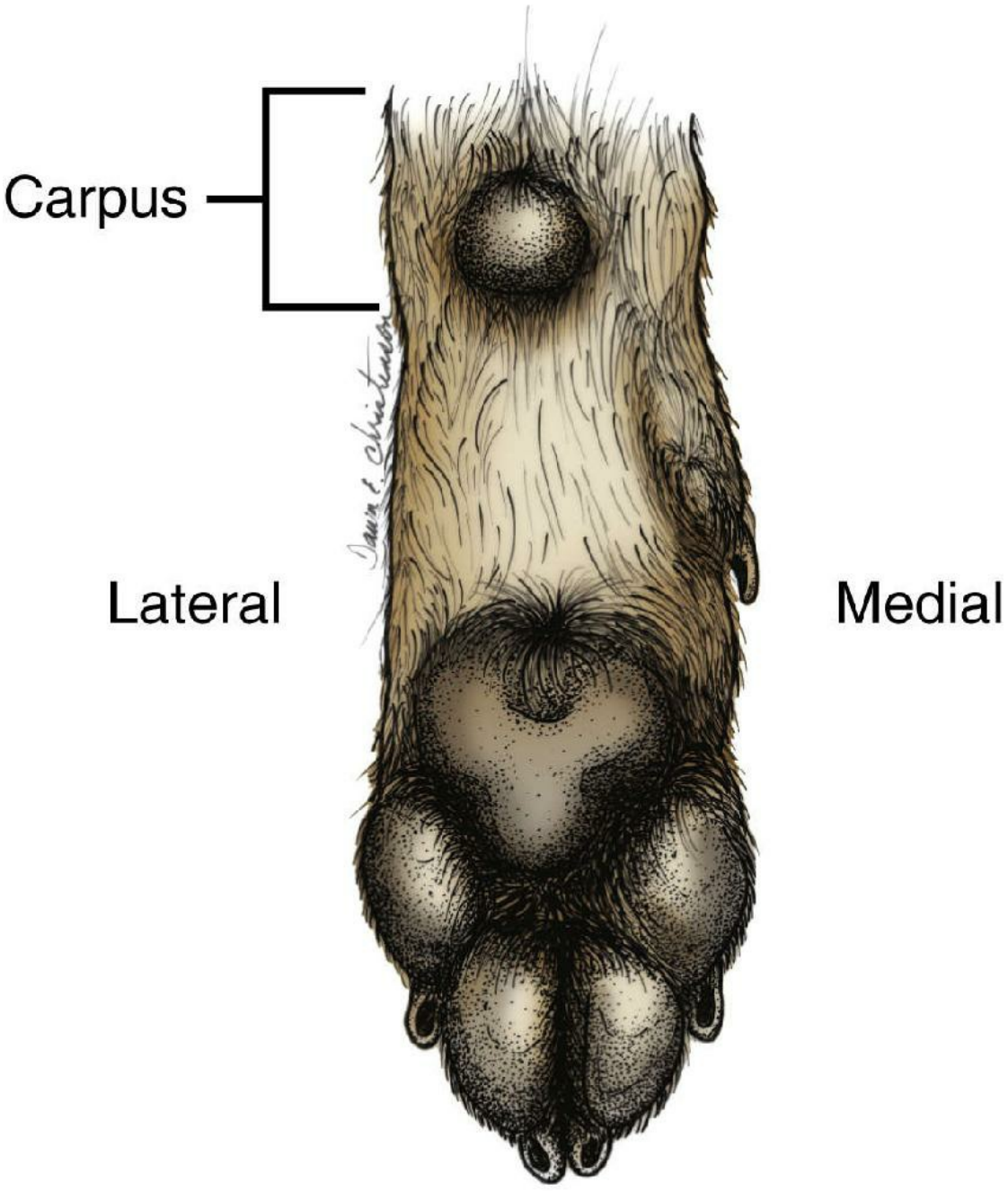

FIG. 1.11 Palmar surface, left front paw of a dog.

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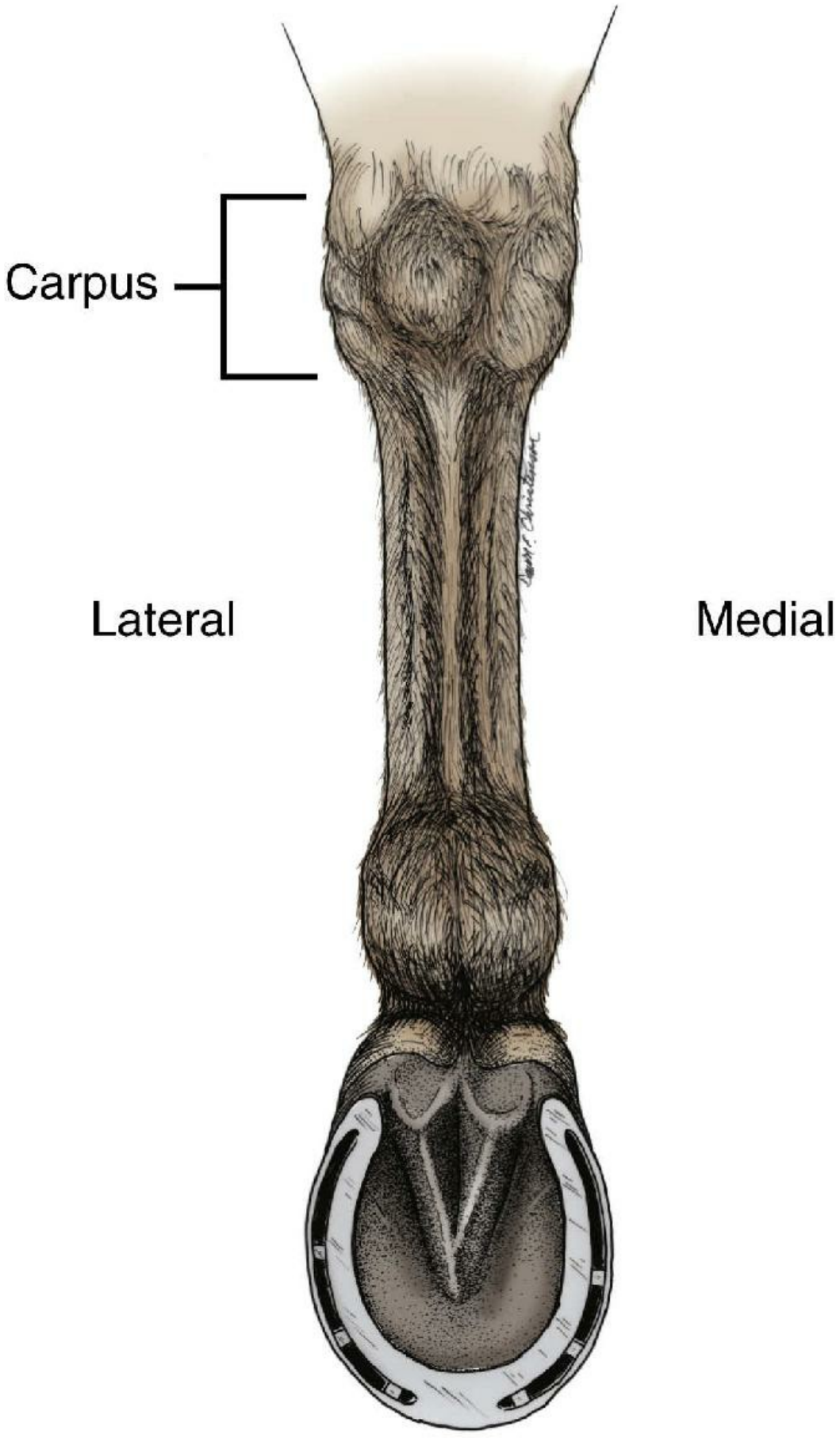

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FIG. 1.12 Palmar surface, left front limb of a horse.

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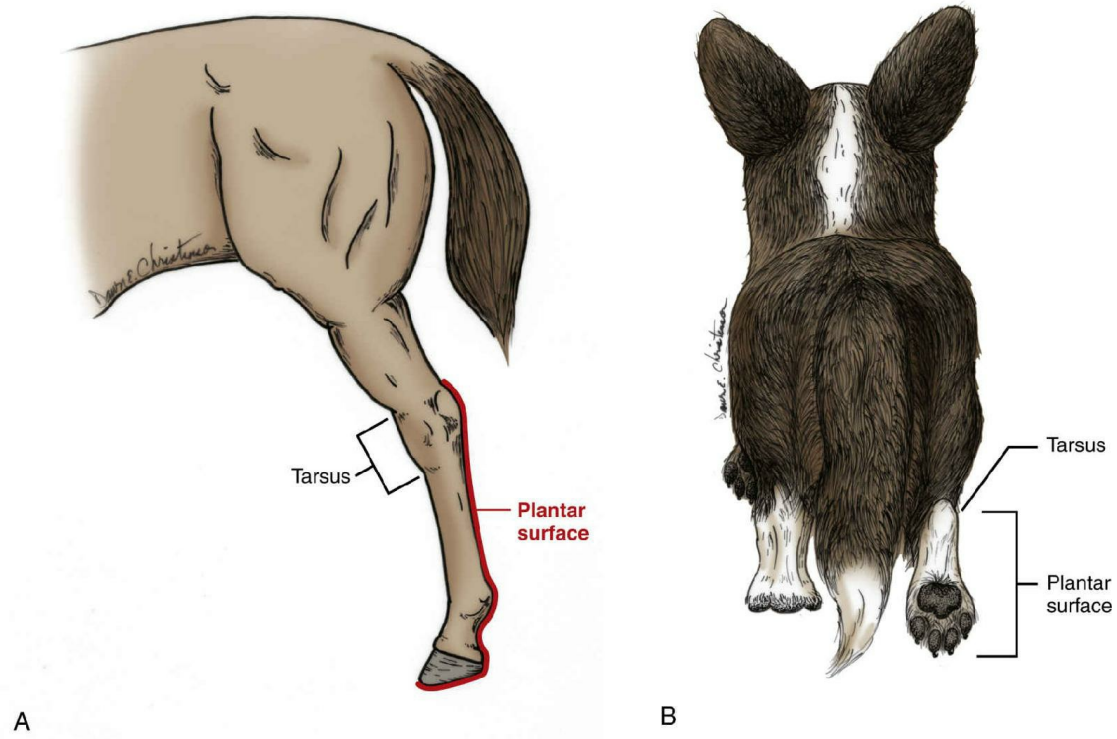

FIG. 1.13 (A) Plantar surface, left rear limb of a horse. (B) Plantar surface, right rear limb of a dog.

Okay, so that takes care of the posterior aspect of the distal part of the limbs. What about the anterior aspect (Fig. 1.14A and B)? That surface is the *dorsal surface* of the paw or hoof. In the forelimb, the dorsal surface extends proximally to include the carpus. In the rear limb, the dorsal surface extends proximally to include the tarsus.

What about the corresponding surfaces that lie proximal to the carpus and tarsus? Proximal to the carpus and tarsus of the limbs we merely use the directional terms *cranial* and *caudal* (see Fig. 1.14A and B). Again, I did not make the rules. I am merely the messenger. Whoever established these rules must have thought that, because the proximal part of each leg is attached to the body, we can reference the head and the tail for these proximal surfaces of the extremities. I hope that the colored schematics in Fig. 1.14A and B will help you to understand the relationships of limb surfaces.

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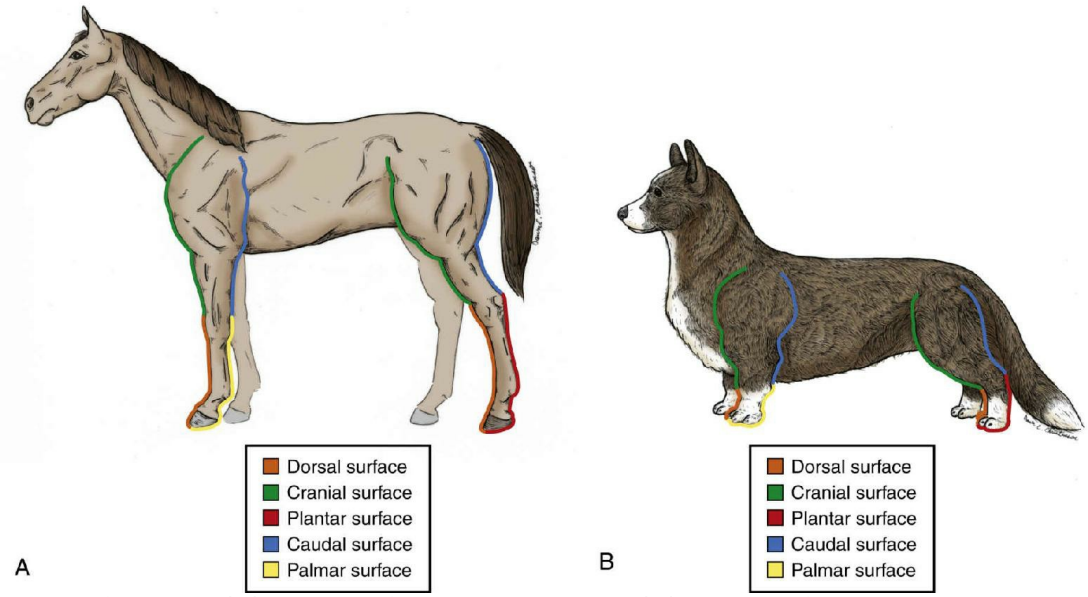

FIG. 1.14 (A) Limb surfaces in the horse. (B) Limb surfaces in the dog.

Applied Radiographic Terms

We have already established the importance of directional terminology in prior sections of this chapter. There is probably no facet of veterinary medicine that relies on directional terminology more than ***radiography*** [*radi(o)-* rays, radiation + *-graphy* process of recording]. In ***radiology*** [*radi(o)-* rays, radiation + *-ology* the study of], gamma radiation (x-rays) is used to penetrate the body. As the rays pass through the body, they interact with the specialized ***radiographic*** [*radi(o)-* rays + *graph(o)-* to record + *-ic* pertaining to] film (standard ***radiograph*** [*radi(o)-* rays + *graph(o)-* a record) or cassette (digital radiograph). The resultant radiograph captures the body parts in varying shades of black, gray, and white, depending on the ease of passage of the radiation (Fig. 1.15A–C).

As you can see in Fig. 1.15A, we are ***radiographing*** [*radi(o)-* rays + *graph(o)-* to record + *-ing* act of] a dog's left front paw and carpus. The *radiographic* view is determined by the order in which the radiation passes through body surfaces. In this example, the radiation from the machine passes through the dorsal surface first, then through the paw, exiting the palmar surface last, before interacting with the radiographic cassette. Because of the body surface sequence, we label this *radiograph* as a ***dorsopalmar*** [*dors(o)-*

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back + *palm(o)-* palm + *-ar* pertaining to] view. Now, it would be way too cumbersome and take up too much space on the *radiograph* to spell out the entire directional term *dorsopalmar*. So, the term is abbreviated to ***DPa***. The human equivalent of this view would be an ***anteroposterior*** [*anter(o)-* front + *poster(o)-* rear + *-or* pertaining to] or ***AP*** view of a person's hand and wrist. A comparable view in the distal rear limb (including the foot and tarsus) would be a ***dorsoplantar*** [*dors(o)-* back + *plant(o)-* sole + *-ar* pertaining to] view, abbreviated ***DPl***. In humans, this would be an *anteroposterior* (*AP*) view of the foot and ankle.

It is also essential to mark which extremity this is, left versus right. In this case, the *radiograph* is labeled "Lt" for left (see Fig. 1.15B and C). Note that some radiographic markers use "L" for left and "R" for right. If by itself, the individual letter is assumed to indicate which side of the body is represented. This is very important, especially if treatment or surgery is required—in our example, for the left paw. Additionally, we often obtain ***bilateral*** [*bi-* two + *later(o)-* side + *-al* pertaining to] radiographs to compare affected versus unaffected extremities. Without right and left radiographic markers, we would not be able to tell which leg we were looking at on the radiograph. You'll also notice that stamped in the upper corner on the medial side of the radiograph (Fig. 1.15C) is the patient's ***signalment*** (i.e., patient name, species, breed, sex, age/date of birth) along with the date of the imaging. This is very important for treating the correct patient.

In Table 1.1, you will find directional terms routinely used in veterinary radiography, along with their abbreviations. Remember that the compound words indicate the order in which the radiation passes through the body. The first root word represents the first body surface penetrated by the radiation. The second root word represents the second body surface penetrated by the radiation. As you look at the table, you may be wondering what an ***oblique*** (o-blīk') view is. It is one that is angled, often at a 30- to 45-degree angle to the body planes. For instance, when bony structures are superimposed (i.e., stacked directly over one another) in a standard ***mediolateral*** [*medi(o)-* middle + *later(o)-* side + *-al* pertaining to] view, an *oblique* view may help us to visualize the individual structures better. We must still use other directional terms along

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with the term *oblique*. For instance, we may position a patient in *right lateral recumbency* for a ***mediolateral oblique (MLO)*** view of the right rear leg.

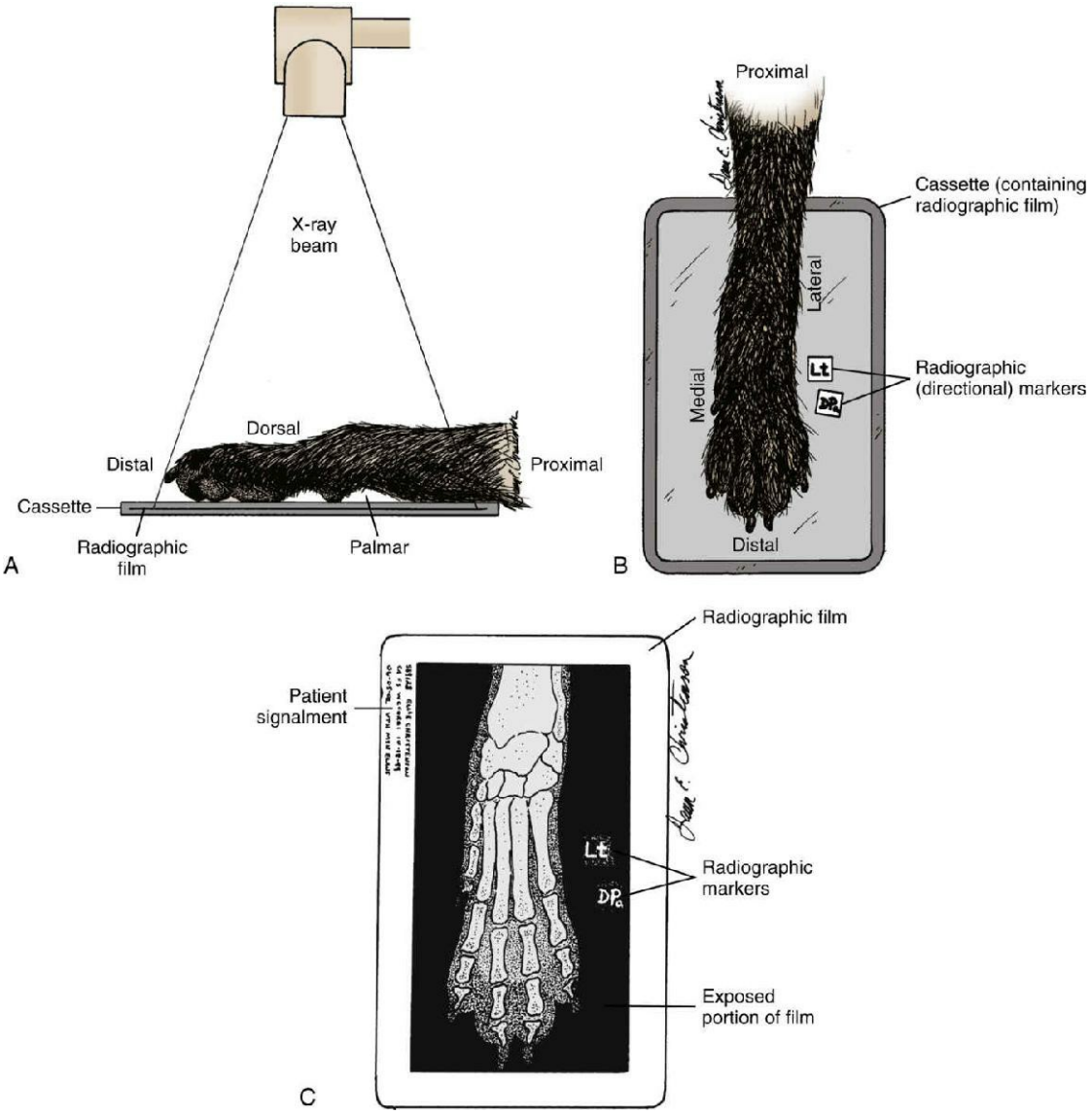

FIG. 1.15 (A) Lateral view of the left (Lt) front paw and carpus for dorsopalmar *(DPa)* radiograph; (B) dorsal view, canine distal left front limb. (C) DPa schematic radiograph of a dog's distal left front limb.

As you look at Table 1.1, are you wondering why we would need mediolateral and ***lateromedial*** [*later(o)-* side + *medi(o)-* middle + *-al* pertaining to] radiographic labels? How about ***dorsoventral*** [*dors(o)-* back + *ventr(o)-* belly + *-al* referring to] versus ***ventrodorsal*** [*ventr(o)-* belly + *dors(o)-* back + *-al* pertaining to]? Does it really make a difference? Yes, it may. First, it may make a difference for the quality of the image produced. The further away a structure is from the radiographic cassette, the more it will be distorted. Have you ever made shadow figures on the wall using your hands and a flashlight? If you hold your hand close to the wall, the shadow created is really crisp and about the same size as your hand, isn't it?

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When you hold your hand further away from the wall, the shadow becomes bigger and fuzzy, right? Similar distortions occur in radiography. If we want to produce a high-quality radiograph of the proximal right rear leg, should the patient be positioned in left or right lateral recumbency? We want the right leg as close to the radiographic cassette under the table as possible. So, the patient should be positioned in *right lateral recumbency*. This dictates a *mediolateral* radiographic view of the right rear leg.

TABLE 1.1

Veterinary Radiographic Directional Terms and Abbreviations

Directional Term	Abbreviation	Directional Term	Abbreviation
Left	Lt or L	Medial	M
Right	Rt or R	Lateral	L or LAT
Dorsal	D	Proximal	Pr
Ventral	V	Distal	Di
Cranial	Cr	Oblique	O
Caudal	Cd	Mediolateral	ML
Palmar	Pa	Lateromedial	LM
Plantar	Pl	Dorsoventral	DV
Rostral	R	Ventrodorsal	VD

TABLE 1.2

Comparison of Standardized Veterinary Radiographic Terminology and Equivalent Human Terminology

Standardized Veterinary Terminology		Equivalent Human Terminology	
Directional Term	**Abbreviation**	**Directional Term**	**Abbreviation**
Craniocaudal	CrCd	Anteroposterior	AP
Caudocranial	CdCr	Posteroanterior	PA
Dorsopalmar	DPa	Anteroposterior	AP
Palmarodorsal	PaD	Posteroanterior	PA
Dorsoplantar	DPl	Anteroposterior	AP
Plantarodorsal	PlD	Posteroanterior	PA

The same is true for *dorsoventral* and *ventrodorsal* views. Let's consider chest radiographs. The heart lies along the ventral border of the chest. So if we want a high-quality radiograph of the heart, we should use a dorsoventral view of the chest. This view may also be necessary for patient comfort. If a dog has fluid accumulating in

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its chest cavity, surrounding its lungs, a dorsoventral view of the chest will be preferred. Why? If we place such a patient in dorsal recumbency, the fluid in its chest will compress the lungs, making it very difficult for the patient to breathe. So even though routine chest radiographs call for a ventrodorsal view, we will put the patient's needs first. This patient will be positioned in *sternal recumbency* (i.e., on its ***sternum*** or breast bone) for a dorsoventral chest radiograph.

Now let's go back and look more closely at that left front paw we radiographed earlier. Compare the dorsal view of the left paw in Fig. 1.15B to the corresponding radiograph shown in Fig. 1.15C. The radiographic markers are made of lead and therefore cannot be penetrated by the radiation. So, they appear bright white on the radiograph. In our patient, the radiographic brightness or darkness is determined by the density of the tissues. That darkness or brightness corresponds to how easy or difficult it is for radiation to pass through those tissues.

In this radiograph (Fig. 1.15C), the bones are the most dense tissue type. Because the radiation had difficulty passing through this dense material, very little radiation passed through to interact with the radiographic film or cassette. Consequently the bones appear brighter or whitish on the radiograph. The surrounding tissue densities appear as varying shades of gray. The more dense the tissue, the lighter the gray will be. Fluids also appear gray (more fluid = a lighter gray). Finally, notice the color of the radiograph surrounding the whole foot. Here the radiograph is solid black. The radiation in these areas passed through the air unobstructed. Therefore it interacted fully with the radiosensitive material in the film or cassette, transforming it to black.

By the way, do you remember, toward the end of the section "Directional Terms Related to Body Surfaces," where we detailed surfaces of the extremities? These surfaces are very important in *radiology [radi(o)-* rays, radiation + *-ology* study of]. The related terms have been standardized for use in veterinary medicine. However, there are times and places where human terms may still be used. So, in Table 1.2, you will find a comparison between the veterinary radiographic terms and their human equivalents. Again, remember that each compound word represents the sequence of radiation

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passage through the extremity's surfaces.

Applied Sonographic Terms

Take a deep breath. We're almost there. The end of the chapter is in sight.

Now that you have a handle on commonly used radiographic terms, let's look at another very important facet of veterinary diagnostic imaging: ***sonography*** [*son(o)-* sound, sound waves + *-graphy* process of recording]. *Sonography* or ***diagnostic ultrasound*** uses sound waves to create images. Think of the way naval ships or oceanographers use sound waves (sonar) to map the ocean floor or to find remnants of shipwrecks. Sound waves are emitted from a probe on the ship. Then the echoes of returning sound waves, bouncing off structures on the ocean floor, are analyzed by a computer to create a map of the ocean floor. *Sonography* uses this basic echo principle. High-frequency sound waves are pulsed from a probe into the body, and the returning echoes are then analyzed by a computer to create images of body structures.

Our goal in this discussion is to merely familiarize you with specific terms used to describe the ***echogenicity*** [*ech(o)-* echo + *gen(o)-* production + *-icity* characteristic of] of structures seen in sonographic images. Of course you can reinforce some of the directional terms you've already learned. Remember the little Corgi who, early in the chapter, rolled onto her back? Well, *dorsal recumbency* is probably the most frequently used positioning for *abdominal ultrasound* in veterinary patients. Because my Corgi loved to lie in *dorsal recumbency*, she was a perfect candidate for students to practice abdominal ultrasound on. She often fell asleep while students scanned her belly. But there are patients who cannot tolerate this positioning. Such patients may be placed in *lateral recumbency* for comfort and cooperation. Abdominal ultrasound may be the most frequent facet of veterinary sonography, but almost any body cavity or body part can be evaluated with ultrasound.

Body tissues, depending on their texture and how they reflect sound waves, will determine their ***echogenic*** [*ech(o)-* echo + *gen(o)-* production + *-ic* pertaining to] properties. Specific terms have been

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developed to describe the *echogenicity* of organs and structures seen in ultrasound images. For instance, to describe the texture of an organ's tissue, we may use the terms ***heterogenous*** [*heter-* different, difference + *gen(o)-* to produce + *-ous* pertaining to] or ***homogenous*** [*homo-* same, sameness + *gen(o)-* to produce + *-ous* pertaining to]. How do we interpret these descriptors? Let's say that the ***sonographer*** [*son(o)-* sound, sound waves + *graph[o]-* to record + *-er* one who] describes a liver lobe using the term *heterogenous*. With that description, she infers that the liver lobe in question has an irregular *echogenic* texture. Whereas the rest of the liver lobes, described as *homogenous*, have a regular or uniform echogenic texture.

Is it possible for anything to *not* create an echo? Yes. Fluid tends to have poor *echogenic* properties. Think back to our brief discussion of sonar. The sound waves emitted from the ship's probe pass easily through the ocean water. They do not echo back until they reach the ocean floor. The same is true in the body. For example, we often use abdominal ultrasound to evaluate the urinary bladder or to guide the collection of a urine sample. The high-frequency sound waves echo off the bladder wall to delineate the structure. But the fluid (i.e., the urine) filling the bladder is ***anechoic*** [*an-* no, absent + *ech(o)-* echo + *-ic* pertaining to]. Without an echo, that urine might as well be a black hole in outer space. Actually, that's a pretty good way to describe anything that is anechoic, because they do appear rather black in sonographic images. Fig. 1.16 shows a dog's urinary bladder filled with anechoic fluid (urine). Make note of the directional labels placed in this figure for your benefit. This patient was in dorsal recumbency for the evaluation. The probe was pressed against the ventral abdomen (top of image) to create this image. Cranial and caudal notations should help orient you to the rest of the patient's body as you look at this sonographic image.

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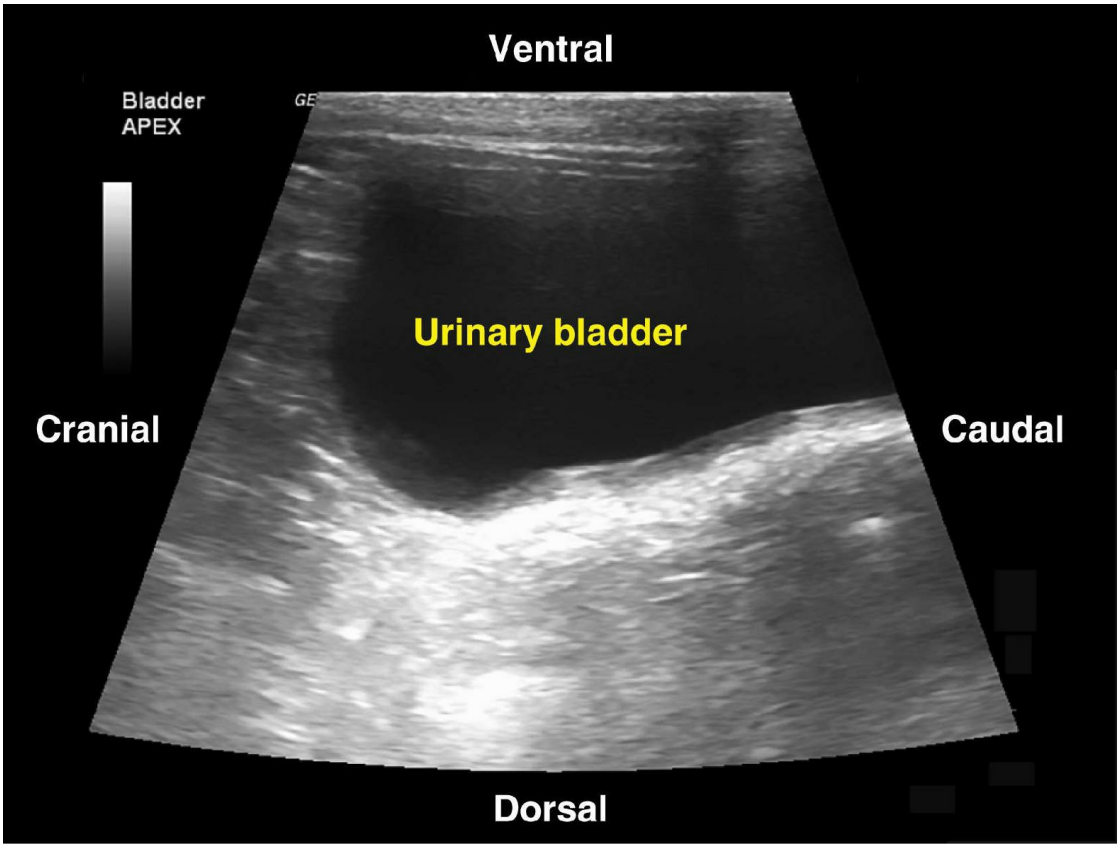

FIG. 1.16 Anechoic fluid-filled urinary bladder of a dog.

Is it possible for organs or structures to have areas that echo too much or too little compared with the rest of the organ? Yes. This is often true in areas of disease within an organ. Let's consider the liver again. Veterinary patients may develop cancer, just as human patients do. Tumors developing in the liver will likely appear different in their *echogenic* properties compared with the rest of the liver. If the *echogenicity* of a tumor appears much brighter than the surrounding liver tissue, the mass will be described as ***hyperechoic*** [*hyper-* excessive + *ech(o)-* echo + *-ic* referring to]. If on the other hand, when we use the term ***hypoechoic*** [*hypo-* decreased + *ech(o)-* echo + *-ic* referring to] to describe the tumor, we are saying that the mass does not echo as much as the rest of the liver tissue. That hypoechoic region would appear a bit darker gray than the rest of the surrounding liver tissue. Comparatively, fluid-filled cysts within the liver may be *anechoic*. Of course, it is also possible to have a nodule of some sort that is ***isoechoic*** [iso- same + *ech(o)-* + *-ic* referring to]. In this case, the nodule may have a *hyperechoic* (bright) capsule (wall) surrounding it, but the interior of the nodule echoes exactly the same (*isoechoic*) as the rest of the liver.

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Case Study

Radiographs

In this case study, I will present clinical information about a patient to you. I will use as many imaging and directional terms as are applicable to the given case. I will not define any of the terms used. Following presentation of the case information, I will ask a series of questions. Those questions will test your knowledge and interpretation of many of the medical terms presented in this chapter. If you have difficulty with the case study and associated questions, you should go back and review the chapter information again.

Our canine patient in this case study is an 8-year-old spayed female Cardigan Welsh Corgi named Ellie. She is brindle and white in color. Her medical record number is 654321.

The owner presented this dog with a primary complaint of intermittent lameness. Ellie has been periodically limping on her left cranial limb for approximately 2 weeks. There are no known injuries to the limb. There is no particular activity that precipitates the lameness. Twice she became three-legged lame, completely refusing to use the affected limb at all. Ellie is not lame today. The owner reports that everything else with regard to the patient is within normal limits (i.e., attitude, appetite, activity level, and elimination habits).

On physical examination, Ellie is found to be in excellent body condition. There is no evidence of injury to the left leg or paw. Bilaterally, her limbs are symmetrical. No pain is elicited on palpation or when putting each joint through its full range of motion. All physical findings are within normal limits. Comparative radiographic evaluation of both front limbs is warranted.

The clinician ordered bilateral forelimb radiographs. These included the following: DPa paw and carpus, ML carpus, L ML elbow, CrCd elbow, and CrCd shoulder. Positioning for some of these views was challenging because of Ellie's Corgi legs. Below are the radiographs.

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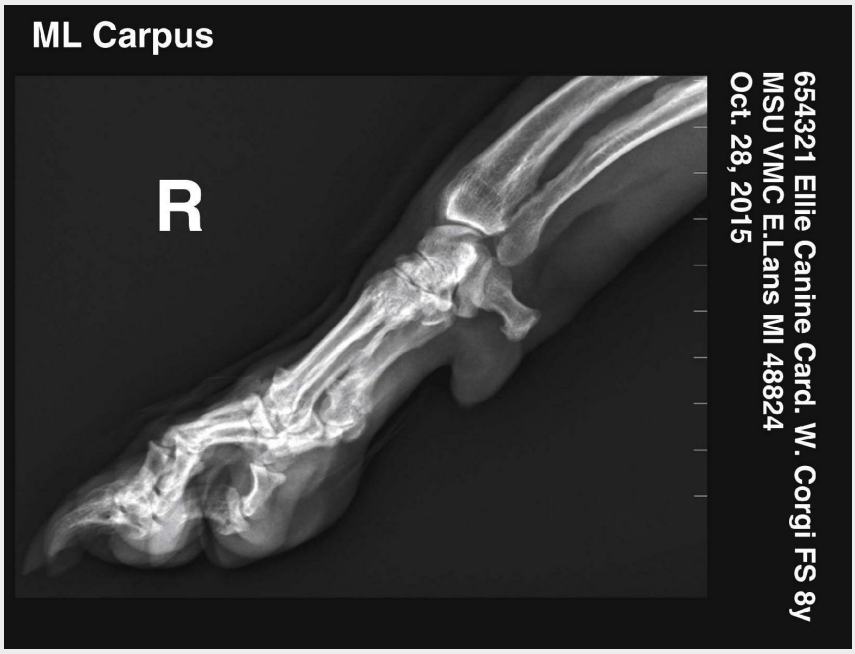
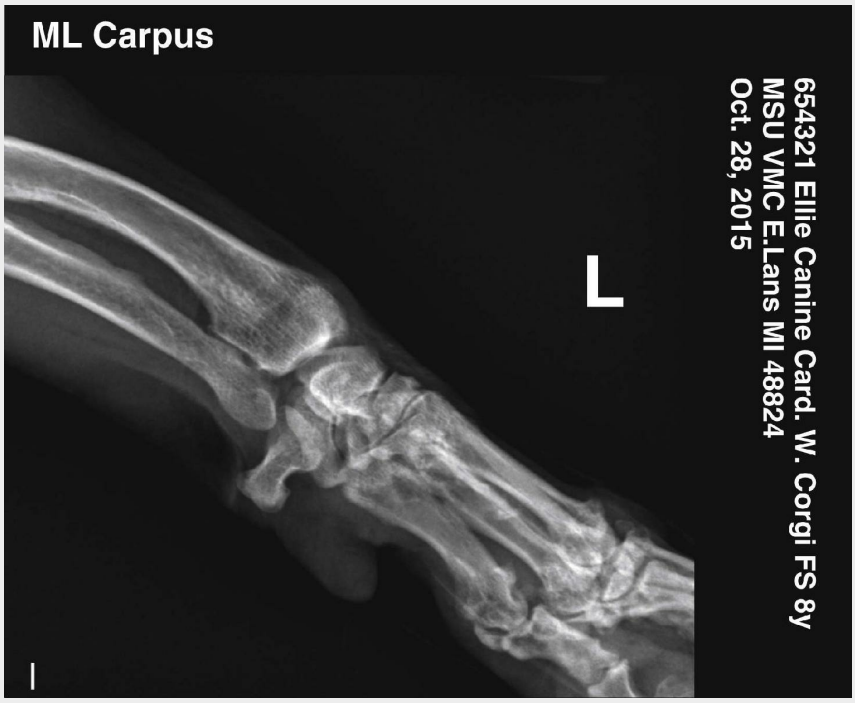
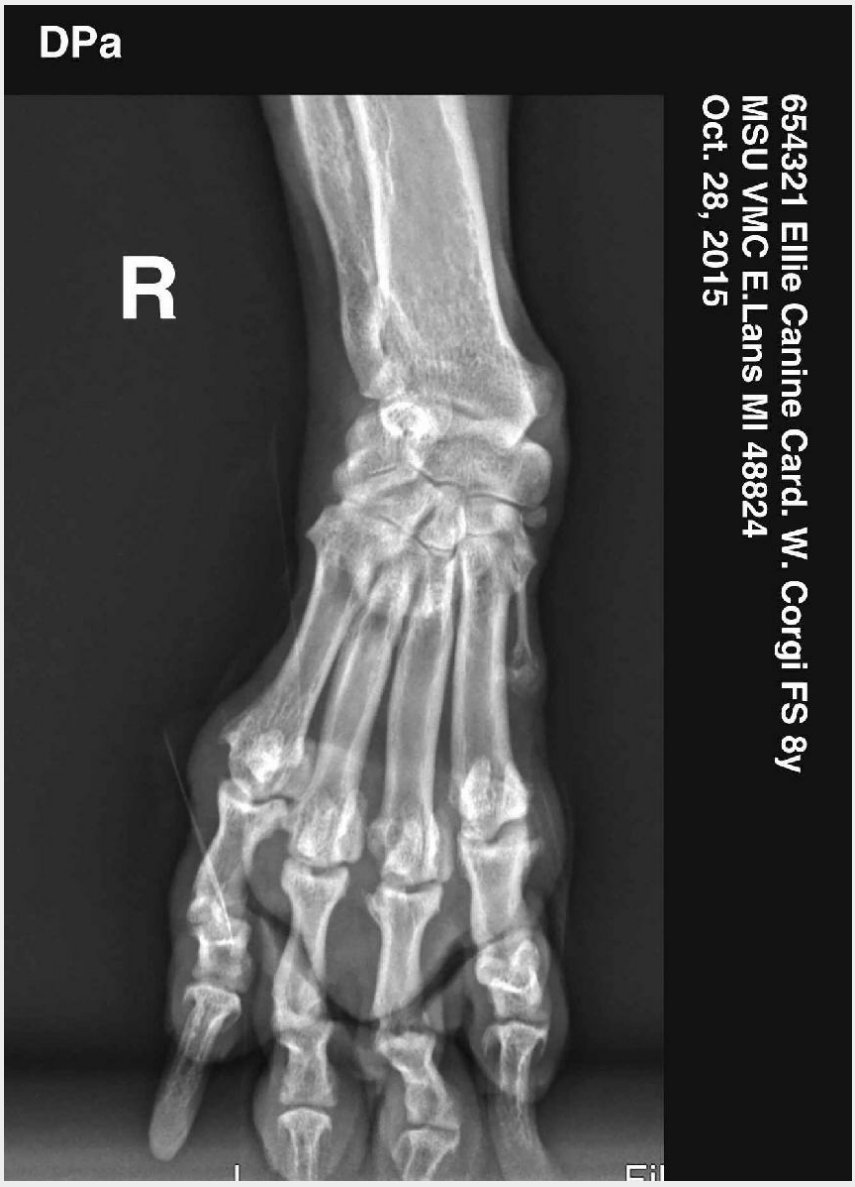
R ML carpus



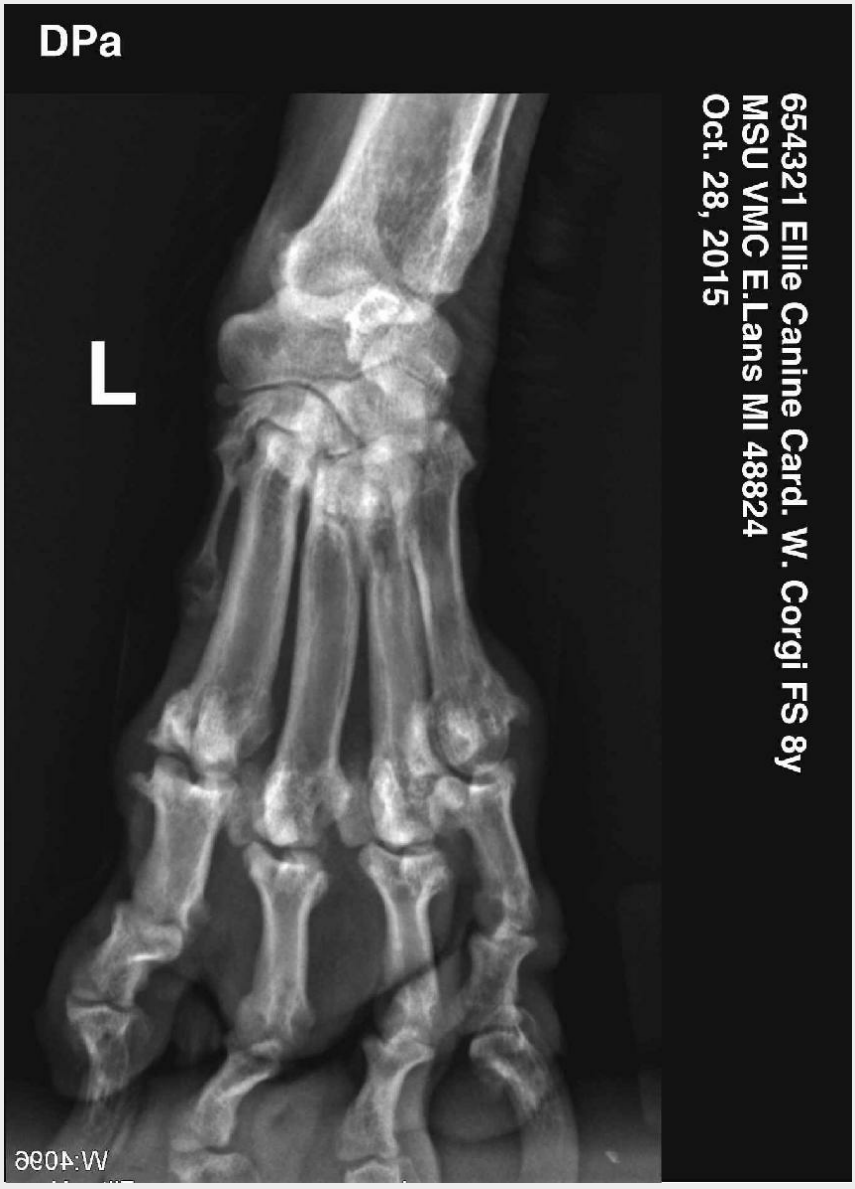
L ML carpus



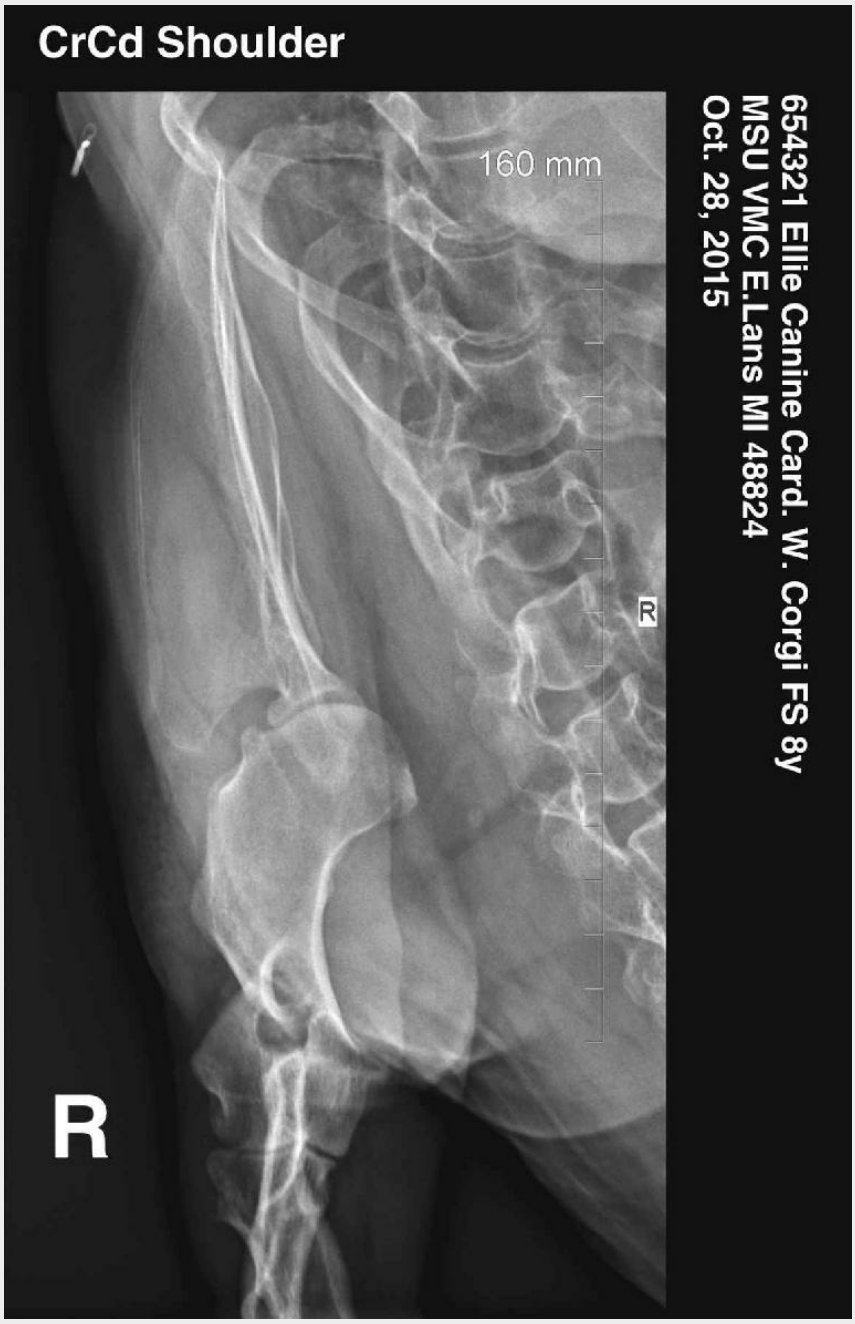
R DPa


DPa
R
654321 Ellie Canine Card. W. Corgi FS 8y
MSU VMC E.Lans MI 48824
Oct. 28, 2015

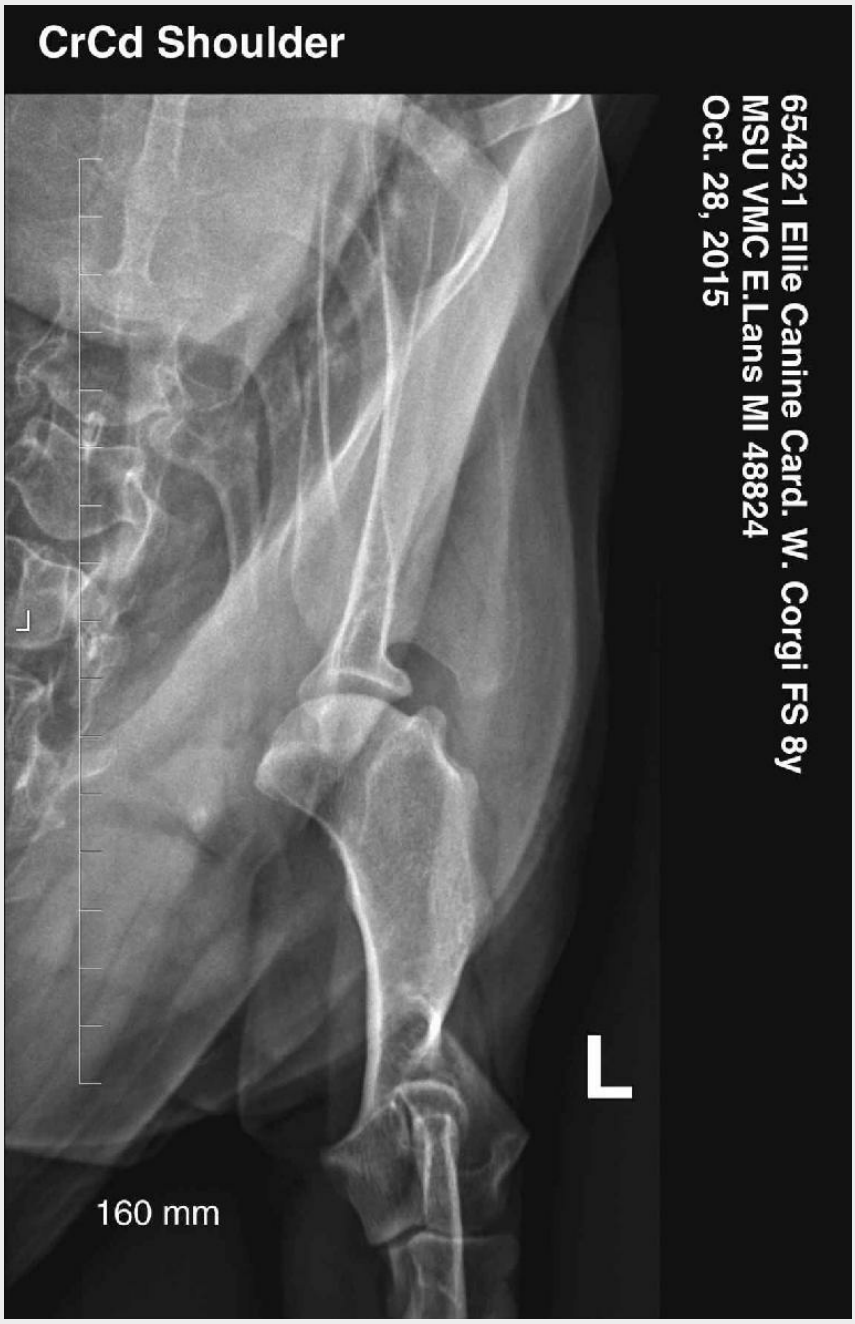
L DPa



R CrCd Shoulder



L CrCd Shoulder


CrCd Shoulder
654321 Ellie Canine Card. W. Corgi FS 8y
MSU VMC E.Lans MI 48824
Oct. 28, 2015
L
160 mm
L

R CrCd Elbow

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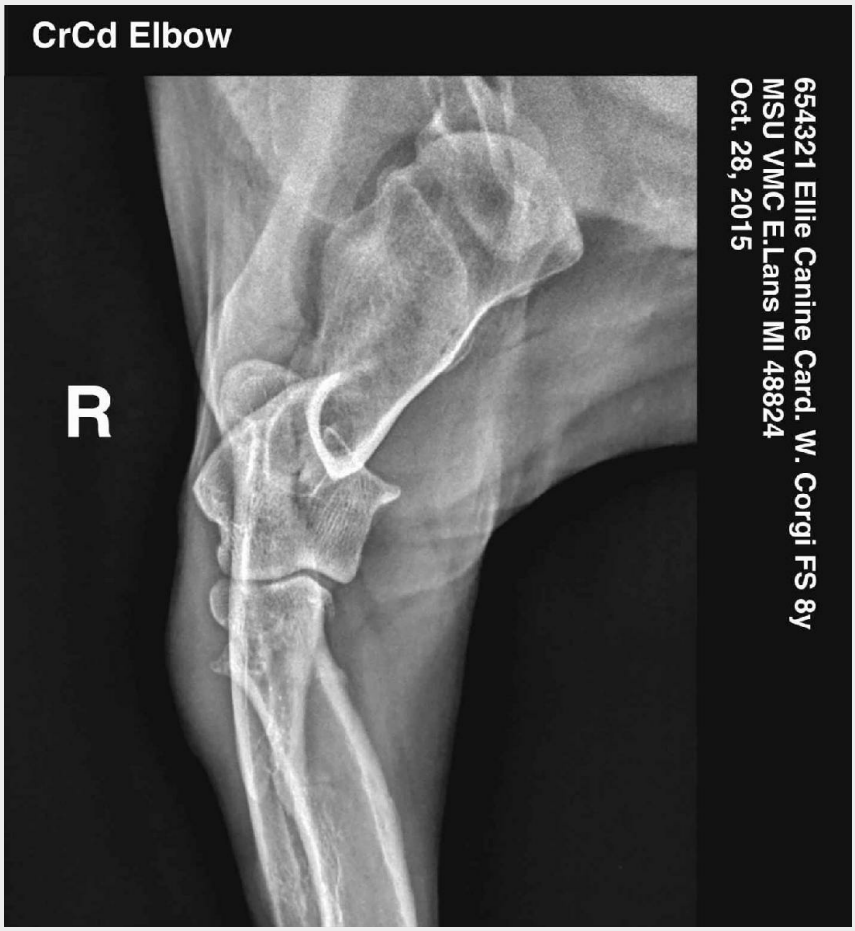

L CrCd Elbow

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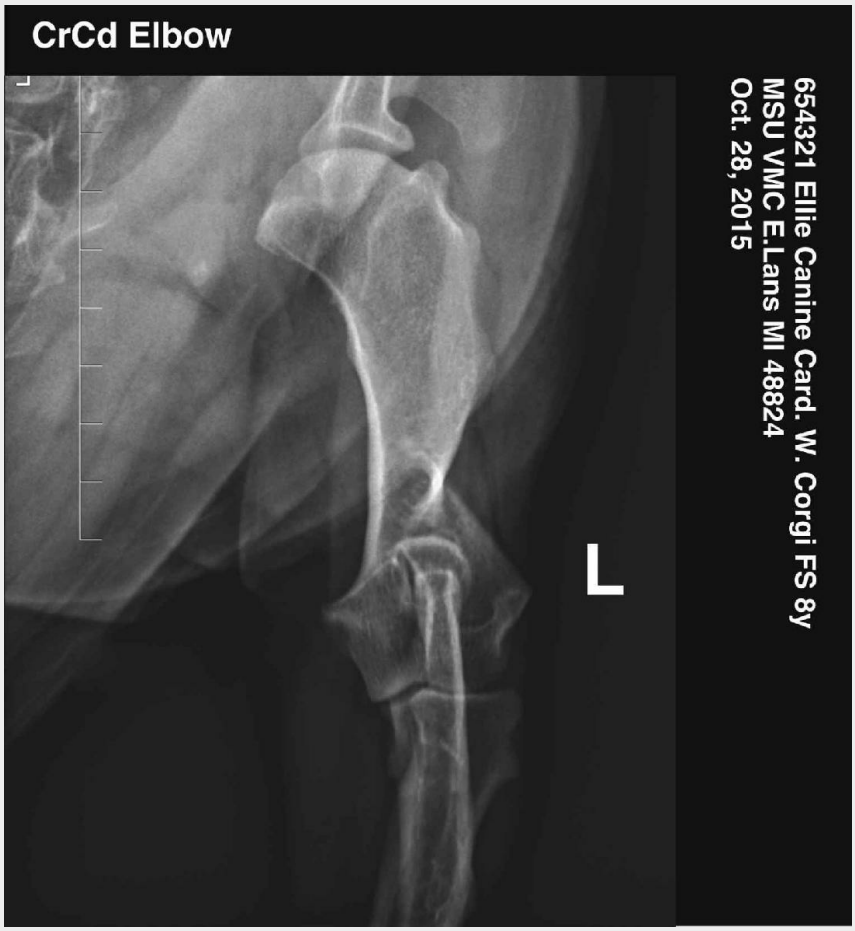

L ML elbow

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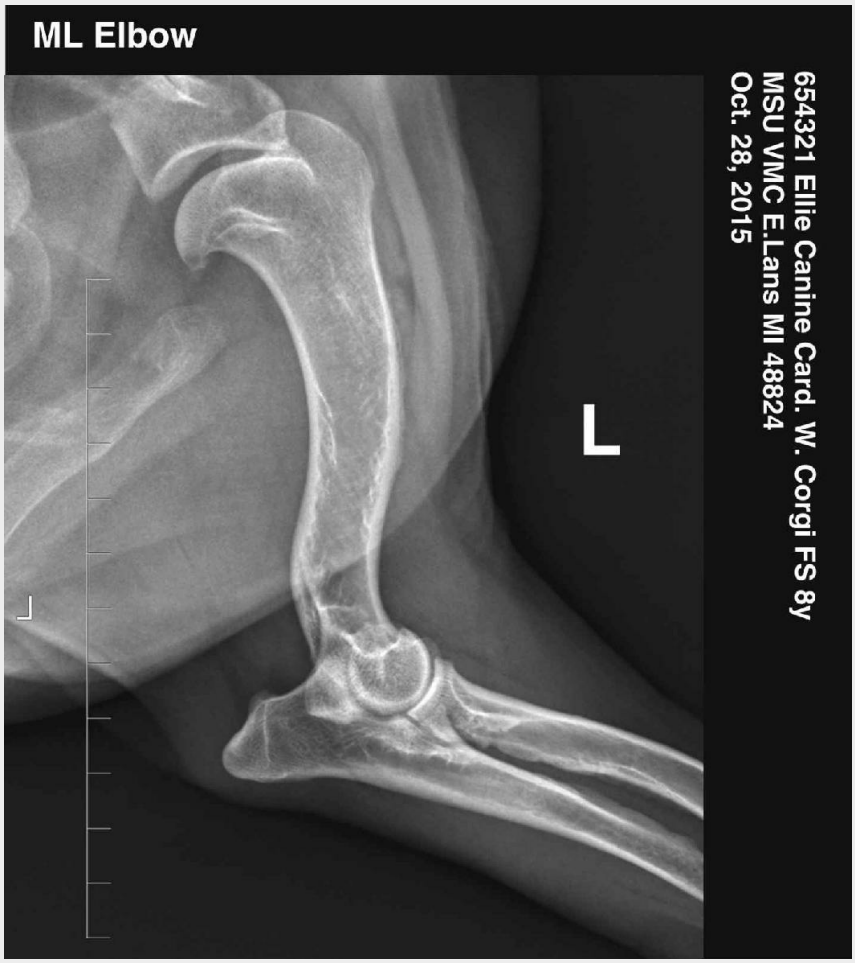

Case Study Questions

1. In the radiographs labeled "ML carpus," what medical term does the abbreviation ML represent? _____
2. In the ML carpus radiographs, which body surface is penetrated last by the radiation? _____
3. For the radiograph labeled "R ML carpus," what was the patient's body position (in three words)? _____ ____ ___
4. Each radiograph is marked with the same information: 654321 Ellie Canine Card.W.Corgi FS 8y. This information represents the patient's. _____
5. Two of the radiographs are labeled "Dpa." What medical

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term does this abbreviation represent? _____

6. What radiographic view is similar to the DPa, but is exclusively for the distal caudal limbs? (medical term not the abbreviation) _____

7. What medical term, meaning front to rear, used in human radiography would be the equivalent of a DPa of a person's hand and wrist? _____

8. For the ML elbow radiograph, the patient's body position was left (in two words)? _____ _____

9. In the radiographs of the elbows and shoulders labeled CrCd, what medical term does the abbreviation represent?_____

10. The CrCd abbreviation should be used only when the _____ portion of any limb is being radiographed.

11. Routine chest and abdominal radiographs were also taken on this patient but not shown. If these radiographs were taken with her in dorsal recumbency, what medical term would best indicate the radiographic view? _____

12. How was this patient most likely positioned for the CrCd elbow and shoulder radiographs (in two words)? _____ ____

13. The medical term for another type of imaging (i.e., diagnostic ultrasound) that was not performed on this patient is _____.

14. Which surface of this patient's limbs is closest to the midsagittal plane of the body?_____

15. The medical term indicating that the clinician requested radiographs of both front limbs is the term _____ literally meaning "two sides."

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The Answer Key to these case study questions may be found in Appendix B.

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Body Building

Applied Atomic, Molecular, and Cellular Terminology

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GOALS AND OBJECTIVES

By the conclusion of this chapter, the student will be able to:

1. Recognize common root words, prefixes, and suffixes related to chemistry, biochemistry, cells, biology, and physiology.
2. Divide simple and compound medical terms into their respective parts.
3. Recognize, correctly pronounce, and appropriately use common medical terms presented in this chapter.
4. Recognize basic anatomic components of animal cells.
5. Demonstrate a basic understanding of applied chemistry, biochemistry, and animal cell structure and function.

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Applied Atomic and Molecular Terminology

Years ago I had a Basset Hound named Sadie (pictured here). People would often look at her and comment on how lazy she was. Yet I had one colleague, a boarded veterinary nutritionist, who always rushed to her defense saying, "She may look lazy on the outside, but at the ***cellular*** [*cellul(o)-* cells + *-ar* pertaining to] level she is very busy". I'll take that one step further by saying that she was even busier at the ***atomic*** [*atom* + *-ic* pertaining to] level.

Sadie, like every ***organic*** [*organ(o)-* carbon-containing + *-ic* pertaining to] ***biologic*** [*bi(o)-* life + *log(o)-* study, knowledge + *-ic* pertaining to] thing, was composed of minute particles of atoms and molecules, specifically arranged to create the whole. Think of how amazing that is! I mean, we're not even talking about cells,

tissues, and organ systems yet. We're talking about basic chemistry. That's right. All of the *organic* and ***inorganic*** [*in-* not + *organ(o)-* carbon containing + *-ic* pertaining to] compounds of the body are made up of various *atomic* elements.

Take a peek at the **Periodic Table of Elements** (Fig. 2.1). If you are unfamiliar with each of the elements and their atomic symbols, refer to Appendix A, near the back of the book. Approximately a dozen chemical elements are most important in body structure and function. Every veterinary professional should minimally be able to recognize the atomic symbols for and understand the importance of these 12 elements: hydrogen (H), carbon (C), nitrogen (N), oxygen (O), sodium (Na), magnesium (Mg), phosphorus (P), sulfur (S), chlorine (Cl), potassium (K), calcium (Ca), and iron (Fe). There are others of importance too, especially those that are significant nutritionally or may result in ***toxicity*** [*tox(o)-* poison + *-city* a state of]. We'll touch on others in subsequent chapters, where appropriate. For now, let's stay focused on the basics.

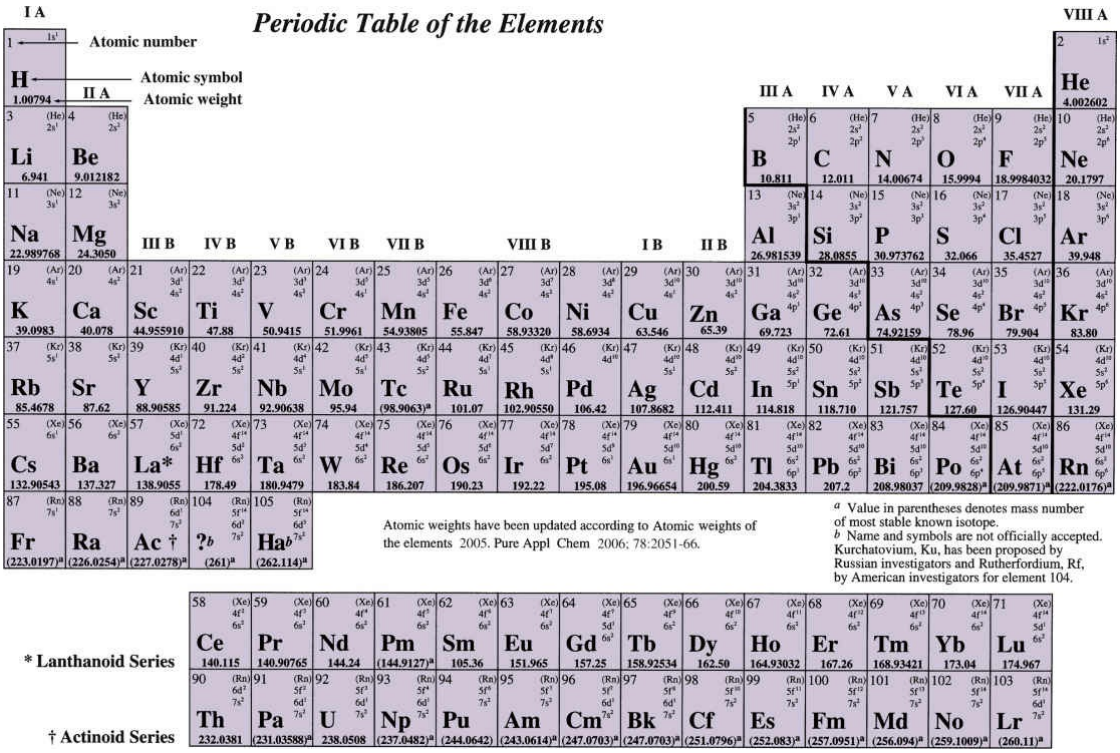

FIG. 2.1 Periodic table of elements.

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Atoms

Consider an atom of hydrogen (Fig. 2.2). It is the lightest of all the elements. Why? Hydrogen's ***subatomic*** [*sub-* under + *atom* + *-ic* pertaining to] particles are pretty limited. It has a single ***electron*** [*electr(o)-* electric + *-on* a unit] orbiting a ***nucleus*** [*nucle(o)-* nut, nucleus + *-us* a, the] containing a single ***proton*** [*prot(o)-* first, primitive + *-on* a unit]. At least it's balanced, with one negatively charged *subatomic* particle (i.e., the *electron*) and one positively charged *subatomic* particle (i.e., the *proton*). That lonely little *proton* is what determines the ***atomic number*** of hydrogen. In fact, the number of protons determines the *atomic number* of any element.

Let's compare an oxygen atom to that of hydrogen. If you look at the periodic table, you'll note that oxygen's *atomic number* is 8. Without even looking at the schematic of the atom in Fig. 2.2, you know from the *atomic number* that oxygen has eight protons. Notice too that oxygen's ***atomic weight*** is considerably more than hydrogen's. Ever wonder how they determine the *atomic weight* of atoms? Well, we can't weigh them on tiny scales. *Atomic weight* is really an estimate. By knowing the number of *protons* and ***neutron****s* [*neutr(o)-* neither + *-on* a unit; i.e., neither positive nor negative], ***nuclear*** [*nucle(o)-* nucleus + *-ar* pertaining to] ***mass*** is estimated. The *nuclear mass* is for all practical purposes the *atomic weight* of a given element. Looking at our hydrogen and oxygen atom comparison (see Figs. 2.1 and 2.2), we find that hydrogen has an *atomic weight* of 1 and "change." That makes sense with only one *proton* and no *neutron*. Oxygen, on the other hand, has an *atomic weight* of almost 16, if we round up. The eight *protons* and eight *neutrons* make up that *nuclear mass*. I hope that this brief explanation makes that periodic table of elements a little less intimidating for you.

What about the *electrons*? If they don't contribute to the *atomic weight*, just what do they do? The number of *electrons* and their arrangement determine, most importantly, how a given atom interacts with others. Now, the number of *electrons* (negatively charged) should equal the number of *protons* (positively charged) in a given atom. With equal numbers of positively and negatively charged *subatomic* particles, you would think that it would render atoms neutral. Not entirely so, because atomic stability is also an important factor. You see, this electrical net neutrality does not

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mean an atom is stable. Stability of an atom is actually determined by the arrangement of its electrons.

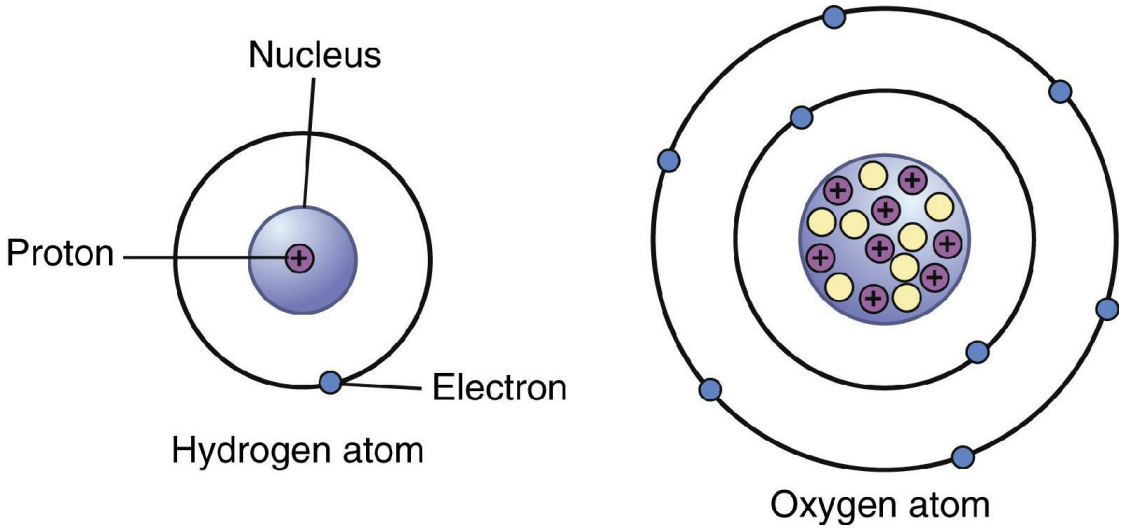

FIG. 2.2 Atomic structure of hydrogen and oxygen atoms.

The arrangement of electrons in their shells (aka "orbits") is quite orderly. If we consider the first 20 elements found in the periodic table, we will find that the maximum numbers of electrons that may be held in each shell of these elements are as follows:

First shell (nearest the nucleus)	2 electrons max.
Second shell	8 electrons max.
Third shell	8 electrons max.

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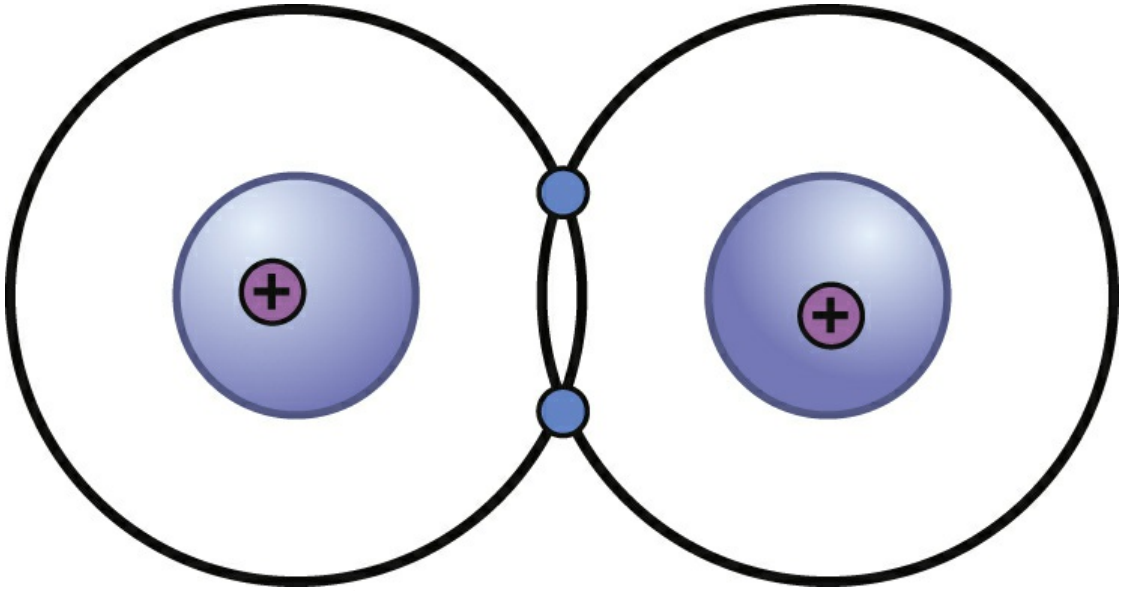

FIG. 2.3 Hydrogen molecule.

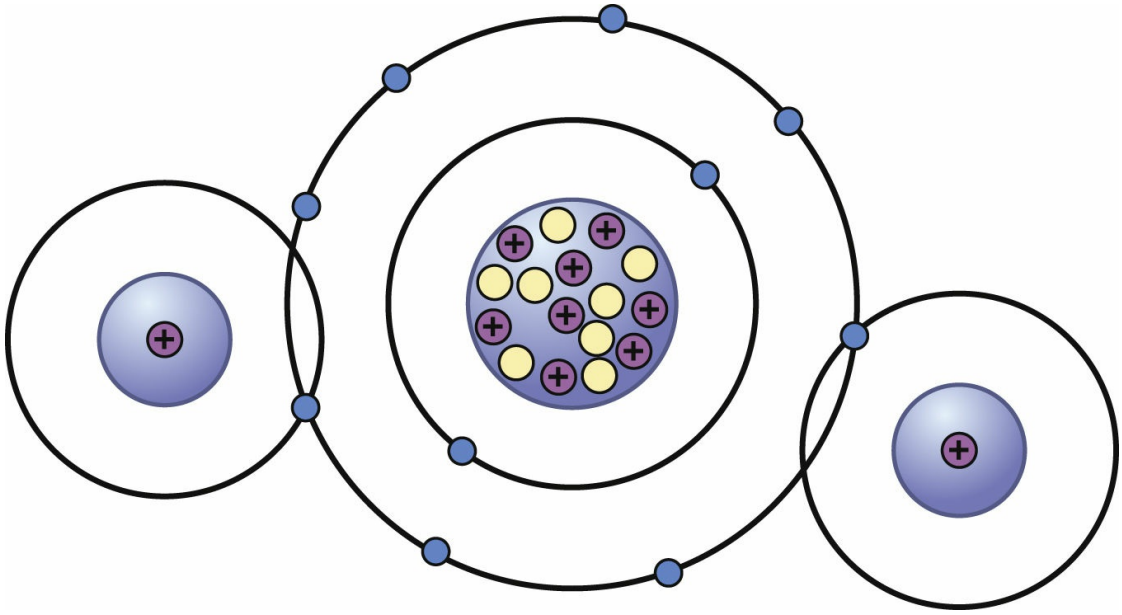

FIG. 2.4 Water molecule. (H_2O)

It is the outermost shell that determines stability. If the outer shell is full, as we find with helium and argon, the atom will be completely stable or ***inert***. *Inert* atoms, like helium and argon, do not interact with other atoms. It is a totally different story for hydrogen and oxygen. Their outer shells are not at maximum capacity of electrons. Therefore these atoms are unstable. Instability means an atom will readily interact with other atoms, all in an

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attempt to fill its outer shell. So, unstable atoms will bond in some way with other atoms to achieve this. Whenever two or more atoms bond—voila!—a *molecule* is formed.

Molecules and Molecular Bonds

A hydrogen *molecule* is formed by two hydrogen atoms (H_2). This molecule is stable because the atoms share their individual electrons (Fig. 2.3). Through sharing, they in essence fill their outer (in this case, only) shells and form a very strong bond. This type of ***molecular*** [*molecul(o)-* molecule + *-ar* pertaining to] bond is called a ***covalent*** [*co-* together + *val(o)-* strong + *-ent* one that] ***bond***. Probably the single-most important *molecular covalent bond* formed in the body and in nature is the one created between hydrogen and oxygen (Fig. 2.4). Oxygen is two electrons short of a full load in its outer shell. So, bonding with a hydrogen molecule (H_2) is a match made in heaven. H_2O is the most abundant *inorganic* molecule found in the body.

Approximately 70% of an animal's body is H_2O (water). Based on that fact alone, we can assume that adequate ***hydration*** [*hydr(o)-* water + *-tion* state of] is essential for life and many ***physiologic*** [*physi(o)-* nature, natural + *log(o)-* study, knowledge + *-ic* pertaining to] processes. That's why animals and people always need access to fresh drinking water. Without it, they cannot maintain adequate *hydration*. ***Dehydration*** [*de-* away from + *hydr(o)-* water + *-tion* a state or condition] can lead to ***pathologic*** [*path(o)-* disease + *log(o)-* to study + *-ic* pertaining to] conditions, like ***hypotension*** [*hypo-* low, insufficient + *tens(o)-* tension, pressure + *-sion* a condition of; i.e., low blood pressure] for instance. Significant, prolonged *hypotension* can be very detrimental to major organs like the heart and kidneys. So remember that H_2O represents one of the most important *covalent molecular* bonds of the body.

There are other types of *molecular* bonds, beyond *covalent* bonds. ***Electrovalent*** [*electr(o)-* electron + *val(o)-* strong + *-ent* one that is] ***bonds*** or ***ionic*** [*ion(o)-* going + *-ic* pertaining to] ***bonds*** are formed when electrons are actually transferred or "going" from one atom to another. In case you're wondering, ***ions*** are nothing more than electrically charged atoms or molecules in a solution. Ions become

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electrically charged by either gaining or losing electrons. Ions with a positive charge are called ***cations*** [*cat-, cata-* down, under + *ion*]. *Cations*, from losing electrons, have more protons than electrons, resulting in a positive charge. ***Anions*** [*ana-* up +*ion*] have a negative charge from gaining electrons. In essence, *anions* have a surplus of electrons compared to protons, creating their negative charge. Get it? A *cation* is "down" (*cata-*) electrons ("get down" with cations) and an *anion* is "up" (*ana-*) electrons. Let's use a sodium chloride (NaCl) molecule (Fig. 2.5) to explain *electrovalent or ionic bonds* a little bit better.

A sodium (Na) atom has 11 electrons and protons, with only 1 electron in its outer shell. So, it is unstable. An atom of chlorine (Cl) has 17 electrons and protons, with 7 electrons in its outer shell. It too is unstable. Remember, the optimal, maximum number of electrons in those outer shells is eight. Because the chlorine atom is closest to that optimum, it will act as a virtual magnet for sodium's lonely electron. Once that electron is captured by the chlorine (Cl^-) atom, the atom develops a negative charge (anion; because it has more electrons than protons). The sodium (Na^+) atom, having lost an electron, now has a positive charge (cation) from having more protons than electrons. As they say, "opposites attract." That is precisely what happens in a sodium chloride (NaCl) molecule. Even in solution, the bond of this NaCl ion will tend to remain because of the net difference in the electrical charge of each atom. To help your understanding of covalent and ionic (electrovalent) bonds, please watch the "Chemical Bonding" animation in the Evolve resources.

Electrolytes and Homeostasis

Did you notice how I wrote the *atomic* symbols for those individual ions (Na^+ and Cl^-)? This is how ***electrolytes*** [*electr(o)-* electron, electricity + *lyte* that may be dissolved] of the body are written, with either a superscript "+" or "–" along with the atomic symbol. An *electrolyte* is a molecule of salt, acid, or base that dissolves or separates into ions in body fluids. Anytime you look at blood work for a patient and see measurements for things like Na^+ (sodium), Cl^- (chloride), K^+ (potassium), HCO_3^- (bicarbonate), or PO_4^- (phosphate), you are looking at values for the patient's *electrolytes*.

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Both water and *electrolytes* are extremely important for ***homeostasis*** [*home(o)-* unchanged + *-stasis* state, condition of standing]. This is the balanced, stable state of the body.

Many *physiologic* mechanisms engage to maintain *homeostasis*. Yes, this is true even at the atomic and molecular levels. For example, nerve impulses rely on the net exchange of ***intracellular*** [*intra-* within + *cellul(o)-* cell + *-ar* pertaining to] K^+ ions and ***extracellular*** [*extra-* outside + *cellul(o)-* cell + *-ar* pertaining to] Na^+ ions. ***Homeostatic*** [*home(o)-* unchanged + *-static* pertaining to standing] mechanisms must maintain just the right quantities of ions like these in order to provide for normal body functions. Just looking at *electrolytes* involved in nerve impulses, it is easy to imagine how conditions like ***hyponatremia*** [*hypo-* low + *natri(o)-* sodium + *-emia* a blood condition] or ***hypokalemia*** [*hypo-* low + *kal(o)-* potassium + *-emia* a blood condition] might impact nerve function. Likewise, because glucose (sugar) is the principal fuel for nerve cells, ***hypoglycemia*** [*hypo-* low + *glyc(o)-* sugar + *-emia* a blood condition] can also have a profound negative impact on nerve function and other cell function for that matter. In short, electrolytes are very important. Too much or too little of key electrolytes results in ***pathology*** [*path(o)-* disease + *-logy* the study, knowledge of].

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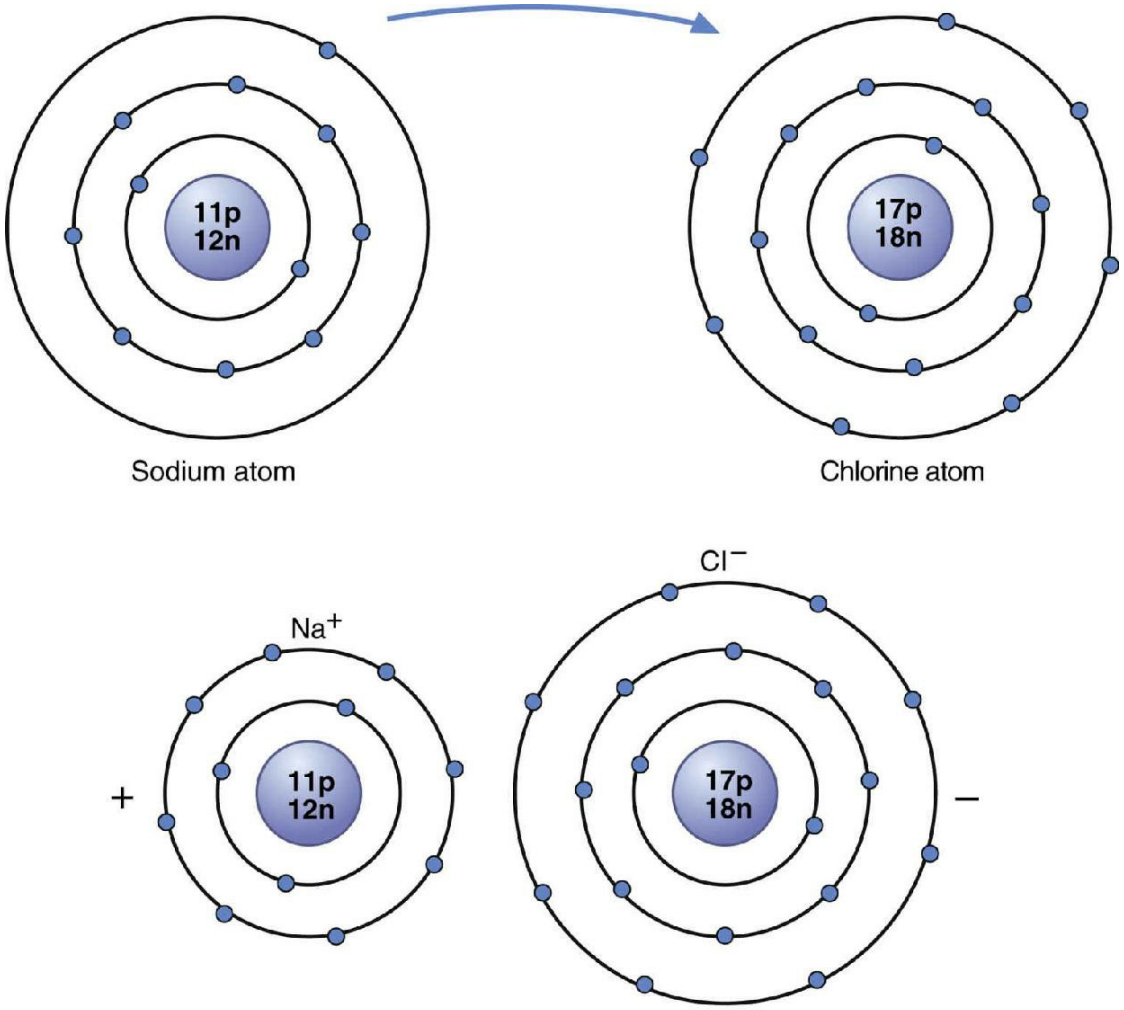

FIG. 2.5 Electrovalent (ionic) bond of a sodium chloride molecule.

Do you see why I can say that Sadie was very busy at an atomic level? Even while she was sleeping, ionic exchange would facilitate nerve impulses and many of her other cells would be producing waste products, like carbon dioxide (CO_2). That CO_2 when combined with the water (H_2O) of the body forms ***carbonic*** [*carbon* + *-ic* pertaining to] ***acid*** (H_2CO_3). As you might assume, too much *carbonic acid* can mess up *homeostasis* by making the body too acidic. In normal ***physiology*** [*physi(o)-* nature, natural + *-logy* the study of], there are many ways for hydrogen ions (H^+) to be removed from that big molecule of *carbonic acid*. We might use H^+ for stomach acid production. Or the kidneys might make the urine more ***acidic*** [*acid* + *-ic* pertaining to] by removing hydrogen ions from circulation. No matter how it is accomplished, removal of one H^+ leaves

bicarbonate (HCO_3^-), a base or alkaline salt ion. Acid–base *homeostasis* is completely tied to numbers of hydrogen ions (H^+) versus bicarbonate ions (HCO_3^-). Too many hydrogen ions (H^+) and too much *carbonic acid* (H_2CO_3) and a patient will develop the *pathologic* condition of ***acidosis*** [*acid(o)-* acid + *-sis* condition of]. Too many bicarbonate ions (HCO_3^-) and a patient will develop the *pathologic* condition of ***alkalosis*** [*alkal(o)-* base, alkaline + *-sis* condition of]. Both conditions can have very negative effects on the function of the body and lead to serious *pathologic* consequences. Extreme *acidosis* or *alkalosis* can be lethal (deadly). Now, this is a very simplistic way of looking at acid-base *homeostasis*. Yet for our purposes here, it is sufficient. We will touch on the topic again in future chapters where appropriate.

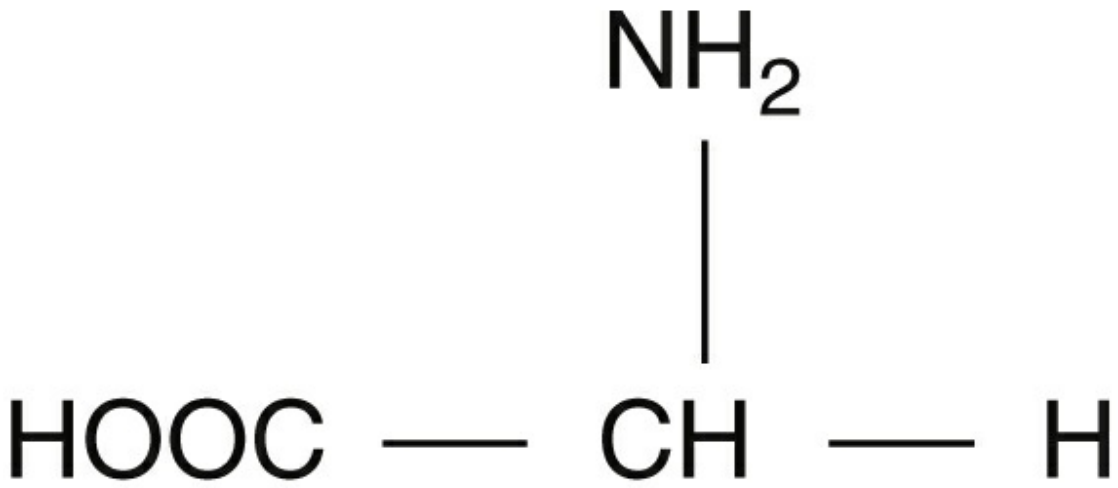

FIG. 2.6 Molecular structure of the amino acid glycine.

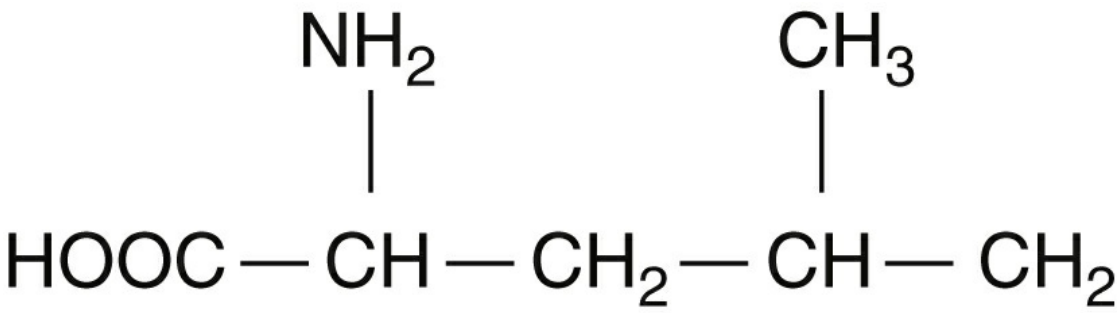

FIG. 2.7 Molecular structure of the amino acid leucine.

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Beyond basic ***biochemistry*** [*bi(o)-* life + *chemistry*], what I find fascinating is how molecules in just the right sequence and combination form the cells and tissues of the body. The invisible become visible and tangible. That's right. Molecules made of various combinations of carbon (C), hydrogen (H), oxygen (O), and nitrogen (N) atoms form ***amino*** (ŭ-me'no) ***acids***. *Amino acids* are the basic structural building blocks of the body. The *molecular* structure of each *amino acid* is different. For instance, the amino acid glycine [gli'seen] (Fig. 2.6) is quite different from leucine [lu'seen] (Fig. 2.7). Yet, these and the other *amino acids* form chains of *molecular* bonds that ultimately create ***proteins.*** Add phosphorus (P) to the *molecular* mix and we form ***nucleic*** [*nucle(o)-* nucleus + *-ic* pertaining to] ***acids***. If you're interested in your ancestry, you may have taken a close look at a particular type of *nucleic acid*: ***DNA—Deoxyribonucleic*** [*deoxy-* less oxygen + *ribonucleic*] ***acid*** to discover your family origins. *DNA* provides basic blueprints for the various cells and tissues of the body, as well as providing genetic links to past and future generations. While portions of our DNA molecular structure may be similar, ultimately DNA is unique to the individual. My DNA sequence is different from yours. Still, DNA is nothing more than well-organized *molecular* chains. Fascinating isn't it that the basis for life as we know it all boils down to atoms and molecules?

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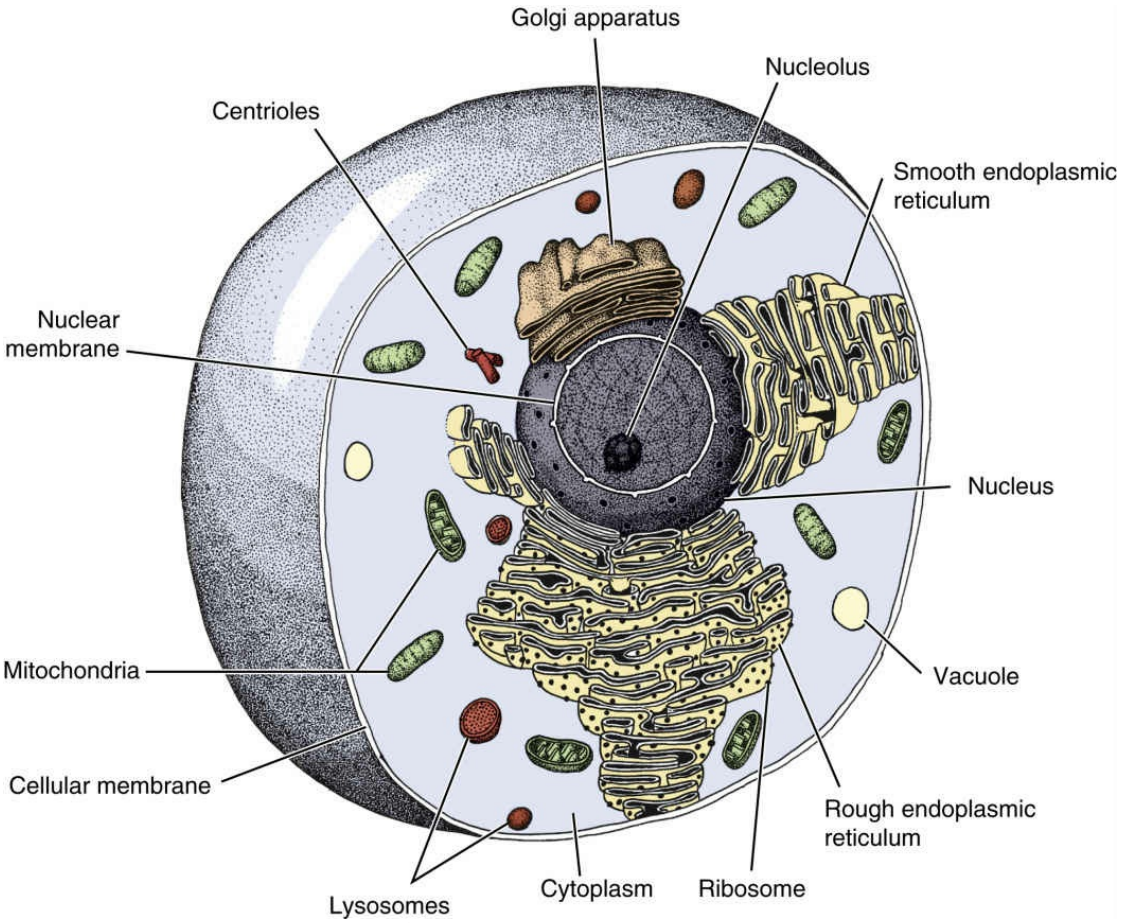

FIG. 2.8 Animal cell.

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Applied Cellular Terminology

In the previous section we discussed atoms, basic *subatomic* structure, *molecular bonds*, and a little *biochemistry* and *physiology*. We ended with a very brief discussion of amino and nucleic acids. We said that *amino acids* through ***intermolecular*** [*inter-* between + *molecul(o)-* molecule + *-ar* pertaining to] bonds formed proteins and DNA. Both are necessary to build cells. And that is the focus of this discussion: cells.

Just as atoms are the building blocks of matter, cells are the building blocks—the smallest functional units—of the body. Both are small and unable to be seen with the naked eye. Yet cells are just big enough that they can be seen through a ***microscope*** [*micro-* small + *scop(o)-* view]. Some ***intracellular*** [*intra-* within + *cellul(o)-* cell + *-ar* pertaining to] ***organelles*** [*organ(o)-* organ + *-elle* a small] are so very small that they can only be seen with ***electron microscopy*** [*micro-* small + *-scopy* to view]. An *electron microscope* uses a beam of electrons, rather than a traditional light source, to form an image of ultra-small *intracellular* structures. Fortunately, ***cytology*** [*cyt(o)-* cell + *-logy* study of] has provided us with a wealth of information regarding *cellular* anatomy (structure) and physiology (function). There are many different types of cells found in the body. For our discussion here, I will simply present a very basic, average cell. Specifics for various cell types and the tissues that they form will be left to subsequent chapters, where the information is most relevant.

Cellular Membrane

Let's begin by looking at the cell in Fig. 2.8, working our way from the outside to its interior. In your Evolve resources, there is an animated overview, entitled "Cells," to help with your understanding of cellular structure and function. Every cell has a ***cellular*** [*cellul(o)-* cell + *-ar* pertaining to] ***membrane*** that encases it. Think of it as a wall or skin that surrounds all of the ***intracellular*** [*intra-* inside, within + *cellul(o)-* cell + *-ar* pertaining to] contents. In fact, if we were to make a cellular analogy to a factory, the *cell membrane* would be the outer walls, roof, and floor. Unlike the rigid,

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impervious [*im-* not + *perve(o)-* passage, penetrate + *-ous* pertaining to] materials of the factory's bricks and mortar, the *cellular membrane* is made of proteins and ***lipids*** [lip(o)- fat]. The ***lipoprotein*** [*lip(o)-* fat + protein] molecules of the cell wall are *impervious* to certain molecules, like water (H_2O). As they say, oil and water don't mix. However, the *lipoproteins* of the cellular membrane are ***semipermeable*** [*semi-* partial + *perme(o)-* penetrate + *-able* possible, capable] to molecules of oxygen (O_2) and carbon dioxide (CO_2). So, like the factory, our cellular membrane has a ventilation system that provides for free exchange and turnover of these important gases. O_2 and CO_2 freely ***diffuse*** (movement across a *semipermeable membrane* from an area of high concentration to an area of low concentration) across the *cellular membrane*. This ***cellular respiration*** ("breathing") between *intracellular* and ***extracellular*** [*extra-* outside + *cellul(o)-* cell + *-ar* pertaining to] fluids is extremely important. ***Intracellular organelles*** [*organ(o)-* organ + *-elle* a small] need an abundant supply of oxygen to function. Likewise, the potentially ***toxic*** [*tox(o)-* poison + *-ic* pertaining to] waste product, carbon dioxide (CO_2), must be evacuated efficiently from the cell (and eventually from the body).

So O_2 and CO_2 can easily *diffuse* across the *cellular membrane*. What about water (H_2O)? We said earlier that water couldn't penetrate the *lipoproteins* in the *cellular membrane*. Fortunately, *cellular membranes* are porous enough to allow for ***osmosis*** [*osm(o)-* impulsion (movement of a fluid) + *-sis, -osis* process of] to occur. *Osmosis* could be thought of as a type of *diffusion*, because something is moving across a *semipermeable membrane*. But in this case, particles or ions are not moving, it's the water that's moving. In *osmosis*, water passes through the porous membrane to dilute a ***hypertonic*** [*hyper-* excessive + *ton(o)-* tension, concentration + *-ic* pertaining to] solution on the other side of the membrane. If it weren't for tiny gaps or pores (little factory windows) in the *cellular membrane*, water would not be able to penetrate the lipids (fats) in the membrane. Oil and water simply don't mix.

By the same token, water isn't simply sloshing from one side of the *cellular membrane* to the other. Forces are involved. The amount of water force needed to equalize solute (particle) concentrations

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(i.e., create an ***isotonic*** [*iso-* equal + *ton(o)-* tension + *-ic* pertaining to] state on both sides of the membrane) is what we call ***osmotic*** [*osm(o)-* impulsion + *-tic* pertaining to] ***pressure***. It's kind of the drawing or attraction force, like that of a sponge that sucks the water away from an area where there is an abundance of water (***hypotonic*** [*hypo-* less + *ton(o)-* tension, concentration + *-ic* pertaining to] solution) to the other side of the membrane where there are a lot of *solutes* (ions, particles) in a *hypertonic* solution, like a concentrated salt (NaCl) solution. In the cellular world, the goal is an *isotonic* state between *intracellular* and *extracellular* environments. Cells tend to dislike extremes. Fortunately, *osmosis* makes ***isotonicity*** [*iso-* equal + *ton(o)-* tension, concentration + *-city* state of] relatively easy. But cells cannot live on water and oxygen alone. What about fuel and energy?

Glucose (sugar) is the main fuel source for cells. *Intracellular organelles* need fuel to do their jobs. Unfortunately, glucose molecules are too big to pass through pores in the *cellular membrane*. Also, glucose molecules cannot freely diffuse through the membrane itself, like oxygen and carbon dioxide can. Glucose molecules require help through ***facilitated diffusion***. To use our factory analogy, we need a service door and conveyor belt to get these big fuel packages inside. Our cellular membranes have carrier proteins (gigantic molecules) that act as tiny conveyor belts to move *extracellular* glucose molecules into the cell. We'll talk about consumption of larger food sources (like bacteria—nice meal there) later because ***phagocytosis*** [*phag(o)-* eating + *cyt(o)-* cell + *-sis, -osis* process of] involves more than just the *cellular membrane*. We need a whole team of *organelles* for *phagocytosis*.

Can you believe it? We've only talked about the *cellular membrane* so far! Just think about all of the important things that the cellular membrane does. It is not simply a container for a cell's "guts." Remember that the next time you look through a *microscope*. In ***cytology*** [*cyt(o)-* cell + *-logy* study of] we tend to focus on the *intracellular* structures. After all, cellular membranes tend to be transparent and pretty boring. We see right through them into the cells. But without a *cellular membrane*, important cellular activities like diffusion, osmosis, and facilitated diffusion can't happen. Literally, without a cellular membrane a cell ceases to exist. So

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don't try to identify cells whose cellular membranes are no longer intact. And without cells we don't have a body because organized cells make tissues, organized tissues make organs and organ systems, and the collective of all of that makes the body. But that's enough philosophical talk; let's look at *intracellular* structures, shall we?

Intracellular Organelles

Cytoplasm [*cyt(o)-* cell + *plasm(o)-* matter] is the ***amorphous*** [*a-* non, not + *morph(o)-* shape + *-ous* pertaining to], generally colorless *intracellular* liquid that suspends all of the other *organelles*. *Cytoplasm* is the stuff that contains *intracellular ions*, providing for chemical reactions and ionic gradients that might promote *osmosis* or *diffusion*. So while it might not look like more than a puddle of liquid, *cytoplasm* is very important in its own way.

Next we have the ***endoplasmic*** [*endo-* within + *plasm(o)-* matter + *-ic* pertaining to] ***reticulum*** [*reticul(o)-* net, network + *-um* a]. This is a complex network, as the name implies, of canals and flattened sack-like structures. In our factory analogy this would be much like the halls, corridors, stairwells, and elevators throughout the building. In the same way that the hallways and such provide structured pathways for factory workers to move things throughout the factory, the *endoplasmic reticulum* provides pathways to transport things throughout the cell. And cells have two different types of *endoplasmic reticula* (*reticula* is the plural of *reticulum*). The ***smooth endoplasmic reticulum*** is purely a transport pathway. The ***rough endoplasmic reticulum***, however, is indirectly engaged in production, as well as the *intracellular* transport of those products. Why is it rough? The *rough endoplasmic reticulum* appears "rough" *microscopically* [*micr(o)-* small + *scop(o)-* view + *-ically* pertaining to] because it is speckled with ***ribosomes*** [*rib(o)-* RNA, ribonucleic acid + *som(o)-* body]. The *ribosomes* often pick up stains that we use in *cytology*, making them and the *rough endoplasmic reticulum* visible *microscopically*.

Ribosomes are the actual production units of the cell. Many factories have various divisions and units that produce different components for the company. In the *cellular* world, *ribosomes* would

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be analogous to the factory's production divisions and units. What do ribosomes produce? Primarily, they produce a wide variety of proteins. Once made, their products enter the *rough endoplasmic reticulum* for transport elsewhere in the cell. Most likely, ***ribosomal*** [*rib(o)-* RNA + *som(o)-* body + *-al* pertaining to] products and by-products will be transported to the ***Golgi apparatus***. In a factory setting, the *Golgi apparatus* could be likened to the packaging department. Of course, cells don't have boxes, bags, or shrink-wrap. However, the *Golgi apparatus* does create a nice, neat little ***vesicle*** [L. *vesicula* small bladder] to contain whatever the product or by-product might be. Those *vesicles* are then transported via the *endoplasmic reticulum* elsewhere in the cell for *intracellular* use. ***Lysosomes*** [*lys(o)-* dissolving + *som(o)-* body], *vesicles* filled with ***lysosomal*** [*lys(o)-* dissolving + *som(o)-* body + *-al* pertaining to] enzymes, are good examples of products kept for *intracellular* use. Of course, some protein-filled *vesicles* may be transported to the *cellular membrane* for ***exocytosis*** [*exo-* out + *cyt(o)-* cell + *-sis, -osis* process of]. Yes, even cells can "export" their goods. Mucus from a secretory cell would be a good example of that.

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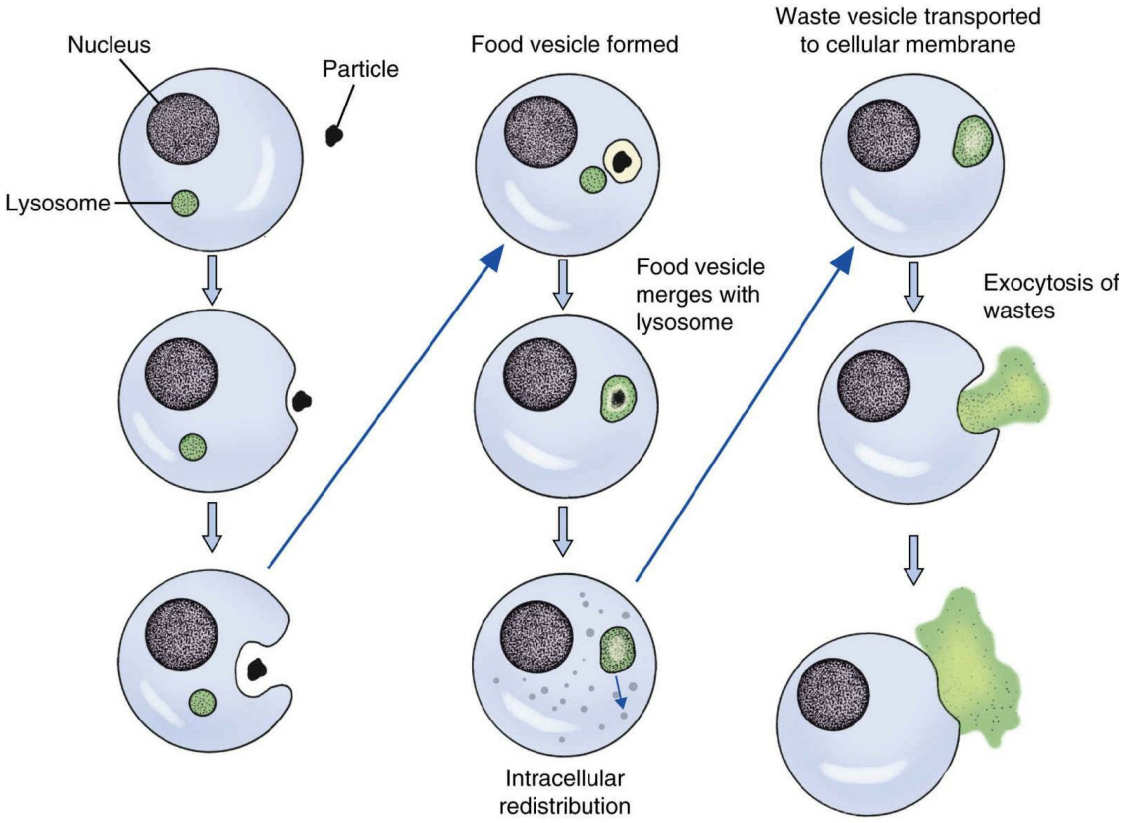

FIG. 2.9 Phagocytosis.

Phagocytosis

Okay, you're probably wondering why a cell would need *lysosomal* enzymes. What on earth would a cell need to dissolve or destroy? To answer that question we need to talk about ***phagocytosis*** [*phag(o)-* eat + *cyt(o)-* cell + *-sis, -osis* process of] (Fig. 2.9). Cells, especially different types of ***macrophages*** [*macro-* big + *phag(o)-* eat, eater, eating], can actually consume large particles and proteins left over from tissue damage, or even organisms, like bacteria. (Note: not all bacteria are bad. There are beneficial bacteria that naturally reside on the skin, along the airways, and in the digestive tract. Through wonderful ***symbiotic*** [*sym-* with + *bi(o)-* life + *-tic* pertaining to] relationships, these bacteria can serve to prevent colonization of ***pathogenic*** [*path(o)-* disease + *gen(o)-* production, producing + *-ic* pertaining to] bacteria.) *Macrophages* are the all-important custodians, sanitation workers, and security guards of the body. Even factories have custodians and perhaps even hazmat (i.e., hazardous material) teams. We'll talk specifically about the security guards in the ***hematology*** [*hemat(o)-* blood + *-logy* study of]

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chapter (Chapter 3).

As you can see in Fig. 2.9, this ultra-simplified version of a *macrophage* is presented with a large particle that needs to be removed from the body. As the *macrophage* approaches the particle, its *cellular membrane* begins to indent to surround the particle. Eventually, a "food" *vesicle* is formed. That *vesicle* merges with a *lysosome*, permitting the *lysosomal* enzymes to break down ("digest") the particle. Any usable by-products of this "digestion event" (e.g., simple sugars or amino acids) will be repackaged and redistributed as needed for use by the cell. These resource vesicles and waste vesicles may appear as ***vacuoles*** [*vacu(o)-* empty, emptiness + *-ole* a small] *microscopically* because they generally don't take up any *cytologic* stains. *Vacuoles* may give a "Swiss cheese" appearance to the *cytoplasm*. Waste vesicles don't remain as *cytoplasmic inclusions*. Eventually, they are transported to the cellular membrane and expelled from the cell via *exocytosis*. It's worth noting that with enough *macrophages* participating in *phagocytosis* in a given area of injury or disease in the body, their waste products in the ***intercellular*** [*inter-* between + *cellul(o)-* cell + *-ar* pertaining to] space can contribute to inflammation in the area. Remember this in subsequent chapters, especially Chapter 3: Applied Terminology for Blood, Lymphatics, and Immunity.

Do you see why my colleague said that Sadie was always very busy at the cellular level? Cells have a lot going on, even on a slow day! What on earth powers all of this *intracellular* activity? After all, factories need power, right? Well, most cells have their own little power generators—***mitochondria*** [*mit(o)-* thread + *chondri(o)-* granule; *mitochondria* is plural]. Microscopically, *mitochondria* are spherical or rod-shaped structures. Their internal structures appear rather thread-like, hence the name. A *mitochondrion* (singular) is one of the most unique and independent *intracellular organelles*. They actually have some of their own *mitochondrial* DNA, RNA, and *ribosomes*. What?! Yeah, most DNA and RNA are found only in the *nucleus*. We'll talk about that later. So, having their own DNA and such makes mitochondria pretty special and shows their importance. What do they do that's so important? Well, these little *mitochondrial* powerhouses don't produce electricity. Electricity is not very useful for the other *intracellular organelles*. No, *mitochondria*

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produce ATP (adenosine triphosphate [*tri-* three + phosphate]) to power *intracellular* activities. All *mitochondria* need are glucose and oxygen, and they will crank out all the ATP a cell could possibly need. By the way, principal by-products of ATP production are CO_2 and H_2O. I just love connections, don't you? You already know mechanisms that rid the cell of these. If you've forgotten, review the prior section of Electrolytes and Homeostasis.

Alright, we're in the final stretch of our *intracellular organelle* discussion. So let's finish up by discussing the ***nucleus*** [*nucle(o)-* nut + *-us* a]. What does a nut have to do with this? Nothing. Probably the first time someone looked at a cell *microscopically*, the scientist thought the *nucleus* resembled a nut. The root *nucle(o)-* comes from the Latin, meaning "nut." Well, nuts! In all seriousness, the *nucleus* may be last in our discussion, but it certainly is not the least. This important *organelle* holds all of a cell's (and our) important blueprints—our DNA. You see, when a cell is not actively reproducing, all of the DNA that makes up our ***chromosomes*** [*chrom(o)-* color + *som(o)-* body] unwinds into a loose ball of ***chromatin*** [*chromat(o)-* color + *-in* the "stuff"]. I know, this is sometimes difficult to grasp. Perhaps this will help you visualize *chromosomes* versus the *chromatin* of the *nucleus*. Think of a tightly knitted scarf as a well-organized *chromosome*. And if we pulled on the loose end of the scarf's yarn, unraveling it and wrapping it into a ball, that ball of yarn would be the loosely wrapped *chromatin*. When we stain *cytologic* preparations, *nuclear* [*nucle(o)-* nucleus + *-ar* pertaining to] *chromatin* accepts the stain readily. So *nuclear chromatin* is literally colorful stuff. The intensity of the staining properties of a given nucleus depends on how tightly or loosely the *chromatin* is wound in the nucleus. Tight, compressed *chromatin* stains dark. Loose *chromatin* stains light. This information will be helpful as you begin to study ***hematology*** [*hemat(o)-* blood + *-logy* study of]. But we'll save that for the next chapter.

Earlier in this section we spent a great deal of time discussing the *cellular membrane*. Well, there is a porous ***nuclear membrane*** that encapsulates the *nuclear chromatin*. The *nuclear membrane* provides separation between the *nuclear chromatin* and the *cytoplasm*. Yet its pores provide controlled passage of various substances between the *nucleus* and *cytoplasmic organelles*. Substances released from the

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nucleus are actually produced by the ***nucleolus*** [*nucleol(o)-* small nucleus + *-us*]. A *nucleolus* is a dense area within the *nucleus* that is made of protein and RNA (ribonucleic [*rib(o)-* ribose, ribosome + *nucle(o)-* nucleus + *-ic* pertaining to] acid). A *nucleus* may contain multiple nucleoli (nu-kle' o-li, plural of *nucleolus*). The primary functions of *nucleoli* are to produce *ribosomal subunits* [*sub-* under, almost + *unit*] and variations of RNA, such as messenger RNA (mRNA). Why would we need mRNA? Well, how else could a factory foreman in the office (*nucleolus*) get detailed instructions to the workers on the production line (*ribosomes*)? A "photocopy" or "pdf" (mRNA) of the instructions or a section of the needed blueprints is made in the office (*nucleolus*) and sent out through a small portal (*nuclear membrane pore*) to the production units (*ribosomes*). Once received, protein production can commence. So *nucleoli* are indirectly responsible for protein production. The more active an individual cell is the more *nucleoli* will be present in the *nucleus*.

Mitosis

There is probably no time of greater activity than immediately before or after cellular reproduction—***mitosis*** [*mit(o)-* thread + *-sis, -osis* a condition of]. Why thread? Particularly during the third stage of *mitosis*, *microscopically* the cell appears to have thread-like structures within it. Through *mitosis*, a cell will create an exact replica of itself. This is where DNA is so important. DNA is encoded ***genetic*** [*gen(o)-* produce + *-tic* pertaining to] information, and it is only found in the *nuclear chromatin*. DNA provides the "blueprint" so that every aspect of a cell can be replicated with precise and minute detail. On a much larger scale, DNA provides the blueprints for organization of cells and tissues into organ systems and the body as a whole. DNA is what dictates the final form of a being, be that a bug, bird, dog, cat, sheep, or human. We keep talking about RNA and DNA. Are you wondering what the basic difference is? Getting back to *molecular* structure, DNA is often referred to as a ***double helix*** [helix, he-liks', *Gr. snail, coil*]—because it has double, parallel strands of molecular chains. Those double molecular chains are the original blueprints for an individual. RNA

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is different because it has only a single strand of molecular chains. It is merely a copy of the original (Fig. 2.10).

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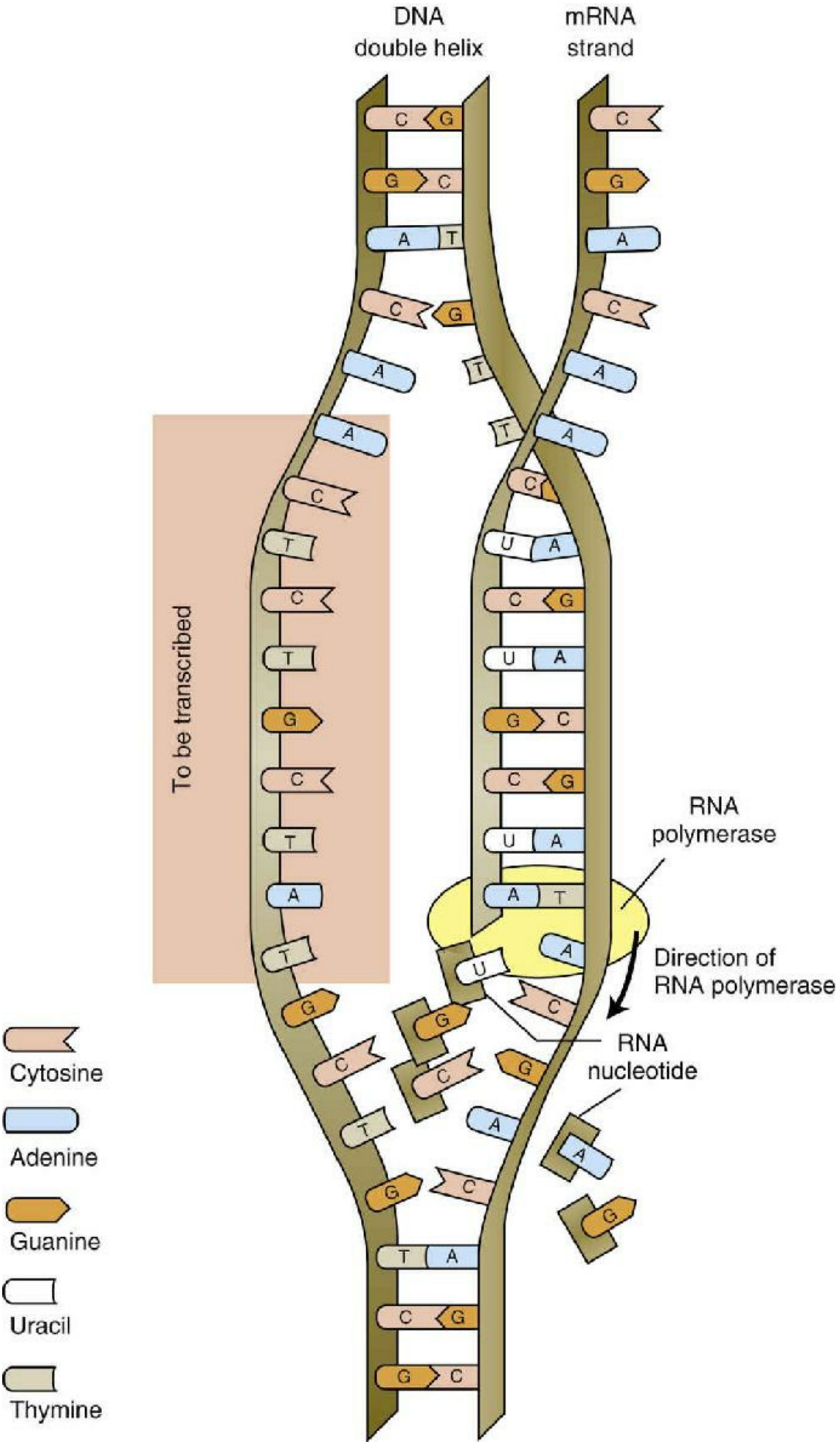
DNA
double helix
mRNA
strand
To be transcribed
RNA
polymerase
Direction of
RNA polymerase
RNA
nucleotide
Cytosine
Adenine
Guanine
Uracil
Thymine

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FIG. 2.10 Deoxyribonucleic acid *(DNA)* transcription of ribonucleic acid *(RNA)*. *mRNA*, Messenger RNA.
From Colville T, Bassert JM. *Clinical Anatomy and Physiology for Veterinary Technicians*. 3rd ed. St Louis, Elsevier Inc; 2016.

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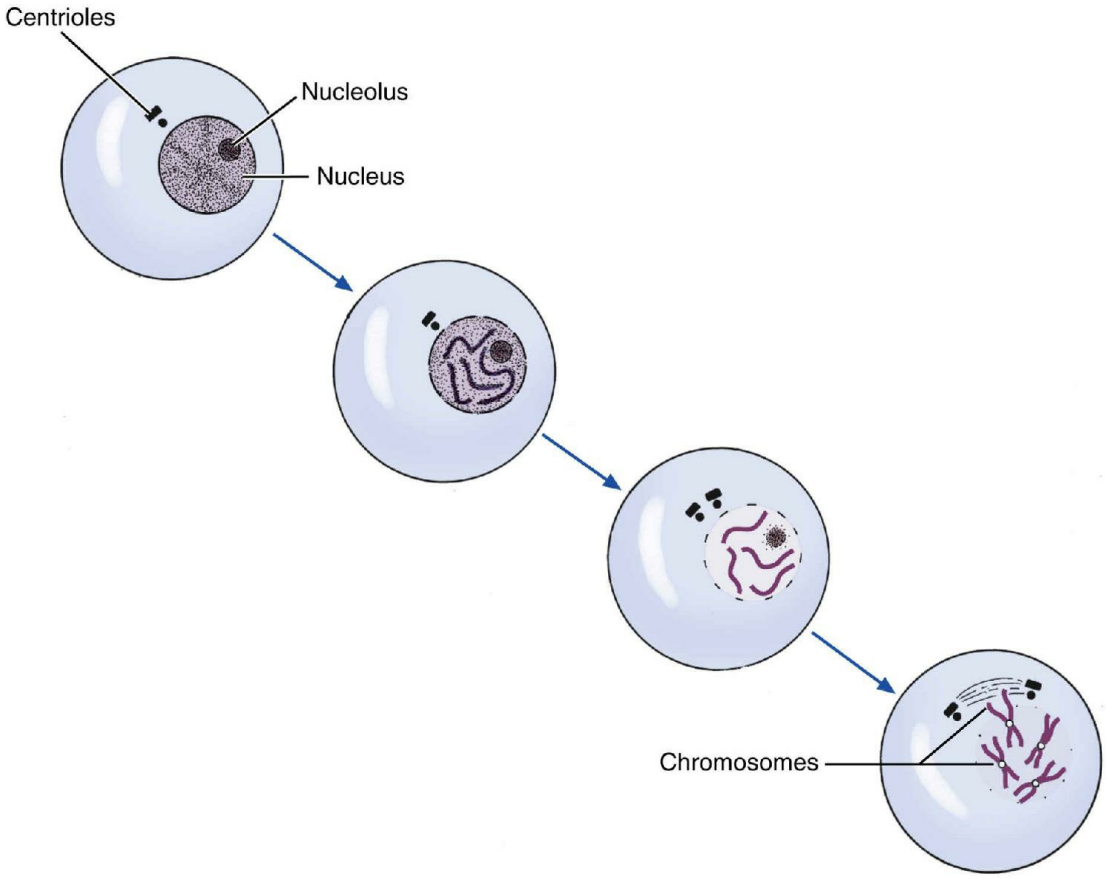

FIG. 2.11 Prophase of mitosis.

There are four principal phases or stages in *mitosis*. The first is the ***prophase*** [*pro-* before + *phase* stage] (Fig. 2.11). Much preliminary work must be done in this stage. The *nuclear membrane* must be disassembled. The *nuclear chromatin* needs to condense and organize into *chromosomes*. Ah, but it's not just a matter of tightly coiling the *chromatin*. No, leading up to this our *nucleolus* had to completely duplicate the molecular DNA chains. Then the original and the duplicate ***chromatids*** [*chromate(o)-* color + *-id* form, shape] are temporarily joined by a ***centromere*** [*centr(o)-* center, middle + *mer(o)-* part]. You guessed it. The *centromere* is the middle part of each *chromosome*. This creates the classic ***chromosomal*** [*chrom(o)-* color + *som(o)-* body + *-al* pertaining to] "X" shape you see in Fig. 2.11. While all of this is taking place the ***centrioles*** [*centr(o)-* center, middle + *-ole* a small] begin to migrate to opposite ends of the cell, forming thin, spindly fibers between them.

Beyond (pun intended) the *prophase* is the ***metaphase*** [*meta-* after, beyond + *phase* stage] (Fig. 2.12). During this brief stage, the *chromosomes* line up along the spindly fibers, midway between the

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centrioles. The *centromeres* of the *chromosomes* actually become attached to the spindle fibers. These *centromere* attachments will provide for the pulling apart of the *chromatids* in the next *mitotic* [*mit(o)-* thread + *-tic* pertaining to] stage.

During the ***anaphase*** [*ana-* back, up, again + *phase* stage] shown in Fig. 2.13, the *chromatids* separate—the original from the duplicate. Once separated, the *chromatids* are drawn by the attached spindle fiber toward their respective *centrioles*. The *centrioles* reel them in like a fishing pole or a kite on a string. By the way, it is primarily during this portion of the process that gives us that *microscopically* visible thread-like appearance and the term *mitosis*. Just remember that *mitosis* is the big umbrella under which all of these *mitotic* stages take place. Now it's not just the *chromatids* that are drawn to opposite sides of the cell. The rest of the *organelles* become divided too. And as the *chromatids* draw close to the *centrioles*, the *cellular membrane* begins to indent near the middle of the cell. This sets us up for the final stage.

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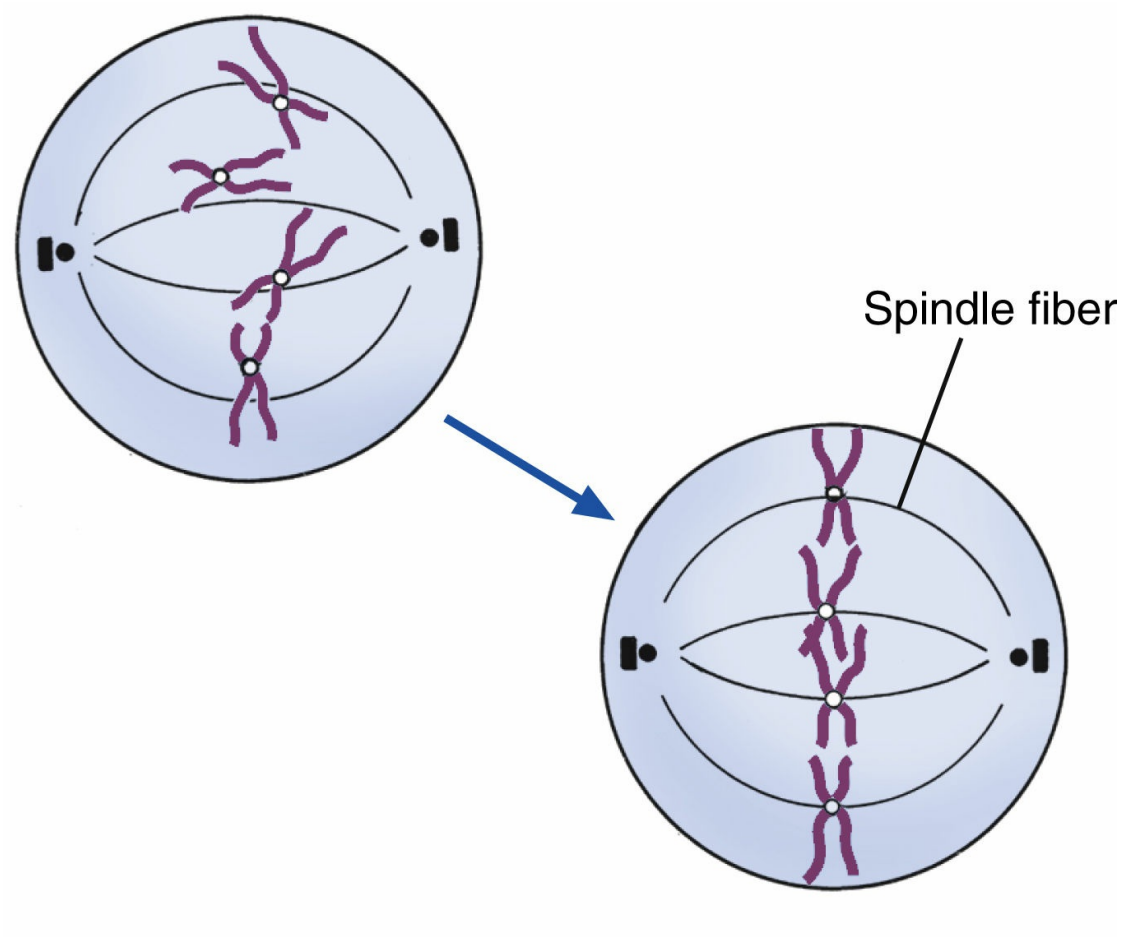

FIG. 2.12 Metaphase of mitosis.

By the time we reach the ***telophase*** [*telo-* end + *phase* stage] (Fig. 2.14), the *chromatids* have reached the *centrioles*. The *cellular membrane* continues to indent further and further, while all of the *cytoplasm* and *cytoplasmic organelles* are carefully divided. The tightly coiled material of the *chromosomes* unravels into loose *nuclear chromatin* and is surrounded again by a *nuclear membrane*. Eventually everything is divided and the *cellular membrane* separates the two new daughter cells—identical twins. A number of finishing touches still need to take place. Each cell will need more *ribosomes*. Protein production needs to increase briefly for rebuilding, restructuring, and development of a fully functional, mature cell. So, *microscopically*, we may see multiple *nucleoli* in the *nucleus* of each cell. As needs diminish, *nucleoli* will fade from sight. They will reappear shortly before and in preparation of the next *mitotic* event.

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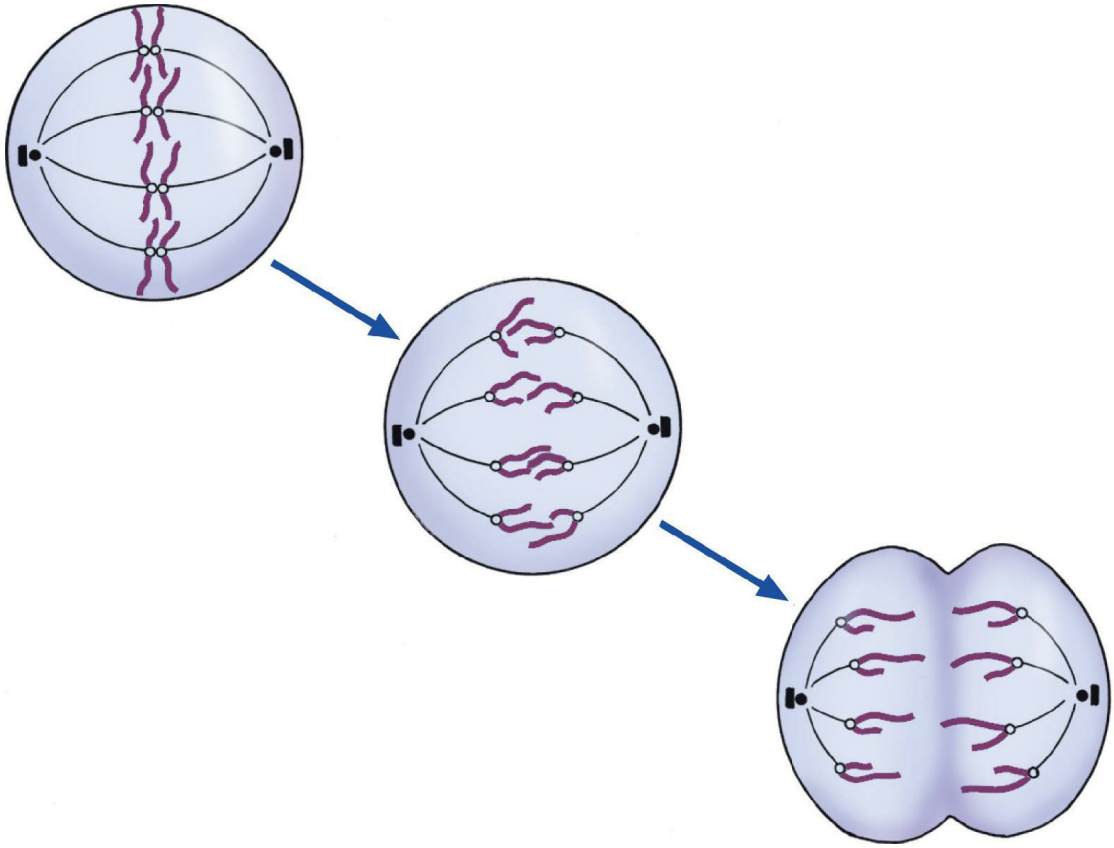

FIG. 2.13 Anaphase of mitosis.

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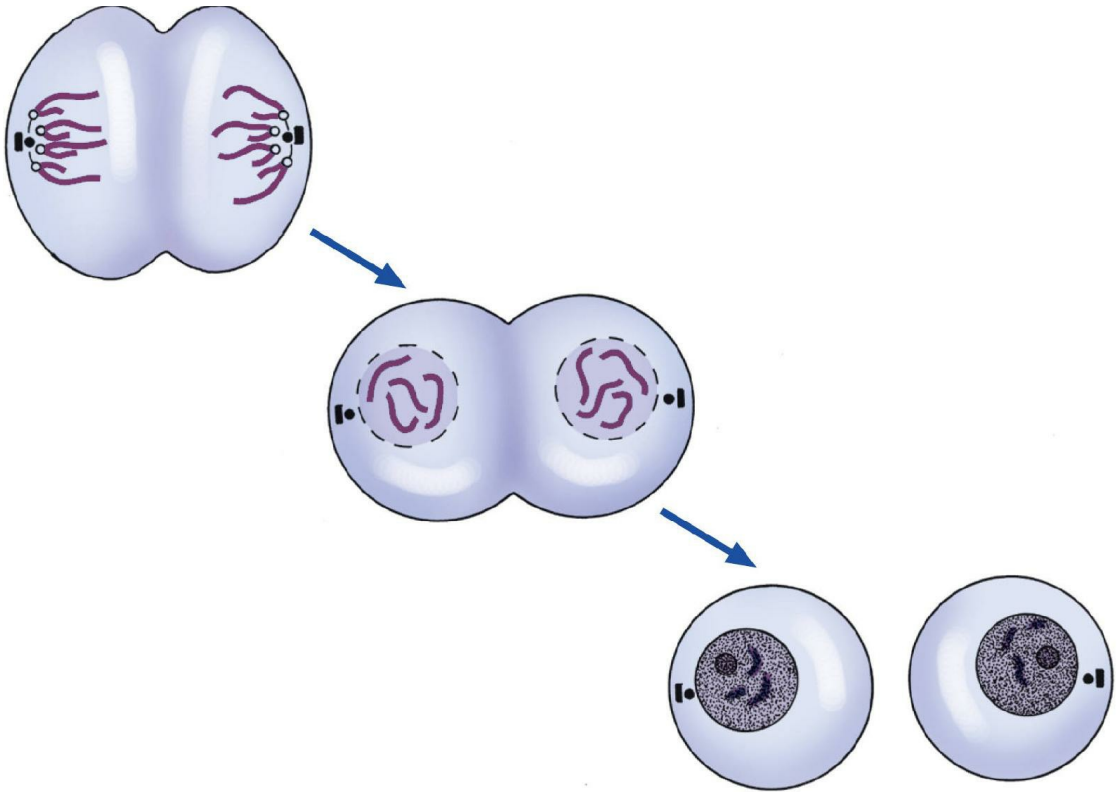

FIG. 2.14 Telophase of mitosis.

Mitosis is a process that repeats time and time again. Some cells undergo *mitosis* more frequently than others. ***Epithelial*** [*epi-* upon + *thele(o)-* nipple + *-al* pertaining to] ***cells*** are a good example of this. There are many different shapes and arrangements of ***epithelium*** [*epi-* upon + *thele(o)-* + *-um* a, the]. Yet, there is a common thread that ties them all together – *epithelial cells* line every surface of the body that is naturally exposed to the environment. So the skin, the interior linings of the airways and lungs, the interior linings of the digestive tract and the urinary bladder, to name a few, are all covered by *epithelial cells*. Now we will talk about these cells and *epithelial tissue* with each body system to which they are important. Suffice it to say that because of the routine loss and turnover of *epithelial cells* from these body surfaces, *mitosis* is a very important part of their existence. And anything that might compromise their ability to undergo *mitosis* could have a profound negative impact on the overall health and well-being of the body as a whole.

Case Study

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Since we've made numerous references to Sadie throughout this chapter, let's use a snapshot of her health history for this case study. Sadie was a chowhound. She loved food. Unfortunately, there were a number of times that she helped herself to things that were not good for her. If we did not latch the pantry door well, she would manage to open it, knock over the kitchen trash container, and consume some of the nasty garbage. Each time resulted in a trip to the emergency room.

Soon after eating the garbage, Sadie would begin to vomit. After a short time, her vomiting would diminish and she would develop profound diarrhea. I would take her in to the emergency room when she would begin to show signs of dehydration.

Most times on presentation in the emergency room, she was markedly dehydrated (estimated at 10% dehydration). She was hypotensive and pale. Based on her electrolyte panel, she was acidotic, hyponatremic, and hypokalemic. All of these factors made her very weak with little energy.

Treatment usually included rehydration with isotonic IV fluids. Antibiotics were also started to eliminate any pathogenic bacteria. Food and oral water were withheld. Electrolytes and other hematology values were monitored closely overnight to make sure that hypoglycemia, did not develop and to ensure that the acidosis, hyponatremia, and hypokalemia did not worsen.

Typically her condition greatly improved and she was discharged after 36 hours in ICU. After finishing her antibiotics, she would develop diarrhea again. It seems that the antibiotics eliminated both the pathogenic bacteria from the garbage and many beneficial bacteria along her digestive tract. Loss of the beneficial bacteria and the symbiotic relationship with them allowed pathogenic bacteria to flourish and cause diarrhea again. Yogurt was recommended to replenish the beneficial bacteria. Eventually homeostasis would be reestablished.

Case Study Questions

1. In the clinical findings for this patient, which medical term indicates that she had low blood pressure?

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_____

2. In the clinical findings for this patient, which medical term indicates that she had a condition of insufficient potassium in her blood?_____

3. Once admitted to the hospital, the patient was closely monitored. Much laboratory data was repeatedly collected as part of this monitoring. The doctor was concerned that she could develop low blood sugar. Which medical term is indicative of this condition?

_____

4. Physical monitoring was used to determine the patient's relative status with regard to water. The goal of fluid therapy was to restore the body to a normal state of water. What is the medical term for this state?

_____

5. The fluids used to restore adequate water and electrolytes were a balanced solution, equal to that of normal body fluids. Which medical term indicates that the fluids were equal with regard to osmotic pressure to bodily fluids?_____

6. If the IV fluids contained excessive amounts of electrolytes compared to body fluids, they would be called _____ solutions.

7. After full recovery the balanced, stable state of the body was finally restored. What is the medical term for this balanced, stable state?_____

8. The medical term that indicates a state of insufficient water in this patient is_____.

9. The medical term that indicates this patient had a blood condition of insufficient sodium is _____.

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10. The medical term that indicates this patient had condition of abundant of H^+ or H_2CO_3 is_____.

11. In solution, ions, bases, and salts are collectively referred to as _____.

12. Beneficial bacteria of this patient's digestive tract had a _____ relationship with her body, because they lived in harmony with her.

13. If Sadie had continued to vomit rather than develop diarrhea, she may have lost excessive amounts of H^+ resulting in _____ (i.e., a condition with too much HCO_3^-, a base salt ion).

14. Sadie consumed large numbers of disease-producing or _____ bacteria from the garbage, resulting in profound clinical disease that required hospitalization.

15. Antibiotics were needed to treat Sadie because the large numbers of disease-producing bacteria would have overwhelmed her cells, making _____ or cellular eating of the bacteria inadequate to rid them from her body.

The Answer Key to these case study questions may be found in Appendix B.

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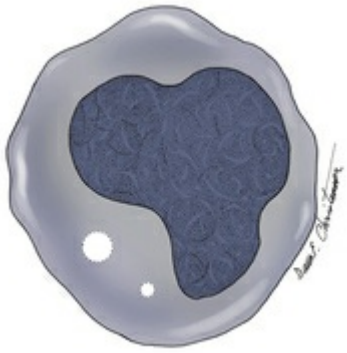

Applied Terminology for Blood, Lymphatics, and Immunity

Applied Whole Blood and Laboratory Terminology,
- Plasma Characteristics,
- Packed Cell Volume,
- Total Solids,
- Buffy Coat,

Applied Red Blood Cell Terminology,
- Erythropoiesis,
- Morphology,
  - Poikilocytosis,
- Hemoglobin,
  - Carbon Monoxide Toxicity,
  - Carbon Dioxide,

Applied White Blood Cell Terminology,
- Leukopoiesis,
- Granulocytes,
  - Neutrophil,

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GOALS AND OBJECTIVES

By the conclusion of this chapter, the student will be able to:

1. Recognize common root words, prefixes, and suffixes related to blood, lymphatics, and immunity.
2. Divide simple and compound words into their respective word parts.
3. Recognize, correctly pronounce, and appropriately use common medical terms related to blood, lymphatics, and immunity.
4. Demonstrate an understanding of the composition of blood.
5. Demonstrate an understanding of the anatomy of the lymphatic system.
6. Demonstrate a basic understanding of blood cell production.
7. Demonstrate a basic understanding of the function of cell types found in whole blood.
8. Demonstrate a basic understanding of the clotting process.
9. Demonstrate a basic understanding of the lymphatic system, as it relates to immunity in health and disease.
10. Demonstrate a basic understanding of structures and mechanisms contributing to edema (swelling).
11. Demonstrate a basic understanding of processes of inflammation and fever.
12. Demonstrate a basic understanding of clinical hematology, including determination of packed cell volume (PCV), total solids (TS), and recognition of mature red blood cells (erythrocytes); common red blood cell size, shape, and color changes; white blood cells (leukocytes); and platelets (thrombocytes).

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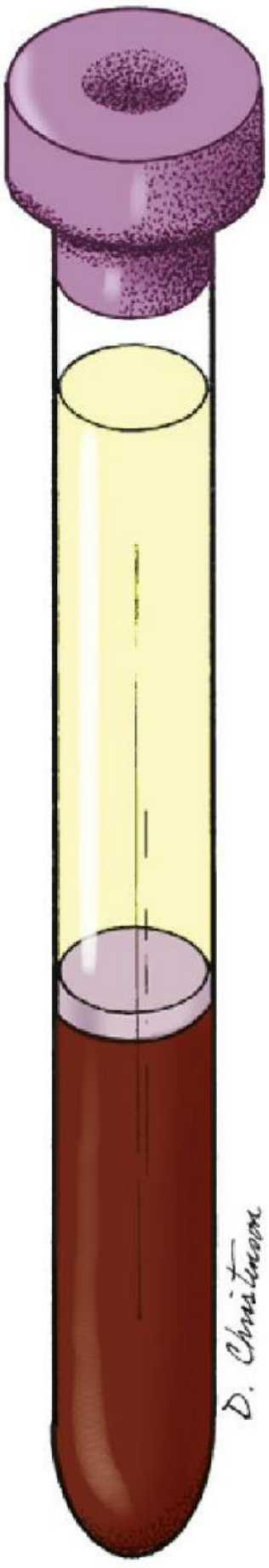

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FIG. 3.1 Whole blood in EDTA (ethylenediaminetetraacetic acid).

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Applied Whole Blood and Laboratory Terminology

We've probably all seen blood, whether our own, someone else's, or the fake stuff of TV shows and movies. Ah, but have you ever thought about what that bodily red liquid really is or what it contains? There is way more to blood than meets the eye. Yes, grossly it is red (no, not gross as in "eeuw, gross!" but gross as in "seen with the naked eye"). Yet not all of it is red. Only ***erythrocytes*** [*erythr(o)-* red + *cyt(o)-* cell; i.e., red blood cells] are red. Other cells and constituents of whole blood are not. Even if we choose to focus exclusively on *erythrocytes*, how do we know if we have enough of them or if they are healthy? We need laboratory analysis to determine that and much more.

Hematology [*hemat(o)-* blood + *-logy* study of] offers an important glimpse into the overall health and well-being of patients. Before we get into the nitty-gritty of blood cells themselves, let's begin with some very basic clinical and laboratory information. First and foremost, we need to know from where to collect blood samples. That information will be found in the ***cardiovascular*** [*cardi(o)-* heart + *vacul(o)-* vessel + *-ar* pertaining to] information in Chapter 5. For our purposes here, we need to know what type of sample is needed. If we want whole blood, for the evaluation of plasma (the unaltered liquid part of blood) and the various cells, we will need the sample to be collected in a tube containing ***anticoagulant*** [*anti-* against + *coagul(o)-* clotting + *-ant* one that is]. The most common *anticoagulant* for whole blood specimens is EDTA (**E**thylene**D**iamine**T**etraacetic **A**cid). Collection tubes containing EDTA have a lavender stopper (Fig. 3.1). There is just the right amount of EDTA in the collection tube to keep the blood from clotting. Of course, as soon as the blood is collected in the EDTA tube, it must be mixed thoroughly with the *anticoagulant* using a gentle rocking motion. This mixing process is important no matter which *anticoagulant* is used. Failure to mix the whole blood with the *anticoagulant* will result in partial or complete ***coagulation*** [*coagul(o)-* clotting + *-tion* process of] of the sample, rendering it

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useless for analysis of cells and plasma. Mixing too vigorously may damage cells, especially red blood cells. *Erythrocytes* are quite fragile. So, rough handling of the sample may cause ***hemolysis*** [*hem(o)-* blood + *-lysis* process of breaking]. A ***hemolyzed*** [*hem(o)-* blood + *-lyzed* broken] sample may interfere with both analysis and interpretation. Remember, regardless of the *anticoagulant* used, handle blood samples gently. Other commonly used *anticoagulants* in *hematology* are heparin (green stopper) and sodium citrate (light blue stopper).

What if we collect our blood sample in a tube that doesn't contain any *anticoagulant*, like a red-stoppered ("clot") tube? Well, after it has been left undisturbed for a period of time (~5 to 10 minutes for 2 to 3 mL tubes), the sample will clot. The clotted sample can then be placed in a ***centrifuge*** [*centr(o)-* center + *-fuge* a driving away from] to separate the solid clot from the liquid ***serum***. Such a sample would be used for ***serology*** [*ser(o)-* serum + *-logy* study of]. *Serology* is a very broad area of laboratory testing that involves chemical analysis of the *serum* for things like proteins, glucose, electrolytes, and antibodies, as well as liver and kidney function. The one protein that cannot be measured from serum is ***fibrinogen*** [*fibrin(o)-* fiber + *gen(o)-* producer]. In the *coagulation* process that we'll talk more about later, *fibrinogen* is converted to fibrin, forming a clot. So, if we want to measure *fibrinogen*, we'll need ***plasma***. Plasma requires an *anticoagulant* and contains *fibrinogen*, while serum (the liquid left after clotting) does not. This is why many commercial benchtop tests call for either plasma or serum. The liquids are very much the same, with the exception of *fibrinogen*.

Let's say that we've collected our whole blood specimen in an EDTA tube and (gently) thoroughly mixed it. Now, we can begin the process of evaluating the specimen. One of the first things we need to know is the relative percentage of *erythrocytes* to plasma and other cells. For this ***packed cell volume*** (**PCV**) determination we will need a *centrifuge*, ***hematocrit*** [*hemat(o)-* blood + *crit(o)-* separation] tubes, and a special clay for plugging one end of each *hematocrit tube*. The filled and plugged *hematocrit* tubes are placed and secured in the *centrifuge*. When the *centrifuge* is turned on, it spins the *hematocrit* tubes at an extremely high rate of speed (i.e., RPMs, revolutions per minute). The ***centrifugal*** [*centr(o)-* central +

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fug(o)- to drive away + *-al* pertaining to] force exerted on the blood in the *hematocrit* tubes drives and separates the heavier blood components from the rest. *Centrifugal force* is basically artificial gravitational force. If you've ever ridden one of those fast-spinning rides at an amusement park or the county fair, you have experienced *centrifugal* forces. Unlike the amusement park ride, our centrifuge places our whole blood under extreme forces that drive the heaviest particles away from the center of the centrifuge. The heaviest particles (*erythrocytes*) are driven away to the clay-plugged end of the *hematocrit tube* (Fig. 3.2). ***Leukocytes*** [*leuk(o)-* white + *cyt(o)-* cells; i.e., white blood cells] and ***thrombocytes*** [*thromb(o)-* clot + *cyt(o)-* cells; i.e., clotting cells or platelets] are a bit lighter and settle in the middle of the *hematocrit tube*, atop the *erythrocytes* in what we call the "***buffy coat***." And the ***plasma***, being lighter than any of the cells, fills the remainder of the *hematocrit tube*.

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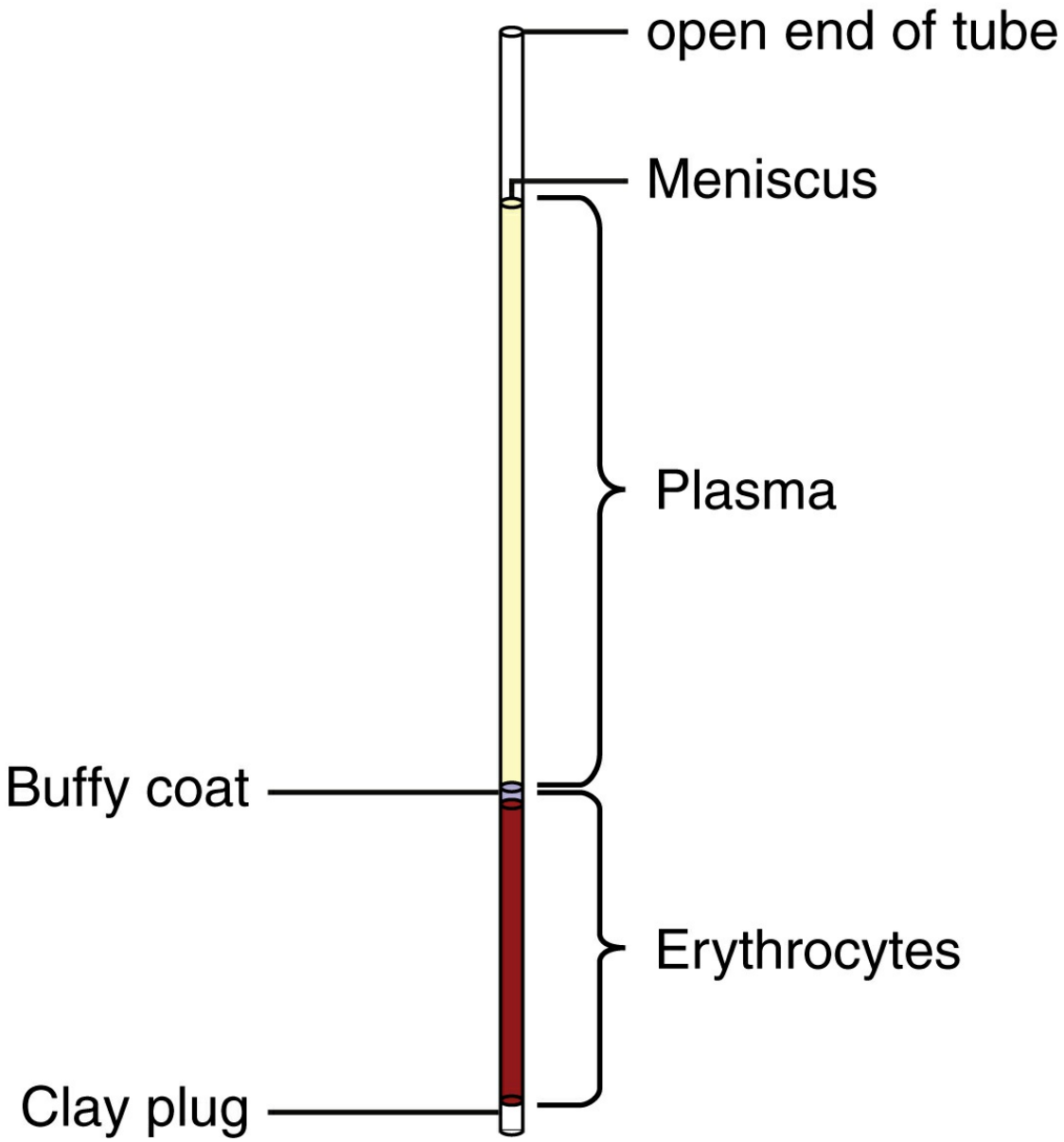

FIG. 3.2 Spun hematocrit tube.

Plasma Characteristics

Before we even begin to measure the packed *erythrocytes*, we can make note of any gross plasma characteristics observed. Plasma is the fluid portion of whole blood that contains dissolved substances like *electrolytes* (e.g., ions and salts; refer to Chapter 2 for more information), important proteins (like clotting factors and antibodies), and other substances. Normal plasma for most of our domestic animals is a transparent, colorless to lightly straw-colored liquid. ***Lipids*** [*lip(o)-* fat + *-id* a/an] suspended in the plasma give it a whitish coloration. ***Lipemia*** [*lip(o)-* fat + *-emia* a blood condition]

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may be a normal transient situation, following a meal. This is usually mild in appearance, with only white *lipids* appearing at the meniscus (i.e., the very top curved edge) of the plasma column, or it may make all the plasma appear ever so slightly cloudy. There are some ***pathologic*** [*path(o)-* disease + *log(o)-* knowledge + *-ic* pertaining to] conditions, like diabetes mellitus, that can create marked *lipemia*, making the plasma appear opaque white, like whole milk. Only further testing and patient evaluation will determine the cause of the ***lipemic*** [*lip(o)-* fat + *em(o)-* blood + *-ic* pertaining to] plasma. Our job in the laboratory is to simply report it, if we observe it.

Now, horses (sometimes cattle and other livestock) often have somewhat yellowish colored plasma. The yellowish colored plasma for them is normal and probably related to dietary pigments. Yellowish colored plasma in anyone else is abnormal and would be reported as ***icteric*** [*icter(o)-* jaundice + *-ic* pertaining to]. *Icteric* plasma is evidence of ***bilirubinemia*** [bilirubin + *-emia* a blood condition]. ***Bilirubin*** is a by-product of the breakdown of ***hemoglobin*** [*hem(o)-* blood + *glob(o)-* glob, stuff + *-in* the], the red-colored, iron-containing protein of *erythrocytes* that is important for transporting oxygen in the body. The liver is responsible for removing *bilirubin*, the yellow-colored waste product from the plasma, recycling it for the creation of bile. We'll discuss this in the digestive chapter (Chapter 7). If we need bile for digestion, then we must always have small amounts of bilirubin in the bloodstream, right? Right. So, when we think of ***icterus*** [*icter(o)-* jaundice + *-us* the], it is probably more accurate to say that ***hyperbilirubinemia*** [*hyper-* excess + *bilirubin* + *-emia* a blood condition] exists. It takes excessive amounts of bilirubin to create grossly visible yellowing of the plasma, as well as mucous membranes (e.g., gums) and skin. If *hyperbilirubinemia* develops to create *icteric* plasma, one of three things is probably happening: (1) there is excess destruction of *erythrocytes* and thereby excess release of *hemoglobin*, creating more *bilirubin* than the liver can handle; (2) the liver is diseased and cannot remove even normal amounts of *bilirubin*; or (3) something is slowing or preventing the flow of bile from the liver, creating a backup and buildup of *bilirubin* in the plasma. Only further laboratory testing and patient evaluation will help us determine the

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cause of the ***icterus***. Our job in the laboratory is to simply report the *icteric plasma*, if we observe it.

Hemolytic [*hem(o)-* blood + *-lytic* pertaining to breakage] plasma may also be seen in our spun *hematocrit* tube. This gives the plasma a pink to reddish coloration. Could this be from our collection and handling of the blood sample? You bet! *Hemolysis* can also be caused by *pathologic* conditions. Diseases that result in *hemolysis* are very serious and may even be deadly. So, if we observe *hemolytic* plasma, we need to review our handling of the sample. Collecting another sample, taking care each step along the way to avoid ***hemolyzing*** [*hem(o)-* blood + *-lyzing* actively breaking] the sample, may be the safe play. If after careful collection and handling the plasma is still *hemolytic*, we must report it. If the subsequent sample is not *hemolytic*, we know that it was an artifact in the previous sample (probably from poor handling). As with *lipemia* and *icterus*, our job in the laboratory is to report *hemolytic* plasma. However, because we may artificially *hemolyze* the sample, we must problem-solve any *hemolytic* sample and do everything in our power to ensure the integrity of the sample. We also need to make sure, from the get-go, that we always collect and handle blood samples with care. Yes, we can often collect another sample, if the first one is *hemolyzed*. However, we should never put our patients through additional stress and trauma unnecessarily. Get my drift? Good.

Packed Cell Volume

Okay, so we've grossly evaluated the plasma in our *hematocrit tubes* and recorded our findings. Now we can measure the **PCV** (**packed cell volume**). The *packed cell volume* is a relative measurement of packed *erythrocytes* to the total volume in the spun *hematocrit tube*. To determine the PCV, we will need a *hematocrit reader*—a card with a bunch of angled lines and percentages associated with them (Fig. 3.3). The lines are angled to accommodate variable total volumes in our hematocrit tubes. (Please note that the hematocrit reader shown in Fig. 3.3 does not show all of the fine 1% lines in between each of the 10% lines shown.) In order to determine the PCV of our sample, we must lay the spun hematocrit tube on the reader in a very specific way. The bottom of the *erythrocyte* column (touching the

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clay) is placed on the "0" line, and the plasma meniscus (lowest point of its arch) is placed on the 100% line. Wherever the top of the *erythrocyte* column falls is our PCV. In this example, the PCV is 40%. The PCV is always reported as a percentage, because the packed erythrocytes are only a portion (percentage) of the total (100%) volume in the hematocrit tube.

Many factors can influence the PCV. ***Dehydration*** [*de-* away from, reduced + *hydr(o)-* water + *-tion* a condition/state of], for example, will tend to increase the PCV reading. In fact, dehydration could artificially make it appear that a patient has ***polycythemia*** [*poly-* many + *cyt(o)-* cell + *hem(o)-* blood + *-ia* condition of; i.e., excess numbers of red blood cells, or RBCs]. This is a good example of why we never rely on a single piece of clinical data to make a diagnosis. It goes without saying that ***anemia*** [*an-* without + *em(o)-* blood + *-ia* a condition of], because of overall reduced numbers of *erythrocytes* in the blood, will lower the PCV. Yet, I have seen *dehydrated* patients who **appeared** to have normal PCVs, due to *dehydration*. After replenishing the patients' fluid deficits, their true ***anemic*** [*an-* without + *em(o)-* blood + *-ic* pertaining to] conditions were revealed. Had we not rechecked the PCV, we would have missed the *anemia*. Believe me, I have seen some very *anemic* patients through the years. I recall a puppy with hookworms (an intestinal parasite that sucks blood) who had a PCV of only 12% (normal average for dogs is ~45%). I also recall an adult cat, with a horrific flea infestation, who had a PCV of only 8% (normal average for cats is ~38%). Without treatment, both of these patients would have died from their blood-loss *anemias*. The good news is, both patients responded well to life-saving blood transfusions and elimination of their parasites.

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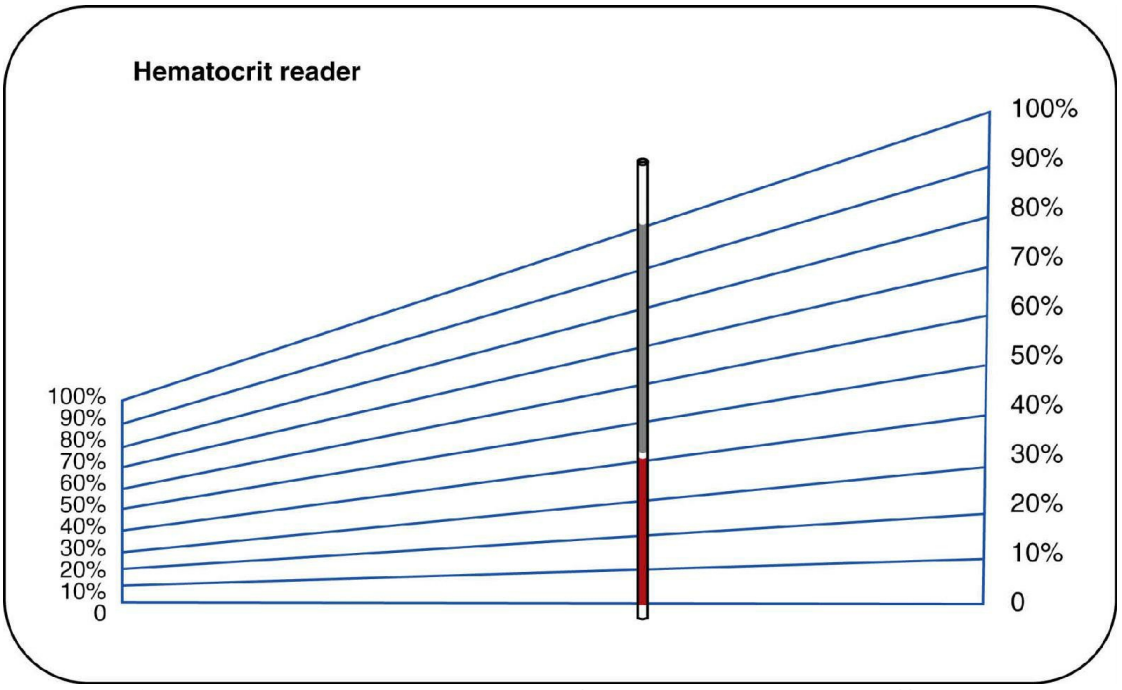

FIG. 3.3 Spun hematocrit reading (packed cell volume 40%).

Total Solids

Let's see, so far with our whole blood sample (using EDTA as the *anticoagulant*), we've spun our *hematocrit tubes* in a *centrifuge*, grossly evaluated the *plasma*, and measured the PCV. About the only thing left to do with our spun hematocrit tubes is to measure the **total solids** (**TS**) in the plasma. Wait, what? Isn't plasma a liquid? Yes. When we began this section, we said that plasma was the liquid portion of whole blood. It is a solution, containing varying concentrations of proteins, electrolytes, and other dissolved particles. Using a ***refractometer*** [*refract(o)*- breaking/bending + *meter* measure] we can roughly measure the concentration of the TS (particles) in the solution (plasma). It does this by refracting (bending) the light through a prism. The greater the concentration of dissolved particles (solids), the more light is bent, and the higher the number on the refractometer's scale. Notice that I did not say we are measuring the **total protein** (**TP**) using the refractometer. There are other particles, in addition to protein, in plasma. So, to say "total solids" is more accurate. TP, including specific concentrations of albumin, globulins, and fibrinogen, requires more intricate laboratory testing, well beyond the capabilities of the refractometer. So, in spite of the fact that the refractometer scale

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(Fig. 3.4) we use to determine TS is labelled “protein,” remember that the instrument is actually measuring all “solids” in solution.

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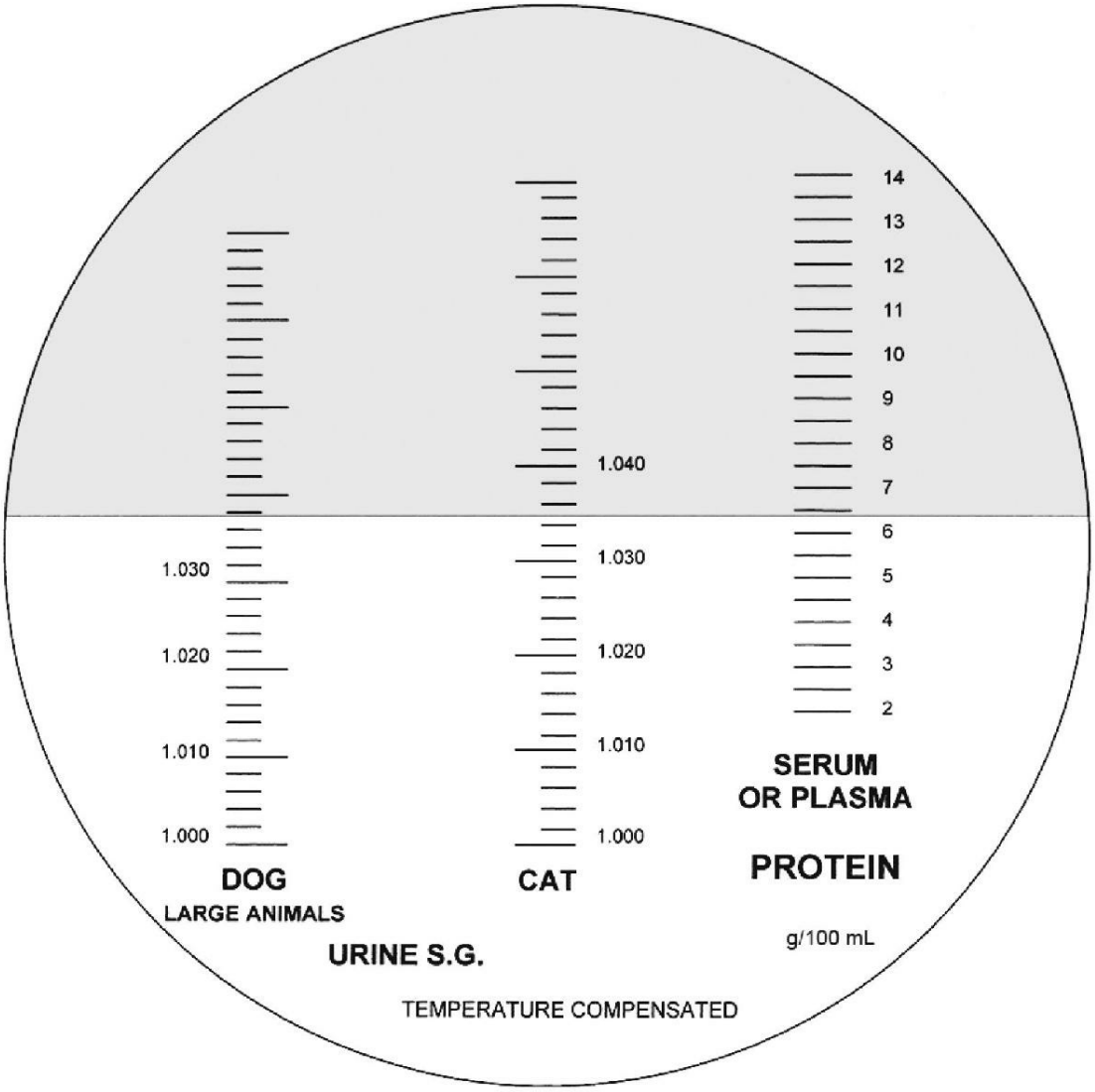

FIG. 3.4 Total solids (TS) determination with a refractometer (TS 6.4 g/dL).

To determine **total solids** (**TS**), we will use one of our spun *hematocrit tubes*. We will break the tube just above the *buffy coat* to access nothing but the plasma. With the lid of the *refractometer* covering the glass prism, we'll press the open end of the broken hematocrit tube in the little notch in the lid. (DO NOT tap the hematocrit tube directly on the glass! You'll ruin the prism!) The plasma will flow under the lid. Then, shining a light source through the lid and plasma, we will see a defined line of demarcation between gray and white fields on the scale (see Fig. 3.4). Where that line of demarcation falls on the "serum or plasma protein" scale (far right) is the TS measurement. It is reported in g/dL (grams per ***deciliter*** [*deci-* one tenth + *liter*]. Notice in the example shown that at the very bottom of the far-right scale it states "g/100 mL". Let's see,

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if there are 1000 mL (mL = ***milliliter*** [*milli-* one thousandth + *liter*]) in a liter, then 100 mL is one tenth of a liter. So, 100 mL is a *deciliter* (dL). And in Fig. 3.4 we find that our TS measurement for this sample is 6.4 g/dL. For most of our domestic animals, 6.0 to 7.0 g/dL is a ballpark average for TS. Now, think back to our discussions of *lipemia* and *hemolysis*. These two plasma abnormalities can really interfere with our TS measurements. The *lipids* in *lipemic* samples refract much differently than other particles. Not only will lipids alter the TS measurement, they also tend to blur the line of demarcation in the *refractometer*. In a markedly *lipemic* sample, the lipids can make it very difficult if not impossible to even read the *refractometer*. With all of the free *hemoglobin* (a protein) found in *hemolytic* plasma, TS readings can be dramatically elevated. This is why our gross plasma evaluation is important—it provides guidance to the veterinarian for the interpretation of the measured values.

Buffy Coat

Finally, you may be wondering if we ever do anything with the buffy coat in our hematocrit tubes. Yes, but not routinely. Don't get me wrong, I always look at the buffy coat grossly. If it ever appears abnormal in any way, I will make note of it and probably bring it to the attention of the veterinarian. If the buffy coat is especially large, I will measure it using the hematocrit reader. In my experience, a normal buffy coat is usually ≤ 1%. I have measured some much greater than that. I think the largest I've measured was 7%. In that particular case, we quickly changed the rest of the submitted blood work from "routine" to "STAT." We wanted fast results for cell counts and cellular ***morphology*** [*morph(o)-* shape + *-logy* study of] to initiate appropriate treatment ASAP. The additional ***hematologic*** [*hemat(o)-* blood + *log(o)-* knowledge/study + *-ic* pertaining to] testing provided important information to guide our care of the patient. As it turned out, that patient had a major infection causing a marked increase in numbers of certain *leukocytes* in the blood. I've seen other buffy coats that were extremely small or absent too. Those patients had too few *leukocytes* and needed to be protected from exposure to ***pathogens*** [*path(o)-* disease + *gen(o)-* producers],

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even normal bacteria on our skin. Again, additional *hematology* was needed to guide patient care. You see, the preliminary blood evaluations that we've discussed here (gross plasma characteristics, PCV, TS, and perhaps buffy coat) are useful but only to a point. They barely scratch the surface of information available in *hematology* and fall short in helping us determine the health status in any patient. We need more information to be sure. That is why we will be spending a great deal of time, in the next sections, looking closely at each cell type and much, much more. If you would like a quick 'preview of coming attractions', please look at the animation: "Types of Blood Cells", in the Evolve resources. Now, let's dig deeper, shall we?

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Applied Red Blood Cell Terminology

Erythrocytes (red blood cells, RBCs) are so very important. Without them, we cannot transport oxygen throughout the body. And without oxygen, all the other cells, tissues, and organs (aw heck—the whole body) cannot survive. *Hemoglobin*, as mentioned earlier, is the iron-containing ***erythrocytic*** [*erythr(o)-* red + *cyt(o)-* cell + *-ic* pertaining to] protein that gives RBCs their red coloration and that provides the molecular vehicle for transporting oxygen. We'll talk more about *hemoglobin* later in this section. For now, suffice it to say that anything that adversely alters *erythrocyte* numbers, structure, and/or *hemoglobin* content will adversely affect bodywide oxygenation. Before we look at these little, oxygen-carrying red "wagons" up-close and personal, let's take a brief look at where and how they are produced.

Erythropoiesis

Erythropoiesis [*erythr(o)-* red + *poie(o)-* production + *-sis* the process of] is the production of red blood cells. This takes place in the bone marrow. Now, you might think: "out of sight, out of mind—no point in knowing this." But that would be a very wrong conclusion. Do you remember the *anemic* patients that I mentioned in the previous section? We discovered that their blood-sucking parasites caused significant blood loss and resultant *anemia*. But what if they didn't have blood loss from parasites or ***hemorrhage*** [*hem(o)-* blood + *-rrhage* escaping; i.e., bleeding]? There are only two other causes of *anemia*: (1) destruction (*hemolysis*) somewhere in the body or (2) decreased production. Understanding *erythropoiesis* and the maturation of *erythrocytes* enables us to look for clues, through *hematology*, physical examination, and other diagnostic testing, to know whether or not a patient's *anemia* is treatable or not. If treatable, our comprehensive knowledge and understanding are essential for restoring the health of our patients. Enough justification—let's get down to the nitty-gritty.

As I said, *erythropoiesis* takes place in the bone marrow. The bone marrow responds primarily to a hormone, produced by the

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kidneys, called (wait for it….) ***erythropoietin*** [*erythr(o)-* red + *poie(o)-* producer + *-tin* the]. Do you see how intricate and interdependent body systems are? Who would think that decreased *erythrocyte* production could be linked to kidney disease? I know, when I was a student, I never imagined such a possibility. Yet, as I learned and as you now know, there is a direct link between the kidneys and RBC production—and that link is the hormone *erythropoietin*. Understanding what happens in response to that hormone requires a little refresher in cellular reproduction or *mitosis*, as well as basic cell structure. If necessary, please refer to the sections on Intracellular Organelles and Mitosis in Chapter 2. I'll wait.

Ready? In the bone marrow are ***hematopoietic*** [*hemat(o)-* blood + *poie(o)-* produce/production + *-tic* pertaining to] stem cells that undergo *mitosis*. Mind you, these stem cells not only replenish themselves, they also create all of the various blood cells (including *thrombocytes*, *leukocytes,* and *erythrocytes*) that will eventually wind up in the blood stream. Of course, in response to *erythropoietin*, these stem cells will increase production of the *erythrocytic* cell line. But this is **not** a quick process that results in rapid replenishment of oxygen-carrying RBCs in peripheral blood. No, just as we needed time to grow and mature from infancy to adulthood, so too do blood cells.

For this discussion, let's get a little crazy and use an analogy that we are all familiar with—human development. To do this, I'll compare each stage of *erythrocytic* maturation to human life stages, like infants and toddlers. So, to begin, let's consider the ***rubriblast*** [*rubri(o)-* red + *blast(o)-* germ/shoot] as an "infant" *erythrocyte*. (Side note: anytime you see a cell type ending in "blast," you know that it is the youngest of that cell type.) Remember from Chapter 2 how the daughter cells resulting from *mitosis* had much work to do organizing, producing proteins for the *intracellular organelles* and such? Well, our young *erythrocytes* need to do this too. That's why if you ever see *rubriblasts* or ***prorubricytes*** [*pro-* before + *rubri(o)-* red + *cyt(o)-* cell], most likely in a bone marrow sample, you will probably see multiple *nucleoli* and very ***basophilic*** [*bas(o)-* blue + *phil(o)-* loving + *-ic* pertaining to] *cytoplasm*. As our *erythrocytes* continue to mature, the nucleus and other intracellular organelles are needed

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less and do less work. So, as we look at the next stage of maturation, the ***rubricyte*** [*rubri(o)-* red + *cyt(o)-* cell], the cytoplasm will be less basophilic and the nucleus will be a bit smaller, perhaps with rare to no nucleoli. By the way, *rubriblasts* being the earliest in the erythrocytic series, also undergo mitosis, along with some of the *prorubricytes* and some first- and second-generation *rubricytes*.[1]

If the *rubriblast* is like an infant, the *prorubricyte* is like a toddler, and the *rubricyte* is like a tween. None of them are prepared to leave the comfort and security of their home, the bone marrow. In fact, even the teen-like ***metarubricyte*** [*meta-* after, beyond + *rubri(o)-* red + *cyt(o)-* cell] should remain in the bone marrow. After all, the *metarubricyte* still has a very condensed (almost blackish) useless nucleus and its endoplasmic reticulum, both of which are going to get in the way of carrying oxygen. Yep, it's too young to get a job yet. However, like some teens, we may find some *metarubricytes* slipping out of the bone marrow prematurely and into circulation. (This is why this is the first one shown in Table 3.1.) Having *metarubricytes* in peripheral blood is not ideal and usually indicates a problem with the bone marrow's "exit doors." Gotta get better locks! If we do see any metarubricytes in a blood smear, we often refer to them as ***nucleate red blood cells*** (**NRBC**s). We also need to keep track of their numbers, because they can make automated counts of other nucleated cells (*leukocytes*) inaccurate.

The youngest *erythrocyte* that we **should** see in peripheral blood is the ***reticulocyte*** [*reticul(o)-* net + *cyt(o)-* cell]. It gets its name from the endoplasmic *reticulum* that is still found in its cytoplasm. They also have some ribosomes and mitochondria, but no nucleus. These young cells and mature erythrocytes are ***anuclear*** [*a-* without + *nucle(o)-* nucleus + *-ar* pertaining to]. Reticulocytes lose their nuclei just before leaving the bone marrow. And because reticulocytes have a few mitochondria, they can still metabolize energy sources. (Lacking these mitochondria, metabolism for mature RBCs is very limited.) Using special stains, we can actually highlight the ribosomes and endoplasmic reticulum so that we can accurately count the *reticulocytes* (see Table 3.1 and Fig. 3.5). We often want to do this, to know conclusively how well the bone marrow is responding in *anemic* patients. Using conventional stains for routine *hematology*, the *cytoplasm* of these young cells will be ever so slightly

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basophilic. Plus, these young cells don't have their full complement of hemoglobin yet. So, they can't stain a beautiful, bright red like fully mature RBCs. Though they do appear somewhat reddish. So, if we see these cells on a blood smear, do we say they are blue or red? Neither. We report ***polychromasia*** [*poly-* many + *chrom(o)-* color + *-asia* condition of]. *Reticulocytes* are *polychromatic* [*poly-* many + *chrom(o)-* color + *-tic* pertaining to] cells in routine blood smears. But visualization of subtle *polychromasia* can be difficult. That is why *reticulocyte* counts, using special stains, may be ordered for *anemic* patients. But without special stains to accurately count *reticulocytes*, *polychromasia* is THE indicator of red blood cell regeneration. That's why it is so important to look for *polychromasia* and report it. But don't expect to see polychromasia or reticulocytes in horses or cattle. They typically only release mature *erythrocytes* into the blood stream.[2] So, *polychromasia* in these animals is extremely rare.

How long does it take from bone marrow stimulation with *erythropoietin* until we have *reticulocytes* (or mature RBCs) released into circulation? It could take about a week. Fortunately, under normal circumstances, mature *erythrocytes* can remain in circulation for months: in the ***canine*** (dog) nearly 4 months, ***porcine*** (pig) ~3 months, ***feline*** (cat) ~2.5 months, ***equine*** (horse), ***bovine*** (cattle), and ***ovine*** (sheep) ~5 months.[3] But if there is ***acute*** (sudden) loss or destruction of red blood cells, we won't have their replacements anytime soon. In *feline* patients, because of the extremely short RBC lifespan, such an event can result in a serious *anemic* crisis very rapidly. With enough loss of *erythrocytes* in anyone, it can leave the body in pretty rough shape for a while. Speaking of shape, let's discuss *erythrocyte* ***morphology*** [*morph(o)-* shape, form + *-logy* the study of] next.

Morphology

Normocytic [*norm(o)-* normal + *cyt(o)-* cell + *-ic* pertaining to] mature *erythrocytes* (see Table 3.1 and Fig. 3.6) for most domestic animals are rather flat, disc-like cells. (They remind me of thumbprint cookies, without chocolate or jam filling the indent in the middle. This *bilateral* or two-sided indent is most pronounced in

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canine erythrocytes, hence the distinct zone of central pallor.) Now, this might seem like an odd shape, but it serves a purpose. First, that shape gives *erythrocytes* lots of surface area—very important for delivering oxygen to body tissues. We need a lot of direct surface contact for that. Plus, that shape provides tons of flexibility, allowing the erythrocytes to squeeze through the smallest blood vessels (capillaries). Again, this is very important for delivery of oxygen to body tissues. When fully mature with a full complement of *hemoglobin*, mature erythrocytes should stain a nice, bright red with a pale center, as shown.

Of course, we will never have just fully mature *erythrocytes* merrily sailing along through the bloodstream, right? We always have a mixture of younger, middle-aged, and geriatric (old) cells in peripheral blood. Most of them will be mature, been-there-done-that, physically fit erythrocytes, holding down their job of transporting oxygen. Like us, there are always youngsters entering the work force and others headed into retirement. So, in any normal patient's blood, we will likely see some ***anisocytosis*** [*anis(o)-* varied + *cyt(o)-* cell + *-sis* a condition of], as shown in Fig. 3.7. Some will be *normocytic*, in size and color. Others will be ***macrocytic*** [*macr(o)-* large + *cyt(o)-* cell + *-ic* pertaining to]. These larger, *macrocytic* cells are probably *reticulocytes*. So, they'll also be *polychromatic* in color. The ***microcytic*** [*micr(o)-* small + *cyt(o)-* cell + *-ic* pertaining to] cells are losing or have lost their *zone of central pallor* (the lighter area in the middle of a *normocytic erythrocyte*). These are the ones headed into retirement. Now, if we begin seeing way more macrocytic and microcytic cells in a blood smear, something may be wrong. Greater numbers of *macrocytic, polychromatic* cells may indicate a response to blood loss. That patient may also have an abnormally low PCV. Large numbers of *microcytic* cells may indicate that the spleen is trying to repair damaged and misshapen *erythrocytes*. In this case, we will also likely see ***poikilocytosis*** [*poikil(o)-* irregular + *cyt(o)-* cell + *-sis* a condition of].

TABLE 3.1

Erythrocytic Maturation Sequence

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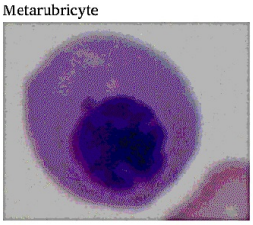
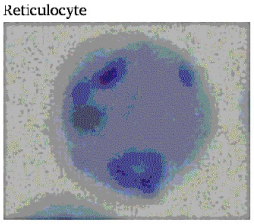
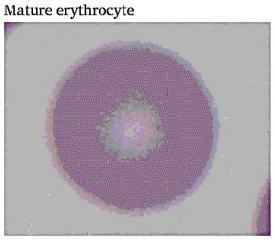
In Bone Marrow			In Peripheral Blood Circulation		
Rubriblast	Prorubricyte	Rubricyte	Metarubricyte	Reticulocyte	Mature erythrocyte
					

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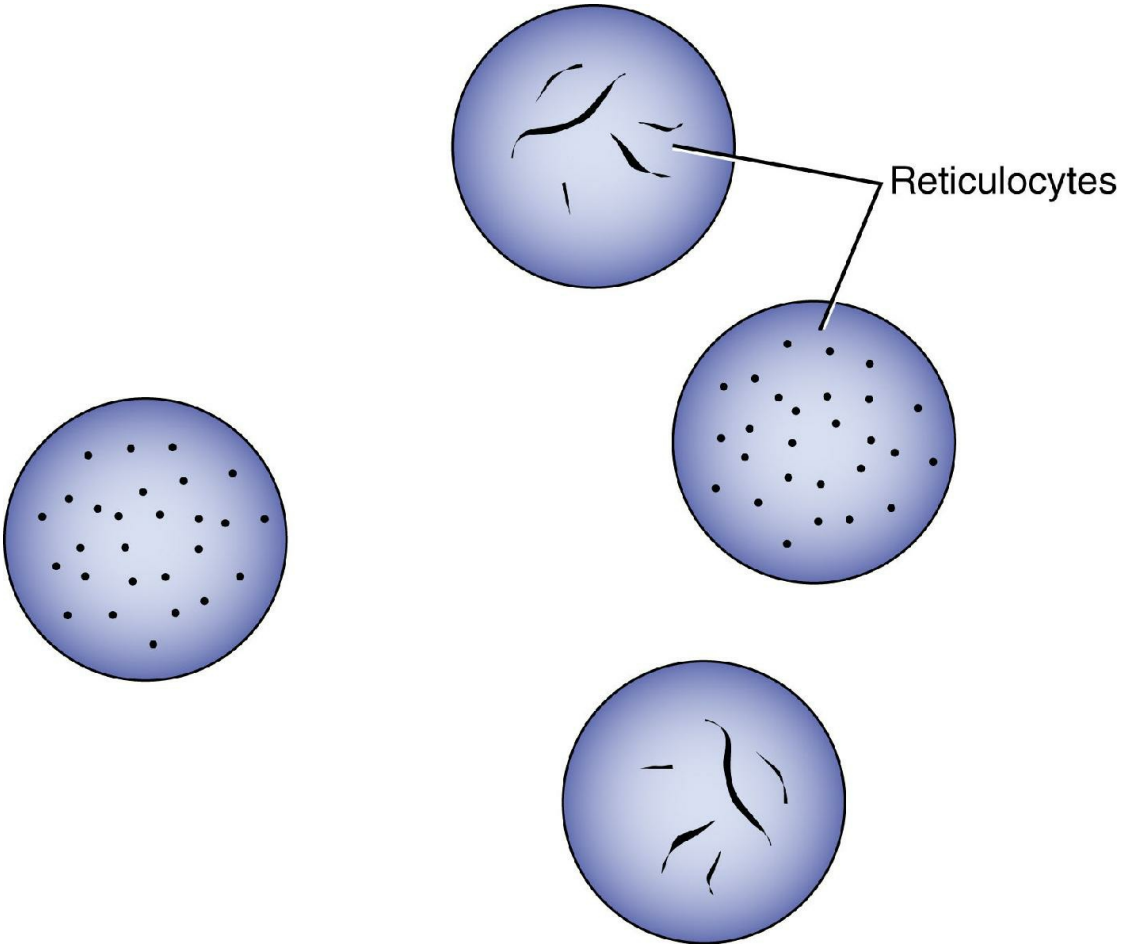

FIG. 3.5 Reticulocytes stained with new methylene blue.

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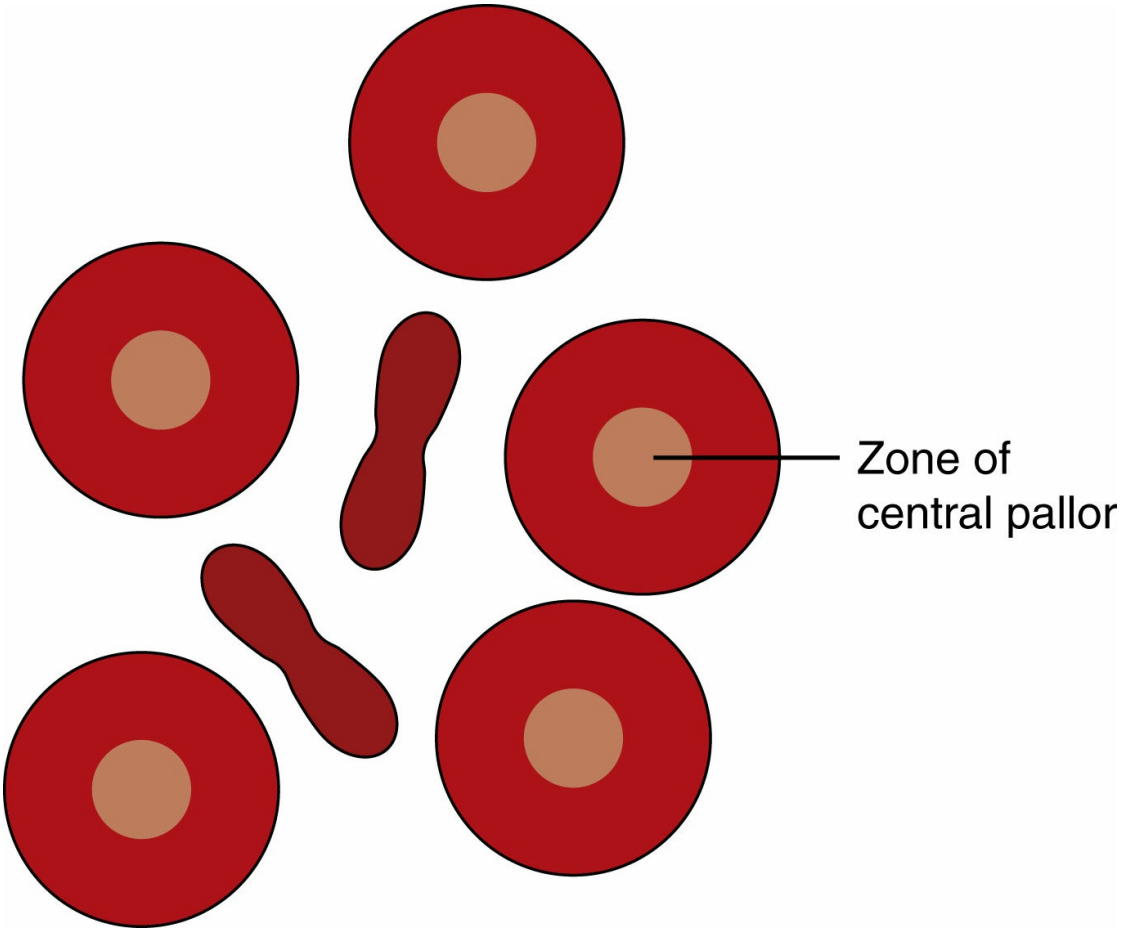

FIG. 3.6 Normocytic erythrocytes in circulating blood.

Poikilocytosis

Poikilocytosis is a very important facet of *erythrocyte morphology* (Figs. 3.8 and 3.9). There are many variations on a theme of ***morphologic*** [*morph(o)-* shape/form + *log(o)-* knowledge/study + *-ic* pertaining to] changes that can be seen in RBCs. Each abnormal shape is distinct and indicates particular abnormalities in the body. This is why we don't simply report *poikilocytosis*. That's too broad a category. It doesn't help the veterinarian arrive at an accurate diagnosis and treatment plan. So, we give special names to each *erythrocytic* shape abnormality.

Spherocytes [*spher(o)-* sphere, ball + *cyt(o)-* cell] are literally *erythrocytes* that are shaped like balls. Remember, a *normocytic erythrocyte* will be ***discoid*** [*disc(o)-* disk + *-oid* resembling], average size, with a zone of central pallor. Well, if there is something wrong with the shape or cellular membrane of an *erythrocyte*, ***splenic*** [*splen(o)-* spleen + *-ic* pertaining to] ***macrophages*** [*macr(o)-* large +

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phag(o)- eater] will attempt to fix it. These *macrophages* will remove irregular and damaged portions of the cell membrane, patch the remainder, and send the repaired RBC on its way. Now we have less cellular membrane stretched around a full complement of *hemoglobin*. So, *spherocytes* are very plump cells. They cannot lay flat on a slide because of their spherical shape, making them appear smaller and a darker red than *normocytic erythrocytes*. We report them. However, they don't tell the veterinarian why repairs were needed to these cells. So, we continue to look for other *poikilocytic* [*poikil(o)-* irregular + *cyt(o)-* cell + *-ic* pertaining to] changes. By the way, *equine*, *feline*, and *bovine erythrocytes* tend to have small, slight, barely discernable zones of central pallor, so it is difficult to identify *spherocytes* in those animals. *Canine erythrocytes*, on the other hand, have a very distinct zone of central pallor. So, *canine spherocytes* are easy to identify.

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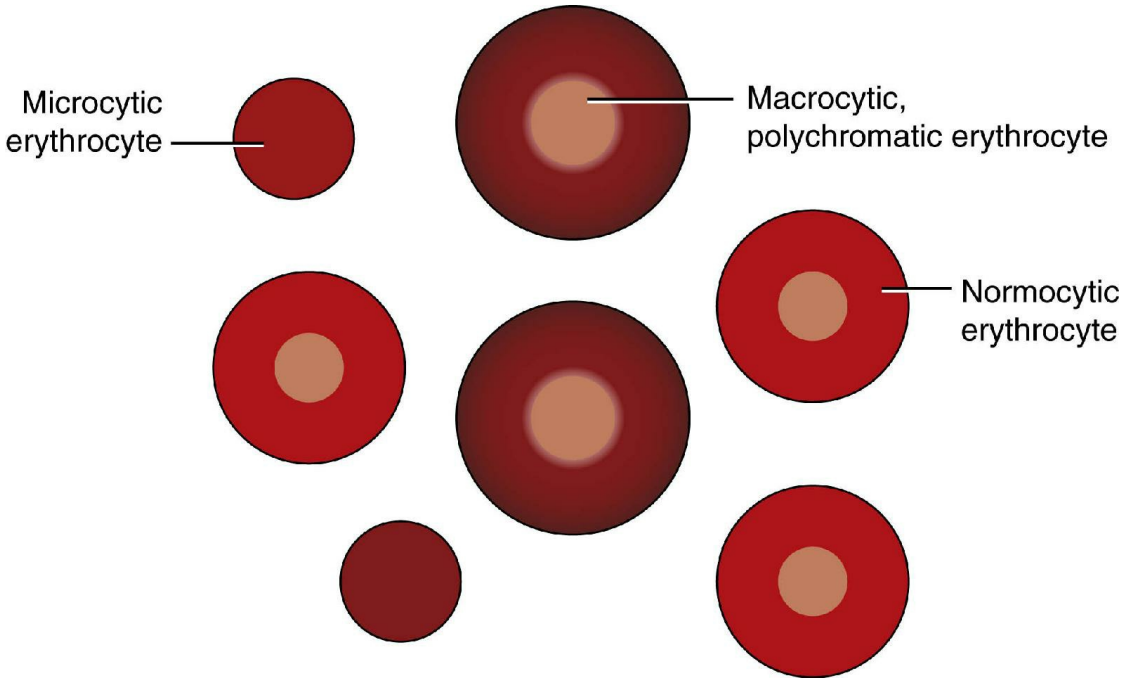

FIG. 3.7 Anisocytosis.

Stomatocytes [*stomat(o)-* mouth + *cyt(o)-* cell], as their name implies, appear to have mouths. We may see this in young, *macrocytic, polychromatic erythrocytes* that simply have a bit too much cellular membrane. Remember, these cells just lost their space-occupying nuclei before leaving the bone marrow. Until they're full of hemoglobin, which tightens up the cellular membrane, that oversized, floppy membrane may form a crease-like indent that makes the cell look like it's smiling. However, there are times when *stomatocytes* develop due to disease, like liver disease. In this case, we see *stomatocytes* without *polychromasia*. Report them if you see them, along with any other *morphologic*, color, or distribution changes that you see. It's up to the veterinarian to analyze all of the clinical information available to accurately interpret laboratory data and make a diagnosis. Without all of the puzzle pieces, that can't be done and may put our patients at risk.

Echinocytes [*echin(o)-* hedgehog + *cyt(o)-* cell] are spiny-looking *erythrocytes*. The whole surface of the cell has evenly distributed little spiny projections, sort of like a hedgehog. It's all in a name, right? Often, we'll see *echinocytes* in blood smears as an artifact. The artifactual change may happen during sample handling and blood smear preparation, due to changes in temperature, pH, or drying. However, there are times when ***echinocytosis*** [*echin(o)-* hedgehog + *cyt(o)-* cell + *-sis* a condition of] is due to ***pathology*** [*path(o)-* disease

+ *-logy* the knowledge/study of; i.e., the presence of disease]. Venomous snake bites and electrolyte disturbances can cause ***echinocytic*** [*echin(o)-* hedgehog + *cyt(o)-* cell + *-ic* pertaining to] changes. So, be careful not to simply blow off *echinocytes* when you see them. They should be reported for very good reason.

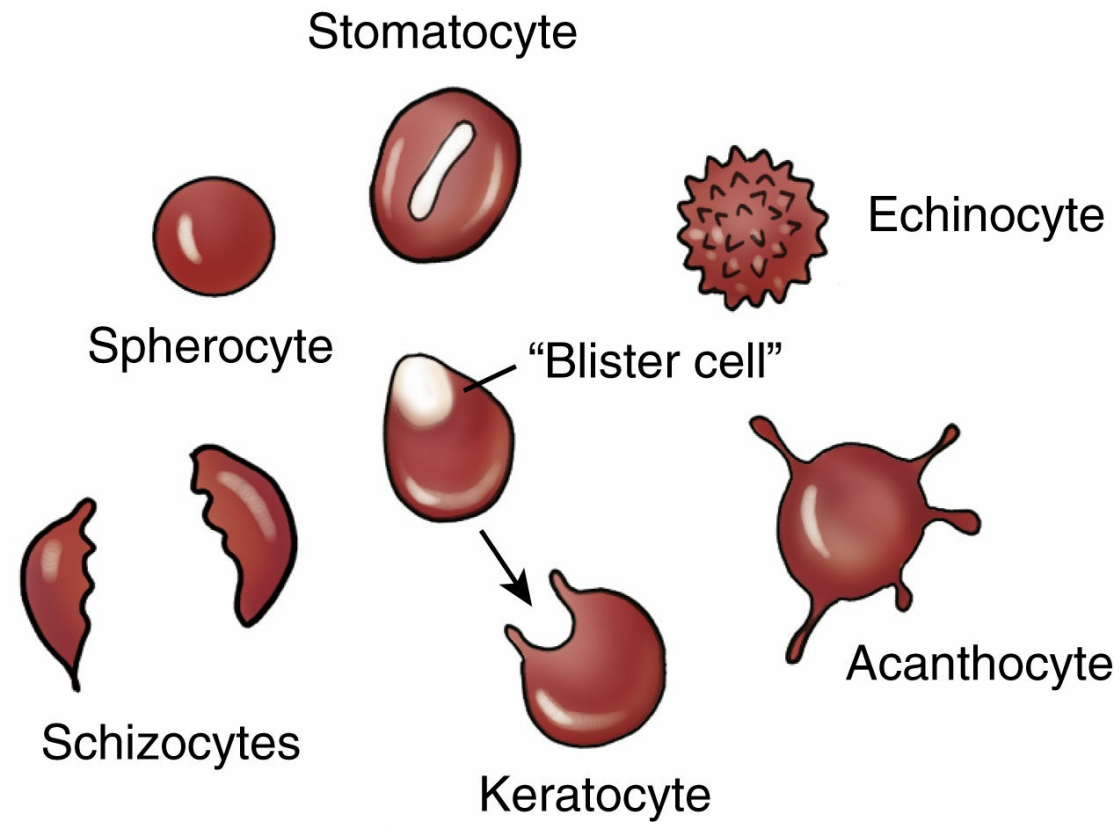

FIG. 3.8 Poikilocytosis.

Acanthocytes [*acanth(o)-* thorn + *cyt(o)-* cell] have projections too. But the thorny projections of *acanthocytes* are very different from *echinocytes*. ***Acanthocytic*** [*acanth(o)-* thorn + *cyt(o)-* cell + *-ic* pertaining to] projections are few and far between and randomly disbursed, like thorns on a rose's stem. Plus, the projections are not uniform in size, as you can see. Now, many of my students frequently had difficulty remembering the characteristics of acanthocytes versus echinocytes. If you remember the meaning of the root word in each name, it will help. *Echin(o)-* means hedgehog, whose bodies are covered in many, many, uniform, prickly little spines, right? So, *echinocytes* are covered in many uniform little projections. *Acanth(o)-* means thorn, like the few found on roses,

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which vary in size and location along the stem. So *acanthocytes* have only a few projections that vary in size and location on the cell. *Acanthocytes* are never artifacts. They most commonly develop in liver disease and certain cancers. As with other *poikilocytic* changes, *acanthocytes* should be reported.

Keratocytes [*kerat(o)-* horn + *cyt(o)-* cell] literally look like they have horns, like a bull. This morphologic change usually follows the rupturing of a blister-like vesicle. Why on earth would an erythrocyte have a vesicle? Well, there are times when a small portion of the RBC's hemoglobin and cellular membrane are damaged, resulting in something called a Heinz body. The hemoglobin in a Heinz body is denatured and the associated cell membrane damaged from oxidation. Eventually, the vesicle may become stretched out and rupture, leaving the two horn-like projections. One of the most common causes of Heinz bodies (and consequently keratocytes) in canine erythrocytes is onion toxicity. Really, ingestion of any member of the onion family (including chives and garlic) can cause this in dogs. If severe, it may even result in *anemia*. Please report *keratocytes*.

Schizocytes [*schiz(o)-* split/divided + *cyt(o)-* cell], also called ***schistocytes*** [*schist(o)-* split/divided + *cyt(o)-* cell], are fragments of damaged *erythrocytes*. Either name will do in your reporting. These *erythrocytes* have either been torn by ***intravascular*** [*intra-* within + *vascul(o)-* vessel + *-ar* pertaining to] fibrin strands or by turbulent blood flow. *Schizocytes* are never artifacts. They always signal serious underlying disease that is physically traumatizing RBCs. Obviously, these *schizocytic* fragments are the lucky survivors. Other *erythrocytes* may not have survived the trauma, resulting in release of free *hemoglobin* in circulation. Yes, this could be a cause of the *hemolytic* plasma that we discussed earlier. That is why it is important to report both *schizocytosis* [*schiz(o)-* split + *cyt(o)-* cell + *-sis* a condition of] and *hemolytic* plasma.

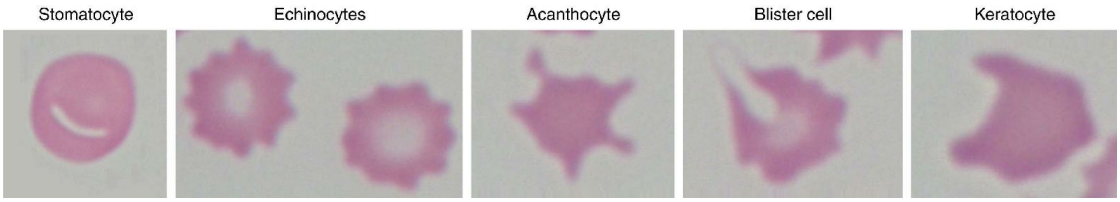

FIG. 3.9 Poikilocytes.

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Hemoglobin

Finally, we get to talk about the wonderful reddish protein that fills *erythrocytes*: *hemoglobin*. It is a very large molecule. Now, I'm going to get into some pretty fine detail here. Please bear with me. These details will help you understand the oxygen-carrying capacity of this stuff.

As I said, *hemoglobin* is a very large molecule. It's made of two distinct compounds: (1) heme—the iron-containing, pigmented portion produced by mitochondria, and (2) globin—the protein portion produced by ribosomes, as you would expect. The ratio of heme to globin in this molecule is 4 to 1. Why is that important? Each heme molecule, because of the iron (Fe^{++}), can carry one molecule of oxygen (O_2). So, with this 4:1 ratio, a single hemoglobin molecule can actually carry four oxygen molecules. This gives each *erythrocyte* tremendous oxygen-carrying capacity! By the way, when oxygen is loosely bound to *hemoglobin*, the *erythrocytes* become bright red. This is because the RBCs are full of ***oxyhemoglobin*** [*oxy(o)-* oxygen + *hem(o)-* blood + *glob(o)-* "stuff" + *-in* the]. Now that you understand the importance of Fe^{++} in hemoglobin, just think for a moment of the impact on the body for a patient who has an iron deficiency. Too little iron would ultimately result in too few heme molecules and too little hemoglobin. Oh, but that doesn't happen very often, does it? Oh yes it does. Iron deficiency anemia is common in piglets. Those little piglets don't thrive and may not survive, because they can't oxygenate their tissues efficiently.

We'll talk more about gas exchange, as well as blood flow throughout the body in Chapter 5. For now, imagine each hemoglobin-packed erythrocyte squeezing through the smallest of vessels in the lungs. The erythrocytes arrive there depleted of oxygen. We refer to this oxygen-depleted hemoglobin as ***deoxyhemoglobin*** [*deoxy-* reduced oxygen + *hem(o)-* blood + *glob(o)-* "stuff" + *-in* the]. Blood with an abundance of *deoxyhemoglobin* will appear a dark red. This is what we collect from veins for most of our diagnostic blood samples. And because there is more oxygen in

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the lungs and less in the erythrocytes, O_2 can freely diffuse across the cell membranes from the lungs to the blood. Once the erythrocytes squeezing through the lungs have picked up a full load of oxygen (oxyhemoglobin), they become bright red again. This is why blood in arteries is bright red. The oxygen-laden blood is delivered all over the body, delivering the oxygen to body tissues by a reverse diffusion process. Because of the loose molecular bond between O_2 and hemoglobin, diffusion from the erythrocytes out into the tissues is quick and easy.

Carbon Monoxide Toxicity

This is an opportune time to talk about a hemoglobin bond that is extremely strong and difficult to break. I'm talking about ***carbon monoxide*** [*mono-* one] ***(CO)***. That's the stuff produced by burning fuel (gas, propane, wood, etc.). It is an odorless gas found in the exhaust of things like automobiles, generators, kerosene heaters, grills, fireplaces, gas ranges, furnaces, and gas water heaters, to name a few. CO is a silent, indiscriminate killer of people and animals. You see, that single oxygen atom makes CO stick like glue to hemoglobin, forming a strong ***carboxyhemoglobin*** [*carb(o)-* carbon + *oxy(o)-* oxygen + *hemoglobin*] (COHb) molecule. Of course, with CO hoarding hemoglobin, our erythrocytes' ability to transport O_2 is significantly reduced. This results in serious tissue ***hypoxia*** [*hypo-* low+ *ox(o)-* oxygen + *-ia* a condition of] throughout the body that can be lethal (deadly).

What's really odd is how this looks when we physically examine patients suffering from ***carbon monoxide toxicity*** [*tox(o)-* poison + *-city* state of; i.e., poisoning]. Usually, if a patient is ***hypoxic*** [*hypo-* low + *ox(o)-* oxygen + *-ic* pertaining to], its mucous membranes (gums and tongue) will be pale or bluish. But in CO toxicity, the mucous membranes will be cherry-red, from that lone oxygen atom making the hemoglobin red. Don't be fooled by those bright red gums, especially in an unconscious patient or one who's having difficulty breathing. We can treat carbon monoxide toxicity if caught early enough. It takes time to treat—with oxygen therapy, of course. But it may be hours before the CO begins to loosen its grip on the hemoglobin. People and animals die every year from CO

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toxicity. That's why carbon monoxide monitors in homes are so important.

Carbon Dioxide

We've talked about *erythrocytes* and *hemoglobin* with regard to oxygen and carbon monoxide. What about carbon dioxide (CO_2)? Does hemoglobin have anything to do with CO_2? We'll get there in a moment, after we review a little about CO_2. In the previous chapter, we learned that CO_2 is a by-product of cellular activity. We even talked briefly about the formation of *carbonic acid* and acid-base *homeostasis*. What does this have to do with *hemoglobin* and *erythrocytes*? Not much. Most CO_2 will diffuse from the tissues and ultimately be transported in the plasma, as *carbonic acid* (H_2CO_3) and its ionized constituents, hydrogen (H^+) and bicarbonate (HCO_3^-). See? Plasma is important for more than just floating red and white blood cells and platelets around the body. In fact, we'll talk more about the importance of plasma later. First, let's look at another important blood cell type: *leukocytes*.

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Applied White Blood Cell Terminology

Leukocytes [*leuk(o)-* white + *cyt(o)-* cell] or white blood cells (WBCs) are so diverse in *morphology* and function. This makes them really interesting and exciting to study! I mean, *leukocytes* don't have a single, simple job to do, like *erythrocytes*. Okay, so transporting oxygen is very important and necessary for life. But I've got to say that's rather boring compared to *leukocytes*. I mean, we're talking about being able to recognize ***pathogens*** [*path(o)-* disease + *gen(o)-* producer], like bacteria and viruses, and then call in the "troops" to attack and destroy them. And there is so much more that *leukocytes* do! Seriously, *leukocytes* in action would make a great action-adventure film! Okay, so maybe I'm the only one who would go to the theater for that one. All I ask is that you try to show a little enthusiasm over these amazing cells. I'm telling you, you are way more likely to see ***leukocytosis*** [*leuk(o)-* white + *cyt(o)-* cell + *-sis* condition of; i.e., excess numbers of WBCs] than *polycythemia*. So, get ready for some exciting and important information.

Leukopoiesis

As you may recall, *erythropoiesis* is the production of red blood cells. Well, using what you already know about the parts of that medical term, you've probably already figured out that ***leukopoiesis*** [*leuk(o)-* white + *poie(o)-* production + *-sis* the process of] is the production of WBCs. Simple, right? Yes and no. You see, when the stem cells in bone marrow respond, they don't simply create one type of cell. Depending on the ***interleukins*** [*inter-* between + *leuk(o)-* white + *-in* a, the] secreted by various WBCs, the bone marrow will step up production and release of particular *leukocytes* that are needed. Did you catch that? *Interleukins* are chemical substances that provide communication, if you will, *between* WBCs and between WBCs and the bone marrow stem cells. Some of these chemicals even work with *erythropoietin*, for kind of a double-whammy effect, to stimulate *erythropoiesis*. These are complicated processes. For our purposes, simply recognize that there are many factors involved in stimulation of *leukopoiesis*.

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Depending on the specific stimulation of the stem cells, their mitosis will create various ***leukocytic*** [*leuk(o)-* white + *cyt(o)-* cell + *-ic* pertaining to] blast cells. These cells then undergo similar *mitotic* activity (like the *rubriblasts* did) to produce their particular type of *leukocyte*. Let's talk about the individual cell lines that result from this process, shall we?

Granulocytes

Granulocytes [*granul(o)-* granule + *cyt(o)-* cell] are *leukocytes* that have *cytoplasmic* granules. Their cytoplasmic granules are basically lysosomes. There are three distinctly different *granulocytes*, made visually distinct and named by the staining properties of their *cytoplasmic* granules. Functionally too, each *granulocyte* has a different purpose.

Neutrophil

Mature ***neutrophils*** [*neutr(o)-* neutral + *phil(o)-* loving] have abundant cytoplasmic granules packed within their cellular membranes (Fig. 3.10). But because the granules don't really pick up the stains that we use in *hematology* (hence their neutrality in terms of color), it takes a discerning ***microscopic*** [*micro-* small + *scop(o)-* to view + *-ic* pertaining to; i.e., viewing using a microscope] eye to see them well. In most domestic mammals, with the exception of ***ruminants*** (e.g., bovine, ovine, and ***caprine*** [goat]) *neutrophils* are the most abundant *leukocyte* in the blood. Why do we need so many? *Neutrophils* are the police, marines, and infantry all rolled into one. They are the first ones to take on *pathogens* in battle. These self-sacrificing cells respond quickly to their call to duty and recruit others to join their ranks. We'll talk about how they take on pathogens and call reinforcements in a moment. First, let's take a close look at their production and maturation (Table 3.2).

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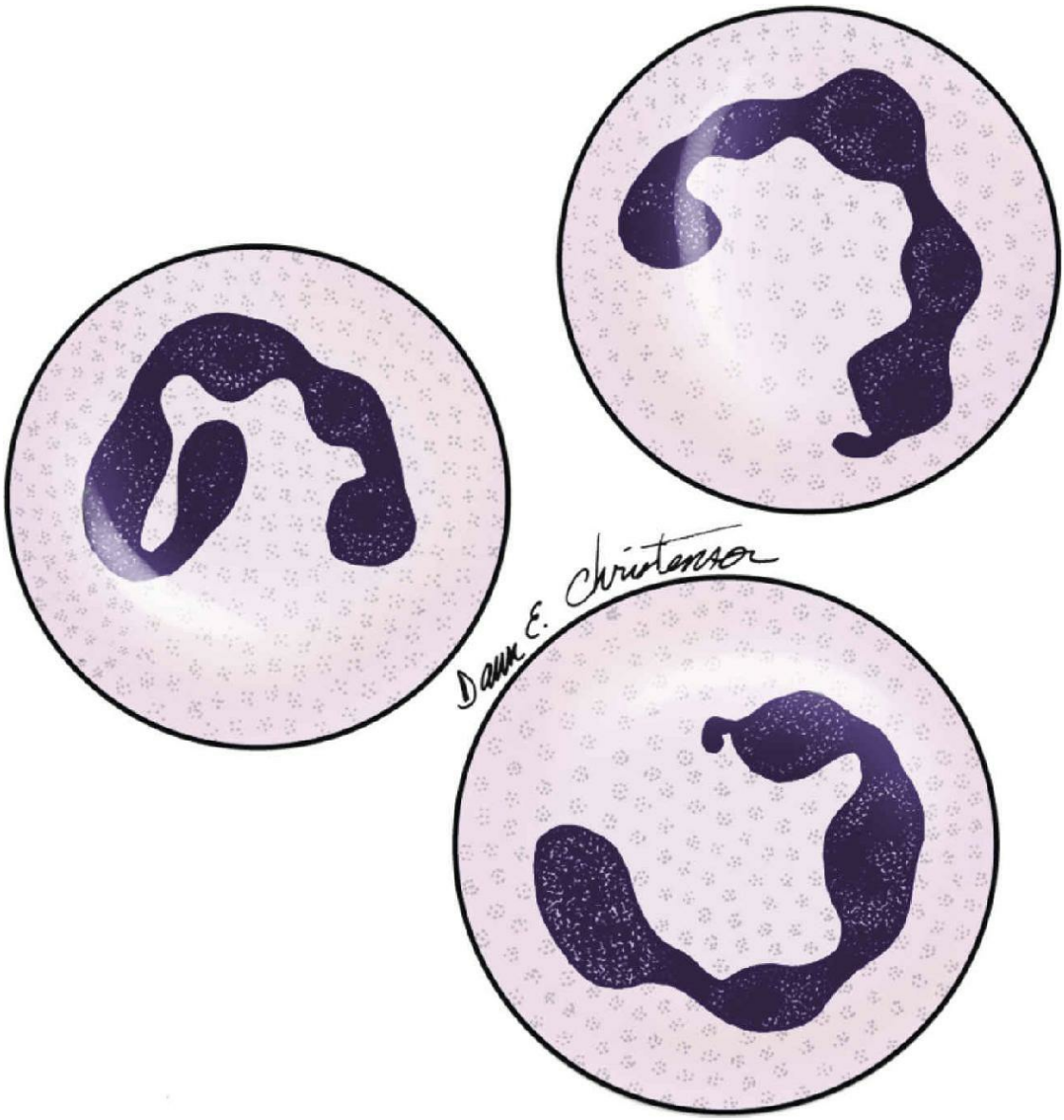

FIG. 3.10 Mature, segmented neutrophils.

In the bone marrow, from *mitosis* of a *hematopoietic* stem cell, we begin with the "birth" of a ***myeloblast*** [*myel(o)-* marrow + *blast(o)-* germ/shoot]. Like the *rubriblast*, the *myeloblast* could be thought of as an "infant" *neutrophil*. Remember, we said earlier that blast cells of any kind are the youngest of any cell line. So, *myeloblasts* are the germinal "seeds" that will undergo *mitosis* to produce an abundant "crop" of *neutrophils*. The direct products of a *myeloblast's mitosis* are ***promyelocytes*** [*pro-* before + *myel(o)-* marrow + *cyt(o)-* cell]. Some promyelocytes will undergo mitosis too, producing ***myelocytes*** [*myel(o)-* marrow + *cyt(o)-* cell]. Even some first- and second-generation *myelocytes* may undergo *mitosis*. Third-generation *myelocytes* and the maturation stages that follow cannot undergo

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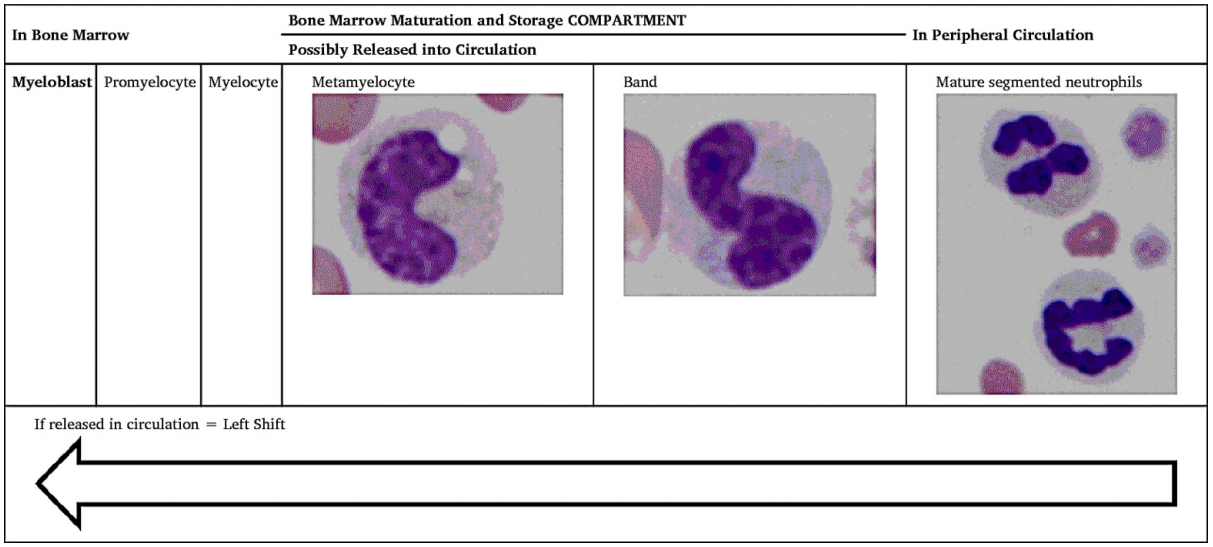
mitosis. Only *myeloblasts*, *promyelocytes*, and first- and second-generation *myelocytes* have that capability. Why bother emphasizing all of this mitosis of these young cells? Think about it. In the face of profound infection, isn't it reassuring to know that the bone marrow can maximize the numbers of *neutrophils* produced? From the mitosis of one stem cell producing two new *myeloblasts*, we can ultimately gain 32 new mature *neutrophils* (max) in circulation.[4] This level of multiplication is very important when fighting *pathogens*.

Before we move on to the next maturation stages, let's talk briefly about the *morphology* of these really young cells. *Myeloblasts* and *promyelocytes* each have a round nucleus with loose (not dense) nuclear chromatin. As you would expect, they likely have nucleoli (multiple nucleoli in *myeloblasts*). First-generation *myelocytes* may appear similar. Second- and third-generation *myelocytes* tend to have areas of increased density in their nuclear chromatin, with no nucleoli. All of these stages should remain in the bone marrow. In *extreme* cases of demand for neutrophils, there may be times when *myelocytes* are released into circulation prematurely. Nothing like sending a child to fight a war. Ideally, all of these cells and the maturation stages that follow should remain in the bone marrow until they have reached maturity, especially if they have *myel(o)-* in their names. Suffice it to say that life is not always ideal.

TABLE 3.2

Neutrophilic Maturation Sequence



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However, in ideal circumstances, *myelocytes* will mature into ***metamyelocytes*** [*meta-* after + *myel(o)-* marrow + *cyt(o)-* cell]. *Metamyelocytes* have a more distinctive nucleus—no longer round or oval but indented a little bit on one side (see photo in Table 3.2). Actually, the nuclear shape of *metamyelocytes* reminds me of butter beans or kidney beans. Plus, the nuclear chromatin is loose, but more condensed (darker) in spots than its predecessors. If it stays in the bone marrow to mature (as it should!), the *metamyelocyte* will eventually mature into a ***band neutrophil***. Band? Really? I know. After throwing all of those huge, tongue-twisting medical terms at us, why on earth would they give this stage such an odd, simple name? Truthfully, I have no idea. All I can say is to count your blessings. If it helps, perhaps we should think of band neutrophils as young adults (late teens, early 20s?) who are inexperienced, yet ready to take on the world and drive the bus for the band. Get it?—A band driving the bus for the band? Never mind. The nuclear chromatin of a band neutrophil is more condensed in areas than the metamyelocyte. The morphology of the nucleus of a band is quite distinct (see photo in Table 3.2). It is far more elongated than any of its predecessors and is usually in the shape of a U, a C, or an S, with uniform thickness. Any indentations in its nucleus and the cell gets booted completely into the mature neutrophil category. When needed, these mature ***segmented neutrophils*** (see photo in Table 3.2) are released into circulation. They are called segmented neutrophils because the nucleus is highly condensed and "pinched" into two to five distinct segments. All of this nuclear "shape-shifting" is precisely why neutrophils are described as ***polymorphonuclear*** [*poly-* many + *morph(o)-* shape + *nucle(o)-* nucleus + *-ar* pertaining to]. Yes, that is a very big word that simply means the nucleus of a neutrophil may come in many twisted shapes. *Polymorphonuclear* condenses what I just said in that whole prior sentence. This is why I love medical terminology—brevity.

Table 3.2 shows the normal linear progress, from left to right, of the ***neutrophilic*** [*neutr(o)-* neutral + *phil(o)-* loving + *-ic* pertaining to] maturation sequence. I've included images for those cells you are **most** likely to see in a peripheral blood smear. Obviously, we would prefer to have the most mature neutrophils released into peripheral circulation. However, in times of peak demand (e.g., a

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severe bacterial infection), we may rapidly deplete all of the available mature neutrophils in the bone marrow's storage compartment. Fortunately or unfortunately, in order to meet the demand for large numbers of neutrophils, we may have to release less mature cells into circulation. This is done in an orderly manner. The oldest cells are always released first. So, mature segmented neutrophils are released first (shown far right in Table 3.2). Then band neutrophils are released (shown to the left of the mature neutrophils). If there is still need for more neutrophils, then *metamyelocytes* may be released (shown to the left of the band). In a last-ditch effort to meet demand we may even see third-generation *myelocytes* released from the maturation compartment. These immature cells shouldn't be released, but the bone marrow has no choice in times of high demand. Each time we dip into younger and younger cells in the maturation sequence, we backtrack further and further to the left (as shown in Table 3.2). In *hematology*, that's what is meant by the term "***left shift***." Reporting of a "left shift" to the veterinarian merely indicates that numerous immature neutrophils were observed in the blood smear. It indicates significant turnover of neutrophils, most often due to infection.

Just what do all of these neutrophils do once they're in the bloodstream? Well, they go wherever the body tissues need and call them. Call? Yes, ***chemotaxis*** [*chem(o)-* chemical + *-taxis* movement stimulated by] happens in response to chemical mediators that may be secreted by body cells and tissues, as well as *pathogens*. Think of it as an "SOS" or 911 call for help. Let's use the example of a dog bite that results in a contaminated, open wound with pockets in deeper tissues. The traumatized tissues and bacterial contaminants in the wound will promote *chemotaxis*. First responders will be mature neutrophils in circulation and those that have been hanging out, *marginalized,* along ***vascular*** [*vascul(o)-* vessel + *-ar* pertaining to] walls. (Yes, marginated neutrophils just hang out along vascular walls, like folks leaning against the wall of subway or bus terminal. They travel, when called.) Once the neutrophils arrive at their destination, they migrate from the bloodstream into the ***interstitium*** [*inter-* between + *stiti(o)-* tissues + *-um* the] and, in this case, the wounded area. The neutrophils will aggressively begin to ***phagocytize*** [*phag(o)-* eat + *cyt(o)-* cell + *-ize* the act of] bacterial

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contaminants.

Remember, we said neutrophils are packed with *cytoplasmic granules*. Those granules contain powerful *lysosomal* enzymes and other chemicals. Some of those chemicals will promote even greater *chemotaxis*, attracting more and more neutrophils to the area. Have you ever had or seen a contaminated wound? Have you observed the thick, creamy-white ***purulent*** [per'u-lent] discharge (i.e., pus) that develops? Part of what creates the opaque, whitish coloration of the discharge are the neutrophils flooding into the area (*leukocytes* are *white* blood cells, remember?). By the way, I like to think of neutrophils as really sloppy eaters, drooling and spilling the contents of their cytoplasmic granules all over. This contributes to ***inflammation*** [*inflamm(o)-* fire + *-tion* the condition of] in the wounded area. We'll talk more about *inflammation* later, when we discuss ***immunology*** [*immun(o)-* protection + *-logy* study of]. For now, simply think of *inflammation* as a turbo-booster for *neutrophilic chemotaxis*.

Now, if you want to talk numbers of neutrophils involved, that's a difficult thing to nail down. The number of circulating neutrophils that we can count, in the blood at any given moment in time, depends on a number of factors. First, the average time any neutrophil will spend in circulation is roughly 10 hours in a healthy animal, with all circulating neutrophils replaced roughly 2.5 times a day.[5] Why? They simply love migrating out into ***extravascular*** [*extra-* outside + *vascul(o)-* vessel + *-ar* pertaining to] tissues. That, by the way, is a one-way ticket. Once out of the bloodstream, they can never return. So, we always have cell turnover (loss and replacement). In health, this should maintain a relatively steady number of circulating neutrophils. Next, we need to consider which species of animal has this infected bite wound. Under normal circumstances, dogs and cats have more circulating neutrophils than any other type of *leukocyte*, with a greater ability to rebound in response to a disease insult, compared to large animals. They also have a nice supply of marginated neutrophils, especially cats. So, in a dog or cat, circulating and marginated cells could meet the initial demands of the contaminated wound, perhaps without a significant impact on total numbers in circulation—especially with those in reserve to quickly replenish what's lost. Make note that large

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animals, especially ruminants, have fewer circulating and marginated neutrophils from the get-go. So, their neutrophils will deplete more rapidly.

Then we need to think about our reserves. Beyond circulating and marginated cells, we do have a large number of neutrophils in the maturation and storage ("holding") area of the bone marrow. Neutrophils in "holding" could represent approximately 80% of those in the bone marrow and may give us about a 4- to 5-day supply, depending on the rate of cell turnover. If the wound infection spirals out of control, we may see an abundance of neutrophils (including immature cells) released into circulation. This would result in a *neutrophilic* ***leukocytosis*** [*leuk(o)-* white + *cyt(o)-* cell + *-sis* a condition of; i.e., increased numbers of WBCs, in this case primarily neutrophils] with a *left shift.* We could also say that the patient has a ***neutrophilia*** [*neutr(o)-* neutral + *phil(o)-* loving + *-ia* condition of; i.e., excess numbers of neutrophils], with a *left shift.* (However, the latter phrase doesn't speak to the overall abnormal increase in WBCs, as *neutrophilic leukocytosis* does.) Hopefully, supply and demand will balance out. If not, we could rapidly deplete most available neutrophils. If this happens, the ***neutropenia*** [*neutr(o)-* neutral, neutrophil + *-penia* deficiency] that develops may leave our patient with little ability to fight the raging infection. Yes, ***granulopoiesis*** [*granul(o)-* granulocyte + *poie(o)-* production + *-sis* process of], especially in response to things like ***interleukins*** [*inter-* between + *leuk(o)-* white + *-in* the] secreted by dying neutrophils, will demand that the bone marrow produce and release more. Unfortunately, once the stem cells are stimulated, there may be a 3- to 5-day delay before we see an appreciable new supply of neutrophils in circulation.[6] As you can see, there is much involved. It all boils down to supply and demand with these "first-responder" defenders of the body. While neutrophils may be the most abundant granulocyte, they are not the only granulocytes of the body.

Eosinophil

Are you thinking that ***eosinophils*** [*eosin(o)-* red + *phil(o)-* loving] are *granulocytes* with red cytoplasmic granules? You are correct! You'll notice, when you look at Fig. 3.11, that the two eosinophils shown

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do not have the same number or size of red granules. There is wide species variation in this regard. *Canine eosinophils* have variably sized round granules. *Feline eosinophils* are full of small rod-shaped granules. *Ruminant* and *porcine eosinophils* have small uniform, round granules. *Equine eosinophils* are my favorite. Their eosinophils are packed full of large granules, making them look like plump raspberries.

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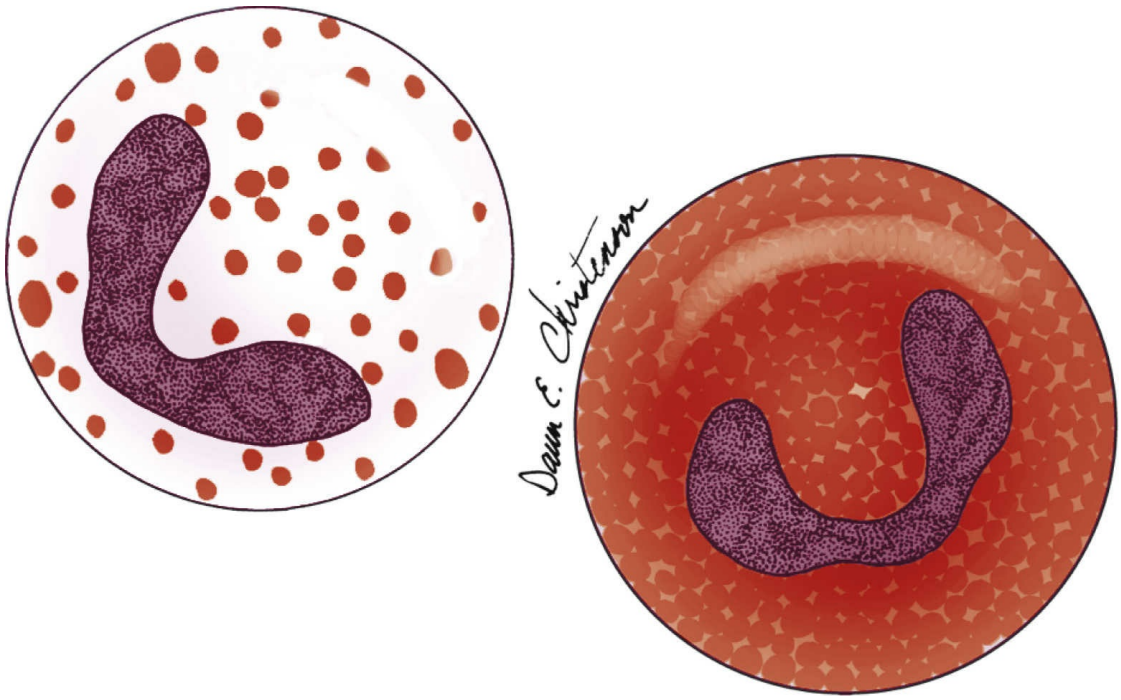

FIG. 3.11 Eosinophils.

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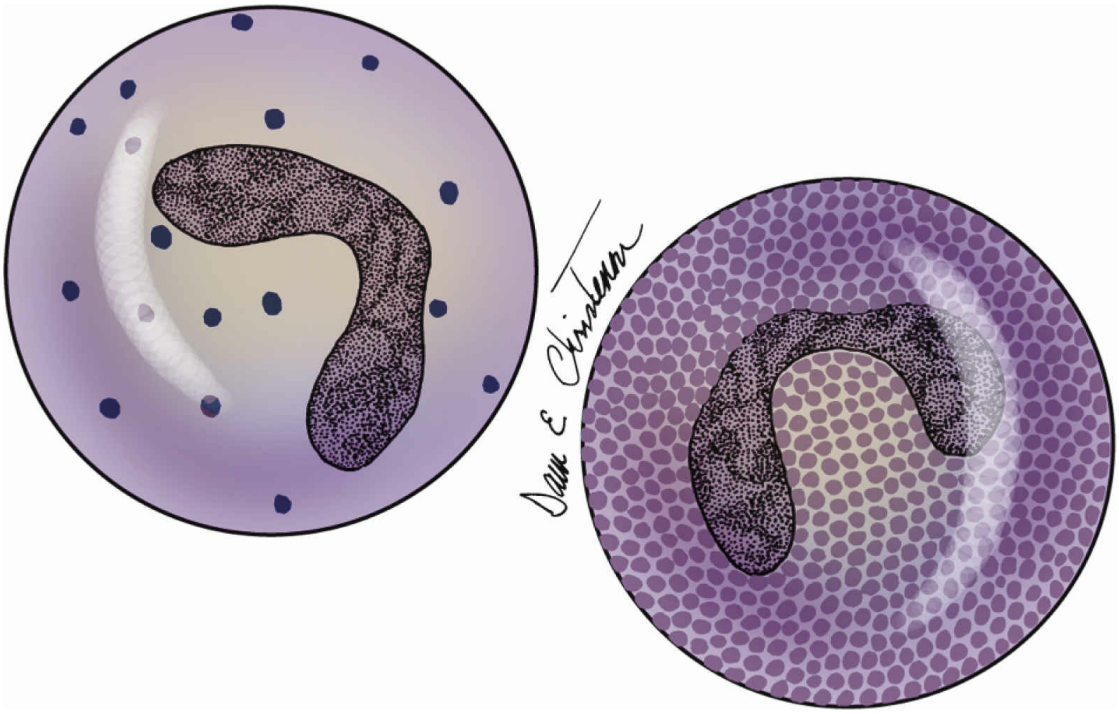

FIG. 3.12 Basophils.

Other than morphology, how do *eosinophils* differ from neutrophils? Well, eosinophils are not seen very frequently. In fact, compared to most other *leukocytes*, eosinophils are few and far between in normal animals. The only leukocyte that we see fewer of than eosinophils are ***basophils*** [*bas(o)-* blue + *phil(o)-* loving]. We'll discuss those, after eosinophils. You see, eosinophils are specialists. Their areas of "expertise" are allergic conditions and parasitic infestations, especially ***helminths*** [hel'minths; from Gr. *helmins* worms]. In these circumstances, we frequently see ***eosinophilia*** [*eosin(o)-* red + *phil(o)-* loving + *-ia* condition of; i.e., increased numbers of eosinophils]. Yes, eosinophils can kill helminths. They also contribute to inflammation associated with allergic conditions. Bacteria are not their thing. They leave the bacterial battles to the neutrophils. By the way, their production and maturation sequence are very similar to neutrophils.

Basophil

The *basophil* is the last *granulocyte* to discuss. As their name implies, basophils have bluish-purple colored cytoplasmic granules (Fig. 3.12). As with eosinophils, there are species variations. Granules of *feline basophils* tend toward a light blue-purple. Other domestic animal basophils have dark purple staining granules. As to

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numbers of granules, the *canine basophil* shows the most variation, with only a smattering of granules. Other domestic animal basophils have abundant cytoplasmic granules.

Basophils are probably the rarest *leukocyte* of the body. Like eosinophils, basophils are specialists. We tend to see ***basophilia*** [*bas(o)-* blue + *phil(o)-* loving + *-ia* condition of; i.e., increased numbers of basophils] most commonly in allergic conditions. Some of their granules contain histamine, contributing to inflammation associated with allergic conditions. Allergic reactions will be discussed with *immunology*, later in this chapter. Like their fellow granulocytes, basophils have a similar production and maturation sequence.

Agranulocytes

Agranulocytes [*a-* without + *granul(o)-* granules + *cyt(o)-* cells] do not have distinctive cytoplasmic granules, as the name implies. Their cytoplasm tends to look quite homogenous and, frankly, rather boring compared to granulocytes.

Monocyte

Monocytes [*mono-* one + *cyt(o)-* cell] are the largest of the *leukocytes* (Fig. 3.13). They tend to have a large amount of homogenous, grayish cytoplasm surrounding a large, ***pleomorphic*** [*ple(o)-* more + *morph(o)-* shape + *-ic* pertaining to] nucleus. The images in Fig. 3.14 demonstrate both cellular size and the *pleomorphic* nature of the monocyte's nucleus. Note how open and loose the nuclear chromatin is, especially compared to the mature neutrophil shown. Monocytes may or may not have vacuoles in their cytoplasm. The longer they are exposed to EDTA, the more likely vacuoles will develop. In fact, prolonged exposure to EDTA can create vacuoles in numerous leukocytes.

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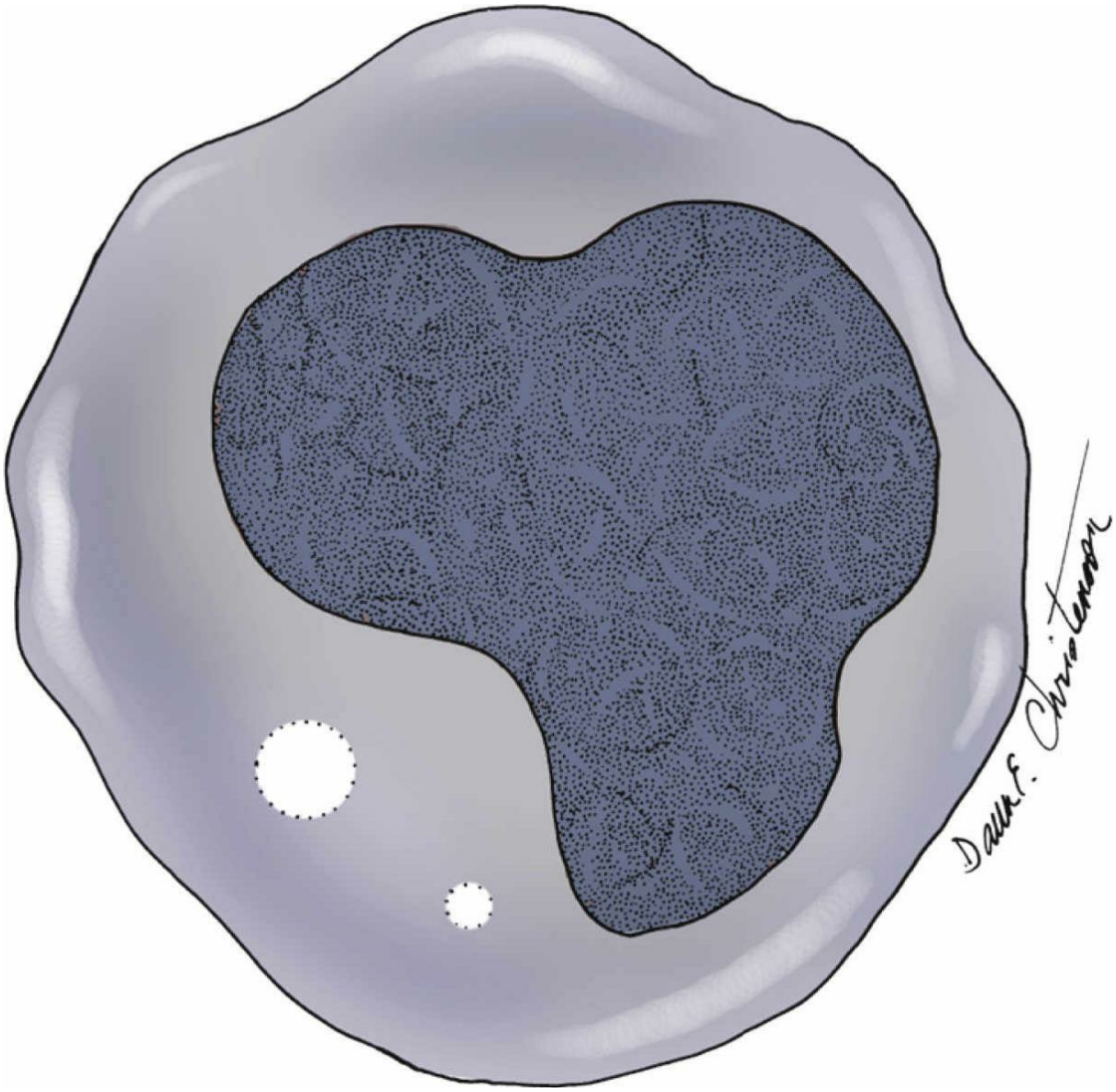

FIG. 3.13 Monocyte.

In *hematology*, the *monocyte* falls in third place, under normal circumstances, with regard to numbers in circulation. Aha, but monocytes do not tend to remain in circulation! They leave the bloodstream in droves, to populate key areas of the body, including the lungs, chest and abdominal cavities, spleen, liver, and lymph nodes, to name a few. Once they leave the bloodstream, we no longer call them monocytes; we call them ***macrophages*** [*macr(o)-* large + *phag(o)-* eat, eater]. Yet they're really the same cell doing the same things, just named differently based on location. Simply remember: *monocytes* are in the bloodstream; *macrophages* are in the tissues.

As the name *macrophage* implies, these cells *phagocytize* pathogens, cellular debris, and foreign particulate matter. As we mentioned in our discussion of *poikilocytosis*, *splenic macrophages* are

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important for *erythrocytic* repairs, as well as removal of really old *erythrocytes*. Do you remember the hypothetical open wound that we discussed with the neutrophil? Well, we would find *macrophages* in that wound too, contributing to the *purulent exudate* (pus). In a wounding situation, macrophages not only mop up the mess left by neutrophils and the trauma itself, they also secrete a number of ***chemotactic*** [*chem(o)-* chemical + *-tactic* pertaining to movement directed by; i.e., chemical to promote *chemotaxis*] substances and growth factors important to tissue repair in wound healing.

As we said in our discussion of *neutrophils*, they are the first responders to injuries and *pathogens*. *Monocytes* will follow, in a somewhat delayed manner, from the initial insult of pathogens or injury. So if we see ***monocytosis*** [*mono-* one + *cyt(o)-* cell + *-sis* condition of; i.e., increased numbers of circulating monocytes], we can generally assume that the *pathology* involved has been there for at least a few days, maybe longer. You see, unlike neutrophils, *monocytes* have no storage holding area in the bone marrow. So any new monocytes entering circulation are purely from stimulation of stem cells and ***monoblasts*** [*mono-* one + *blast(o)-* shoot/germ]. Replenishment takes time. Looking at the flip side of the coin, we can assume that ***monocytopenia*** [*mono-* one + *cyt(o)-* cell + *-penia* deficiency of; i.e., deficiency of monocytes] could leave a patient with a limited defense system. It most certainly will delay healing of a wound, like the one discussed earlier. This could be problematic, especially for a surgical patient. Fortunately, monocytes tend to remain in circulation for nearly a day.[7] Under normal circumstances, then, we can maintain relatively stable numbers in circulation, before they eventually migrate out into the tissues.

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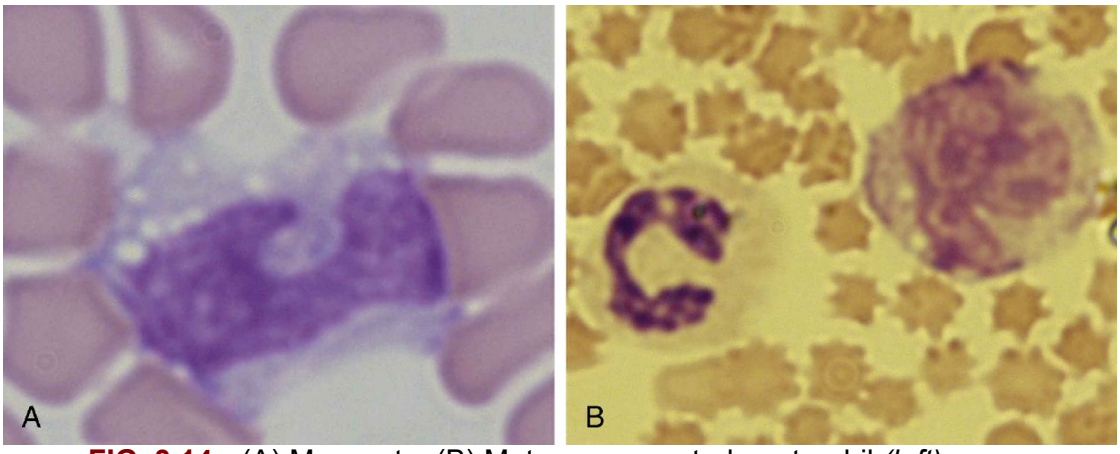

FIG. 3.14 (A) Monocyte. (B) Mature segmented neutrophil *(left)* and Monocyte *(right)*.

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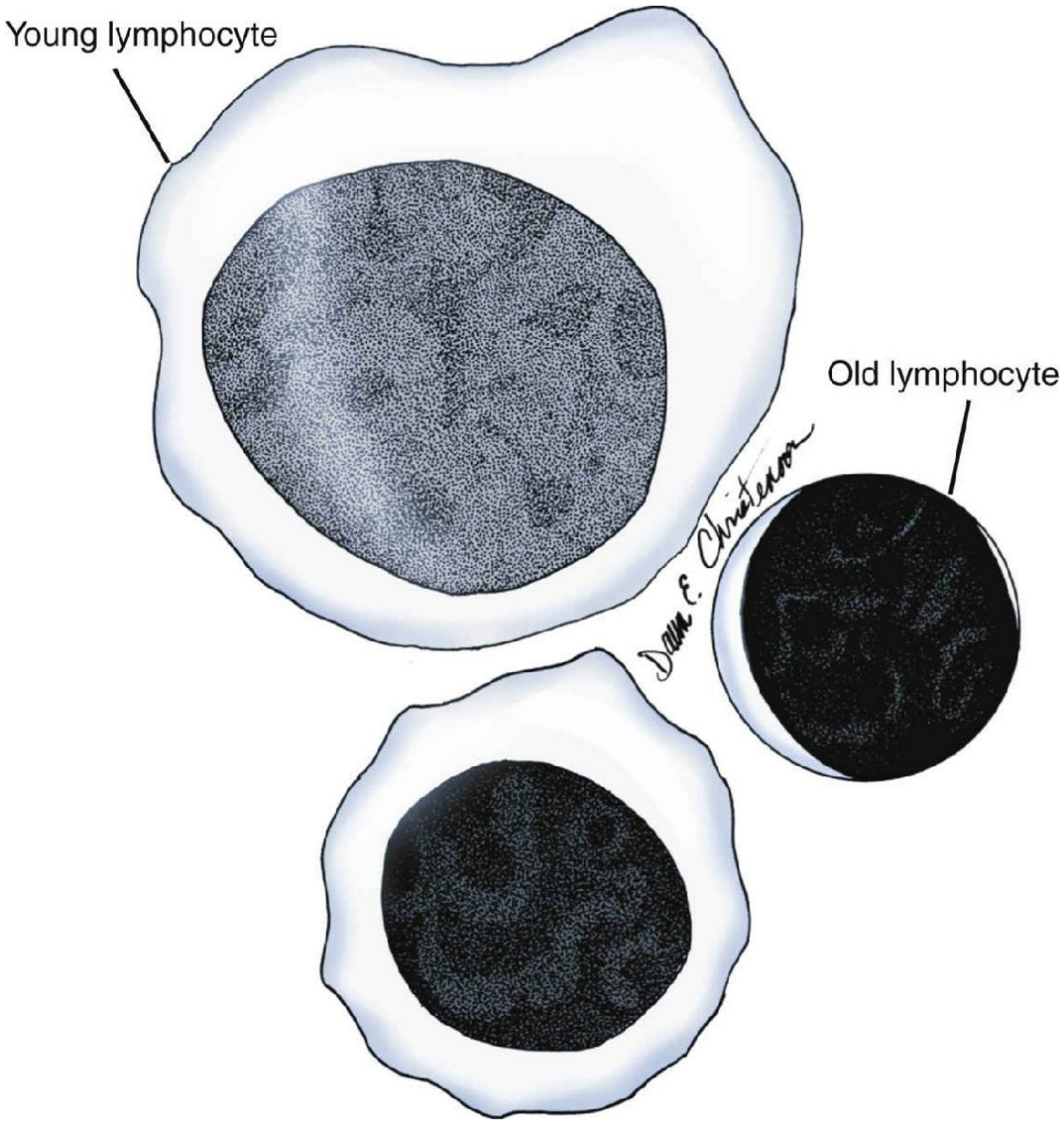

FIG. 3.15 Lymphocytes.

Lymphocyte

Lymphocytes [*lymph(o)-* lymph/"water" + *cyt(o)-* cell] are extremely important in ***immunity*** [*immun(o)-* exemption/protection + *-ity* the state of]. Remember this, when we get to *immunology* in a little while. We're almost there. Right now, looking at the basic definition of *lymphocyte*, you might be wondering what water has to do with it. Well, lymphocytes are closely associated with the ***lymphatic*** [*lymph(o)-* "water" + *-tic* pertaining to] *system*, which just happens to transport a watery fluid (lymph) through lymphatic vessels. But before we dig deeply into that system, let's stay focused on the cell in question. So, as with the other *leukocytes*, let's begin by looking at

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lymphocytic morphology.

Lymphocytes in peripheral blood vary in size, nuclear chromatin, and nuclear-cytoplasmic ratio (Fig. 3.15). Don't panic. These are merely variations on a theme that are related to the age of the cell. If you want consistency, it's worth noting that regardless of age, the nucleus of lymphocytes is usually round or oval. Younger lymphocytes tend to be larger cells, with more cytoplasm and a larger nucleus (i.e., greater nuclear-cytoplasmic ratio). The nuclear chromatin of these young cells is less condensed, giving it a little lighter staining quality. As lymphocytes age, they tend to "shrivel up" in a way. The nuclear chromatin becomes very condensed and dark. Likewise, the cytoplasmic volume is reduced. In fact, really mature (i.e., old) lymphocytes may have only a scant (tiny) amount of cytoplasm, like the smallest one shown in Fig. 3.15. How old is old in lymphocytic terms? Well, most lymphocytes only live about 2 weeks.[8] That's pretty long, compared to all of our other leukocytes! Other lymphocytes, like memory cells and those that periodically engage in mitosis, may live months to years. That's a mighty long time!

The cytoplasm of lymphocytes is usually transparent and colorless, often with a thin *basophilic* rim near the outer edges of the cellular membrane. That marginal *basophilia* (blueness) tends to become more pronounced where the lymphocyte's cell membrane touches other cells. This is probably because that portion of the cytoplasm and cellular membrane are a bit thicker, where they're touching the other cell. It's a different story for ***reactive lymphocytes***. *Reactive lymphocytes* have intensely basophilic cytoplasm. Why are they reactive? They're working hard, responding to ***antigenic*** [*anti-* antibody + *gen(o)-* producing + *-ic* pertaining to] stimulation. Think of it this way. When you're physically working really hard, you probably get really flushed (red) looking, right? Well, when lymphocytes are working really hard, they turn blue. This makes sense when I think of all of the activity of ribosomes and their production of proteins (for antibodies). Those things tend to pick up the basophilic stains typically used in hematology.

As to production and maturation of lymphocytes, this is very different from our other leukocytes. All of the leukocytes discussed

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thus far are produced in the bone marrow. Not all lymphocytes originate from the bone marrow. *Embryologically* [*embry(o)-* embryo + *log(o)-* knowledge, study + *-ically* pertaining to], B lymphocytes originate there, while T lymphocytes originate primarily in the thymus (thi'mus). (We'll discuss the thymus more, when we talk about lymphatic anatomy and physiology.) Beyond that, most of the lymphocytes seen circulating in adult animals originate in secondary ***lymphoid*** [*lymph(o)-* lymph + *-oid* resembling] tissues, like the spleen and lymph nodes. For now, simply remember that there are two main functional types of lymphocytes (B lymphocytes and T lymphocytes). B lymphocytes handle the big job of ***immunoglobulin*** [*immun(o)-* protection + *globul(o)-* "blob", globule + *-in* the; i.e., antibody] production, including IgG, IgA, IgM, and IgE. The "I" in each of the antibodies listed stands for immunoglobulin. T lymphocytes produce *chemotactic* substances to guide movement of other lymphocytes, as well as attract other leukocytes. T lymphocytes are also involved with cellular immunity.

Wow, lymphocytes sound unique, don't they? And that's just the tip of the iceberg! All of our other leukocytes were produced in the bone marrow and put into circulation on a one-way ticket to eventually go out into *extravascular* tissues where they likely die. Not lymphocytes—they earn "frequent flyer miles" because they recirculate! That's right. A typical lymphocyte's journey would be enter the bloodstream from lymphoid tissue (e.g., spleen); leave the bloodstream, eventually leave to be picked up by lymphatic vessels; travel through lymphoid tissue (e.g., lymph node); and, at some point, go back into the bloodstream. Most of the lymphocytes recirculating are probably T lymphocytes. B lymphocytes tend to be homebodies, hanging out in lymphoid tissue. They get out and about too, just not as much or as regularly as T lymphocytes. There are also some **T** lymphocytes that **T**arget virus-infected cells. Of course, they need to be sensitized to the virus involved, for recognition. If they recognize it, the T cells (lymphocytes) will **T**arget the infected cell and be ***cytotoxic*** [*cyt(o)-* cell + *tox(o)-* poison + *-ic* pertaining to], eliminating the infected cell AND the virus inside it. This is part of the cellular immunity I mentioned earlier. Hmmm, maybe that's a good way to keep B and T lymphocytes straight—**T** lymphocytes **T**ravel and **T**arget, and **B** lymphocytes are

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home-**B**ound. Yeah, I like that.

One last discussion about *lymphocyte* numbers and I'll shut up about them. We said earlier that *neutrophils* are the predominant *leukocyte*, in normal, healthy dogs, cats, and horses. *Lymphocytes* are second in numbers for those animals. The opposite is true in adult ruminants, in whom *lymphocytes* are the predominant WBC and *neutrophils* are second. I point this out because through the years I have had many students report ***lymphocytosis*** [*lymph(o)-* lymph + *cyt(o)-* cell + *-osis* a condition of; i.e., abnormally increased numbers of lymphocytes] after evaluating ruminant blood smears. Those patients had normal numbers of lymphocytes. The students simply got too used to seeing a predominance of neutrophils in other types of animals. You always need to keep species variation in mind, even in the laboratory. Do I memorize normal *hematology* values for domestic animals? Heavens no. What if I remember the wrong numbers for the wrong species? I have always kept hematology charts with species normal ranges close at hand for my reporting. Accuracy is important. Without it, I may lead the veterinarian astray in her or his diagnosis. What if I worked in a large-animal veterinary practice and was very used to looking at bovine and ovine blood smears? On the rare occasion that I might look at a canine sample, I could easily mistakenly report ***lymphopenia*** [*lymph(o)-* lymph + *-penia* deficiency of; i.e., deficiency of lymphocytes]. This could lead the veterinarian to look for some sort of immunodeficiency that just doesn't exist. Never lose sight of the importance of accuracy.

Do you see why I got excited at the beginning of this section? Look at all of the amazing things that *leukocytes* do. Each one has its area of expertise, but they all work collectively to protect the body. Now, just imagine a patient with ***leukopenia*** [*leuk(o)-* white + *-penia* deficiency; i.e., a deficiency of WBCs]. Think of how that condition would leave the patient at risk for infectious disease. Relatively harmless pathogens, for healthy individuals, could become lethal (deadly) for ***leukopenic*** [*leuk(o)-* white + *-penic* pertaining to deficiency] patients.

Well, that covers *erythrocytes* and all of the *leukocytes*. I know that's an awful lot of information, with so many details. I am sorry, but I'm just the messenger. Unfortunately, this is the nature of the

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body and veterinary medicine. If you haven't done so yet, take a break before we move on to the next section. Clear your mind, stretch, eat or drink something, and maybe do a little deep breathing to relax. There, do you feel refreshed and ready to move on? Good. Let's look at the last cell type found in blood—platelets.

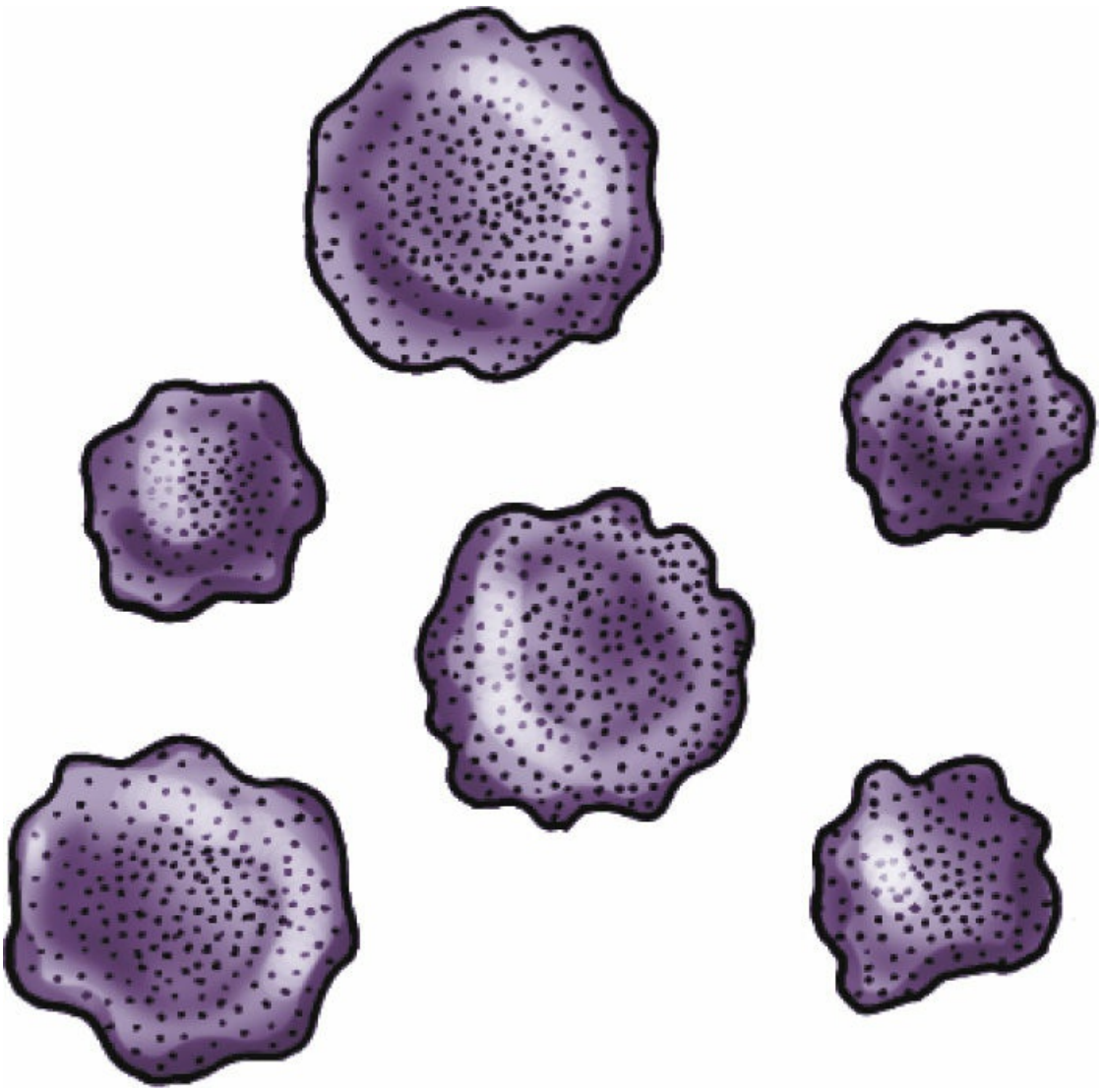

FIG. 3.16 Thrombocytes.

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Applied Platelet Terminology

Thrombocytes [*thromb(o)-* clot + *cyt(o)-* cell] or platelets in mammals are not actually complete cells. (In birds, they are whole cells.) We're going to focus exclusively on mammals here. In mammals, *thrombocytes* found in the blood are *cytoplasmic* fragments of ***megakaryocytes*** [*mega-* large + *kary(o)-* nucleus + *cyt(o)-* cell]. *Megakaryocytes* are found in the bone marrow. Like most of the other blood cells we've discussed so far, megakaryocytes are produced by stem cells. After much mitosis and maturation, the final-stage megakaryocytes in the bone marrow are huge, with abundant granulated cytoplasm. Fragments of their cytoplasm are released into circulation. These tiny fragments are what we see in peripheral blood smears, as *thrombocytes* or *platelets*.

The *morphology* of *thrombocytes* resembles the cytoplasm of the megakaryocytes: pinkish-lavender with numerous granular specks (Fig. 3.16). Size of platelets is variable, yet all should be much smaller than an erythrocyte. *Large forms* of platelets, approximately half or more of the size of an erythrocyte, are newer, recently released fragments. Increased numbers of large forms may indicate increased loss of platelets, which may indicate bleeding. Food for thought, when we see them.

Functionally, platelets are involved with ***hemostasis*** [*hem(o)-* blood + *-stasis* stoppage; i.e., clotting]. We'll discuss *hemostasis* in detail in the next section. For now, I will try to briefly explain the role platelets play in that whole process. Let's use a relatable example, like a paper cut. We've probably all had those. We know that the paper has damaged blood vessels, because we bleed, right? Well, the injury to the vessels exposes the blood to tissue, like ***collagen*** [Gr. *kolla* glue + *gen(o)-* produce, producer]. Platelets exposed to the *collagen* will become activated and stick to it and to each other like glue. (Note: von Willebrand's factor [vWF] is a protein needed for platelets to actually stick to the collagen.) Activation of these platelets will stimulate activation of more platelets in the area, until enough platelets have aggregated (i.e., clustered, crowded) in the site as a *platelet plug*. This literally plugs the open ends of the severed vessels, stopping the bleeding. Have

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you ever scratched or picked at the scab that forms in an injury like that? (You know who you are.) When that scab is removed prematurely, the platelet plug is removed with it and the clotting process needs to start all over again. So don't do it. Let things heal in their own good time. That scab will eventually deteriorate and fall off on its own.

Beyond disturbing the platelet plug that forms in an injury, there are ways that we can actually interfere with platelet function. Commonly used medications, like aspirin, many other nonsteroidal anti-inflammatory drugs (NSAIDs), and others can interfere with platelet function. These drugs interfere with platelet adhesion (sticking), necessary for aggregation and formation of the platelet plug. With this kind of interference in platelet function, bleeding will be prolonged. This is why surgeons always want patients off of drugs like these long enough prior to surgery. If patients (human) or owners of veterinary patients don't comply with the surgeon's directive, there could be significant, even life-threatening ***hemorrhage*** [*hem(o)-* blood + *-rrhage* escaping; i.e. bleeding] during the surgical procedures. We'll talk more about this in our discussion of *hemostasis*. Before we go there, let's talk about platelet numbers.

Thrombocytopenia [*thromb(o)-* clot + *cyt(o)-* cell + *-penia* deficiency; i.e., a platelet deficiency] can result in prolonged bleeding. Why would we have a platelet deficiency? Oh, there are a number of things that could cause that. ***Oncology*** [*onc(o)-* tumor + *-logy* knowledge, study of; i.e., cancer] patients receiving certain chemotherapy drugs often develop *thrombocytopenia*. Some chemotherapy drugs suppress the bone marrow and platelet production. This reduces our supply. We've talked about supply and demand before. Really, any imbalance of supply and demand of platelets could cause *thrombocytopenia*. On the demand side, we could have a ***coagulopathy*** [*coagul(o)-* clot, clotting + *-pathy* disease process/condition], like ***disseminated*** (widespread) ***intravascular coagulopathy*** (**DIC**), that uses up all available platelets and clotting factors. It may take close to 5 days before we have new platelets in circulation. Of course, in DIC, there is much more to worry about than simply a platelet deficiency. It depends on the underlying cause of the DIC, but most animals don't survive it. Another

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possibility would be an immune-mediated (i.e., caused by the immune system) problem. I have seen quite a number of patients with *immune-mediated thrombocytopenia*. These patients produce *antibodies* against their own platelets, resulting in their destruction. We'll talk more about ***autoimmune*** [*auto-* self + *immune*] disorders in the immunology section.

Finally, we could have a problem with supply, caused by a ***myelopathy*** [*myel(o)-* marrow + *-pathy* disease process/condition]. This could affect just the *megakaryocytes*, resulting in *thrombocytopenia*. Of course, with some ***neoplastic*** [*ne(o)-* new + *plas(o)-, plast(o)-* to form + *-tic* pertaining to; i.e., cancer] *myelopathies*, all cell lines could be affected, resulting in ***pancytopenia*** [*pan-* all + *cyt(o)-* cell + *-penia* deficiency]. Obviously, none of these scenarios are good. In fact, they are very serious conditions. Do you remember our discussion early in this chapter about blood handling and adequate mixing of blood samples with an *anticoagulant*? We need to be absolutely certain that the blood has been collected and handled (mixed) appropriately before we go down the path of true *thrombocytopenia*. Delayed or inadequate mixing of blood with the anticoagulant could result in platelet clumping and partial clotting in the sample. This will artificially reduce numbers of platelets observed and counted, and that could lead to an incorrect diagnosis, costing the animal its life. Remember, in veterinary medicine, if owners receive a grave diagnosis for their animal, they may opt for ***euthanasia*** [*eu-* good + *thanas(o)-* death + *-ia* process of] to prevent or minimize suffering. So we need to be sure that we always collect and handle blood samples appropriately, to prevent misdiagnosis due to faulty laboratory data.

As you can see, platelets are important to *hemostasis*. But they are only one part of the larger ***hemostatic*** [*hem(o)-* blood + *-static* pertaining to standing, inhibiting] process. There is much more to that story, to be discussed next.

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Applied Terminology for Bleeding and Hemostasis

Hemostasis, as you already know, is the process of stopping bleeding. (Watch the Evolve animation, "Hemostasis," for a basic overview of the process.) If there is bleeding, we know that there is *vascular* injury. Of course, *vascular* injury doesn't always mean that there is blood dripping all over the floor. Have you ever bumped a knee or other body part against an immovable object like a dresser or cabinet? The skin didn't break, but the ***hematoma*** [*hemat(o)-* blood + *-oma* swelling] that developed at the site of impact was a clear indicator that vessels were damaged. Bleeding in a *hematoma* is simply less messy than slicing a finger with a kitchen knife. Seriously, though, *hemorrhage* from some injuries can be life-threatening. That in mind, it seems like it would be good idea to have built-in redundancies and multiple players involved in *hemostasis*. That way, if one element fails or is inhibited by something (like platelets are by aspirin), then we'll have enough other mechanisms available, hopefully, to ultimately stop the bleeding. For this discussion, keep Fig. 3.17 close at hand. We'll refer to many of the elements demonstrated in that schematic as we move along. I find the visual of that schematic helpful. I hope you do too.

We've already established that if vessels are damaged, bleeding will occur. One important event, in response to the injury, will help minimize the size of the clot needed to seal off the vessel. That event, noted at the top of the schematic, is ***vasoconstriction*** [*vas(o)-* vessel + *constriction* contracting (i.e., making the *vascular* opening smaller)]. Think about it. It is much easier to seal off the end of a drinking straw than a fire hose.

Now, if you're looking at the schematic you see two *hemostatic* pathways that eventually converge into a single path and the final clot formation. Here is where redundancy is of value. In a normal animal, when a vessel is damaged, both pathways will be engaged. I can't think of a single situation, in a normal, healthy animal, that only one pathway would be stimulated independently of the other.

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That said, I am going to discuss the ***extrinsic*** [*ex-* out, outside; *extrinsic* pertaining to being situated outside] and ***intrinsic*** [*in-* within, inside; *intrinsic* pertaining to being situated inside] pathways separately. Just bear in mind that they are simultaneous events. Once set in motion, these events tumble along, like rows of falling dominos. Please note that the most important organ in the body for protein and clotting factor production is the liver. Patients with severe liver disease will likely have impaired *hemostasis*, due to clotting factor deficiencies.

It is also important to note that each of the clotting factors produced by the liver are found in circulating plasma in their inactive form. Each inactive factor must be activated to its active form before the next "domino" can be activated and fall, so to speak. For instance, the inactive form of clotting Factor 2 is ***prothrombin*** [*pro-* before + *thromb(o)-* clot + *-in* the]. Once it is activated, it becomes ***thrombin*** [*thromb(o)-* clot + *-in* the]. ***Fibrinogen*** [*fibrin(o)-* fiber, fibrin + *gen(o)-* producer] is another good example of an inactive form of a clotting factor. If you remember, we said that *fibrinogen* is the difference between plasma and serum. As you can see from the schematic, once *fibrinogen* is activated, it becomes fibrin, in the final clot. And in terms of a blood sample, this leaves us with serum.

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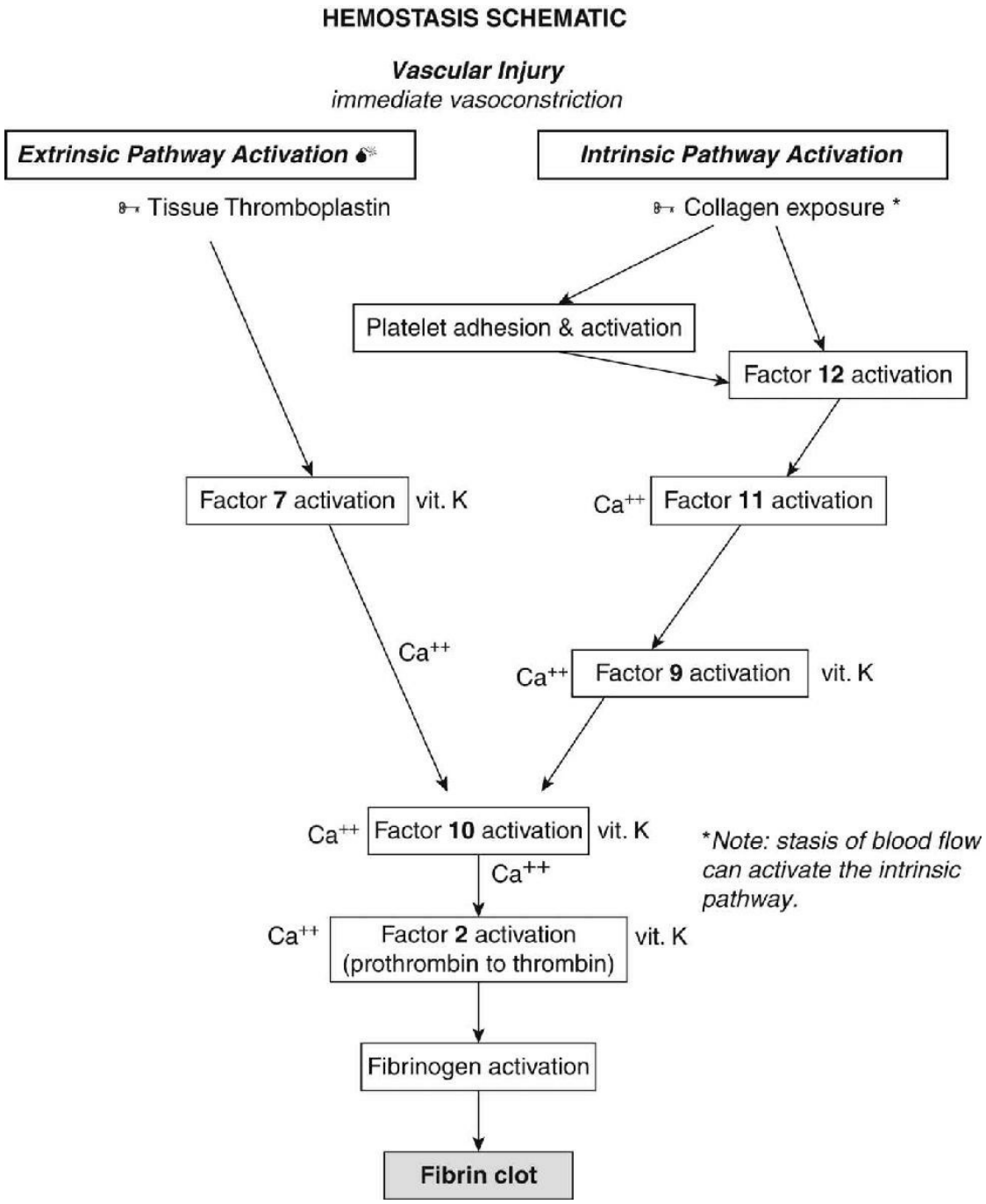

FIG. 3.17 Hemostasis schematic.

Because we have already talked about *platelets* and *collagen*, let's begin with the ***intrinsic pathway*** (far right of the schematic in Fig. 3.17). Why intrinsic? Because we are talking about those things within the blood vessel. The key to activating the intrinsic pathway is exposure of the blood to collagen, as we learned earlier. Understand, that underneath the slippery ***endothelial*** [*end(o)-* within + *thel(o)-* nipplelike + *-al* pertaining to] cell lining of every blood vessel is collagen. Notice that exposure to collagen activates

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both platelets and clotting Factor 12. And once platelets are activated, they contribute even more to activation of other platelets and Factor 12. From this point forward, Factor 12 activation leads to sequential activation of subsequent clotting factors, through the common pathway.

The ***extrinsic pathway*** (far left of the schematic in Fig. 3.17) tends to be way more explosive. Notice that it is exposure of blood to *tissue thromboplastins* that initiates the cascade of events along this pathway. The more cells and tissues are damaged, the more *tissue thromboplastins* are released from the cells, creating an explosive progression of ***coagulation*** [*coagul(o)-* clot + *-tion* the process of; i.e., clotting]. This is partly the reason why crushing injuries don't bleed as much as clean cuts—there is much more tissue thromboplastin exposure. The other reasons crushing injuries don't bleed as much are (1) there is more vascular damage, exposing more collagen for activation of the intrinsic pathway, and (2) the vessels have been crushed and occluded. All of these influences contribute to the value of *hemostatic forceps* in surgery. Regardless, when thinking of the explosive nature of the extrinsic pathway, it helps that we only have to activate Factor 7 and proceed to the common pathway. Once we reach the common pathway, we are only a few Factor activations away from a clot.

Did you notice, at various places in Fig. 3.17, that both calcium (Ca^{++}) and vitamin K (vit. K) are listed? We must have sufficient quantities of both for normal *hemostasis*. As you may recall, we talked briefly in Chapter 2 about various electrolytes. Ca^{++} is an important electrolyte for many activities in the body, not the least of which is *hemostasis*. Of course, we can and do use its importance in *hemostasis* to our advantage. For example, *the anticoagulant* EDTA chelates (binds up) calcium. That's how it prevents blood from clotting in that lavender-top tube. Warfarin, on the other hand, is an *anticoagulant* that interferes with vitamin K. You see, vitamin K is needed for the liver to produce clotting Factors 7, 9, and 10. With warfarin tying up vitamin K, those Factors (obviously essential for clotting) cannot be produced. Believe it or not, warfarin is the active agent in the drug Coumadin. In human medicine, Coumadin is frequently used to treat ***thrombosis*** [*thromb(o)-* clot + *-osis* a condition of; i.e., abnormal clotting somewhere in the vasculature].

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Most commonly people develop ***thrombus*** [*thromb(o)-* clot + *-us* a] formation in a large deep vein (DVT—deep vein thrombosis) in a leg. Those being treated with Coumadin need to avoid certain foods, like spinach, that are rich in vitamin K, because too much dietary vitamin K may diminish the effectiveness of the drug. In veterinary medicine, we don't treat patients for *thrombosis* as frequently as they do in human medicine. Most of the time when we deal with warfarin, it's a poisoning situation. Some ***rodenticides*** [*rodent(o)-* rodent (i.e., mice, rats) + *-cide* to kill] contain warfarin. These agents kill rodents by interfering with hemostasis, causing the animals to bleed to death. If a dog or a cat inadvertently eats the warfarin-based *rodenticide*, it can experience life-threatening *hemorrhage*. If pet owners seek treatment immediately, we can minimize absorption of the warfarin and prevent *hemostatic* complications. We may need to follow up with supplemental vitamin K for an extended period of time. Unfortunately, sometimes our intervention is too late to save them.

I hope these examples show you how our knowledge of hemostasis can be valuable. It's valuable with diagnostic testing too. In any patient presenting with a *coagulopathy*, we can do various diagnostic tests to try to isolate where the problem lies along those pathways. If we're suspicious of platelet deficiency or dysfunction (like with Von Willebrand's disease, vWF deficiency), we'll do a ***buccal*** [*bucc(o)-* cheek + *-al* pertaining to] ***mucosal*** [*mucos(o)-* mucus + *-al*] ***bleeding time*** (**BMBT**). The beauty of a BMBT is that it doesn't require much time or any fancy equipment. In short, we make a small cut in the gum of the patient and time how long it takes to clot. This is an easy, inexpensive, and effective diagnostic test. Yet it is limited. If the BMBT is normal, we know that platelets are functioning okay. But it tells us nothing about the other portions of the pathways. To look at Factors 12, 11, and 9, we use an **activated partial *thromboplastin*** [*thromb(o)-* clot + *plast(o)-* formation + *-in* the] **time** (**APTT**). If the APTT is prolonged, we know that we likely have a deficiency of one or more of those three clotting factors. If the APTT is normal, we may need to run a **prothrombin time** (**PT**). The PT evaluates Factor 7 by replicating the tissue thromboplastins. If only the PT is prolonged, then we know that we likely have a Factor 7 deficiency.

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Of course, other things may need to be evaluated too, like electrolytes, TP, and fibrinogen levels. That last one is very important. Even if we have all of the other pieces to the puzzle but have no fibrinogen, we can't form a clot. I've seen this happen in heat stroke patients. The extreme heat precipitates out all of the fibrinogen, effectively creating DIC (*disseminated intravascular coagulopathy*). Heat stroke patients may survive the extreme heat event, only to die days later from DIC. That is so sad, because heat stroke is preventable. (Never ever leave a dog in a car during warm weather, especially summertime! It takes only minutes to reach lethal levels, even with the windows partially open.) Finally, severe ***hypoproteinemia*** [*hypo-* low + *protein* + *-emia* a blood condition of] could create deficiencies of fibrinogen and other clotting factors. In either one of these examples (DIC and severe hypoproteinemia), probably all of our coagulation tests will be abnormally prolonged.

Wow! Did you ever dream there was so much to know about blood? Well, congratulations, you made it through the hematology portion of this chapter! We'll touch on some of this information periodically throughout the lymphatic portion of the chapter. So, when you're ready, let's get started.

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Applied Terminology for Lymphatic Anatomy and Physiology

The lymphatic system has many facets. I'll get us started with some brief discussions of important anatomic features, like lymph nodes and the spleen. Then we'll get down to business with immunology. Please note that the case study at the end of the chapter will apply information from the entire chapter (hematology and lymphatics/immunology). Because you are now familiar with hematology and the spleen is intimately involved with blood, let's begin our discussion with the spleen.

The Spleen

The spleen is a remarkable, tongue-shaped organ found in the abdominal (belly) cavity. It lies alongside the stomach (*rumen* in ruminants), in the left cranial abdomen (Fig. 3.18). It may extend along the curvature of the stomach, to approximately the ventral midline. The head of the spleen is somewhat protected by the caudal ribs. Sometimes, especially with ***splenomegaly*** [*splen(o)-* spleen + *-megaly* enlargement of], the spleen may extend well beyond the ventral midline, to the right side of the body as well as caudally. You'll find a brief overview of splenic structure and function in the Evolve animation "How the Spleen Operates."

The spleen is extremely *vascular*. Plus, it has a spongelike quality that permits it to store large amounts of blood. In the dog, for example, roughly 30% of the total blood volume may be stored there. (Remember this when we talk about GDV in Chapter 7.) This blood storage can come in real handy when there is a sudden need for it, like high-stress, fight-or-flight situations. In these situations, *splenic* smooth muscle contracts and squeezes blood back into circulation. As you know, the more *erythrocytes* in circulation, the greater our oxygen-carrying capacity. So, whether we're running for our lives (like I do when I see a spider) or standing our ground to fight a foe, the spleen can turbo-boost our circulating blood volume. How cool is that? But wait, there's more!

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The spleen is a one-stop-shopping destination for the blood. In blood storage sinuses, we also have *splenic macrophages*. We mentioned those earlier. These splenic macrophages *phagocytize pathogens*, as well as damaged, diseased, or old worn-out erythrocytes. Remember when we spoke about *poikilocytosis*? Well, here in the spleen is where macrophages will make necessary repairs to RBCs, sort of like a body shop and car wash. The macrophages "detail" the erythrocytes, making them all shiny-pretty (and functional), so they can "hit the road" in circulation for transporting more oxygen. But that's not all!

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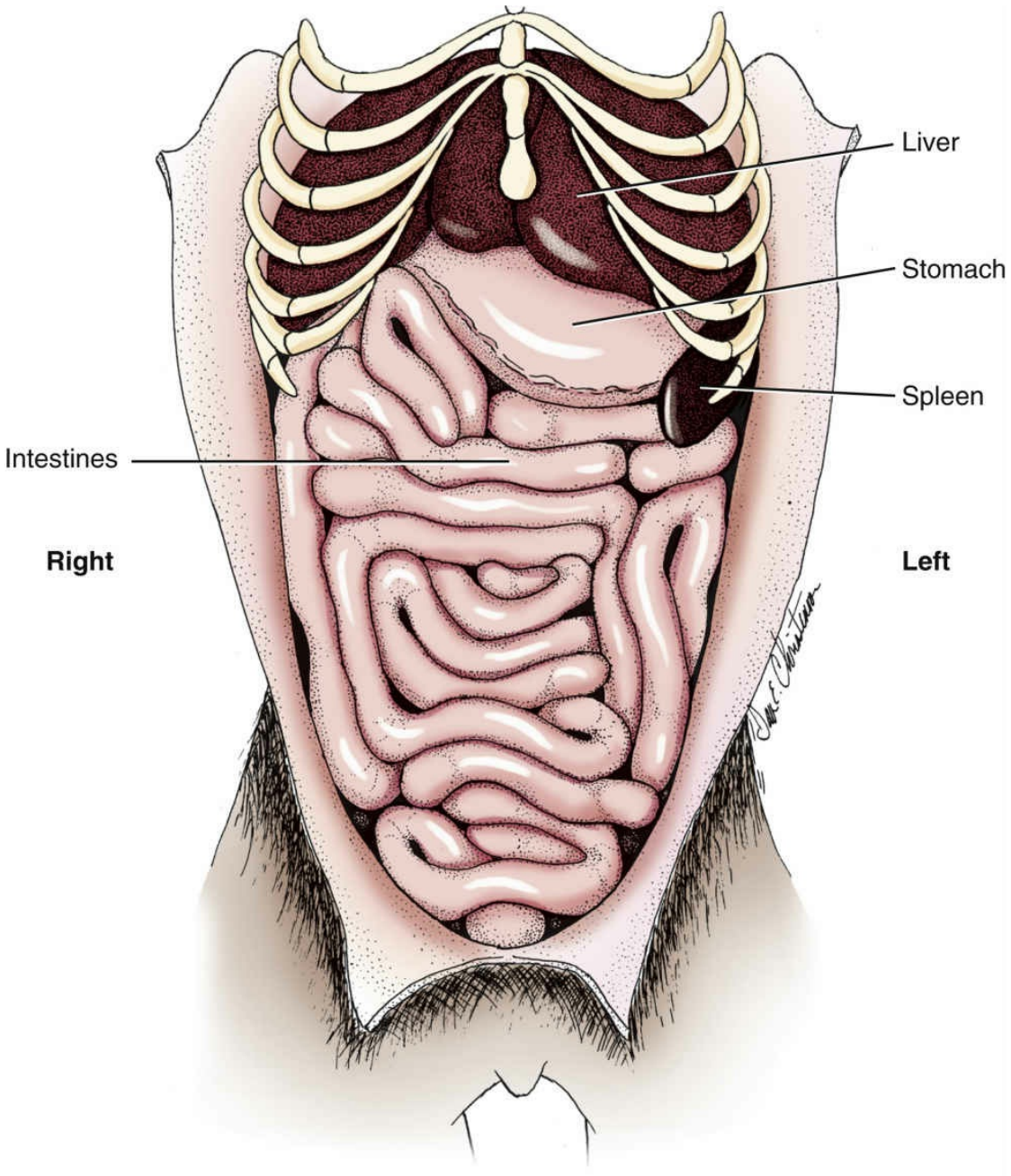

FIG. 3.18 Spleen in a dog's abdomen.

The spleen also has ***lymphoid*** [*lymph(o)-* lymph + *-oid* resembling] tissue (commonly called ***white pulp,*** because of its white appearance). You can actually see these areas grossly, scattered throughout the deep-red vascular tissue and sinus areas (i.e., ***red pulp***). The lymphoid tissue has lymphocytes, as you might expect. In immune (protective) responses, these lymphocytes, with their ***antigen***-specific [*anti-* antibody + *gen(o)-* producer; i.e., pathogen] receptors, will clone themselves. The new population of lymphocytes will provide protection against specific pathogens.

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More on that later, when we talk about immunity in detail.

It sounds like the spleen is a pretty important organ, doesn't it? It is. In spite of that, animals and people can actually live quite well following a ***splenectomy*** [*splen(o)-* spleen + *-ectomy* act of cutting out, removal of]. Why would we want to remove it? Well, sometimes it is damaged by severe trauma, putting the patient at risk of bleeding to death from the damaged spleen. *Splenectomy* may be the only way to stop the hemorrhage. Sometimes splenic macrophages become over-achievers in ***autoimmune*** [*auto-* self + *immun(o)-* protection] disorders, like *immune-mediated hemolytic anemia*. In this example, ***autoantibodies*** [*auto-* self + *antibody*] have been produced against the body's own erythrocytes. By removing the spleen, we significantly slow or stop immune-targeting and macrophagic destruction of erythrocytes. Finally, *splenic* ***neoplasia*** [ne(o)- new + plas(o)- to form + -ia condition of; i.e., cancer] is relatively common, especially in certain *canine* breeds. Splenic tumors are highly vascular and often rupture, putting the patient at risk of bleeding to death. Like the trauma situation, a *splenectomy* is a life-saving surgical procedure. Even if the tumor is ***malignant*** [*mal-* bad; malignant tumors are often aggressive and spread], the splenectomy may buy important quality time for the owners with their beloved pet. Bottom line: animals can live following a splenectomy. Lymphoid tissue and macrophages elsewhere in the body will pick up the spleen's duties.

Lymphoid Tissue

We've already addressed the white pulp found in the spleen. We also have patches of lymphoid tissue along portions of the digestive tract. Most lymphoid tissue, however, is found in well-organized, grossly recognizable structures, like the thymus, tonsils, and lymph nodes.

Thymus

The ***thymus*** is very important in immunity early in life. It is found in the cranial ***thoracic*** [*thorac(o)-* chest + *-ic* pertaining to] cavity (Fig. 3.19), in the ***mediastinal*** [*medi(o)-* middle] membrane. (The ***mediastinum*** is a connective tissue membrane that divides the

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thoracic cavity into right and left halves.) The thymus is most important early in life, because that is when the immune status of the individual is developing. As you might expect, during this time of peak activity, the thymus is large. With age, thymus activity and size diminish. This is why the immune status of ***geriatric*** (old) patients decreases, leaving them more susceptible to infectious disease. Remember those bookends: very young and very old. Very young patients haven't fully developed immunity yet, and very old patients are losing or have lost it. Both are at risk when exposed to ***pathogenic*** [*path(o)-* disease + *gen(o)-* producing+ *-ic* pertaining to] organisms. In fact, they may be so ***immunodeficient*** [*immun(o)-* protection + *deficient* (reduced)] that they can't even keep ***commensal*** [*com-* with + L. *mensa* table + *-al* pertaining to; i.e., living with the host without causing injury; cf. *symbiosis sym-* together, with + *bi(o)-* life + *-sis* process/state of; i.e., living together, in harmony] organisms from colonizing and causing disease.

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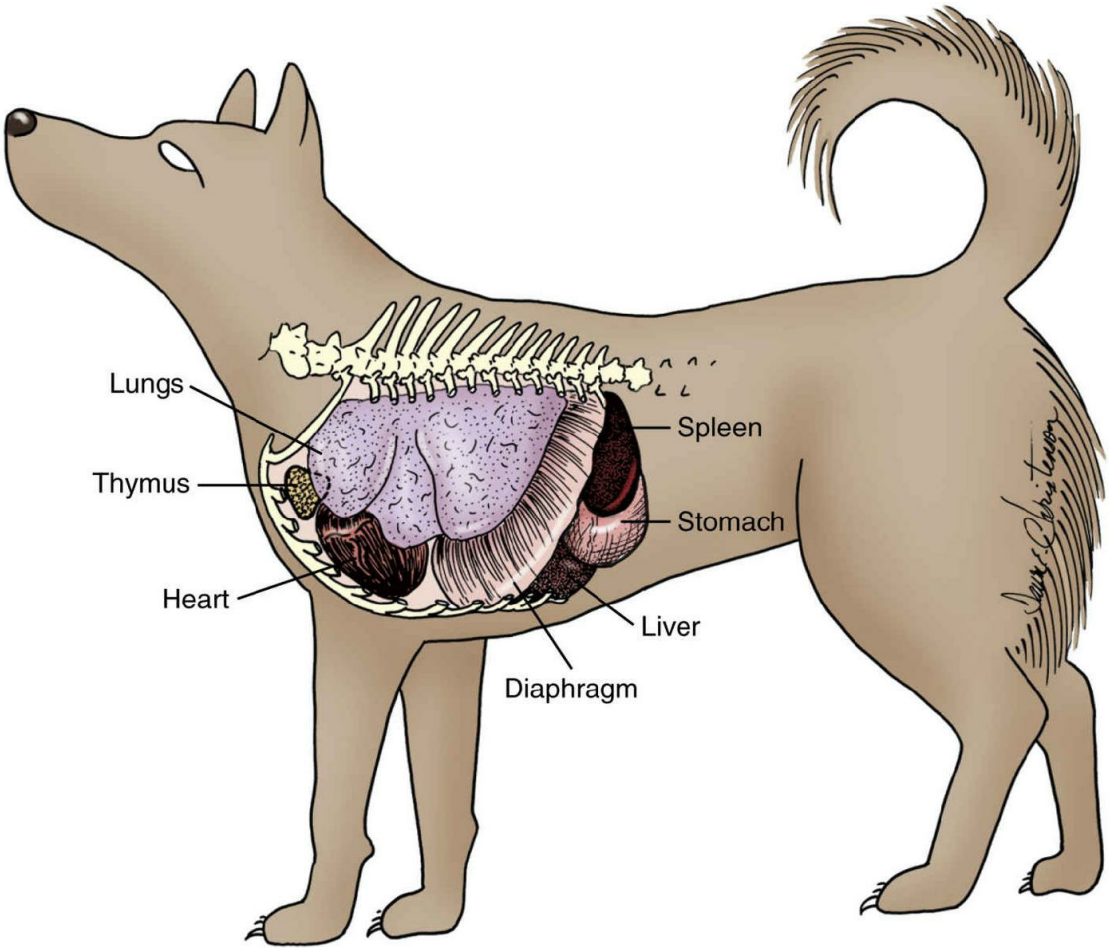

FIG. 3.19 Thymus and spleen in situ (left ribs removed).

If you remember earlier, when we discussed lymphocytes, we mentioned T lymphocytes. We said that the "T" could stand for "Travel" and "Target," right? Well, it can also stand for Thymus, because most T lymphocytes come from the thymus. The ***lymphoblastic*** [*lymph(o)-* lymph + *blast(o)-* germ/shoot + *-ic* pertaining to] cells in the thymus are incredibly important for the production of T lymphocytes and maturation of T lymphocytes for *immunoglobulin* production. This is key for developing immunity in young animals. Many of the T lymphocytes produced will be distributed to other lymphoid tissue in the body, including tonsils and lymph nodes.

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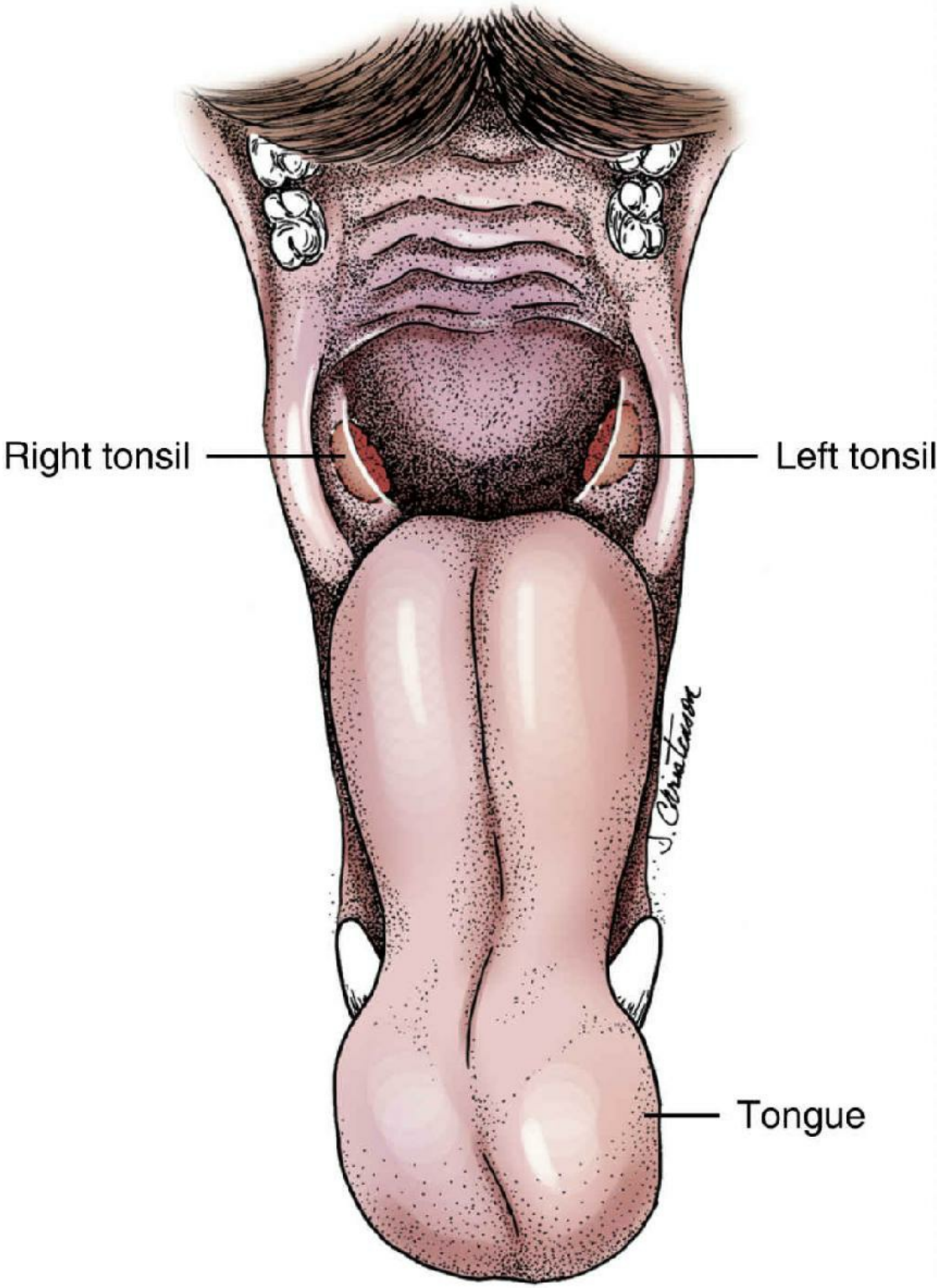

FIG. 3.20 Tonsils.

Tonsils

Tonsils are lymphoid tissue structures found bilaterally in the ***pharynx*** [Gr. "the throat"]. Under normal circumstances the tonsils

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will be tucked away in the ***tonsillar*** [*tonsill(o)-* tonsil + *-ar* pertaining to] ***crypts*** [*crypt(o)-* hidden] (Fig. 3.20). In cases of ***tonsillitis*** [*tonsill(o)-* tonsil + *-itis* inflammation of] they become enlarged and bulge out of their *crypts*. Normally, we cannot see them when performing a ***pharyngeal*** [*pharyng(o)-* throat + *-al* pertaining to] examination. Now, tonsils appear nodular like lymph nodes, but they are not considered lymph nodes. For one thing, unlike lymph nodes, tonsils are not enveloped in a connective tissue capsule. Plus, lymph doesn't pass through tonsils the way it does through lymph nodes.

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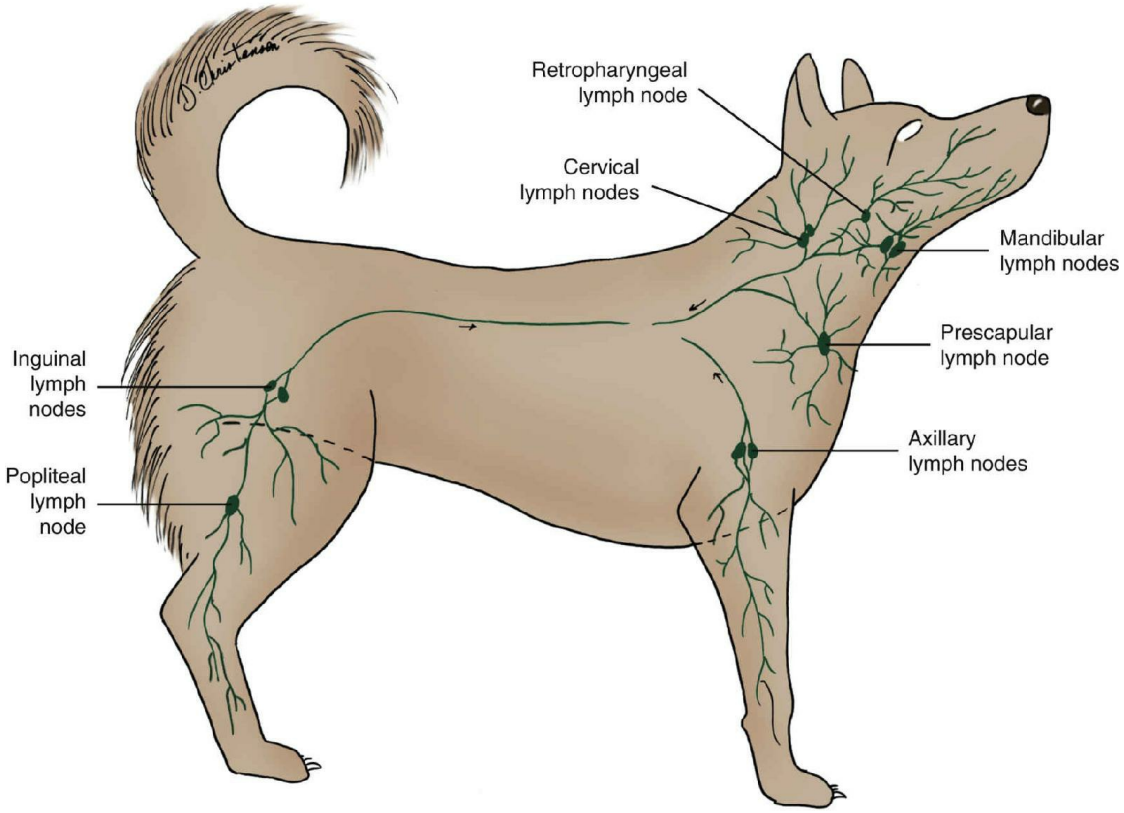

FIG. 3.21 Superficial lymph node schematic.

Why do we have tonsils? For any of us who have experienced *tonsillitis*, they may seem to be more trouble than they're worth. When they're inflamed, they can make swallowing incredibly difficult. And, certainly for dog breeds like English Bulldogs and Pugs, they can even make breathing difficult. Those sorts of difficulties often make a ***tonsillectomy*** [*tonsill(o)-* tonsil + *-ectomy* to cut out; i.e., surgical removal of the tonsils] necessary. All true. Yet, tonsils provide a very valuable protective service. They have prime real estate, in the *pharynx*, armed with T lymphocytes offering first-line defense against *pathogens* entering the respiratory or digestive tracts. Sure, we can live without them after a *tonsillectomy*. But that leaves no one watching the "front door," so to speak. In that case, we'll have to rely on regional lymph nodes and other immune mechanisms for protection. So, in the *pharyngeal* area, the ***mandibular*** [*mandibul(o)-* mandible, jaw + *-ar* pertaining to] and ***retropharyngeal*** [*retro-* backward, behind + *pharyng(o)-* throat + *-al* pertaining to] lymph nodes will have to pick up the slack.

Lymph Nodes

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Lymph nodes ("glands") are located throughout the body. They're not really glands. But we use the root *aden(o)-*, meaning gland, when we refer to them in medical terms, like ***lymphadenectomy*** [*lymph(o)-* lymph + *aden(o)-* gland + *-ectomy* to cut out; removal of; i.e., surgical removal of lymph node(s)]. Why aren't they glands? Lymph nodes are not glands because their tissues don't actively secrete substances, like enzymes or hormones. Instead, lymph nodes are filters of sorts, strung out strategically along all of the lymphatic vessels of the body. Within their tissues, lymph nodes have populations of *lymphocytes* and *macrophages*. Some of the lymphocytes engage in *mitosis* to produce more lymphocytes for the body. Both T and B lymphocytes may be produced in lymph nodes. The lymphocytes work with their teammates (macrophages) to target and destroy *pathogenic* organisms passing through in the lymph. And whatever *pathogens* one lymph node may miss will be caught by the lymph node next in line along the lymphatic vascular flow. That's why there are so many lymph nodes strung out along lymphatic vessels, like a string of pearls. It's called teamwork.

During physical examinations, we try to focus on some of the strategically placed superficial lymph nodes (Fig. 3.21). Recognize that there are many more lymph nodes in the body than just these. These are merely those that we try to palpate during a physical exam. Can we always feel each of them? No. And there are some that we don't even attempt to palpate, especially in large animals. Can you imagine trying to palpate ***inguinal*** [*inguin(o)-* groin + *-al* pertaining to] lymph nodes in a horse or a cow? You might not live to tell about it. Plus, let's face it, with the amount of bulky muscle and tissue in large animals, unless some of the other lymph nodes are markedly enlarged, we'll never feel them. In fact, this is true even in the dog or cat. However, in the dog and the cat, we should always try to palpate most of them, except the *retropharyngeal* nodes. Those can't be palpated in anyone. But if a ***pathologist*** [*path(o)-* disease + *log(o)-* knowledge + *-ist* one who specializes in] is trying to find evidence of *bovine tuberculosis* (TB), on ***postmortem*** [*post-* after + *mortem* death] examination, *retropharyngeal* lymph nodes will be collected for *microscopic* evaluation.

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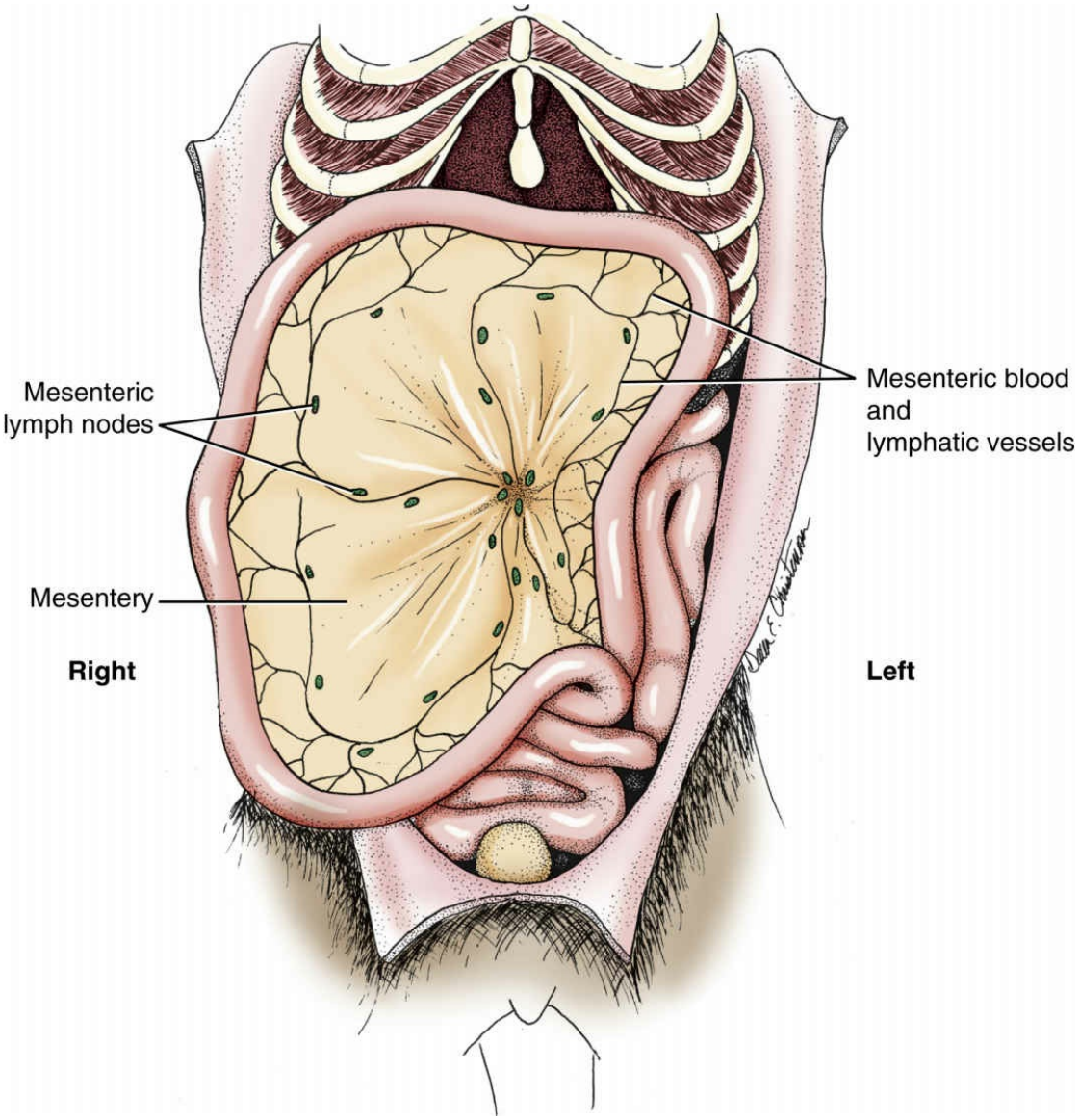

FIG. 3.22 Mesenteric lymph nodes.

In the dog and cat, we should always palpate lymph nodes in a ***symmetrical*** [Gr. *symmetros*; *sym-* with + metron measure + *-al* pertaining to; i.e. equal, same] manner (i.e., palpating like lymph nodes bilaterally simultaneously). If the nodes are ***asymmetrical*** [*a-* without + *symmetry* same + *-cal* pertaining to], it may indicate pathology in the region. For instance, we should always be able to palpate the ***popliteal*** [*poplit(o)-* "ham" + *-al* pertaining to] lymph nodes. These nodes are loosely attached caudal to the stifle, at the base of the hamstring muscles (caudal thigh). Let's say the right *popliteal node* is enlarged. Because we know that the popliteal lymph nodes filter lymph from the distal extremity, we will be suspicious of injury or disease in this patient's distal right rear limb.

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Mandibular lymph nodes can always be palpated too. However, students frequently confuse *mandibular* lymph nodes with salivary glands found in the same region. So, before you panic thinking that you've found ***unilateral*** [*uni-* one + *later(o)-* side + *-al* pertaining to] or ***bilateral*** [*bi-* two + *later(o)-* side + *-ally* pertaining to] huge mandibular lymph nodes, have an experienced clinician or technician double-check for you. Finally, ***prescapular*** [*pre-* before, in front + *scapul(o)-* scapula (shoulder blade) + *-ar* pertaining to] lymph nodes can and should also be palpated routinely. They are a little more challenging than *popliteal* and *mandibular* lymph nodes to palpate. Still, if you grab deep enough into the tissues between the point of the shoulder and the body wall, you should be able to feel them.

Cervical [*cervic(o)-* neck + *-al* pertaining to], ***axillary*** [*axill(o)-* axilla ("arm pit") + *-ary* pertaining to], *inguinal*, and ***mesenteric*** [*mesenter(o)-* mesentery + *-ic* pertaining to] lymph nodes will probably only be palpable if they are enlarged. The ***mesentery*** is a sheet of supportive connective tissue in the abdominal cavity associated with the intestines (Fig. 3.22). Vessels, including lymphatic vessels and *mesenteric* lymph nodes, are encased and supported by the *mesentery*. Obviously, there is an awful lot of stuff in the abdomen. Trying to palpate even mildly enlarged lymph nodes is like trying to find a needle in a haystack. That's why we often rely on abdominal ultrasound to evaluate mesenteric lymph nodes. If you can easily palpate cervical, axillary, inguinal, or mesenteric lymph nodes, you know that ***lymphadenomegaly*** [*lymph(o)-* lymph + *aden(o)-* gland + *-megaly* enlargement of] exists. By the way, some veterinary professionals use the term ***lymphadenopathy*** [*lymph(o)-* lymph + *aden(o)-* gland + *-pathy* a disease of] to indicate lymph node enlargement. This is probably because if lymph nodes are involved in a disease process, they are likely enlarged. However, *lymphadenomegaly* is a more accurate way of referring to their enlargement. I point this out because you may hear one or both in reference to lymph node enlargement.

Lymphatic Fluid and Vascular Flow

As we've already established, lymph is that colorless, watery liquid

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that is transported via lymphatic vessels and filtered by lymph nodes. But where does it come from and ultimately where does it go? This could be perceived as one of those "which came first, the chicken or the egg?" discussions. But we're going to start with the *interstitium*, simply to have a clear starting point.

All ***interstitial*** [*inter-* between + *stiti(o)-* tissue + *-al* pertaining to] space is bathed in lymph. Most of it actually originates from the bloodstream, leaking out from *capillaries* (the smallest of blood vessels). Blood pressure contributes to this, literally pushing fluid out through the thin capillary wall. A minor contributor to interstitial fluid are cells. (You may remember from Chapter 2 that we said two of the by-products of cellular activity are water and carbon dioxide.) So, there are the sources of interstitial fluid. Lymphatic vessels in the area pick up any excess interstitial fluid. But movement of lymphatic fluid is completely passive. You see, blood moves through blood vessels because the heart pumps it around. Not so in lymphatic vessels. So, how does it move? It relies entirely on gravity and movement of surrounding tissues. For instance, body movements and muscle contractions push on and compress the lymphatic vessels, passively causing lymphatic fluid to flow. In the abdomen, pressure from contraction of abdominal muscles and movements of the intestines cause mesenteric lymph to flow. Mind you, regardless of location, this is a one-way street. ***Unidirectional*** [*uni-* one + *directional*; i.e., one-way] valves in larger lymphatic vessels prevent any backward flow of lymph. If you look at Fig. 3.21, the tiny arrows show you the direction of lymphatic flow, all moving in a ***centripetal*** [*centri(o)-* central + *-petal* directed; i.e., moving to the center] direction. That *centripetal* movement carries all lymphatic fluid to the *thoracic duct*, where it re-enters the blood stream. So, now we've come full circle. Do you see why I said that this is kind of a "chicken and egg" scenario?

The most important thing to take away from this brief discussion is that the production and flow of lymphatic fluid, under normal circumstances, is a constant and dynamic give and take. What is produced should be taken away. This equilibrium keeps the *interstitium* bathed in fresh fluid all the time.

Edema

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Edema [Gr. *oidema* swelling] is an excess accumulation of interstitial fluid. How can this happen? There are a number of things that can contribute. We might think about edema as a faucet and drain situation. Whether we turn on the faucet too high and/or block the drain, we may wind up with a sink that's overflowing.

Looking at the input side (our faucet), we need to look at the blood capillaries, right? There are a number of forces at play here. First, there is *osmotic pressure*. We talked about osmotic pressure in Chapter 2. Putting it in context of the blood, we need to think about what components of the blood will be most attractive to water, to keep it in the bloodstream. (Hint: think about the plasma.) What does plasma have in abundance that will act like a sponge to attract and hold water in the bloodstream? Proteins. That's right, proteins in plasma help keep water in the bloodstream. If that's true, then what happens in cases of *hypoproteinemia* [hypo- low + protein + -emia blood condition]? The blood has less drawing and holding power—less osmotic pressure. So, water may leak from capillaries more readily, especially when there is more protein in the interstitium than there is in the plasma. This is why in *hypoproteinemic* animals, they may develop ***edematous*** [*edema* + *-tous* pertaining to] fluids in the interstitium and/or body cavities. A good example of this is in ruminants who are heavily infested with blood-sucking parasites in their digestive tracts. Anemia and hypoproteinemia frequently develop from these infestations, especially in younger animals. Gravity often dictates where the edematous fluids collect. We call this ***dependent edema*** because it depends on gravity. So, these animals may develop *dependent edema* along their *ventral* chest and abdomen and perhaps distal extremities. But where the edema is most noticeable, especially early, is in the throatlatch area (i.e., ventral jaw and neck). Their grazing habits probably contribute to the gravitational forces making it collect there. Because of the location of the *edematous* fluids, we call this *"**bottle jaw**."* If you ever observe *bottle jaw*, you can probably safely assume that the animal is *hypoproteinemic*.

Second, ***hydrostatic*** [*hydr(o)-* water + *-static* pertaining to standing] ***pressure*** can contribute to edema formation. We said earlier that blood pressure can push water out of the capillaries into the interstitium. That pressure is greatest on the ***arterial*** [*arter(o)-*

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artery + *-al* pertaining to] side of the capillary. (Arteries carry blood from the heart to all parts of the body. So, pressure within them is high.) Lymphatic vessels pick up most of the interstitial fluid produced by the arterial end of blood capillaries. Blood capillaries, on the ***venous*** [*ven(o)-* vein + *-ous* pertaining to] side of the capillary, reabsorb a little fluid, by virtue of osmotic pressure. (Veins return the blood to the heart. Pressure within veins is quite low. Like lymphatic vessels, veins too have *unidirectional* valves to keep venous blood moving toward the heart.) With all of this, we should maintain equilibrium with regard to volumes of interstitial fluid gained and lost. Ah, but what if we have extremely high blood pressure? That opens up the faucet, pouring excessive amounts of fluid into the interstitial space. But high blood pressure is not common in animals like it is in people. So, how does *hydrostatic* pressure play a role in edema formation in animals? It's typically faulty venous and/or lymphatic vascular flow. If venous blood flow is impaired or obstructed, say by a deep-vein thrombus or a bandage that's too tight, blood leading up to that obstruction will back up, like a river that's been dammed by beavers. This will eventually increase hydrostatic pressure in the capillaries from the *venous* side, producing excess amounts of interstitial fluid. Looking at lymphatic vessels, if we impair lymphatic flow, from immobility or *lymphadenopathy*, we may not be able to remove even normal volumes of interstitial fluid produced. This is why in paralyzed patients we use things like massage and passive movements of their limbs to enhance *centripetal* venous and lymphatic return. If we don't, they will likely develop dependent edema (i.e., where gravity dictates; usually on the downside).

Third, *inflammation* combines many of these mechanisms, resulting in edema. We'll talk more about inflammation in detail a little later. For now, understand that inflammation increases capillary permeability, resulting in more leakage of fluid into the interstitium. It also increases cellular activity, resulting in more of the cellular by-product—water. Inflammation provides for *chemotaxis*, attracting various *leukocytes*, like *neutrophils*, to the inflamed tissue, and they in turn will increase ***inflammatory*** [*inflamm(o)-* fire, inflammation + *-tory* pertaining to] effects in the area. Inflammation may impair movement of the body part,

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resulting in poor venous and lymphatic return, compounding edema formation. Inflammation may result in extreme losses of plasma proteins, like in inflammation resulting from extensive second- and third-degree burns. Loss of ***serous*** [*ser(o)-* serum + *-ous* pertaining to] fluid in large burns can cause severe hypoproteinemia. You already know that hypoproteinemia can result in edema formation, and in this case even in areas apart from the burn. There are many factors that contribute to edema formation in the presence of inflammation. That is why edema is one of the cardinal signs of inflammation. More on that later.

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Applied Terminology for Immunology

The immune system provides protection for the body. The word ***immune*** comes from the Latin, *immunis*, that literally means free or exempt (i.e., free or exempt from disease; protected). A moment of full disclosure: ***immunology*** [*immun(o)-* immunity/protection + *-logy* the study of] is a complex subject. I do not intend to cover it in depth. My goal is to give you a brief, functional understanding of the major facets of *immunology*. I'll leave the rest to your coursework dedicated to the topic.

Humoral Immunity

Humoral [*humor(o)-* L. *humor* liquid] ***immunity*** has nothing to do with comedy, in case you were wondering. Body humor is the fluid component of the body. You already know that ~70% of the body is water, with large portions of that precious liquid found in plasma and lymph (interstitial and lymphatic). The body's humor contains, among other things, antigen-specific immunoglobulins (antibodies). So, when we talk about humoral immunity, we are talking about a systemic (i.e., body-wide) response to antigenic (pathogenic) exposure.

Whenever the body is exposed to a *pathogen/antigen* (e.g., viral, bacterial, fungal, etc.), there may be an immediate nonspecific response at the point of invasion. It will be recognized as foreign and cells (e.g., macrophages and neutrophils) in the area may try to eradicate it at the point of entry. But the initial battle may not be sufficient to win the war. When we wage war against a *pathogen*, we need to be able to call in all the "troops" and give them the tools to recognize this specific invader. This is where B lymphocytes come into play. B lymphocytes produce *immunoglobulins* (Ig) against *(anti-)* the specific *pathogenic/antigenic* invader. (B lymphocytes produce most of our immunoglobulins. And they are probably doing it from the comfort of lymphoid tissue somewhere else. They're home-bodies, remember.)

Let's say that a dog is exposed to the respiratory virus, Parainfluenza, for the first time. Exposure may have been from

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another dog at the dog park, who was coughing and spewing the pathogen onto objects (like toys and the grass) and perhaps even directly in the face of our unprotected and unsuspecting dog. The point of entry, then, is likely be the mouth and nose of our furry friend. We may have an ineffective response of sentinels (neutrophils and macrophages) in the nose and pharynx. That response is limited, because everyone cannot recognize the virus yet. So the exposed dog will become clinically ill. Humoral immunity takes time. But just because the dog is becoming ill does not mean there is nothing going on. During the course of the illness, B lymphocytes will be producing a lot of "good" immunoglobulin (**IgG**), specifically for the Parainfluenza virus. These antigen-specific antibodies fit together with the Parainfluenza virus like perfectly fitting puzzle pieces. I like to think of these antibodies as creating "handles" on the pathogen so that our other body defenders (T cells and macrophages) can capture, destroy, and remove the invading organism. Eventually, if the dog is immune-competent, it will fully recover.

IgG persists in the body for a long time. So, if the dog is ever exposed to the Parainfluenza virus again, it will already have Parainfluenza-specific IgG. B lymphocytes may also produce **IgM** rapidly during this subsequent exposure. (IgM is produced more rapidly than IgG, especially during secondary exposures to the pathogen.) Parainfluenza-specific **IgA** is probably already present along the mucous membranes of the respiratory tract, giving us an immediate and aggressive response at the point of re-entry of the virus. (IgA is typically found out in the tissues, along mucous membranes, providing for local immunity on re-exposure to antigens.) So between all of these immunoglobulins and the cells responding, this dog will be protected and not become ill when it is exposed to Parainfluenza again.

By the way, there are B and T lymphocytes that have extraordinary memories of particular pathogens. We actually call some of them "***memory cells.***" True *memory cells* are found in circulation as well as hanging out in lymph nodes and lymphoid tissue. They can also "teach" other lymphocytes to recognize antigens. The memory cells of our Parainfluenza patient will help mount a very rapid and aggressive response to any subsequent

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exposure to the virus.

Passive vs. Active Immunity

The previous Parainfluenza virus scenario is a good example of ***active immunity***. The body was exposed to the viral pathogen and responded by actively producing antibodies. Unfortunately, very young animals (such as newborns) aren't capable of doing this. But they are not completely unprotected. You see, when newborns nurse for the first time, their mothers' initial milk (***colostrum***) contains tons of ***maternal antibodies***. Mom produced those antibodies either from exposure to the live pathogen or to the pathogen via vaccines. The newborns passively receive the maternal antibodies when they suckle. Their bodies don't need to do anything. That's why we call this ***passive immunity***. Immunoglobulins are passively received from mom. But what if the newborns don't have access to the *colostrum* during the critical first 12 to 24 hours after they're born? Well, if we have frozen colostrum, we could thaw it and feed it to them. If not, plasma from an immune-competent donor could be given to them through an ***intravenous*** [*intra-* within + *ven(o)-* vein + *-ous* pertaining to] infusion. Passively received antibodies will offer temporary protection, hopefully, until the young animal begins to produce its own antibodies. This is where vaccines are very important.

Immunizations

Immunizations [*immuniz(o)-* giving protection + *-tion* the process of] or *vaccinations* are used to stimulate immunity against specific *pathogens*. Vaccines contain pathogens in a form that prevents them from causing disease. The antigen in the vaccine may be killed or attenuated (altered). So the organism in question cannot cause disease. But it is still foreign to the body, so an immune response will be mounted.

Why do we repeat immunizations, especially in young animals? Remember those *maternal antibodies* that we mentioned earlier? Well, if an animal has a lot of maternal antibodies, it's protected and has no reason to actively produce any of its own antibodies.

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Eventually, those maternal antibodies will fade away to unprotective levels. Unfortunately, we don't know exactly when those maternal antibodies will drop, leaving the youngster unprotected. So, we give youngsters immunizations at regular intervals, playing the odds that at some point maternal antibodies will be extremely low or completely gone, so that we can stimulate *active immunity*. Plus, when we give a "booster," we're going to stimulate an ***anamnestic*** [*anamnes(o)-* memory + *-tic* pertaining to] ***response***. We may have gained protective levels of antibodies from the first immunization. But the *anamnestic response* from a booster will put protective levels of *immunoglobulins* "over the top," so to speak. This is, in part, due to the *memory cells* we talked about earlier.

Do immunizations provide 100% protection? No, they may not. For one thing, what is the immune status of the individual receiving the vaccine? We can't predict the exact response of each individual to a given vaccine. If the individual is capable of an immune response to the vaccine, at least some protection will be gained. Even if 100% protection is not achieved, if the individual is exposed to the actual disease and becomes ill, the illness will not be as severe and won't last as long. Why? Because exposure to the "real McCoy" should stimulate an *anamnestic* response, similar to a booster immunization.

Allergic Responses

An allergy or ***hypersensitivity*** [*hyper-* excess, excessively + *sensitiv(o)-* sensitive + *-ity* state of being] is an overreaction by the body. This is different from the antigenic response, discussed earlier, in a number of ways.

First, we're not talking about *pathogens, but allergens*. ***Allergens*** [*allerg(o)-* allergy + *gen(o)-* producing/producer] are often foreign substances, like pollen or particular foods, not organisms. What about dogs or cats with flea allergies, you ask? Well, those animals are allergic to the flea's saliva (a substance) not the flea itself (an organism). Second, the individual must have a predisposition to hypersensitivity. Not everyone develops allergies. Third, allergies generally involve an **IgE**-histamine reaction that involves mast

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cells.

Type I Hypersensitivity

I'll use my basset hound, Sadie, to help you understand Type I hypersensitivity reactions. I swear, sometimes I thought she was allergic to life. Not really. But she was allergic to many, many things. I knew this from the get-go, when I adopted her from the Humane Society. I asked why she was surrendered at 6 months of age. I was told that it was due to allergies. Of course, I assumed (bad thing to do) that the family was allergic *to her*. Nope. It was Sadie who had the allergies, creating medical issues that the family was not able to manage. So, from a young age, Sadie developed her allergies. And this is typical. Most dogs develop allergies early, often before 6 months of age. Sadie, like many allergy sufferers, inherited a genetic predisposition to overreact to potential allergens.

Sadie's primary allergens included some tree pollens (maple), ragweed, corn, and chicken. Let's use her ragweed allergy to explain the process. She could not have an allergic reaction to ragweed pollen the first time her body was exposed to it. She was born in September–peak ragweed season here. That provided her initial exposure. There is a period of time necessary to *"sensitize"* the body. How long and how many exposures are needed for *sensitization* varies. Who knows, maybe by the end of her first ragweed season, she was already showing outward signs of her ragweed allergy. Maybe it took until her second ragweed season. I'll never know. What matters is that there was a sensitization period. When her body was sensitized to ragweed pollen, it produced immunoglobulins for this substance. But instead of IgG, she produced IgE. And because she had the predisposition to hypersensitivity, she overproduced IgE. (To help you link Ig**E** to allergic reactions, just think: Excessive and Excitatory.)

IgE "arms" the ***mast cells*** of the body. Mast cells are found throughout the body. Their cytoplasmic granules contain a number of inflammatory mediators, principally histamine. In most domestic animals, mast cells are found in greatest abundance in the skin. Mast cells are also found in large numbers along the digestive tract. They are also found along the respiratory tract, but not as

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abundantly as in people. (That's why we sneeze when exposed to respiratory allergens and dogs, like Sadie, don't.) The skin is the place that we find the most mast cells in animals. This is why most allergies in animals cause ***dermatitis*** [*dermat(o)-* skin + *-itis* inflammation of]. So, during the sensitization process, Sadie armed the mast cells of her skin with more and more IgE for ragweed. Once fully sensitized, all it took was a single *"challenge exposure"* with sufficient ragweed pollen to be ***allergenic*** [*allerg(o)-* allergy + *gen(o)-* produce + *-ic* pertaining to] and result in *dermatitis*. The keyword there is "sufficient." You see, her body had a threshold, like a line drawn in the sand. Once the line is crossed, with enough allergen, the reaction begins. And by the way, exposure can take many forms. In this example, the pollen could be inhaled and/or make direct contact with her skin. Either way, dermatitis would result.

During the threshold-breaching *challenge exposure*, mast cells fully armed with IgE ***degranulate*** [*de-* away from + *granul(o)-* granules + *-ate* the act of; i.e., they release their granules]. Remember, the cytoplasmic granules of mast cells contain powerful *inflammatory* mediators, principally histamine. Once the histamine is released, inflammation is set in motion. (Eosinophils and basophils may also contribute to escalation of the inflammation. Most allergic individuals have higher than normal numbers of circulating eosinophils and basophils.) Of course, Sadie experienced generalized ***pruritus*** [*prurit(o)-* itch + *-us* an, the; i.e., itching] from the allergic dermatitis. But she always had a predictable pattern. First, one ear would become inflamed and ***pruritic*** [*prurit(o)-* itch + *-ic* pertaining to], then the other ear, then her face and feet, and finally her whole body. Hopefully, I caught it early and gave her an ***antihistamine*** [*anti-* against + *histamine*]. If I didn't, she might lick, rub, and scratch herself raw, because of the *pruritus*. It's funny, she held true to that pattern, whether she was reacting to pollens or food. Corn was her worst dietary allergen. Within minutes of consuming just a little piece of a corn chip, her left ear would flare. From there, things rapidly went downhill. Isn't it amazing how the body works?

How did I manage her? Well, I'll admit, I never started her on ***hyposensitization*** [*hypo-* low, decreased + *sensitiz(o)-* sensitivity + -

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tion process of; i.e., process of lowering sensitivity] injections ("allergy shots"). These work by exposing the body to low doses of the allergen, well below the threshold, so there's not enough allergen to cause an allergic reaction. Instead, the body is "tricked" into a more appropriate response. IgG is ultimately produced (see why I call it the "good" antibody?). Without excessive amounts of IgE, the IgE-histamine cycle is broken. Sadie's environmental allergies were seasonal and not that severe, so I could easily manage them with antihistamines. I also avoided ***immunosuppressive*** [*immun(o)-* immune/protection + *suppress* reduce + *-ive* pertaining to] drugs, like steroids, because I didn't want to open her to risk of infectious disease, along with other side effects. As to her food allergies, once I determined the specific foods she was allergic to, I could avoid them. She was on a restricted, ***hypoallergenic*** [*hypo-* low + *allerg(o)-* allergy + *gen(o)-* producing + *-ic* pertaining to] diet. It was only during the holidays and in public places that she managed to consume little bits of her nemesis foods that inadvertently fell within her reach. And she was a scent-hound, remember. So she could find every crumb. Ah, but she was loveable.

Contact Allergies

Most contact allergies are delayed hypersensitivity reactions. In people, poison ivy hypersensitivity is a good example of this. I know many people with this allergy, so I'll stick with it for this discussion. (Knock on wood, I haven't developed this allergy yet. "Yet" is probably the operative word.) The sequence of events for contact allergies, like poison ivy, are a bit different from the Type I hypersensitivity reactions described earlier. We still need a series of exposures to sensitize a predisposed individual to the allergen (poison ivy in this example). But this time, sensitization involves lymphocytes, most likely T lymphocytes.

Once sensitized, the next exposure will set the wheels of the allergic reaction in motion. But there are a number of factors involved. First, there needs to be sufficient contact time with the poison ivy oils. The protein structure of the oils is incomplete. They are completed by binding with skin epithelium and that takes time, perhaps 30 minutes or so. So, if you are allergic to poison ivy, wash

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it off with liberal soap and cool water immediately. If removed quickly and completely, binding cannot take place, preventing an allergic reaction.

But you won't know if you removed it all for a while. Delayed hypersensitivity reactions, as with poison ivy, can take days to over a week before allergic symptoms become apparent. Because of the delay, many folks can't remember when or where they may have come in contact with the allergen. Why the delay? Well, we're relying on lymphocytes, remember. We probably don't have very many, if any, T lymphocytes at the point of contact. They'll need to make their way there. That takes time. Once we have them in place, they can kick off the inflammatory reaction. By the way, if you are allergic to poison ivy, you need to know that you can have an allergic reaction from skin contact with the plant oils, even in the middle of a snowy winter. You can also have an allergic reaction if you inhale smoke from burning wood that has vines attached or that is contaminated with the plant's oils. Imagine the blister-like, oozing skin lesions developing in your lungs, from inhaling the smoke. That could be very serious.

Fortunately, most of our domestic animals don't develop contact allergies as much as people do. Perhaps, if we were furry, we wouldn't either. Go ahead, you can chuckle at that idea. Chuckle now, because our next topic is no laughing matter.

Anaphylaxis

Anaphylaxis [an"uh-fuh-lak'sis; *ana-* back, up, again + *phylaxis* guarding] is a life-threatening allergic reaction. Like all allergic reactions, the individual needs to be sensitized to the allergen. Unlike the classic Type I hypersensitivity reaction discussed earlier, in this situation a *challenge exposure* results in massive numbers of mast cells degranulating, releasing histamine simultaneously throughout the body. Remember, domestic animals have large numbers of mast cells in their skin, along their digestive tracts, and along their airways. If we have massive amounts of histamine released, profound inflammation will rapidly develop in those areas. Profound inflammation causes ***acute*** (sudden) *edema* formation in all of the areas mentioned. That is a massive amount of fluid suddenly lost to the interstitium, and this can cause an *acute*

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drop in blood pressure. Edema in the lungs, along with contraction of smooth muscle along the airways, will create difficulty breathing and impair gas exchange of oxygen and carbon dioxide. Death can come very quickly in ***anaphylactic*** [*ana-* back, up, again + *phylact(o)-* guarding + *-ic* pertaining to] reactions.

Do we ever see *anaphylaxis* in veterinary patients? You bet we do! Some of the most common situations that we see it in include vaccine reactions, plasma or blood transfusion reactions, medication reactions (e.g., antibiotic allergies), and, much less frequently, bee or wasp stings. If the animal is in our care, we will watch closely for any adverse reactions. This is especially true during transfusions. But many times, like after immunizations during a wellness visit, we have to rely on the owners' power of observation. That is why we need to be very clear in our instructions for things that they need to watch for. Initial vaccine reactions are usually subtle (e.g., unusual swelling at the injection site, hives, facial swelling, pruritus, vomiting, diarrhea, etc.). Many owners won't correlate most of those symptoms with the immunization. They'll chalk them up to something else. That's why we need to tell them what to watch for. Each subsequent booster may compound the unusual symptoms, until finally the animal goes into anaphylaxis following another immunization. If it's going to happen, it will typically develop in less than 30 minutes. If we have a patient in whom we are suspicious of having hypersensitivity to a vaccine, we should have them stay in our facility for at least 20 to 30 minutes after being immunized. That way we can render life-saving treatment immediately. Hopefully, our vigilance and emergency care will save a life.

I remember a kitten, early in my career, who died from anaphylaxis. She had a *Cuterebra* (type of bot fly) larva in the skin of her neck. These bots grow for a long time under the skin of the host animal (about a month), before they finally crawl out to complete their lifecycles. This gives the body a lot of time to become sensitized to the bot. Well, the little kitten didn't appreciate our restraint, or our attempt to remove the larva. She squirmed at the wrong moment and the larva was crushed in the struggle. This released a sudden ***bolus*** (concentrated mass) of allergen into the kitten's body. She immediately went into anaphylaxis. Despite our

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best efforts, she didn't make it.

If you remember anything at all about anaphylaxis, remember this: it is a life-threatening allergic reaction that can rapidly result in death, all because of profound inflammation.

Inflammation

Throughout this immunology section, I have repeatedly mentioned inflammation. But what is it exactly? The Evolve animation, "Inflammatory Response", provides a nice introduction to inflammation. We'll go into more detail. First, to be clear, inflammation is a normal, protective response to disease and injury. Inflammatory mediators come from a number of sources, including damaged cells and tissues, pathogenic organisms, hypersensitivity reactions, as well as various leukocytes (especially granulocytes). As you may recall, we said that neutrophils, with their sloppy phagocytic activity, really help to increase inflammation in a wounded area. In fact, let's revisit that bite wound that we talked about in the neutrophil section.

With the dog bite example, we have damaged tissues and pathogens (bacteria) in the wounded area. Chemical mediators of inflammation are released initially from the damaged tissues and toxins produced by the bacterial activity. This will result in inflammation (go figure). Inflammatory mediators are also chemotactic for neutrophils and macrophages. Inflammation, regardless of its cause, has five cardinal signs: heat, redness, edema, pain, and loss of function. Let's talk about each of these individually.

Heat:

Since the root word for inflammation means "fire," there must be heat. Where there is fire there is heat, right? But why is there excess heat in an inflamed area? Inflammatory mediators cause dilation of vessels in the area. This localized increased blood flow distributes heat from the core of the body to the local area. Also, there is typically increased cellular activity in the area. This is certainly true in the case of our bite wound. In that scenario, we have a lot of cellular activity for phagocytosis and tissue repair. Cellular activity

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generates heat. And remember, once we have numerous neutrophils in the bite wound, their phagocytic activity will magnify the inflammation. More inflammation means more heat. So, in the acute stage of injury (first 24 to 48 hours), how can we minimize the hot side effect of inflammation? That's simple: Apply cold.

Redness:

Redness goes hand in hand with increased heat. If we have increased blood flow to an area, we'll be able to actually see that. Blood, after all, is red, right? So, inflammatory mediators cause dilation of blood vessels, resulting in increased blood flow, and that causes the ***erythematous*** [*erythema* redness + *-tous* pertaining to] appearance in the inflamed area. More inflammation means more *erythema*. In the acute stage of injury (first 24 to 48 hours), how can we minimize vascular dilation? Apply cold. Cold tends to cause blood vessels to constrict.

Edema:

We've already established the fact that inflammatory mediators cause vascular dilation and increased blood flow to the area. This alone may result in greater fluid loss into the interstitium. Add the heat associated with inflammation and capillary permeability (leakage) increases dramatically. All of these three effects (heat, erythema, and edema) are protective. Edematous fluids in a wounded area can help dilute noxious elements, like bacterial toxins from our hypothetical bite wound. Fluid pouring into the wound (both lymph and serous fluid) can serve to flush debris and contaminants from the wound. And increased blood flow provides a speedy pathway for neutrophils and eventually macrophages to enter the area, to phagocytize debris and bacterial contaminants. As we've already said, neutrophilic activity heightens inflammation. Greater inflammation means greater edema. We also know from our earlier discussion of edema that it can be counterproductive. Excessive edema can impair movement and create pain. So, in the acute stage of injury (first 24 to 48 hours), how can we minimize edema? Apply cold. Cold reduces heat, reduces cellular activity, reduces capillary permeability, and causes constriction of blood

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vessels. All of that helps minimize edema formation.

Pain:

Most of us probably don't like pain. It is a very unpleasant sensation. Unfortunately, pain and inflammation go together like peanut butter and jelly, bread and butter, and … well, you get the idea. Now, in the case of our bite wound, the injured tissues will directly stimulate sensory nerve pathways for the perception of pain. Initial inflammatory mediators contribute to stimulation of pain pathways. Have you ever accidentally hit your thumb with a hammer or closed your finger in a car door? I have, on both counts. The crushing injury and tissue damage immediately triggers a pain response. That's important. Otherwise we might keep doing the same foolish thing, resulting in even greater injury. Ah, but have you noticed how after a few minutes or hours pass, the injured area is pounding so much that you can count your heart rate? We are now in a catch-22. Inflammatory mediators and their side effects of increased blood flow contributing to heat and edema formation exacerbate stimulation of pain pathways. Especially as edema increases, even tissues not directly traumatized become stretched and painful. And both pain and edema lead to our final symptom of inflammation—loss of function. So, in the acute stage of injury (first 24 to 48 hours), how can we minimize pain? Apply cold. Cold application minimizes many of the inflammatory side effects of heat and edema that can increase pain. Cold application also slows nerve activity, reducing impulses along pain pathways. In fact, with cold applied correctly, we can actually temporarily remove pain completely.

Loss of Function:

Whether we are talking about our injured thumb or finger or our hypothetical patient with the bite wound, pain alone will cause the injured individual to protect the area. If the bite wound we've been talking about is on an extremity, the patient may limit use of the limb or not use it at all. Eventually, edema will impede movement of the extremity. Loss of function, seen as lameness, makes perfect sense when we think of inflammation in a finger or limb. But what if the inflammation involves an organ like the kidney or the liver?

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Will organs like that also experience some degree of loss of function due to inflammation? Yes. The same inflammatory symptoms develop, we simply can't see them because they are concealed in the abdomen. Yet, I assure you inflammation of those organs results in increased blood flow, in spite of the fact that we can't directly feel the heat or see the redness. Those inflamed organs will develop edema and become very painful. Whatever the cause of the inflammation, coupled with edema, collectively will damage the functional cells and structures of the organs. The biggest difference, when major organs like that lose function compared to an extremity, is the organ dysfunction can have major systemic consequences. Lameness from inflammation probably won't be lethal, but organ failure from inflammation will be. But short of that, inflammation of an organ like the liver will probably cause the individual to slow down and rest more. Both of these responses will allow the body to focus its energies on fighting pathogens (if they're the cause of the inflammation), repair, and healing. Rest is restorative. With all of our other symptoms of inflammation, we said that cold application would be beneficial. It's pretty difficult to apply cold to major organs. But in all other areas of the body, when acute injury happens, cold application will be beneficial, especially during the first 24 to 48 hours. And by minimizing and preventing all of the other inflammatory symptoms (heat, erythema, edema, and pain) we can minimize loss of function.

Fever

We have probably all experienced fever, with common colds or the flu. But what exactly is fever? I like to think of fever as a systemic (bodywide) inflammatory response. Most of the time fever or ***pyrexia*** [*pyrexia* is derived from Gr. *pyr* fire] develops from pathogenic invasions. Going back to our humoral immunity example, the unvaccinated dog exposed to the Parainfluenza virus for the first time will become clinically ill. One of the clinical manifestations of the illness is *pyrexia*. Fever in infectious diseases like this is EXTREMELY beneficial. Fever is an indicator that the body's immune system is kicking into high gear and *that* is a very good thing. But how does fever actually result in the increased body temperature? Good question.

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First, we need ***pyrogens*** [*pyr(o)-* fire (fever) + *gen(o)-* producers]. *Pyrogens* are more or less specialized inflammatory mediators. Pyrogens may be produced by bacteria, viruses, leukocytes, and macrophages, to name a few. So, in the case of the dog with Parainfluenza, we probably have a number of pyrogens contributing to the fever. Ultimately, these ***pyrogenic*** [*pyr(o)-* fire, fever + *gen(o)-* producer + *-ic* pertaining to] substances enter the brain. Body temperature regulation (the thermostat) is located in a specific part of the brain, called the hypothalamus. We'll talk more about that region of the brain in Chapter 11. The name of the location is not as important as the fact that it houses the ***thermoregulatory*** [*therm(o)-* temperature + *regulatory* regulating; i.e., thermostat] center. When certain pyrogens (most have very limited access to the brain) reach the "thermostat," they change the thermostatic setting. If our dog with Parainfluenza has a normal body temperature setting of 101°F, the new thermostatic setting may be 104°F. With that new set-point, all of the body temperature sensors now perceive that the body is cold. So mechanisms to increase body temperature are engaged, like shivering, vascular constriction in the periphery to conserve body heat at the core (gums will look pale), and seeking heat (under blankets, over a heat duct, etc.). Once the body temperature reaches the new setting of 104°F, the heating mechanisms will be disengaged, temporarily. But this is an abnormally produced thermostatic setting, so maintaining it is challenging. Sometimes, we may overshoot the goal. Then we have to engage cooling mechanisms—for the dog this is primarily panting, plus superficial vascular dilation to dissipate some heat. And the dog is on a roller coaster ride, trying to maintain the new thermostatic setting. We've all been there—hot, cold, hot, cold . . .

Like localized inflammation, bodywide inflammation of fever creates pain too. We recognize this as generalized aches and pains. All of this makes us feel lousy and lethargic (lazy)—sort of generalized loss of function. Sleep and rest are important to conserve energy for our mounting immune response. Unfortunately, we respond to the heat of fever just like heat of a summer day—we have reduced or absent appetites. We need energy. In fact, we burn much more energy and oxygen in a ***pyretic*** [*pyret(o)-* fever + *-ic* pertaining to] state. And while we may look

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lazy on the outside, we are very busy at the cellular level. T lymphocytes and macrophages are doing their best to destroy the pathogen, while B lymphocytes are producing loads of IgG. Fever enhances all of this activity. That's why we don't give medications to artificially reduce fever, unless the fever is approaching dangerous levels. Think about that the next time you catch a cold. If you take something to reduce the fever (and the aches and pains), you may be prolonging your illness. Let the body do what it was designed to do when faced with invading pathogens. Part of that design is fever. Just sayin'.

Of course, once the pathogen is eradicated, we no longer have pyrogens to force the unusual thermostatic setting. In this example, our Parainfluenza dog's thermostatic setting will return to a normal 101°F. This is what people commonly refer to as the fever "breaking." Now that the set-point is normal, the body's sensors perceive that it is hot. So, cooling mechanisms are engaged, until a normal body temperature is achieved. Inflammation is gone—the dog feels better, no more aches and pains, and its appetite returns. The dog may have residual respiratory symptoms and lethargy. Rest will still be important through the remainder of its recovery. While immune activity may be winding down, it will still be increased compared to normal.

By the way, mild fever may develop following immunizations. These fevers don't last long (hours to maybe a day). And it may be just enough of a temperature elevation to reduce an individual's appetite and activity level. This fever is simply an indication that the immune system is actively responding to the vaccine. And that is precisely what we want to have happen.

Autoimmune Disease

Autoimmune [*auto-* self + *immune*] disease is when the immune system actually targets one (or more) of the body's own components as a foreign entity. There are a number of circumstances thought to trigger autoimmune disorders, but much of the time they are ***idiopathic*** [*idi(o)-* spontaneous, unknown + *path(o)-* disease + *-ic* pertaining to; i.e., disease of unknown cause]. There is a plethora of *idiopathic autoimmune* disorders that target the

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skin, blood, joints, kidneys, and thyroid, to name a few. We certainly can't cover them all. So, let's look at a couple of the more common *canine* disorders: *immune-mediated hemolytic anemia* and *immune-mediated thrombocytopenia* [*thromb(o)-* clot + *cyt(o)-* cell + *-penia* deficiency of].

Immune-Mediated Thrombocytopenia

We mentioned *immune-mediated thrombocytopenia* when we discussed platelets in the hematology section of this chapter. Immune-mediated thrombocytopenia, as you might expect, results in a *coagulopathy*. In my experience, it's one of the more common causes of spontaneous bleeding in dogs. *Autoantibodies* are formed that promote targeting and destruction of *thrombocytes*. Owners may have noticed nosebleeds (***epistaxis***, ep″ĭ-stak'sis), blood in the urine (***hematuria***, he″mat-ūr'e-uh, [*hemat(o)-* blood + *ur(o)-* urine + *-ia* condition of]), and/or unusual bruising. When we collect a blood sample from a patient with this disease, we may notice that the collection site tends to bleed longer than normal. The patient may be *anemic*. On physical examination we may discover *splenomegaly* and superficial bruising (e.g., ***petechiae***—pet-eek'e-a; pinpoint speckles; ***ecchymoses***—ek″ĭ-mo'sēz; larger spots, usu. ≤ 1 cm in diameter).

Numerous diagnostic tests to rule out other *coagulopathies* will be needed. *Coagulation* tests that focus on clotting factors (APTT and PT) will likely be normal. The platelet count will be quite low, and the *buccal mucosal bleeding time* will be prolonged. Of course, none of this evidence tells us the specific cause of the *thrombocytopenia*. If the veterinarian is highly suspicious of *immune-mediated thrombocytopenia*, he or she may put the patient on *immunosuppressive* doses of steroids. If the patient responds to the ***immunosuppressant*** [*immun(o)-* immune, protection + *suppress* + *-ant* one that], a *diagnosis* of *immune-mediated thrombocytopenia* is made. If the patient does not respond, further diagnostic testing may be needed, like a bone marrow ***biopsy*** [*bi(o)-* life + *-opsy* to view; i.e., collection of tissue for microscopic viewing]. This helps determine the health and numbers of *megakaryocytes*.

Because immune-mediated thrombocytopenia results from a "confused" and overzealous immune system, immunosuppressive

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therapy is the main course of action to treat it. Treatment may be temporary or ***chronic*** ([Gr. *chronos* time]; i.e., long-term). During the early stages of the disease, hospitalization and supportive care will be important. Patients need to be monitored closely for bleeding. Anemia, if severe, may require blood transfusions.

Immune-Mediated Hemolytic Anemia

Immune-mediated hemolytic anemia (IMHA) also results from production of *autoantibodies*. In this case, the autoantibodies promote the targeting and destruction of *erythrocytes*. Obviously, this can result in profound *anemia* and *hyperbilirubinemia*. So, we expect these patients to have pale and *icteric* mucous membranes. Our blood samples will have a low PCV and icteric plasma. And there is a unique, grossly visible characteristic that we may see in the EDTA collection tube: ***agglutination*** [*agglutin(o)-* glue, gluing + *-tion* process or state of]. You see, the autoantibodies make the *erythrocytes* actually stick together in clumps. Some of these clumps may be grossly visible. Instead of a nice homogenous coating of blood on the side of the tube when we mix the sample, the blood appears to separate into clumps. If we mix a drop of blood with some saline and look at it under the *microscope*, these clumps and clusters of *erythrocytes* will remain *agglutinated*. The saline can't break the bonds. This is how we tell *agglutination* apart from *rouleaux* (roo'lo). Plus, *microscopically*, *agglutination* looks more like grape clusters (Fig. 3.23), whereas *rouleaux* looks like neatly stacked coins. Make note that in the image of agglutination, there is plenty of room for those erythrocytes to spread out individually. So, something is obviously gluing them together. Rouleaux is often seen in thick areas of a blood smear, where cells don't have room to spread out. And, using the saline test, rouleaux will dissipate when placed in the saline solution.

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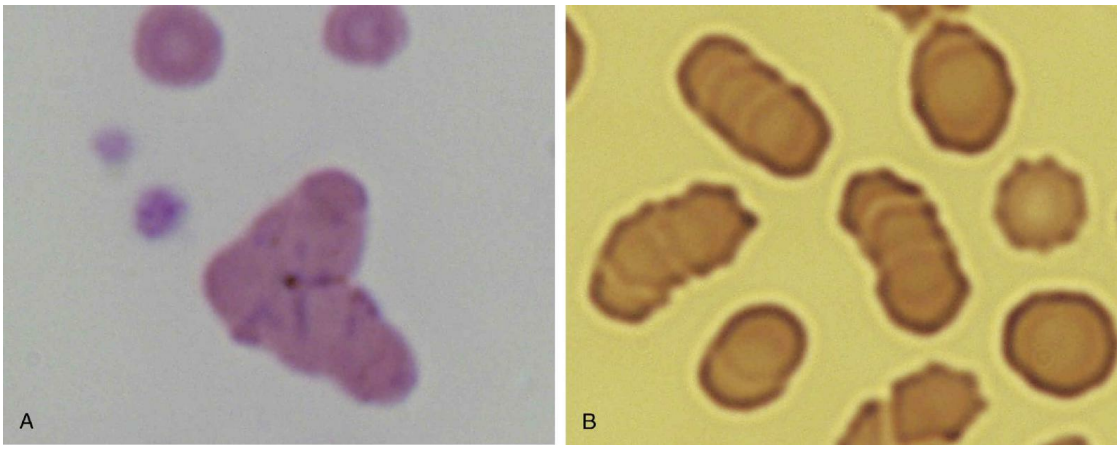

FIG. 3.23 (A) Agglutination and (B) rouleaux.

Now, *erythrocytic* parasites could prompt immunoglobulin production that would result in agglutination. We may need to do additional testing to rule out those pathogens. When those are ruled out, we are left with probable autoimmune disease. Okay, so we've got autoantibodies gluing erythrocytes together. Where is *hemolysis* taking place, and who is doing it? *Splenic macrophages* do the dirty work. And because they are so busy with a backlog of *immunoglobulin*-tagged and *agglutinated* RBCs to destroy, we often see *splenomegaly* in IMHA patients. And boy are these *splenic macrophages* relentless! Sometimes it seems like they *hemolyze* blood as quickly as we can transfuse it. Yes, transfusions are usually necessary. In fact, multiple transfusions are usually required. For critical dogs who have never received a transfusion before, we may risk giving the first transfusion from a universal donor, without a ***cross-match***.

What is cross-matching? It is a method of exposing the recipient's blood and serum to a donor's blood and serum. Think about it. The donor's blood is technically foreign to the recipient's body, so the recipient may mount an immune response to the donor's blood. The risk of a profound, life-threatening reaction is relatively low, with the first transfusion. That's why we may risk giving the first one to a dog without cross-matching. After that, it would be foolish to transfuse without cross-matching. And because the cells and plasma/serum proteins are *antigenically* distinct, we should cross-match to both. Remember our discussion in immunology—whenever the body is exposed to an *antigen*, lymphocytes will produce *immunoglobulins* specific to that antigen. So, in the case of

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transfusions, our risk of transfusion reactions increases with each subsequent transfusion. And if large quantities of IgE are produced, we could precipitate *anaphylaxis* with the next transfusion. That is why cross-matching is so important, especially if the patient has already received a transfusion. (By the way, many cats have what we call ***alloantibodies*** [*all(o)-* other + antibody; naturally occurring antibodies against other individuals of the same species]. These can result in major transfusion reactions even to the very first transfusion. So, cats should *always* be cross-matched to their donors.)

Beyond supportive care and life-saving transfusions, we need to reduce and hopefully stop the autoimmune attack on the dog's own erythrocytes (as well as possibly the donor's). The key to winning this battle is to put the patient on *immunosuppressants*, like steroids. Until we slow and eventually stop the production of *erythrocytic autoantibodies*, we are fighting a losing battle. Even so, sometimes the only way to stop the over-the-top *macrophagic* activity is to do a *splenectomy*. Believe you me, this is a risky undertaking. The *anemia* alone makes surgery risky, because this patient cannot afford to lose any blood. You already know that the spleen is a highly vascular organ that may be holding up to 30% of the dog's blood volume. Plus, these patients are at tremendous risk of *hemorrhage* during surgery. You see, in addition to *immunosuppressants*, we often treat these patients with *anticoagulants*. Why? Well, the *agglutination* and ***thrombocytosis*** [*thromb(o)-* clot + *cyt(o)-* cell + *-sis* condition of; i.e., excess numbers of platelets] typically seen in IMHA put them at risk of *thrombosis*. So, to prevent life-threatening *thrombus* formation, we use *intravenous* infusions of *anticoagulants*, like heparin. With heparin on board, we have major interference with *hemostasis*. Do you see why we typically make a splenectomy a last resort? It is extremely risky. But it may be the only way to stop the *hemolysis*, so the patient has a fighting chance at recovery.

These patients do not recover quickly. They are critical for days to weeks. Sadly, some do not make it, despite our best efforts. Those who do survive face a slow recovery period, even after they go home. They are typically sent home on oral *immunosuppressants* and *anticoagulants*.

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Case Study

Our patient is Buz. He is a 6-year-old, neutered male, 20-pound mixed-breed dog. Over the last couple of days, his owners noticed that Buz seemed lethargic and had a poor appetite. Concerned, they've brought him in to be evaluated. Historically, the owners state that he was fine until a couple of days ago. He has not travelled, been around other dogs, or gotten into any possible toxins. There is no history of vomiting, diarrhea, coughing, or sneezing. His stool is normal consistency and color.

On physical examination, we notice that Buz is not his typical, rambunctious self. He's usually jumping up trying to slobber our faces. Today, he's just lying there like a lump. He is mildly pyretic, with a rectal temperature of 103.9°F. His mucous membranes are pale and mildly icteric. We could not find any evidence of lymphadenopathy, but on abdominal palpation splenomegaly was noted. Heart and respiratory rates, at rest, are elevated.

The veterinarian has ordered a complete blood count (CBC), serum chemistry, and coagulation tests (APTT and PT). Because it will take a little while to get all of these test results, she asks you to do a quick PCV and TS. Your PCV/TS results are 18%, 6.8 g/dL with mildly icteric plasma. You also noted, on mixing the EDTA sample, that there appears to be gross agglutination. When informed of this, the doctor asks you to perform a blood-saline evaluation. On doing this, you confirm agglutination microscopically. This preliminary information makes the veterinarian put erythrocytic parasites and autoimmune disease at the top of her pathologic rule-out list.

Laboratory findings: (Note: abnormal elevations marked with "**H**" and abnormally low values marked with "**L**"):

CBC results: RBC 2.1 × 10^6/μL (**L**), PCV 18% (**L**), Hb 5.8 g/dL (**L**), RBC morphology: anisocytosis, polychromasia, spherocytosis, 6 NRBC/100 WBC (**H**), agglutination noted; platelets 250 × 10^3/μL; corrected WBC 42.1 × 10^3/μL (**H**), Segmented Neutrophils 80% (**H**), Band Neutrophils 8% (**H**), Lymphocytes 4% (**L**), Monocytes 9% (**H**), Eosinophils 0%, Basophils 0%, WBC morphology normal.

Serum Chemistry results: BUN 15 mg/dL, Creatinine 0.5 mg/dL,

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TP 6.7 g/dL, Albumin 2.9 g/dL, ALP 636 U/L (**H**), ALT 542 U/L (**H**), glucose 92 mg/dL, Na^+ 146 mmol/L, K^+ 4.5 mmol/dL, Cl^- 118 mmol/L, Ca^+ 9.8 mg/dL, Total bilirubin 9.8 mg/dL (**H**).

Coagulation assays: APTT 72.0 sec., PT 12 sec.

Based on all available information, the veterinarian makes a tentative diagnosis of IMHA. Just in case splenic macrophages are masking the presence of erythrocytic parasites, she orders immunoglobulin titers for common parasitic pathogens and DNA tests for tick-borne blood pathogens. It will take 24 to 48 hours to receive results to these latest laboratory tests. Buz has already been hospitalized and placed on intravenous fluids (an isotonic electrolyte solution). This therapy has further reduced his PCV to 12%. This prompts the doctor to order Buz's first transfusion of packed RBCs, from a universal donor. She also orders a constant-rate infusion of intravenous heparin, as well as doses of intravenous dexamethasone sodium phosphate (steroidal immunosuppressant). With the fluid therapy, his body temperature has returned to normal (101.8°F).

Buz receives the first half-unit of packed RBCs over 4 hours. During the transfusion, we monitor him closely for any signs of a transfusion reaction. He does well. In fact, because he begins to oxygenate better, we see a gradual decrease in his heart and respiratory rates over the 4-hour period. His PCV an hour after completing the transfusion is 24%. Over the next 6 hours, we begin to see his heart and respiratory rates gradually increase. So, we recheck a PCV. It has dropped to 14%. A second transfusion is ordered. We administer second half-unit of packed RBCs from the same donor. Like the first, it is transfused over 4 hours. Two hours into the second transfusion, Buz develops pyrexia (104.2°F) and vomits. Concerned that these may be signs of a reaction, the transfusion rate is slowed. His body temperature decreases to an acceptable level, maintaining at 102.4°F. He experiences no further episodes of vomiting. His PCV 1 hour after the second transfusion is 20%. Hemolytic plasma is noted, another possible sign of transfusion reaction.

Buz continues this way over the next few days. He is requiring transfusions each day. Because of suspicion over reaction to his second transfusion, he is cross-matched to all subsequent donor

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blood. Still, whatever ground appears to be gained in his PCV after each transfusion quickly fades. So, the doctor gains informed consent from the owners to perform a splenectomy. All risks, including severe hemorrhage, are discussed with the owners to help them make this difficult decision. He is cross-matched to several units of blood, in anticipation of requiring transfusions during surgery. He actually receives a whole unit during the procedure. Buz survives the splenectomy. From this point forward, it seems that we gain ground daily. His final transfusion of a half-unit is delivered several hours after the splenectomy.

After 10 days of hospitalization, Buz is well enough to go home. He is discharged on oral medication, an anticoagulant, and prednisolone (an oral steroid). He is to be rechecked in 1 week. Eventually, Buz fully recovers and is weaned off of the anticoagulant and steroids. At his final recheck, a month after stopping his medications, all laboratory data is within normal limits. And he is back to his old self, jumping up to slobber our faces.

Case Study Questions

1. The physical examination abnormality of _____ was a gross indicator of the presence of hyperbilirubinemia in Buz.
2. Which medical term indicates that Buz had an enlarged spleen? _____
3. From Buz's laboratory reports, _____ indicated that there was a variation of erythrocyte size observed on the blood smear.
4. Based on the extremely low RBC count, PCV, and Hb, what medical term would best be used for this condition? _____
5. From Buz's laboratory reports, lymphocytes were shown to be low in numbers. What medical term would be used

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to indicate this condition? _____

6. In spite of low lymphocyte numbers, his WBC count was actually quite high. What medical term is used to indicate this condition? _____

7. A corrected WBC count was necessary, due to the six nucleated RBCs found on the blood smear evaluation. Which stage of erythrocyte maturation was most likely observed as NRBCs? _____

8. Buz's laboratory reports also showed that he had _____ or the condition of abnormally high numbers of monocytes.

9. Which medical term indicates a condition in which some young erythrocytes picked up multiple staining properties? _____

10. The medical term _____ is indicative of erythrocytes being stuck together by immunoglobulins. This was seen grossly and microscopically in Buz's blood sample.

11. Based on all of the available information, the veterinarian was highly suspicious of IMHA, an _____ disorder, in which the body produces antibodies against itself.

12. As part of his treatment, Buz received heparin, which is an _____, used to prevent clotting.

13. If heparin was not administered, Buz would have been at risk of _____ or a condition of clotting.

14. Buz's risk of clotting was greater than normal due to his erythrocytes sticking together in clumps and _____ or a condition of excess platelets.

15. Buz also received _____ agents, to reduce the

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production of antibodies against his own erythrocytes.

16. Antibodies are also called _____.
17. When Buz presented and during one of his transfusions, he developed a condition of fever, which is medically termed _____.
18. Medications and blood transfusions were inadequate to stop macrophagic destruction of his erythrocytes. So, he underwent surgical removal of his spleen, a procedure that is medically termed a _____.
19. Because of his heparin infusion, Buz was at risk of _____ or extreme blood loss during the surgical procedure.
20. Buz had excess numbers of neutrophils, medically termed _____, with a left shift. This information alone would be insufficient to make an accurate diagnosis in Buz's case, because those abnormalities are common in many conditions with inflammation or infection.

The Answer Key to these case study questions may be found in Appendix B.

[1] Latimer K.S., *Duncan & Prasse's Veterinary Laboratory Medicine Clinical Pathology*, 5th ed., Wiley- Blackwell Publishing; 2011: 9.

[2] Ibid., 9

[3] Ibid., 11

[4] Ibid., 49-50

[5] Ibid., 51

[6] Ibid., 50

[7] Ibid., 53

[8] Ibid., 57

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Applied Terminology for Muscles, Bones, and Joints

Applied Muscle Terminology,
- Muscle Types,
  - Cardiac Muscle,
  - Smooth Muscle,
  - Striated Muscle,
- Skeletal (Striated) Muscle Anatomy and Physiology,
  - Myocytes and Muscle Contraction,
  - Muscle Metabolism and Fatigue,
  - Neuromuscular Junction,
  - Muscle Hypertrophy and Atrophy,
  - Connective Tissues,
- Muscles and Muscle Groups,
  - Adductors and Abductors,
  - Extensors and Flexors,
  - Muscles of Respiration,
- Intramuscular Injection Sites,

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- Canine and Feline Intramuscular Injection Sites,
- Equine Intramuscular Injection Sites,
- Bovine and Caprine Intramuscular Injection Sites,
- Porcine Intramuscular Injection Sites,

Applied Bone Terminology,

- Basic Bone Anatomy,
- Bone Growth,
- Fractures and Fracture Repair,
  - Fractures,
  - Fracture Repair,

Applied Joint Terminology,

- Joint Types,
  - Fibrous Joints,
  - Cartilaginous Joints,
  - Synovial Joints,
- Joint Form and Function,
  - Hinged Joints,
  - Ball and Socket Joints,
  - Gliding Joints,
  - Pivot Joints,

Comparative Skeletal Anatomy,

- Canine and Feline Skeletal Anatomy,
- Equine Skeletal Anatomy,
- Ruminant Skeletal Anatomy,

Case Study,

GOALS AND OBJECTIVES

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By the conclusion of this chapter, the student will be able to:

1. Recognize common root words, prefixes, and suffixes related to muscles, bones, and joints.
2. Divide simple and compound words into their respective parts.
3. Recognize, correctly pronounce, and appropriately use common medical terms related to muscles, bones, and joints.
4. Demonstrate an understanding of various muscle types.
5. Demonstrate an understanding of basic muscle anatomy and contraction.
6. Recognize major muscles and muscle groups based on function.
7. Recognize comparative intramuscular injection sites commonly used in domestic animals.
8. Demonstrate an understanding of basic bone anatomy.
9. Demonstrate an understanding of bone growth.
10. Demonstrate an understanding of fractures and bone repair.
11. Demonstrate an understanding of joint anatomy, physiology, and movement.
12. Recognize major bones and joints of domestic animals by scientific and common names.
13. Demonstrate an understanding of comparative musculoskeletal anatomy of dogs, horses, and cattle (and other ruminants).

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Applied Muscle Terminology

Muscle Types

Muscle is muscle, right? Not so. We have three distinctly different types of muscle tissue. Their unique differences make each type well-suited for the jobs they do. Think about it. Do you really want your heart muscle to fatigue like muscles in your arms and legs? I didn't think so. So, pay close attention to gain appreciation for the value of each type.

Cardiac Muscle

Cardiac [*cardi(o)-* heart + *-ac* pertaining to] ***muscle*** (Fig. 4.1) is built like a tank, making it able to take a lickin' and keep on tickin'. Seriously, *cardiac* muscle starts contracting in order to pump blood when an animal or human is just an embryo, and it doesn't stop until death. Generally, there are years between birth and death. That is amazing endurance!

Cardiac muscle activity is ***involuntary*** [*in-* against + *volunt(o)-* the will + *-ary* pertaining to; i.e., against/independent of the will]. Thank goodness for that! If I had to consciously think about keeping my heart beating at a regular rate while walking or driving and chewing gum…well, let's just say that would not be pretty. Talk about distracted driving!

Structurally, *cardiac* muscle is ***striated*** (striped) tissue, similar to skeletal muscle. But there is one very unique feature that makes cardiac muscle completely different from its striated skeletal "cousin." That feature is something called ***intercalated*** [*inter-* between + *calat(o)-* to call + *-ed* to be, being; i.e., to be "called," inserted between] ***discs***. But they don't simply give cardiac muscle unique stripes. *Intercalated discs* are the roughest, toughest connections to be found between muscle cells. There isn't anything else like them anywhere else in the body. These are what give cardiac muscle its outstanding strength. They also provide for seamless delivery of nerve impulses from one cardiac muscle cell to the next. This makes contraction of cardiac muscle extremely coordinated, which is necessary for efficient pumping of blood from

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the heart. We'll talk much more about the heart and cardiac muscle in the ***cardiology*** [*cardi(o)-* heart + *-logy* study of] portion of Chapter 5.

Smooth Muscle

Like cardiac muscle, ***smooth muscle*** tissue (Fig. 4.2) is *involuntary* as well. Smooth muscle does not have striations, giving it a "smooth" appearance. (It's all in a name.) You'll notice in Fig. 4.2 that smooth muscle cells are tapered at each end. They contain actin and myosin filaments (the actual stuff that contracts) crisscrossed over the surface of each cell (Fig. 4.3). When they contract, pulling from each end of the cell, they shorten and bunch the cell up in the middle. In muscle tissue, this is a pretty unusual method of contraction. But it works! In fact, smooth muscle cells organize their contractions so that they appear to do "the wave," like at a sporting event. Seriously, smooth muscle contractions create smooth, rhythmic, wavelike movements. That's important, considering where it's located.

Where do we find smooth muscle? Well, because it's involuntary, it will be found in those areas of the body that we don't need to think about moving. So, that includes places like the digestive tract, respiratory tract, blood vessels, uterus, and urinary bladder. We'll discuss each of those in detail in their respective chapters. But let's focus on the digestive tract for just a moment. Think about what it takes to move food through the digestive tract. We certainly can't have intestinal muscles contracting in a chaotic manner. Nothing would move. We need the smooth muscle to contract slowly, methodically, and rhythmically, starting at the "front door" and progressively squeezing and moving stuff to the "back door." And those smooth, slow, rhythmic and methodical, one-way, wavelike movements successfully move food along at a pace that allows us to digest and absorb important nutrients. That's the beauty of smooth muscle.

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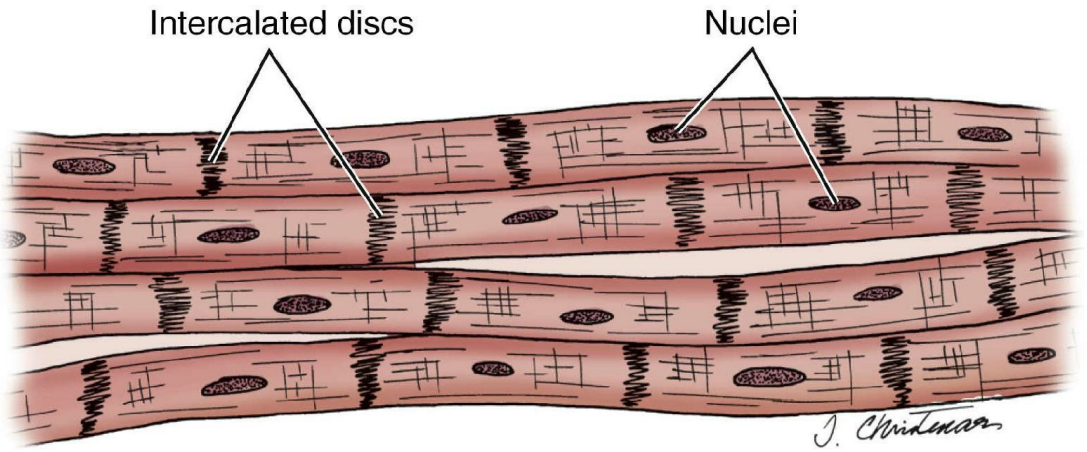

FIG. 4.1 Cardiac muscle.

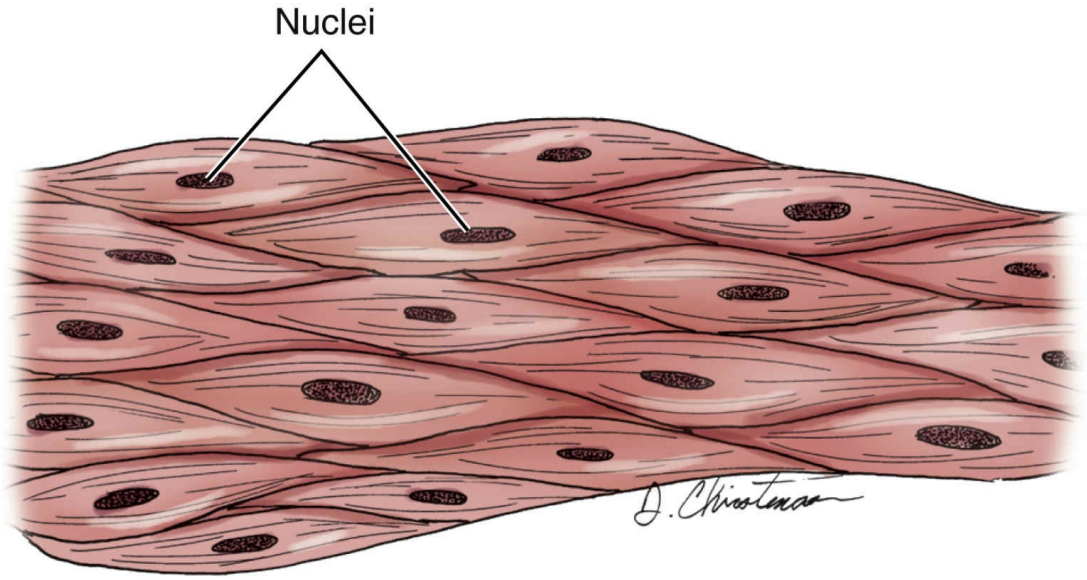

FIG. 4.2 Smooth muscle.

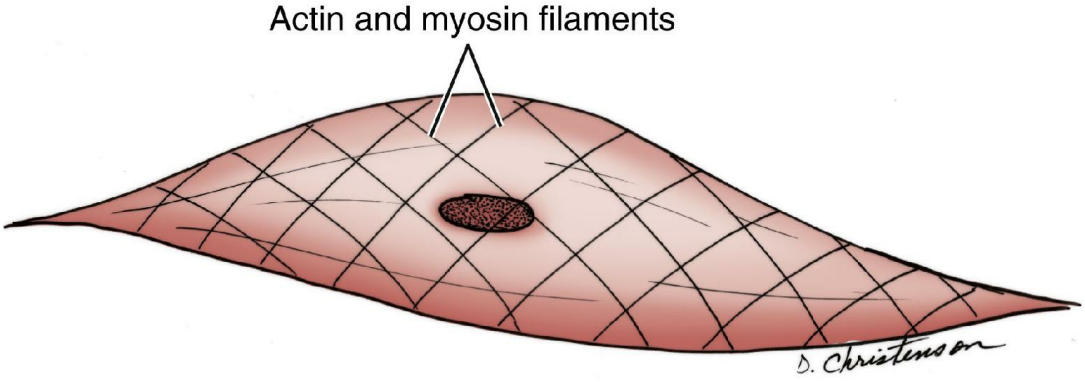

FIG. 4.3 Smooth muscle cell.

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Striated Muscle

Striated muscle tissue (Fig. 4.4) makes up the majority of the muscle tissue in the body. It's classically what we think of when we think of muscle tissue, because most of it is in ***skeletal muscle***. And you've probably already noticed from Fig. 4.4 that it is definitely striped; hence the name: *striated muscle*. Unlike the other types of muscle tissue we've discussed, striated skeletal muscle is ***voluntary*** [*volunt(o)-* the will + *-ary* pertaining to]. With the exception of things like protective reflexes or shivering to generate body heat, most skeletal muscle contractions for body movements are under conscious control. Because the bulk of ***somatic*** [*somat(o)-, soma* body + *-ic* pertaining to] tissues are composed of striated muscle, we're going to look at it in detail. It's structure and function are very important.

Skeletal (Striated) Muscle Anatomy and Physiology

Myocytes and Muscle Contraction

As you look at the skeletal (striated) ***myocyte*** [*my(o)-* muscle + *cyt(o)-* cell] in Fig. 4.5, you've probably already noticed that it doesn't have just one nucleus, it has multiple nuclei. This is much different from *cardiac* and *smooth muscle* cells, and most cells of the body. Part of the reason for this is the overall length of skeletal *myocytes*. They're not as microscopic as most other cells. Skeletal myocytes can actually be several inches long! That whole thing sure won't fit on our microscope! Because of that length it makes sense to have numerous nuclei for a single myocyte, doesn't it? And because myocytes are so long, we often refer to them as *muscle fibers*, rather than cells. Still, they are cells … long and skinny, but cells nonetheless.

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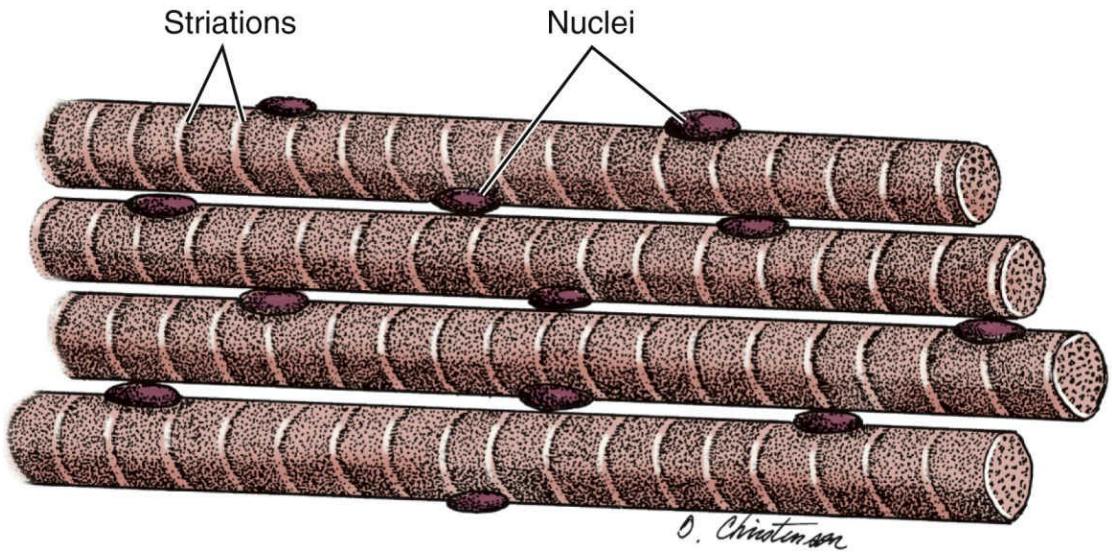

FIG. 4.4 Skeletal muscle.

Another thing you probably noticed in the cross section of the *myocyte* is that it looks more like the cross section of an electrical wire or braided metal cable. This is because each myocyte actually contains multiple long, skinny, cylindrical ***myofibrils*** [*my(o)-* muscle + *fibr(o)-* fiber + *-il* a, the] packed alongside one another, each and collectively surrounded by ***sarcoplasm*** [*sarc(o)-* flesh + *plasm* matter; i.e., cytoplasm]. Everything is held together by the ***sarcolemma*** [*sarc(o)-* flesh + *-lemma* a sheath; i.e., cellular membrane]. You know fromChapter 2 that a cell's organelles, like its *nucleus* and *mitochondria*, are suspended in the cytoplasm. It is similar in *myocytes*. The nuclei and mitochondria of myocytes are suspended in *sarcoplasm*. In fact, we find sarcoplasm not only around the whole bundle of *myofibrils*, but in between them too. Finally, kind of woven around and throughout all of the myofibrils is the ***sarcoplasmic*** [*sarc(o)-* flesh + *plasm(o)-* matter + *-ic* pertaining to] ***reticulum***. The *sarcoplasmic reticulum* of myocytes is similar to the *endoplasmic reticulum* of most other cells. (Please see Chapter 2 if you need a refresher on *intracellular organelles*.) The biggest difference between these two is that the sarcoplasmic reticulum contains high concentrations of Ca^{++} ions when the muscle fiber is at rest. That calcium will be very important for contraction of the ***sarcomeres*** [*sarc(o)-* flesh + *-mer(o)-* part; i.e., a contractile unit in skeletal muscle]. More on that later. Have you noticed a common theme here? *Sarcoplasm*, *sarcolemma*, *sarcomere*, *sarc(o)-* this and that. The root word *sarc(o)-* seems to precede many of those parts. Don't

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let it trip you up. At least you'll know whenever you see the root word *sarc(o)-* that it will have something to do with muscle and its surrounding tissues. It's just one of those things we have to accept and move on. Move on, we shall.

If we take a closer look at the myofibrils, we find that each myofibril is made up of numerous protein filaments. There are two different types of proteins making up those filaments: ***actin*** and ***myosin***. They say, "a picture is worth a thousand words" and that was never more true than now. So, we'll refer to Fig. 4.6 for this discussion. You may also watch the Evolve animation, Muscle Structure, to see it in action.

As you can see, the *actin* and *myosin* filaments are arranged in a neat, orderly fashion, forming little units for contraction called *sarcomeres* (see Fig. 4.6A). Considering the length of the myofibrils making up each long *myocyte*, we couldn't possibly have just one *sarcomere* per *myofibril*. No, we have countless contractile units, strung end to end along each one. Each muscle fiber has innumerable *sarcomeres*, and each muscle has innumerable muscle fibers. That provides for a great deal of strength for contraction.

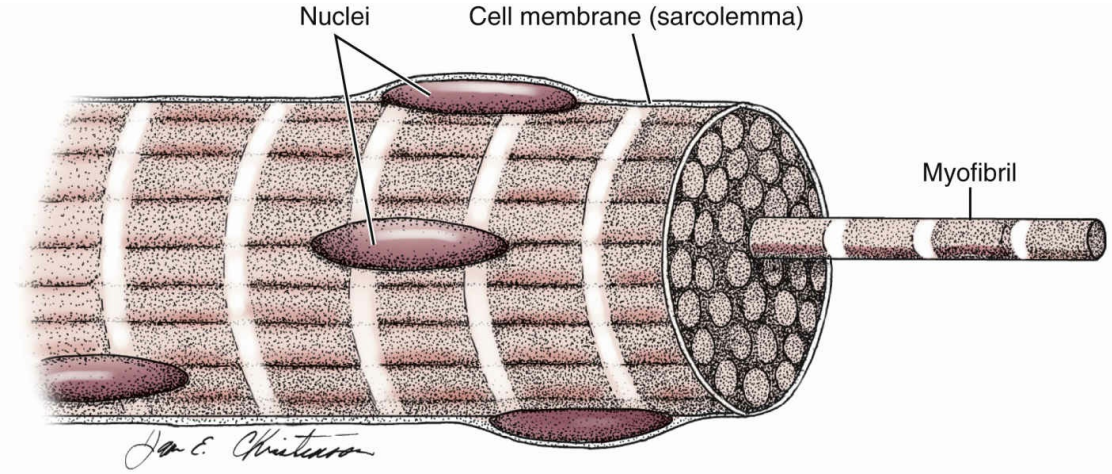

FIG. 4.5 Skeletal myocyte.

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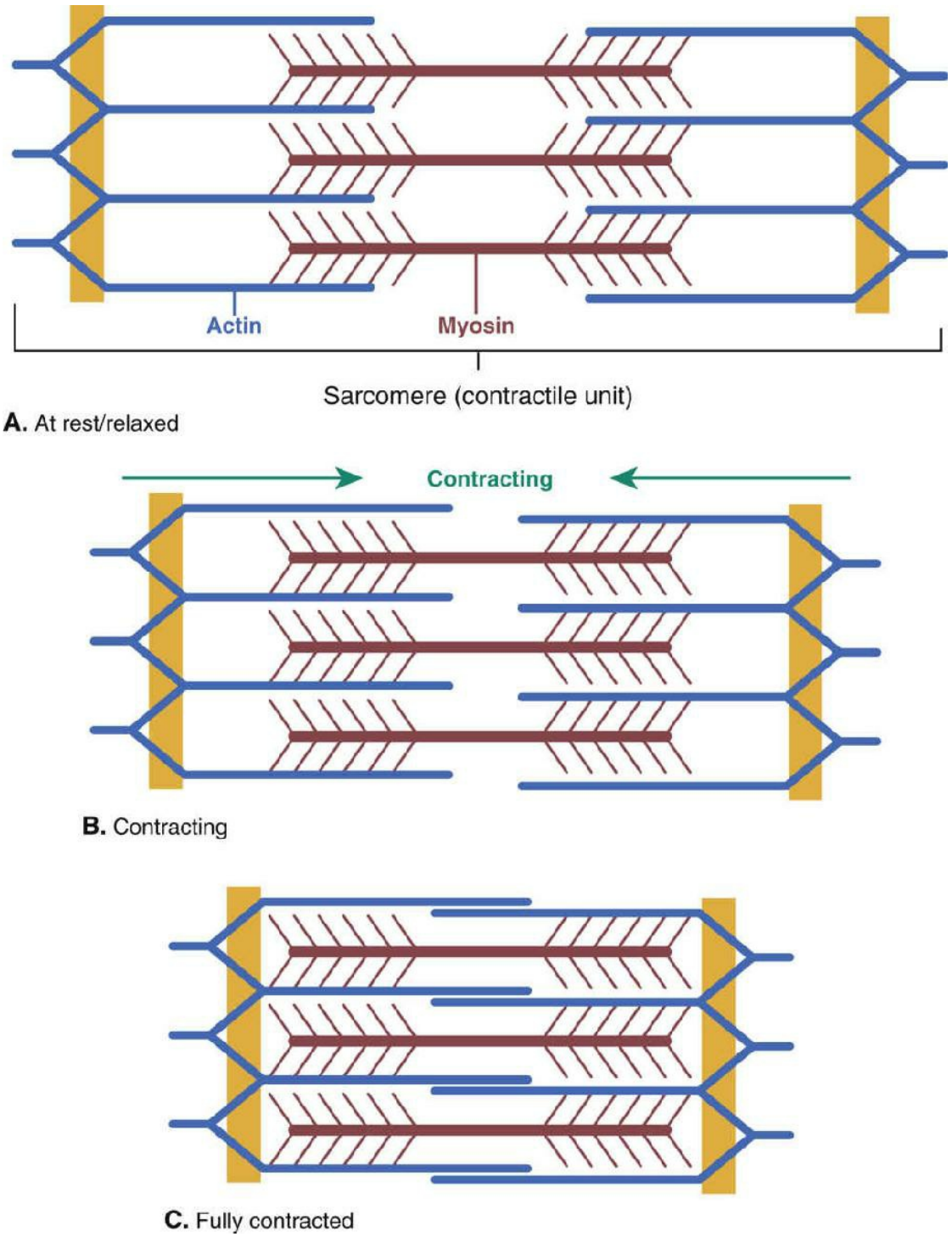

FIG. 4.6 Actin and myosin filaments.

So, how does a muscle fiber actually contract? Well, first we need stimulation from a nerve. We'll talk about that in detail in the section on the ***neuromuscular*** [*neur(o)-* nerve + *muscul(o)-* muscle + *-ar* pertaining to] ***junction***. Let's stay focused here on the muscle tissue. **Contraction** begins with nerve stimulation, causing Ca^{++} to diffuse **out** of the *sarcoplasmic reticulum* **into** the myocyte's

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sarcoplasm. (You'll find that calcium is important for many, many things, from *hemostasis* [*hem(o)-* blood + *-stasis* state of standing/stopping; i.e., clotting] to muscle contraction and much more. Remember that.) During contraction (see Fig. 4.6B), the actin and myosin filaments move along one another, progressively shortening each sarcomere. It's kind of a ratchet effect that doesn't stop ratcheting until the sarcomere is fully contracted (Fig. 4.6C). It's all or nothing when it comes to muscle fiber contraction. Then, it can relax to its original elongated state (see Fig. 4.6A) until the next contraction is stimulated. For **relaxation**, calcium diffuses **from** the *sarcoplasm* and back **into** the *sarcoplasmic reticulum*. (The Evolve animation, Muscle Fiber Contraction, reviews this entire process. It has much more detail than we need for our purposes here. Overall, it is a very good review.)

Golly, that makes it sound like muscle contraction is a jerky, wham-bam sort of movement. So, how can we explain the graceful movements of a cat stalking its prey, a bird taking flight, or a horse performing dressage? Well, muscle fibers are grouped into ***motor units***. All of the fibers within a given motor unit contract consecutively. The key to graceful movement is in the nervous system orchestrating all of the activity among the various *motor units*. In fact, an orchestra is probably a good analogy to use here. And let's focus on the human animal for some movements that we can really relate to. We have all drawn pictures or written notes with a pen or pencil, right? We don't need to use any large muscles or tremendous strength. Our dexterity of fine motor movements to create each stroke of the pen is orchestrated by our nervous system. The brain is the conductor, with each instrument (motor unit) playing its notes when directed to do so.

I promise, we will talk in detail about the nerve pathways and integration of sensory input and motor output in the ***neurology*** [*neur(o)-* nerve + *-logy* study of] portion of Chapter 11. For now, simply recognize that the motor impulses originate in your brain (conductor) and travel through your spinal cord and peripheral nerves, until the impulses finally reach the muscle fibers and motor units associated with each fine movement (notes played). I don't know about you, but I no longer have to think about how to hold the pen or how to move it to create each letter and word. Once a

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given motor task like that is learned and committed to memory, we can go on "autopilot" to carry it out again and again. The brain remembers the muscular requirements for the task. This is what some call "***muscle memory.***" It's the nervous system (brain), not the muscles, that remembers to orchestrate the needed muscle activity. This same type of *neuromuscular* activity takes place in animals, too, giving birds the ability to fly, horses to perform dressage, and cats to stalk stealthily.

Now, a message from our sponsor: the body. You'll find as we move along through this book that the body is a highly-integrated, well-orchestrated collection of tissues, organs, and body systems, all cooperating ***synergistically*** [*syn-* with + *ergist(o)-* one that works + *-cally* pertaining to]. Sure, we're taking the body apart in each chapter and focusing on individual parts and systems. Just remember the importance of ***synergism*** [*syn-* with + *erg(o)-* work + *-ism* a process or condition of]. We need every part of the body working in harmony with the whole to survive and live a healthy life. This *synergism* among the body's organs and systems results in the ***homeostasis*** [*home(o)-* unchanged + *-stasis* state of standing; i.e., the stable state of the body] we talked about inChapter 2. Now, we return now to our regular programming about muscles.

Muscle Metabolism and Fatigue

Of course, the more strength needed for a task, the more muscle fibers and collective muscles are required to make the movement happen. Plus, we need enough glucose and oxygen for efficient muscle activity. The more activity, the more glucose and oxygen are required. Most major muscles have an excellent blood supply to meet their needs of glucose and oxygen. Still, under times of extreme need, say for extraordinary strength and endurance, muscles may require more. Fortunately, we can actually store a bit of both molecules in muscle tissue, for just such occasions. ***Glycogen*** [*glyc(o)-* sugar + *gen(o)-* producer] is the storage form of glucose found in muscle tissue. When energy is needed, *glycogen* is pulled out of storage, "dusted off," and converted to a usable, ready-to-burn glucose molecule. Ah, but in order to "burn" it, we need sufficient oxygen. This is where ***myoglobin*** [*my(o)-* muscle + *glob(o)-* glob/"stuff" + *-in* the] is valuable.

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Myoglobin is the muscular equivalent of ***hemoglobin*** [*hem(o)-* blood + *glob(o)- glob* "stuff" + *-in* the]. Both are pigmented protein molecules. *Hemoglobin*, as discussed inChapter 3, is the red pigment of red blood cells that transports oxygen in the blood. *Myoglobin* is the red pigment of *myocytes* that can temporarily store oxygen. Here's a fun fact: myoglobin is way more attractive to oxygen than *hemoglobin*. What? It's true. And it's a very good thing because myoglobin acts like an accelerator to rapidly diffuse oxygen from the blood into muscle tissue. Whatever oxygen is left unused won't be wasted. It will be temporarily stored in the myoglobin. How cool is that?!

Ah, but now you're probably wondering why we experience muscle fatigue, right? Assuming we have sufficient calcium, glucose, and oxygen, why can't muscles just keep going and going? Cardiac muscle does, right? Right. But skeletal muscle is not the same, by any stretch (no pun intended). Plus, there are individual differences in athletic ability and endurance. Let's face it, there is a huge difference between a couch potato cat or Labrador, compared to a racing Greyhound or Standardbred horse. The couch potatoes' biggest activity of the day may be getting up to go to their food bowls. They may have plenty of glycogen stored, but they'll be hard-pressed to oxygenate well enough to run away from visiting grandkids. And because of that, their muscles will rapidly build up ***lactic acid***. Lactic acid is the by-product of ***anaerobic*** [*an-* without *aer(o)-* air/oxygen + *-bic* pertaining to] ***glycolysis*** [*glyc(o)-* sugar + *-lysis* process of breakdown, i.e., sugar metabolism]. The more *lactic acid* builds up, the less responsive the muscle fibers become to stimulation. Plus, we know how when we're in poor physical condition, overexertion makes our muscles burn and ache—all due to lactic acid. The ***myositis*** [*myos(o)-* muscle + *-itis* inflammation of] may linger for a few days, if we're really out of shape.

Do athletes experience lactic acid buildup and muscle fatigue? Yes. However, through regular conditioning, athletes develop more robust ***intramuscular*** (IM) [*intra-* within + *muscul(o)-* muscle + *-ar*] blood supplies, providing better delivery of glucose and, most importantly, oxygen. So they will not develop lactic acid as rapidly as the nonathletes. Over time, athletes also build up a tolerance to lactic acid, allowing them to "go the distance" better than average.

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Bottom line: athletes will not fatigue as rapidly as those who do not exercise on a regular basis. Now, if the owners of the couch potatoes would simply gradually increase their activity, they could effectively increase their endurance (owner and animal alike). No, they may never achieve the level of activity and endurance as the Greyhound or Standardbred, but they'll at least be able to outrun the grandkids, without muscular consequences.

Neuromuscular Junction

Previously we mentioned stimulation of muscle fibers, but we have not explained it in detail. Again, we'll cover much more of this in Chapter 11. However, it may be useful to have a preview of this portion of the *neuromuscular* discussion now. It will be helpful for understanding particular ***myopathies*** [*my(o)-* muscle + *path(o)-* diseases], like ***myasthenia*** [*my(o)-* muscle + *asthen(o)-* weakness + *-ia* a condition of] ***gravis*** [L. "heavy"]. We'll discuss that disease later. Right now, let's focus on the *neuromuscular junction*.

For this discussion, please refer to Fig. 4.7. As you can see, we have the terminal end of a motor nerve fiber inserting into the outer border of a myocyte. Beneath the sarcolemma and sarcoplasm is a portion of a myofibril, with the actin and myosin filaments just visible. But it's at the ***synapse*** [L. *synapsis* "connection"] that we really want to zoom in. In case you're wondering, a *synapse* is literally a *connection* between two nerves or between a nerve and a target tissue or organ. In this case, our target tissue or organ is muscle. We've enlarged one of the synapses shown for you to see it in greater detail.

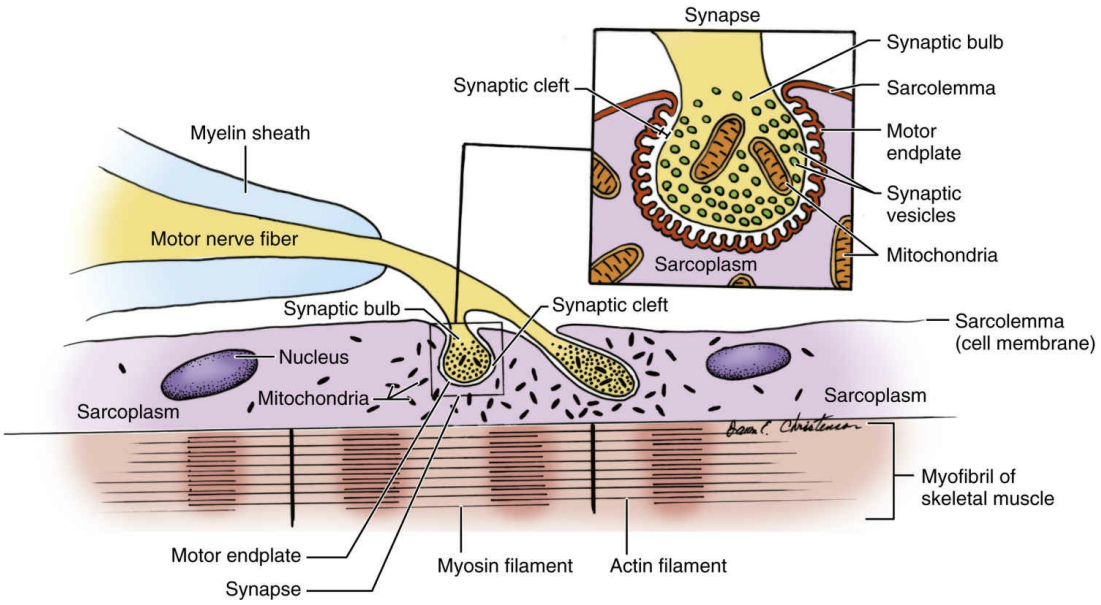

FIG. 4.7 Neuromuscular junction.

In the enlarged view of the *synapse*, you can see that the ***synaptic*** [*synap(o)-* connection + *-tic* pertaining to] ***bulb*** has numerous vesicles. These vesicles are full of the ***neurotransmitter*** [*neur(o)-* nerve + *transmit* send + *-ter* a, the], ***acetylcholine*** (uh-se'tul-ko"lēn). *Acetylcholine* is a chemical substance produced by nerves. There are other *neurotransmitter* substances in the body, but acetylcholine is the exclusive neurotransmitter used to stimulate skeletal muscle. Now look at the opposite side of that tiny ***synaptic cleft*** (i.e., gap). That is the motor endplate. Do you see how it has all of those little dips? Those will act like catchers' mitts, to catch the acetylcholine when it is released into the *synaptic cleft*.

So, this is what happens. As the nerve impulse reaches the synaptic bulbs, some of the vesicles merge with the margin of the bulb. The vesicles open up, releasing acetylcholine into the synaptic cleft. The acetylcholine is caught by the dips in the motor endplate, binding with receptors there. At this point, the myocyte is stimulated. Calcium diffuses from the sarcoplasmic reticulum into the sarcoplasm. With this ionic shift, all of the sarcomeres within the associated motor unit contract. When contraction is complete and calcium diffuses back from the sarcoplasm into the sarcoplasmic reticulum, the sarcomeres disengage and the myofibrils relax.

What happens to the acetylcholine? Well, we need to remove it from the synaptic cleft, especially the excess. We always release

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more than is needed for stimulation. And if we don't remove it from the cleft and motor endplate, stimulation will continue. A portion of the acetylcholine will be reabsorbed into the synaptic bulb and repackaged into vesicles for future use. But that can't take care of all of it. So, we have an enzyme that is released into the synaptic cleft. That enzyme is ***acetylcholinesterase*** (uh-se'tul-ko-lin-es"ter-ās). Okay, that's a really big word. Acetylcholine is a chemical ester. So, there's the *acetylcholinester (o)-* part. The suffix ***-ase*** means an ***enzyme***. No matter on what word you see the suffix *-ase*, you know it is some kind of enzyme. Enzymes are used to 'digest' or breakdown particular substances into smaller bits, for other uses or to be discarded. That is a long explanation to simply say that acetylcholinesterase is the enzyme designed to break down and remove acetylcholine from the synaptic cleft.

What would happen if acetylcholinesterase wasn't released into the synaptic cleft? Well, we would continue to have stimulation of the muscle fibers. That would create muscle tremors and shaking. If all of the ***cholinergic*** [*cholin(o)-* acetylcholine + *erg(o)-* working + *-ic* pertaining to] synapses are affected this way, we could have serious, whole-body seizure activity. This does happen. I've seen it many times. One of the most common scenarios usually involves an ***insecticide*** [*insect* + *cid(o)-* killing; i.e., a chemical used to kill insects]. Guess what—insects have *cholinergic* synapses too. So, if the *insecticides* kill bugs by preventing the release of acetylcholinesterase, then they can kill animals and people that way, too. Sure, the lethal (deadly) dose for a bug is probably less than that of a dog or a human. But we need to remember that some individuals and certain species of animals are more susceptible to these chemicals than others. Cats and birds tend to be highly sensitive to many chemical agents like these. **CAUTION!**—(1) We need to be VERY careful to read all label directions of any ***pesticides*** [*pest* + *cid(o)-* killing]. (2) They need to be applied appropriately, minimizing risk of exposure to nontarget organisms (animals, people, and valuable insects, like honey bees). (3) We need to take care to give detailed instructions for use to our clients, if those products are sold in our veterinary facilities. (4) In emergency exposure situations, we need to tell the owners to bring the product container or packaging with the animal, so that we know what the

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chemical is in order to know how to appropriately treat the animal. Failure to do these things can have deadly consequences. Okay, I'll get off of my soapbox for now.

At the other end of the spectrum, it is possible to have insufficient acetylcholine for muscle contraction. We mentioned *myasthenia gravis* at the beginning of this section. In this disease, we have three possible causes of the *myasthenia* (muscle weakness). Now, if we think back to the structure and function of the neuromuscular synapse, it seems like there are three places to break it: (1) insufficient acetylcholine in the synaptic bulb, (2) removing acetylcholine too quickly from the cleft, or (3) defective cholinergic receptors on the motor endplate. All could be reasonable possibilities. But *myasthenia gravis* in animals tends to be an acquired ***autoimmune*** [*auto-* self + *immun(o)-* protection; i.e., a disease in which the immune system attacks oneself] disorder. (Please refer toChapter 3 to review immunity and autoimmune disorders.) ***Autoantibodies*** [*auto-* self + *antibody*; i.e., antibodies directed against self] disable many of the *cholinergic* receptors. What we see is a weak animal whose weakness progresses and worsens with activity. A dog, for example, may be able to get up and begin walking across a room. But the dog rapidly fatigues, slowing down and finally collapsing, perhaps before reaching the other side of the room. Because this is an *autoimmune disorder*, we can treat patients with medications to suppress the immune system. This will ultimately free up cholinergic receptors blocked by the autoantibodies. In the meantime, we still have a disabled patient. So, to improve motor function, we can also give medications to partially block release of acetylcholinesterase. This prolongs available acetylcholine in the synaptic cleft for better motor endplate stimulation and muscle contraction. Even after all the years that I have been in veterinary medicine, I am still amazed at how we can apply our understanding of anatomy and physiology to benefit the health and well-being of our patients. Knowledge is a very good thing.

Muscle Hypertrophy and Atrophy

I am no stranger to muscle ***hypertrophy*** [*hyper-* excess + *troph(o)-* development] or ***atrophy*** [*a-* without + *troph(o)-* development]. I've

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experienced both, especially the latter. And I have certainly seen considerable muscle *atrophy* in veterinary patients. While I have seen some athletic animals and their bulging muscles, when it comes to hypertrophy, most of the hypertrophy I've seen involved cases of ***hypertrophic*** [*hyper-* excessive + *troph(o)-* development + *-ic* pertaining to] ***cardiomyopathy*** [*cardi(o)-* heart + *my(o)-* muscle + *-pathy* a disease of]. *Hypertrophic cardiomyopathy* is common in cats. Suffice it to say that bulging *cardiac* muscle reduces the size of the heart chambers, ultimately adversely affecting the volume of blood pumped. We'll talk more about that in Chapter 5. For our discussion here, let's stay focused on skeletal muscle.

If you've ever done any regular exercise, you've probably experienced some muscular *hypertrophy*. If you're a bodybuilder, you have really experienced a great deal of hypertrophy. But do you know how you managed to bulk up on muscle mass? With regular exercise, to meet the metabolic needs of the muscle fibers (i.e., with sufficient glucose and oxygen), new blood vessels form. In addition to the new ***vascular*** [*vascul(o)-* vessel + *-ar* pertaining to] networks, more mitochondria develop in the sarcoplasm. Okay, so this will help us fatigue less rapidly, but where does the bulk come from? Well, don't be disappointed when I tell you that you don't actually create new muscle fibers. No, the muscle fibers themselves actually become bulky. Within the myofibrils, we actually develop new actin and myosin filaments. So, the myofibrils themselves increase in diameter. Collectively then, the muscles experiencing *hypertrophy* due to exercise become much larger. Ah, but if you become a couch potato, you will experience *disuse atrophy* and lose all of the mass you gained.

I have experienced *disuse* and ***denervation*** [*de-* away from + *nerv(o)-* nerve + *-tion* a state of] *atrophy*. Leading up to and following my total knee replacement surgery, I had limited use of the affected leg. Because of that my thigh muscles experienced a great deal of *atrophy*. The denervation atrophy developed in my left shoulder and arm muscles because of ***cervical*** [*cervic(o)-* neck + *-al* pertaining to] spine issues. Motor nerves to the affected arm and shoulder muscles were compressed and damaged, so the muscles grew weak. Muscle weakness and loss of sensation in my hand all played a part in limiting my use of the arm. All of that led to profound

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atrophy. In both situations, muscle activity and use were significantly reduced. This in turn caused reduction of capillary (smallest of blood vessels) networks and mitochondria throughout those muscle tissues. Actin and myosin filaments were also reduced. Ultimately, the overall size (bulk) of the muscles were much, much smaller. In fact, the atrophy was so great that I actually experienced significant weight loss. Now that the causes of the atrophy are gone, I am slowly and steadily rebuilding my muscles. It takes time. This is something to remember with our veterinary patients, too.

I have cared for numerous veterinary patients with both disuse and denervation atrophy. If the inciting cause of atrophy can be corrected, it will take much time and rehabilitation for recovery. This is especially true for ***neurologic*** [*neur(o)-* nerve + *log(o)-* study, knowledge + *-ic* pertaining to] patients. We'll talk much more about this in Chapter 11. But whether we are talking about patients with *neurologic* or ***orthopedic*** [*orth(o)-* straight, straightening + *ped(o)-* child + *-ic*; i.e., patients needing correction of musculoskeletal abnormalities] disorders, we also need to take care of musculoskeletal connective tissues. This is especially true in recovery because connective tissues can become quite fragile.

Connective Tissues

Most individual muscles are separated from one another by sheets of ***fibrous*** [*fibr(o)-* fiber + *-ous* pertaining to] ***connective tissue*** called ***fascia*** [L. "band"]. Some of this *fascia* extends beyond the muscle tissue forming strong, cord-like bands of connective tissue called ***tendons***. Tendons firmly connect the muscles to bone.

Sometimes muscles require tough connections with other muscles. For this there is a special sheet of *fibrous connective tissue* called an ***aponeurosis*** [ap"o-nuh-ro'sis; Gr. "sinew"; "solid resilient strength"]. Don't be confused by this word. Do **not** try to subdivide it. It has nothing to do with nerves. It is simply one of those centuries-old words that cannot be fully explained. It is what it is. Just take it and run with it. Where might we find *aponeurosēs* (plural)? They can be found between muscles of the abdominal wall. Obviously we don't have any bones there to connect to the muscles. (Wow, that would be weird and uncomfortable, now that I

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think about it.) Probably the most prominent *aponeurosis* is found on the ventral midline between abdominal muscles on either side of the abdomen. This aponeurosis is an extremely thick, tough, long, white line of connective tissue called the ***linea*** [L. "line"] ***alba*** [L. "white"]. When performing abdominal surgery, we most often enter the abdomen by incising (cutting) through the *linea alba*. It provides an excellent anchor for the suture material used to close the abdomen.

I will mention ***ligaments*** (lig'uh-ment) here. While they have nothing to do with muscles, they are made of fibrous connective tissue. *Ligaments* are the tough, cord-like bands of connective tissue that connect one bone to another. Less frequently, ligaments may also anchor and support internal organs, like the suspensory ligament of the spleen.

Whether we are talking about ligaments, tendons, fascia, or aponeuroses, they are all made of dense, fibrous connective tissue. Because of that they all share similar characteristics. First, fibrous connective tissue is not as vascular as other tissues, like muscle tissue. Connective tissues do not have heavy cellular populations. Fibrous connective tissue is largely made of ***collagen*** [*coll(o)-* glue + *gen(o)-* producer] fibers. This makes these tissues very tough, resilient, and somewhat flexible. However, because these connective tissues have limited blood supplies and cellular populations, if they are injured, these tissues will take longer to heal than other tissues. The principal cells for repair are ***fibroblasts*** [*fibr(o)-* fiber + *blast(o)-* germ, shoot]. There may be a few *fibroblasts* in the area of an injury, but not enough to facilitate repairs. Inflammatory mediators, discussed inChapter 3, will attract *fibroblasts* to the injured tissues. But it will take many days for them to migrate and arrive there in numbers high enough to facilitate repairs. Once there, they need to rebuild the collagen fibers of the connective tissue and then reorganize and reorient the fibers in appropriate alignment. This takes time. If you've ever sprained an ankle, you know this all too well. And it is worth noting that the reparative collagen is never as strong as the original connective tissue.

Our *orthopedic* patients often experience not only trauma to ligaments and tendons, but we also subject their limbs to

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immobilization. During immobilization, even if the connective tissues surrounding the joints are not damaged, they will experience changes. You see, while immobilized, the connective tissues are not under normal bending and stretching forces. It will be perceived by the handful of fibroblasts in the area that the connective tissues need to be debulked and reorganized. Shortening of the collagen fibers is a principal tactic for reorganization. What does this mean for our patient when its limb is removed from the splint or cast? The connective tissues of ligaments, tendons, joint capsule, and so forth have shrunk and have been weakened. The longer the immobilization, the greater the connective tissue consequences. By the way, *neurology* patients who are paralyzed are immobilized by their neurologic injury. Part of our care of such patients is to maintain their connective tissues surrounding joints by performing ***passive range of motion*** (PROM). If we don't, when they recover from the neurologic injury, they may have difficulty getting up and walking due to their joints simply being too stiff. Ligaments and tendons of the toes can undergo such dramatic contracture (shrinkage) that the toes literally curl under, making it impossible for the dog or cat to walk on its foot pads. This is preventable. Even if the patient doesn't experience such extreme changes, it will likely need rehabilitation therapy.

Rehabilitation for stiff, weakened connective tissues surrounding joints is frequently necessary for many types of patients. But we must be careful and remember that these connective tissues are more fragile than normal. If we force a joint beyond its current range, we could actually rip and tear the connective tissues. This will be extremely painful for the patient and will delay its recovery. Gentle, sustained, stretching, repeated over time will achieve the needed result. If we apply warmth to the connective tissues first, we will improve the range of the stretch. Warmth also helps the connective tissues sustain the stretch better.

So, later on when we talk about bones and fractures, and paralysis in Chapter 11, remember the nature of connective tissues. They are very necessary to hold us together. They also take special care to recover from disease and injury.

Muscles and Muscle Groups

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Alrighty, we are finally ready to begin talking about various skeletal muscles and muscle groups. To begin this discussion, let's think about functional groups of muscles. I say "functional groups" because I want us to think about the actions or motions that contraction of those muscles will actually create. So, let's talk about limb movements. We'll talk about these movements again when we discuss joints. The repetition should help your understanding.

Adductors and Abductors

Nearly all of our patients are ***quadrupeds*** [*quadru-*, from *quadri-* four + *ped(o)-* foot, feet]. In order to stand in an upright position on all four feet, these animals need strong muscles to draw their legs toward the median plane. This movement is called ***adduction*** [*ad-* toward + *duct(o)-* draw, lead + *-tion* the act of]. Muscles of the medial thigh, for example, are responsible for adduction of the rear limb. The most logical way to name the group of medial thigh muscles, based on their function, is to call them ***adductors*** [*ad-* toward + *duct(o)-* draw, lead + *-or* the]. We have *adductors* of the front limbs too. Many of the front limb adductors are trunk muscles, not only *adducting* the legs, but also securing the legs to the chest wall. ***Pectoral*** [*pector(o)-* chest + *-al* pertaining to] ***muscles*** are principal *adductors* of the front limbs. They are found bilaterally along the cranial ***sternum*** ("breast bone").

Most muscle groups have opposing muscle groups that counter their movements. It's sort of a yin-yang thing. So, if we can ***adduct*** [*ad-* toward + *duct(o)-* drawing] a limb, we must be able to ***abduct*** [*ab-* away + *duct(o)-* drawing] it too, right? Absolutely! Both movements are shown in Fig. 4.8A. So, the movement of ***abduction*** [*ab-* away + *duct(o)-* draw, lead + *-tion* the act of] is facilitated by contraction of ***abductor*** [*ab-* away + *duct(o)-* draw, lead + *-or* pertaining to;] ***muscles***.

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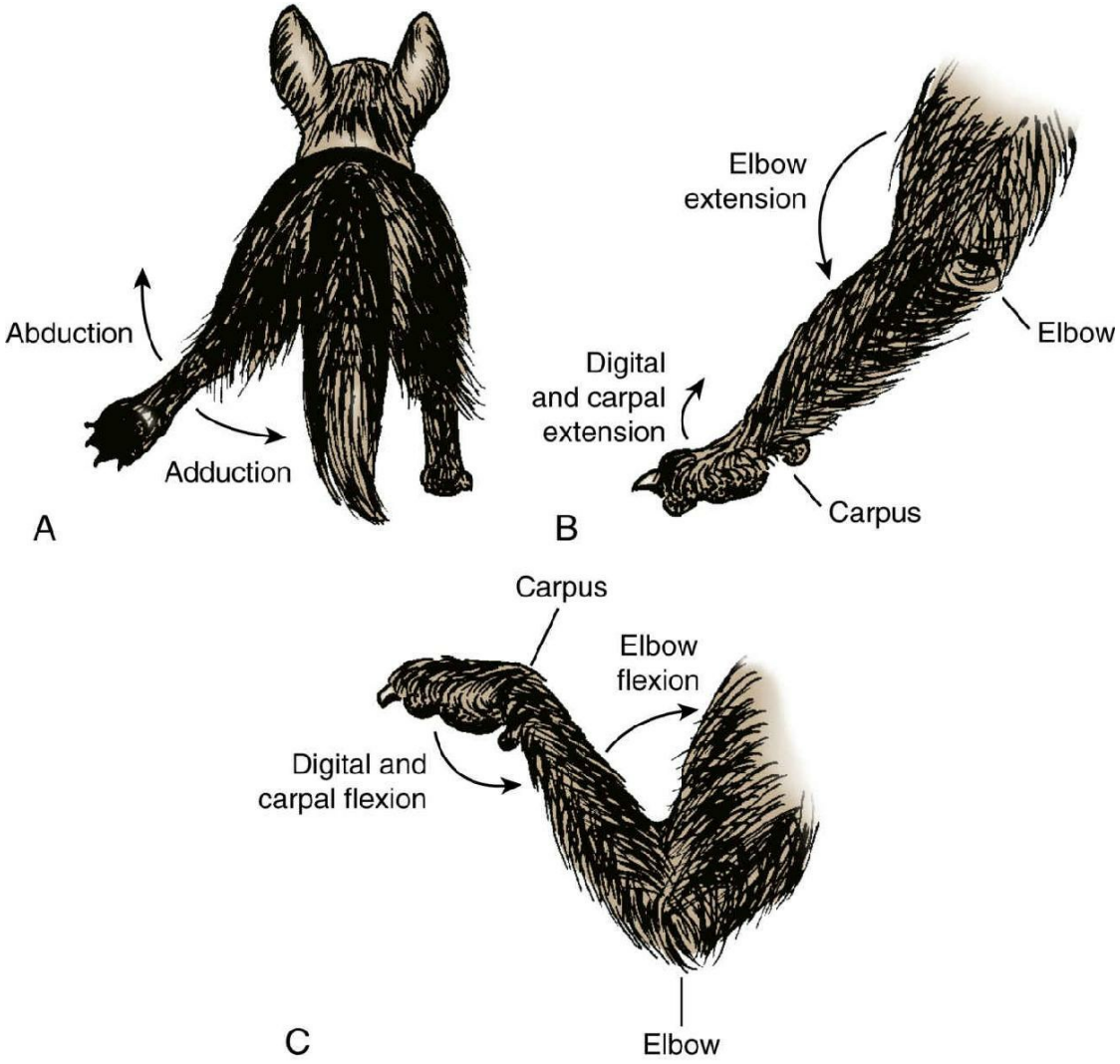

FIG. 4.8 (A to C) Joint movements.

Time out. Did you notice that the suffix *-or* in the prior sentence was defined as "pertaining to"? Earlier it was defined as "a, the," to create a noun. This is one of those suffixes that's kind of a "switch hitter." It can be used to create nouns or adjectives. It all depends on use of the word. When I wrote "abductor muscles," abductor was modifying the word muscles. So, abductor in that sentence was an adjective, therefore the definition of the suffix *-or* had to be "pertaining to." If I had simply written *abductors* to refer to these muscles, that word standing alone would have to be a noun because abductors are things. I could talk about an individual *abductor*—again, a noun, a thing. I don't want to belabor this. I just want to address a possible question that the flip-flop in suffix use may have created for you. If this little side discussion was not the least bit helpful for you, then flush it and move on.

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By the way, are you wondering why *quadrupeds* have abductors, when most of the time they have all four legs straight underneath them? Good question. Think about it. How else would a squirrel reach wide to latch onto the side of a tree? And among domestic animals, well I guess you've never had a dairy cow try to nail you with a powerful side kick. Believe me, you want to stand close to those ladies. It is far better to merely be pushed by their *abduction*, then to be the recipient of the powerful blow at the end of that "sucker-punch." The ***gluteal*** [*glute(o)-* "butt", buttocks + *-al* pertaining to] ***muscles*** play a big role in that abduction movement. That's another good reason to stand close. Then you can feel the *gluteals* begin to contract as she gets ready to take that swing.

Extensors and Flexors

Okay. So *quadrupeds* have opposing *adductors* and *abductors* that help them stand on their four feet. Obviously, these opposing muscle groups are insufficient for standing. We need to maintain the legs in ***extension*** [*ex-* out + *tens(o)-* stretching + *-sion* the act of]. So we have *extensor muscles* in key locations, like the cranial thigh and the caudal forelimb just proximal to the elbow (see comparative Figs. 4.9A to D). These muscles extend the joints distal to them, as shown in Fig. 4.8B, for example. ***Digital*** [*digit* toe + *-al* pertaining to] ***extensors*** of the front foot are located on the cranial ***antebrachium*** [*ante-* before + *brachi(o)-* "arm" + *-um* the]. The *antebrachium* is that portion of the foreleg distal to the elbow and proximal to the ***carpus*** [*carp(o)-* "wrist" + *-us* the]. In fact, these muscles extend the digits and the carpus both. The ***triceps*** [*tri-* three + *ceps(o)-* heads], located on the caudal ***brachium*** [*brachi(o)-* arm + *-um* the; i.e., caudal foreleg between the elbow and shoulder], are the extensors of the elbow. Why call them *triceps*? Well, because they actually have three distinct "heads," making up the triceps muscle group. Extensors of the rear limb, as we've already mentioned, include the cranial thigh muscles. Here we have distinct "heads," too—four to be exact. The *extensors* of the stifle joint are the ***quadriceps*** [*quadri-* four + *ceps(o)-* heads] muscles. What about hip extension? Well, the *gluteals* play a large role in that, too. Distal to the stifle, we have separate extensors for the digits and the tarsus ("hock"). This is just a bit different from the front leg. Digital

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extensors are in a similar location and actually named appropriately for their location: ***cranial tibial muscle***. But the ***tarsal*** [*tars(o)-* tarsus + *-al* pertaining to] extensors are found along the caudal tibia. Because these muscles collectively are shaped to look like a "stomach" on the distal leg, they have been aptly named the ***gastrocnemius*** [*gastr(o)-* stomach + *cnem(o)-* leg + *-us* the].

We need opposing muscles to counter the extensors, permitting us and our furry friends to walk. Our ***flexors*** [*flex* to bend + *-or* a, the] will be found on the opposite side of the leg from our *extensors* (comparative Figs. 4.10A to D). In the front limb, the muscles providing ***flexion*** [*flex* to bend + *-ion* the act of] (see Fig. 4.8C) of the elbow are the ***biceps*** [*bi-* two + *ceps(o)-* heads] muscles. Yep, you guessed it. The *biceps* have two distinct muscular "heads." They are found along the *cranial brachium*. Our ***digital flexors*** of the front limb are found along the *caudal antebrachium*. In the rear limb, some of the *quadriceps muscles*, along with others in the area, serve to flex the hip. Flexors of the stifle are found in the caudal thigh. The names of these flexors really don't make as much sense as some of the other muscles we've named. Perhaps that's why so often folks commonly refer to them as the "ham" or "hamstring." The ***semimembranosus*** [*semi-* partial + L. *membranosus* membrane] is the deeper and larger of the two muscle groups. The ***semitendinosus*** [*semi-* partial + L. *tendinosus* tendon] is thinner and more superficial over the caudal thigh. Typically, we think of both of these flexors as a functional unit. That's why you'll often hear folks refer to them as simply the *sĕmīs*. No, these semis have nothing to do with the trucking industry. As to the rest of the flexors of the rear leg, we need to look again at some muscles we've already mentioned as extensors. Which is which depends on the effect of the associated joints. We said that the *cranial tibial muscle* would provide digital extension, right? Well, it also *flexes the tarsus*. And we said that the *gastrocnemius* muscles provide *extension* of the tarsus. Well, those muscles are also the *digital flexors*. Functionally, context is everything.

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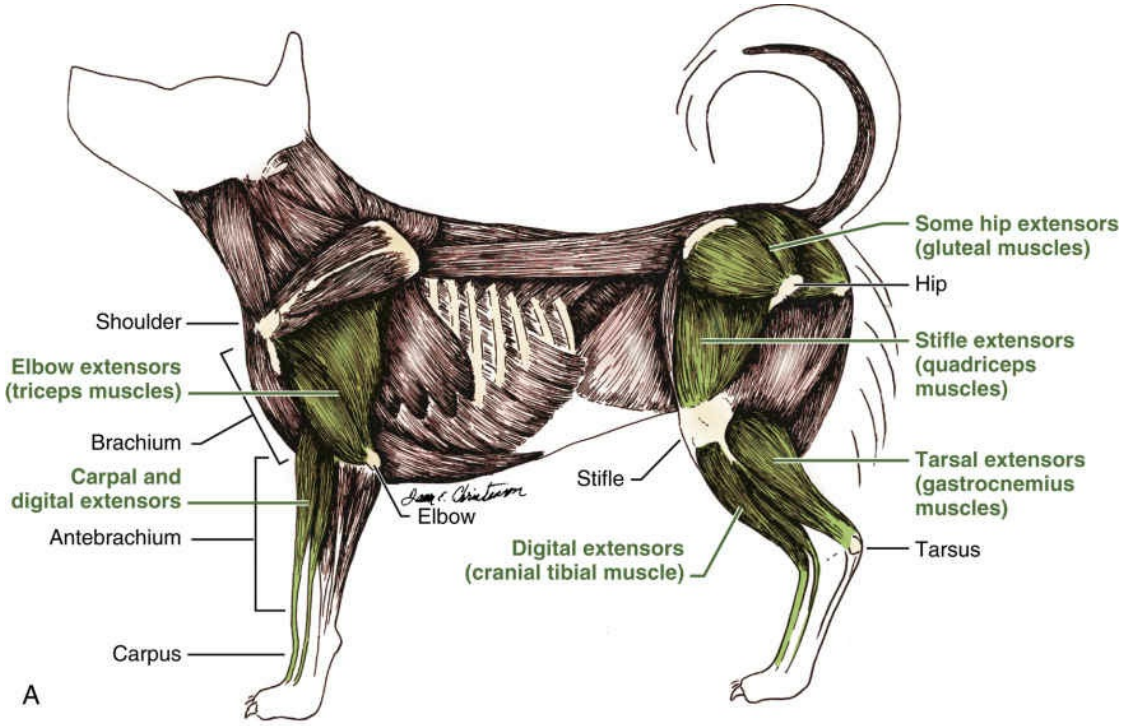
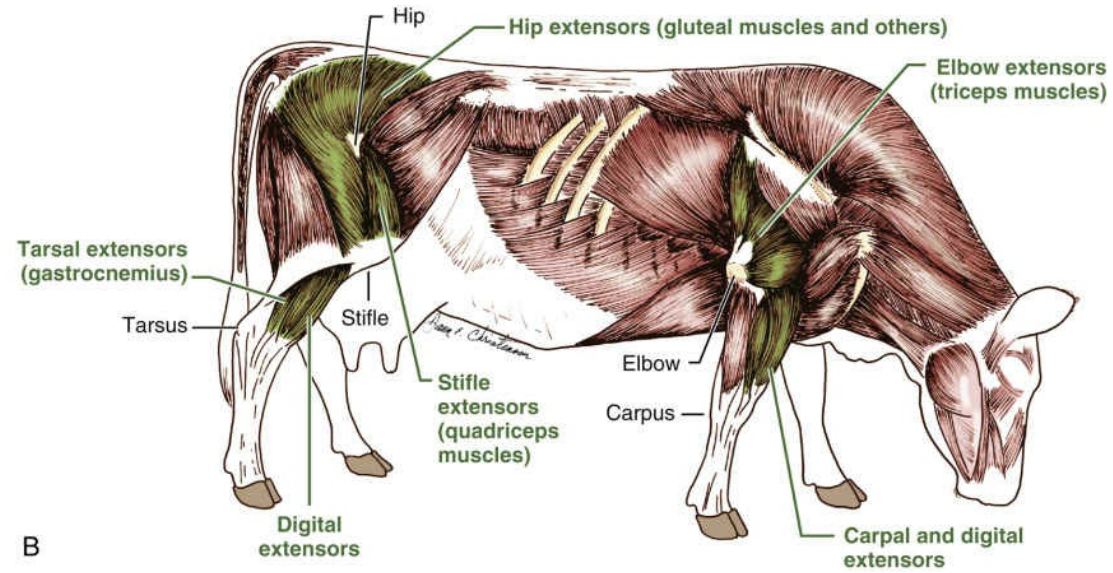

Some hip extensors (gluteal muscles)
Hip
Shoulder
Stifle extensors (quadriceps muscles)
Elbow extensors (triceps muscles)
Brachium
Tarsal extensors (gastrocnemius muscles)
Carpal and digital extensors
Stifle
Elbow
Antebrachium
Digital extensors (cranial tibial muscle)
Tarsus
Carpus
A


Hip
Hip extensors (gluteal muscles and others)
Elbow extensors (triceps muscles)
Tarsal extensors (gastrocnemius)
Tarsus
Stifle
Elbow
Stifle extensors (quadriceps muscles)
Carpus
Digital extensors
Carpal and digital extensors
B

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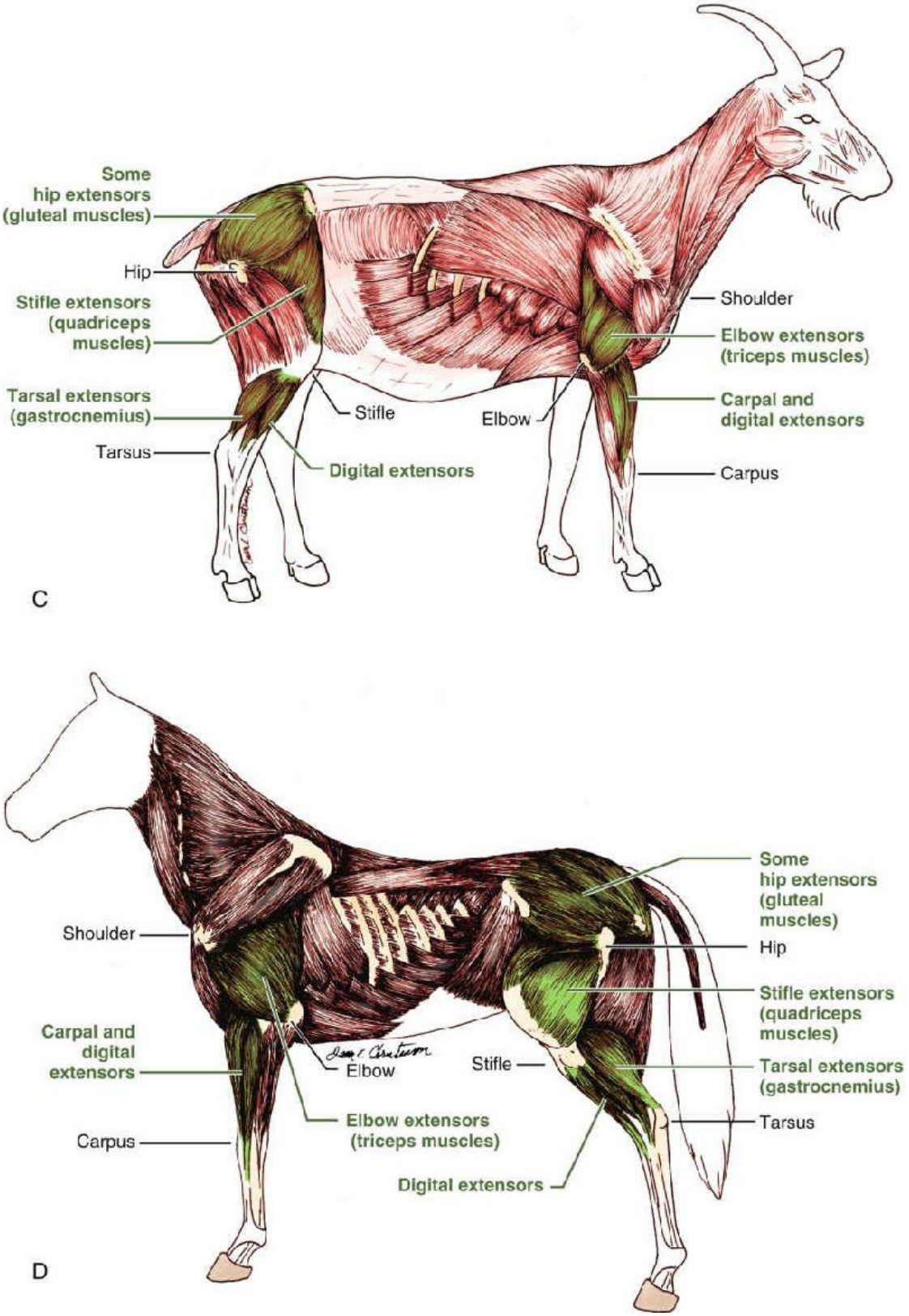

FIG. 4.9 (A) Canine extensor muscles. (B) Bovine extensor muscles. (C) Caprine extensor muscles. (D) Equine extensor muscles.

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Muscles of Respiration

We will discuss these again in Chapter 5. Because breathing is so important, it's worth discussing them twice. The principal muscle of respiration is the ***diaphragm*** [di-uh-fram', Gr. *diaphragma* "a partition-wall"]. As early Greek anatomists indicated, this muscle forms a wall between the ***thoracic*** [*thorac(o)-* chest + *-ic* pertaining to] and abdominal cavities (see Fig. 3.19 inChapter 3 for its location). No, this is not skeletal, striated muscle tissue. It's involuntary smooth muscle. But we certainly can't talk about respiratory muscles without including this one. As there are no joints involved, we can't talk about any of the previous functional movements here. What happens when the diaphragm contracts? It transforms from being domed cranially into the caudal thoracic cavity when it's relaxed, to flattened while pushing and squishing abdominal organs caudally when it contracts. This is largely responsible for ***inspiration*** [*in-* in + *spir(o)-* breathing + *-tion* the act of].

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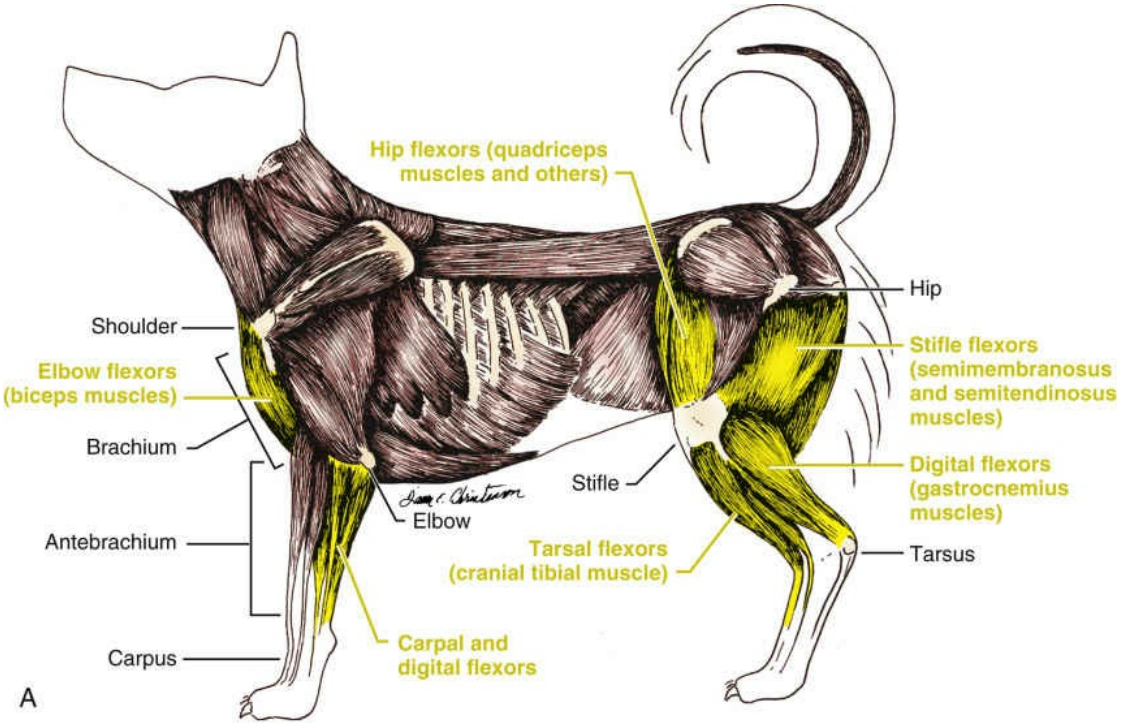
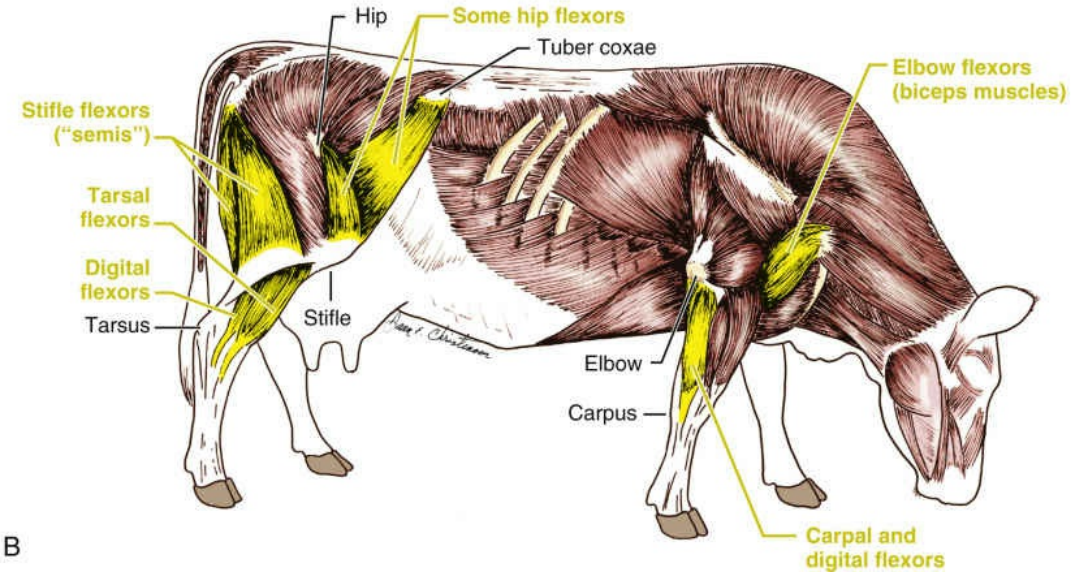

Hip flexors (quadriceps muscles and others)
Hip
Shoulder
Stifle flexors (semimembranosus and semitendinosus muscles)
Elbow flexors (biceps muscles)
Brachium
Digital flexors (gastrocnemius muscles)
Stifle
Elbow
Antebrachium
Tarsal flexors (cranial tibial muscle)
Tarsus
Carpus
Carpal and digital flexors
A


Hip
Some hip flexors
Tuber coxae
Elbow flexors (biceps muscles)
Stifle flexors ("semis")
Tarsal flexors
Digital flexors
Tarsus
Stifle
Elbow
Carpus
Carpal and digital flexors
B

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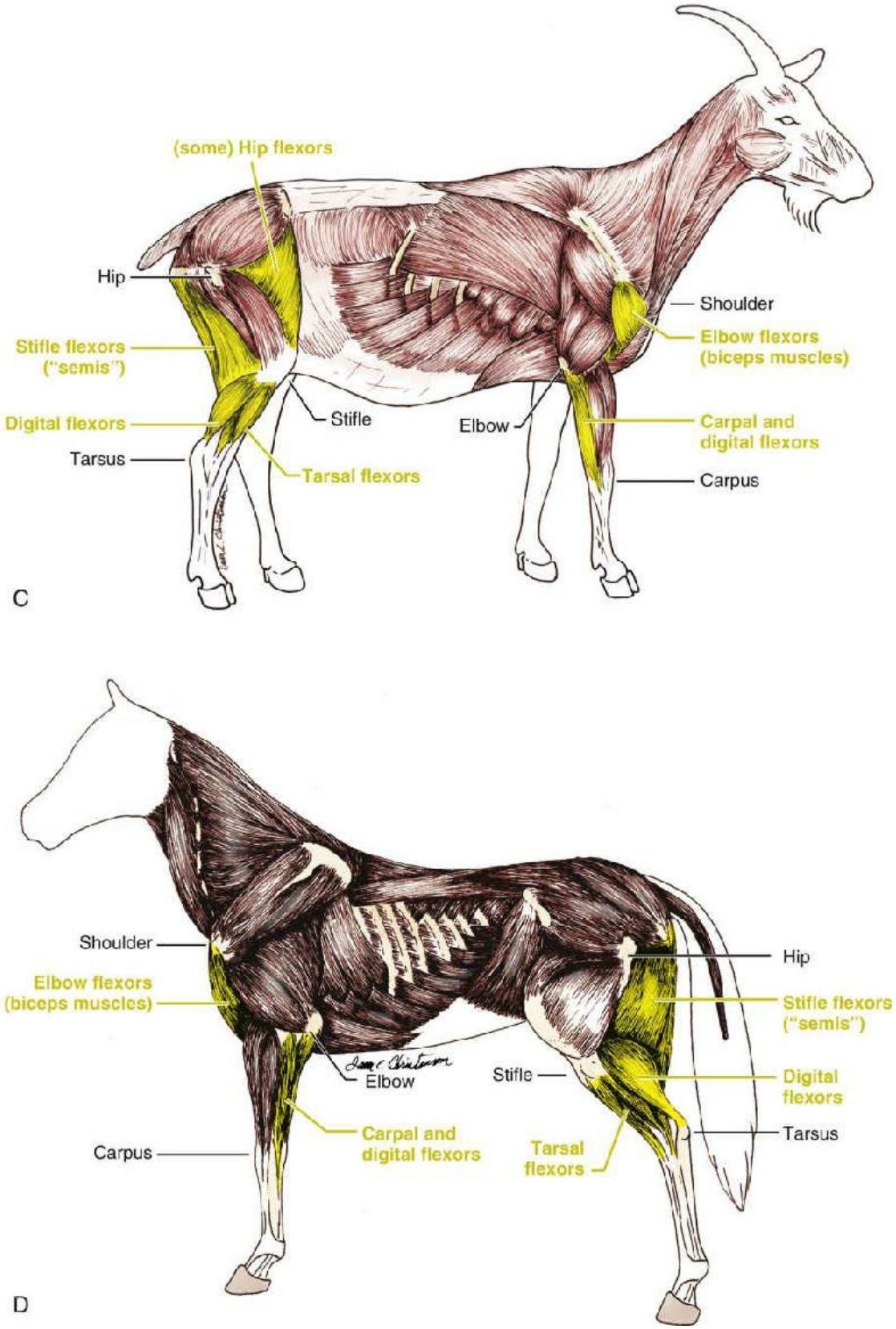

FIG. 4.10 (A) Canine flexor muscles. (B) Bovine flexor muscles. (C) Caprine flexor muscles. (D) Equine flexor muscles.

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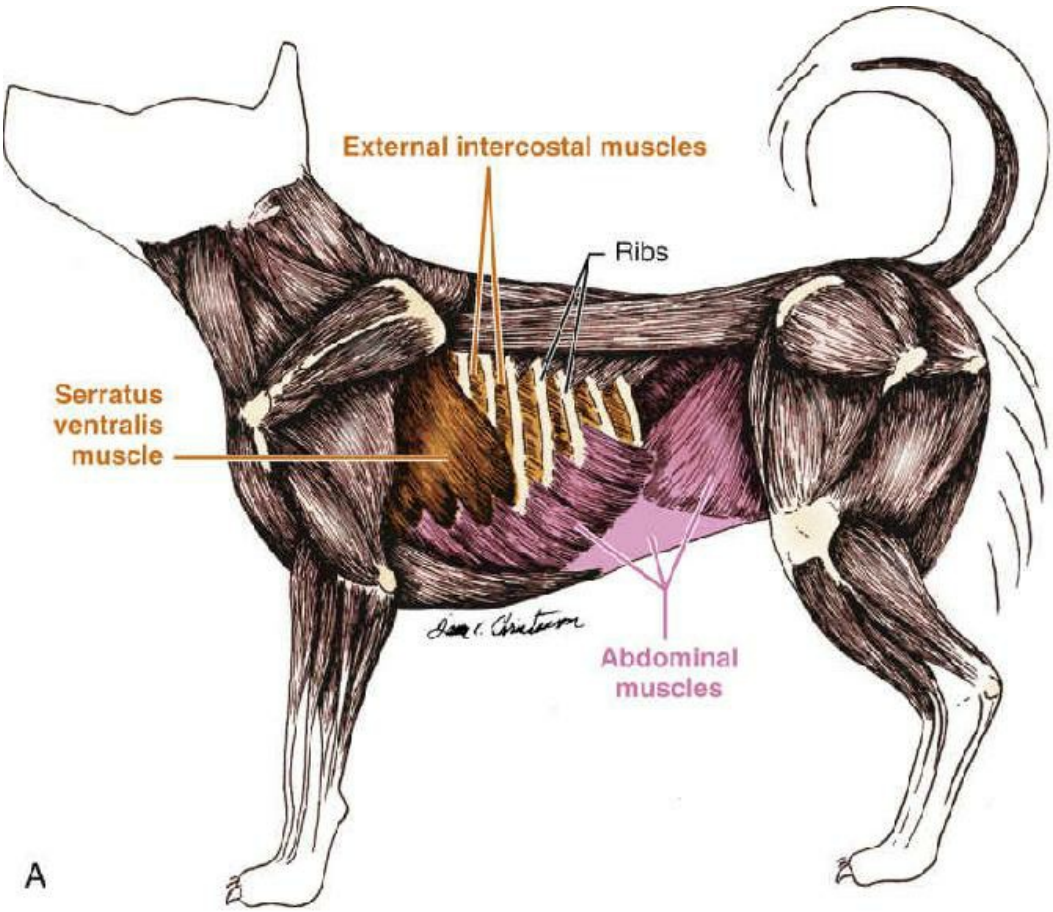
External intercostal muscles
Ribs
Serratus ventralis muscle
Abdominal muscles
A

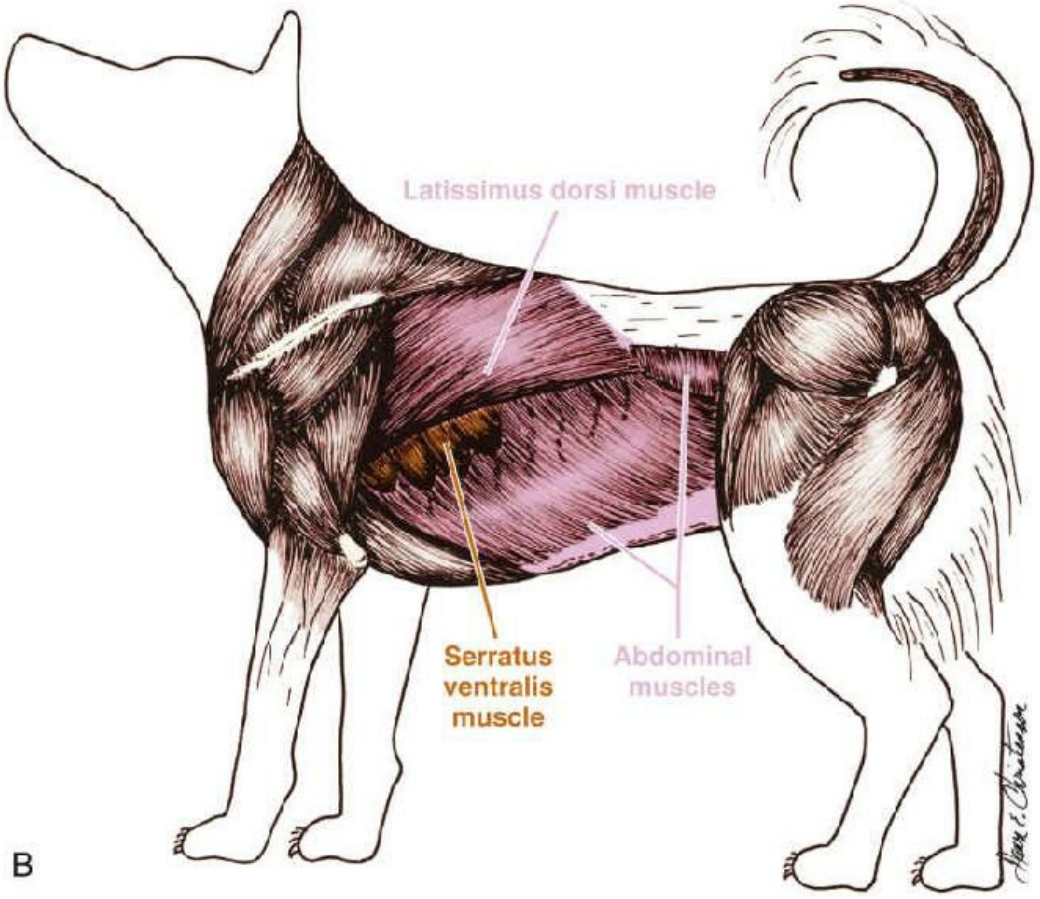
Latissimus dorsi muscle
Serratus ventralis muscle
Abdominal muscles
B

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FIG. 4.11 (A) Muscles of respiration. (B) Superficial muscles of respiration.

External intercostal [*inter-* between + *cost(o)-* ribs + *-al* pertaining to] ***muscles*** (Fig. 4.11A) also help expand the thoracic cavity for breathing in, under normal circumstances. During times of extreme need (exertion or disease), the ***serratus*** [sĕ-ra'tus L. "serrated"] ***ventralis*** [*ventral* + *-is* the] ***muscles*** help expand the rib cage. If you look at the *Serratus*, in Fig. 4.11A, you can see how the muscle got its name. It looks kind of like a serrated knife, doesn't it? But why have *ventralis* in its name? This muscle isn't anywhere near the belly. True. But whoever named it must have thought of the ventral aspect as being the "underside" and the *Serratus ventralis* is "under" some other superficial muscles.

So, what muscles do we use when we breathe out? Actually, none. Breathing out, under normal circumstances, is for the most part a passive movement. It's kind of like a rubber band springing back to its original position. The diaphragm relaxes, and all of the guts squished in the abdomen bounce back to help push it back into the caudal thoracic cavity. Of course, when we are trying to run away from an angry bull or dog, passive movements for breathing out are insufficient. We need to empty our lungs as quickly as possible so that we can take in that next full breath. To assist the ***expiratory*** [*ex-* out + *spir(o)-* breathing + *-tory* pertaining to] movement, we can use many muscles. First, we have ***internal intercostal muscles***. Yes, they are between the ribs, just like the external intercostals. But they lie "under" the *external intercostal muscles*, nearer the interior of the thoracic cavity. Plus, their fibers are aligned in a different direction so that when they contract they draw the rib cage down. Notice all of the abdominal muscles in Figs. 4.11A and B. If the diaphragm is largely responsible for *inspiration*, then it only makes sense to push it back hard and fast, for forced *expiration*. That requires ***abdominal muscles***. We won't list each individual muscle here. Just recognize that there are layers of them that draw down on the caudoventral rib cage and squeeze the abdominal contents dorsal and cranial. In Fig. 4.11B is a large superficial muscle, the ***Latissimus*** (lă-tis'ĭ-mus) ***dorsi*** (dor'si). It covers the *Serratus* and a large part of the rib cage. When it contracts, it rapidly compresses the rib cage for forced expiration.

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We use all of these accessory muscles during exertion, as well as in disease (like when coughing or in cats with asthma). We'll talk about a number of *cardiopulmonary* [*cardi(o)-* heart + *pulmon(o)-* lungs + *-ary* pertaining to] diseases that require use of accessory respiratory muscles in the Chapter 5.

Intramuscular Injection Sites

We have many options for delivery of medications. There are many things to be taken into account when a veterinarian selects a particular route of delivery, including but not limited to, the medication itself, the volume of medication required, the rate of absorption needed, and the animal species. We'll discuss routes of medication administration in Chapter 12. The value of ***intramuscular*** [*intra-* within + *muscul(o)-* muscle + *-ar* pertaining to] medication delivery is the blood supply to the muscles. This provides a reasonably fast rate of absorption, second only to ***intravenous*** [*intra-* within + *ven(o)-* vein + *-ous* pertaining to] injections. If a medication is approved by the manufacturer and the FDA (Federal Drug Administration) for *intramuscular* (IM) administration, then we need to choose an appropriate IM site. Please note that there are species variations for IM site selection. For example, in well-muscled dogs and in horses, the *gluteal muscles* may be used. In "food animals" (e.g., beef cattle or pigs), the gluteal muscles and many of the other IM injection sites would be inappropriate because IM injections may damage the muscle tissue (i.e., beef and pork). Also, some IM injection sites are more painful than others or have risks associated with them. So while I will be explaining and showing various IM sites, please understand that some of these will be left to "last resort" use, after having used up other principal sites. I will focus our attention by species, in order to be clear regarding appropriate use. In all of the figures for IM sites (Figs. 4.12-4.14A & 4.14B), primary injection sites are highlighted in blue. Secondary sites are highlighted in pink.

Canine and Feline Intramuscular Injection Sites

We have a plethora of IM injection sites in dogs and cats. In most dogs and cats we are very limited in volumes of medication that

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can be delivered IM. The smaller the dog or cat, the more limited we become regarding actual sites and volumes. Much of the time, we tend to use the IM route for sedation of patients to help the animal relax for minor procedures like radiographs (x-rays), abdominal ultrasound, or examination of minor injuries. If we need to give repeated doses of an injectable medication for treatment, and if it is approved for other routes of delivery, we will generally choose another route. It makes no sense to make multiple muscles really sore.

For small medication volumes, the ***epaxial*** [*ep-, epi-* on, upon + *axi(o)-* axis, axle + *-al* pertaining to] muscles are an excellent choice (Fig. 4.12). In my experience, they tend to hurt the least in most dogs and cats. We simply need to be sure that we have adequate restraint and isolate the muscle mass correctly. After all, we are extremely close to the spine. The *epaxials* are found along the entire *thoracic* and ***lumbar*** [*lumb(o)-* loin + *-ar* pertaining to] spine. But we don't use their entire length. We only use the ***lumbar epaxial muscles***. To isolate the muscle group, we stay caudal to the last rib, lateral (about 1 to 2 finger widths) to the dorsal spinous processes of the lumbar vertebrae and cranial to the ***iliac*** [*ili(o)-* ilium + *-ac* pertaining to] ***crest***.

A second favorite IM site is the ***gluteal muscle group***. It is useful even in smaller dogs and cats. To isolate this muscle group, we need to find a number of boney protuberances (bumps). So we locate the muscle caudal to the *iliac crest*, dorsomedial to the *greater trochanter* (tro-kan'ter [L. "bump"]) of the femur (thigh bone), dorsocranial to the ***ischiatic*** [*ischi(o)-* ischium, "hip" + *-tic* pertaining to] ***tuberosity*** [*tuber(o)-* bump, bumpy + *os(o)-* bone + *-ity* state of], and lateral to the sacrum. I always locate all four of these ***osseous*** [*osse(o)-* bone + *-ous* pertaining to] points. The ischiatic tuberosity is often easier to find than the greater trochanter of the femur, especially in chubby animals. By rolling my thumb cranial off the ischiatic tuberosity, I feel a little dip and then bump up against the greater trochanter. I always maintain triangulation of the iliac crest, sacrum, and greater trochanter while injecting into the middle of muscle mass.

If a dog is large enough, we can use the ***semimembranosus-semitendinosus*** (i.e., caudal thigh) ***muscles***. In medium to small

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dogs, and certainly most cats, this is a risky muscle mass to use for IM injections. You see, a major nerve is found in this muscle mass, just caudal to the femur. That nerve is the ***sciatic*** (si-at'ik) ***nerve***. If we hit that nerve with our needle, or the medication infused around it irritates the nerve, we can create temporary or permanent paralysis in that leg. The sciatic nerve and its branches supply the semis and all muscles distal to the stifle. Damage to this nerve will result in profound disability. So if this IM site is used, it must be done so with extreme care and caution. The needle should never be directed cranially toward the femur. Plus, we should avoid proximal and distal portions because they tend to be more painful and the nerve is more superficial. To isolate this IM site, we stay caudal to the femur, distal to the ischiatic tuberosity, and proximal to the stifle.

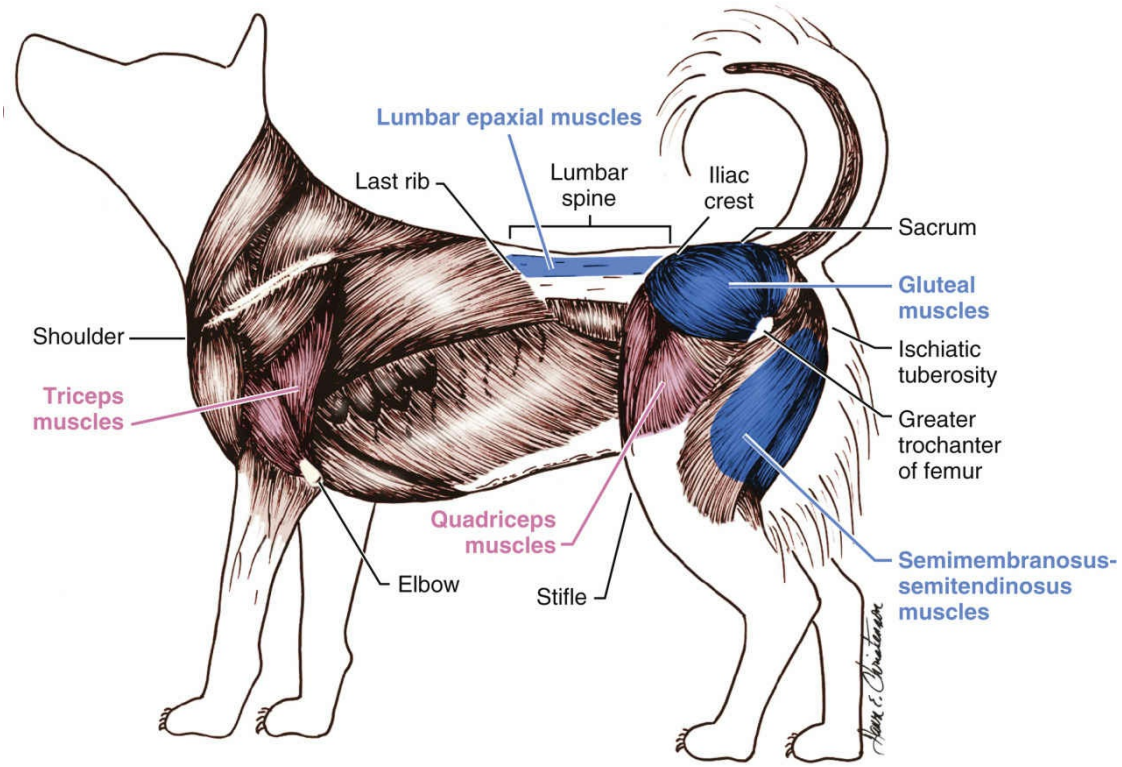

FIG. 4.12 Common canine and feline intramuscular injection sites.

The last two IM injection sites I mention only in case the rest are unavailable. The ***quadriceps muscles*** are often well developed. However, because of the tension that they are typically under, they tend to be painful for IM injections. They lie cranial to the femur,

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proximal to the stifle, and distal to the greater trochanter of the femur and hip. On the rare occasion that I use the quadriceps, I want the animal lying in lateral recumbency. This is the best way to ensure relaxation of the muscle, reducing discomfort during the injection. I most certainly don't want the animal standing for a quadriceps injection! The final site that is rarely used is the ***triceps muscle group***. You already know that it is found in the *caudal brachium*. In my long career, I recall using this muscle group twice. (That's pretty rare use because I've been at this a VERY long time!) We need to be careful to stay in the middle and caudal portion of the triceps muscle. Too close to the humerus and we could risk injury to the radial nerve. Like its "cousin" the sciatic nerve, the radial nerve provides motor nerve fibers to the triceps and many of the muscles distal to the elbow (especially the digital and carpal extensors). So if you do use it, stay in the mid-caudal muscle—distal to the shoulder, cranial to the elbow, and caudal to the humerus.

Equine Intramuscular Injection Sites

Horses have a few large muscle masses that are frequently used for IM injections. For small to moderate volumes of medication, the ***lateral neck muscles*** (Fig. 4.13) provide an excellent and well-tolerated IM site. We typically inject in the middle of a triangle in the lateral neck. That triangle lies dorsal to the *cervical* vertebrae (they lie along the ventral neck), cranial to the scapula, and ventral to the crest of the neck formed by the ***ligamentum*** [L. *the ligament*] ***nuchae*** (noo'ka [L. "the nape"]). Please remember that this IM site should ***not*** be used in nursing foals. If we make those muscles sore, the foal may have difficulty nursing. I'm sure that I don't need to explain why a non-nursing foal is a bad thing.

For larger volumes of medication, the *gluteal* and *semimembranosus-semitendinosus muscles* are often used. Some folks prefer to avoid the *gluteals* in horses. Their rationale for avoidance revolves around infection. You see, if an infection were to develop in a gluteal IM injection site, it would not be able to drain easily. Others contend that provided the injection is performed in an ***aseptic*** [*a-* without + *sept(o)-* bacteria + *-ic* pertaining to; i.e., clean, free of bacteria] manner, the gluteal muscles are okay to use. If you

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do use this IM site, stay caudal to the ***tuber*** [L. bump] ***coxae*** (kok'sa [L. hip]), dorsomedial to the greater trochanter of the femur, dorsocranial to the ischiatic tuberosity, and lateral to the sacrum. And of course, clean the site well first. You could avoid the gluteals, as some folks do, and use the *semis* instead. There are a couple of advantages to using the semis. First, if an infection were to develop, it will drain easily. For me, the advantage of this IM site is that I am less likely to be kicked. I always stand leaning my body snug to the lateral aspect of the rear leg. When the needle is inserted into the caudal thigh, horses tend to kick in the direction of the pain (straight back and I'm not there). Since this is such a large muscle mass, our 1-inch or even 1.5-inch needle won't venture anywhere near the sciatic nerve. So we don't need to worry about creating paralysis. Just stay well distal to the ischiatic tuberosity and well proximal to the stifle. It hurts quite a bit near those borders. If you hurt the horse, he or she will likely hurt you. Just sayin'.

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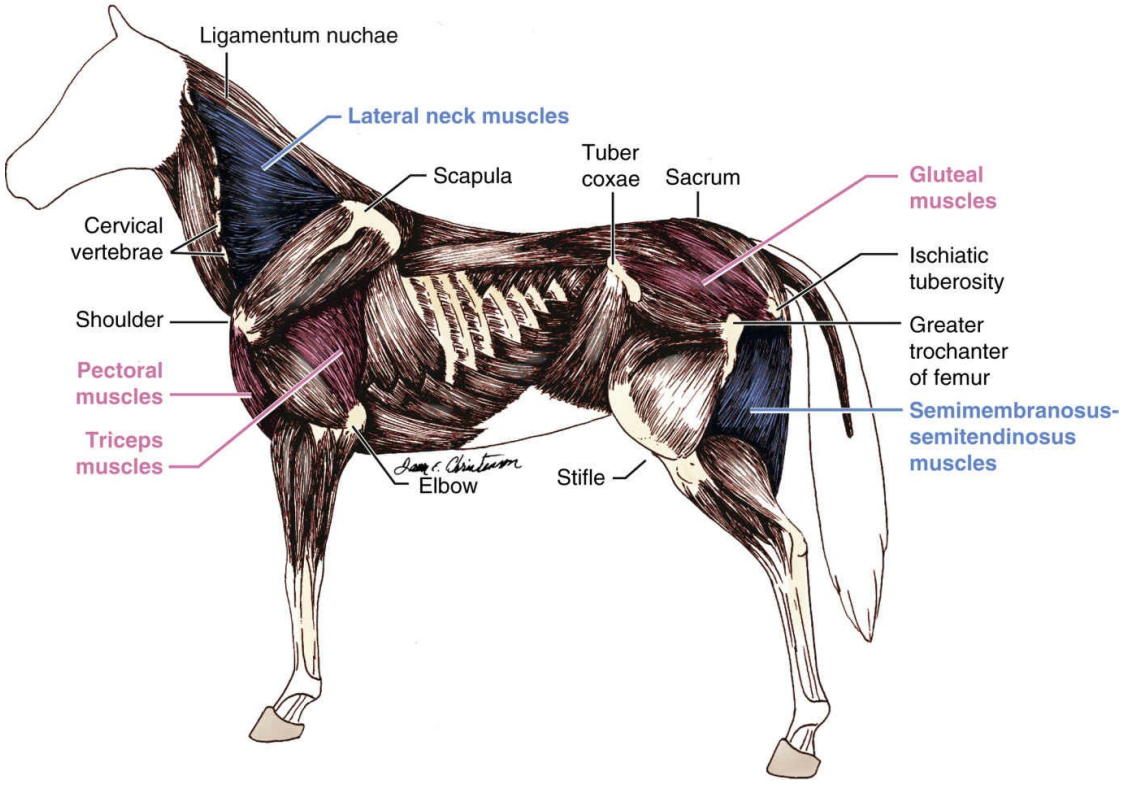

FIG. 4.13 Common equine intramuscular injection sites.

Now, there are times, especially with sick foals, that we may need to rotate multiple IM injection sites to minimize trauma and soreness. (Remember, we do ***not*** use the lateral neck muscles in nursing foals.) So, to give each muscle group more of a break in between injections, we may also use the *triceps muscles* and the *pectoral muscles*. Landmarks for the triceps are as we described them in the dog. If the pectoral muscles are used, we typically only use the cranial-most portion of them, where they are well exposed on the cranial chest. They lie just lateral to the sternum.

Bovine and Caprine Intramuscular Injection Sites

Most of the IM sites that I discuss here are fair game for goats (Fig. 4.14A), provided those goats are not being used for meat. By the same token, there are differences in use between dairy and beef cattle. In beef cattle, IM injections are simply avoided. If we need to give medications IM to beef cattle, we will limit those injections to the *lateral neck*.

For the most part, in goats and in dairy cattle (see Fig. 4.14B), the semis are a preferred IM injection site. Gluteals may also be used. However, as with the horse, some folks prefer not to use *gluteals* in

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dairy cows. For nursing young, we should avoid the lateral neck so that we don't interfere with nursing. As with foals, if we need to rotate IM sites in a dairy calf or a goat kid, we may need to use *triceps* or *pectorals*. This tends to be quite rare.

Notice that I did not include sheep here. We don't tend to give IM injections to sheep because there is simply too much wool in the way. Even if the sheep are used primarily for wool, we should not give IM injections to lambs. Most lambs will be slaughtered for meat. I know this sounds gruesome, but this is the reality of "food animal" medicine.

Porcine Intramuscular Injection Sites

Please note that pigs are only used for meat. Pork producers cannot afford to have any of the meat, especially prime-cuts, damaged from IM injections. So, suffice it to say that we avoid IM injections in pigs. If we must administer medications IM, we will probably only use the *lateral neck muscles*. Landmarks for the lateral neck are the same as for the horse. The difference here is that most pigs have necks built like linebackers. We cannot see or palpate the landmarks as easily as we can in other large animals. It almost becomes an "intra-neck" injection, eyeballing where that triangle for the lateral neck muscles is located. By the way, good luck with that. Let's just say that pigs do not cooperate well and need much restraint.

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Applied Bone Terminology

Archimedes once said, *"Give me a lever long enough and a fulcrum on which to place it, and I shall move the world."* Of course, he was speaking from his perspective as a physicist and engineer. Yet this statement is very applicable to skeletal anatomy and ***biomechanics*** [*bi(o)-* life + *mechanics*; i.e., machine/mechanical principles applied to movements of living things]. So, in spite of the fact that Archimedes lived in antiquity, some of his assertions as a Greek scientist are actually applicable today. You see, without our bones and joints, we and the animals we serve in veterinary medicine would be unable to stand and move the way we do. All of the muscles that we've spent so much time discussing in the previous sections rely on the bones and joints for leverage. That is how *biomechanical* movement is created. Sure, an octopus or a jelly fish moves without a skeleton. But they are not terrestrial creatures, having to stand against gravity and walk across the face of the earth. No, without our skeletal anatomy, we would be limp lumps of tissue. Our internal organs (brain, heart, lungs, etc.) would lack protection. Bones are a very necessary structural part of mammals (reptiles, amphibians, fish, and birds, too!).

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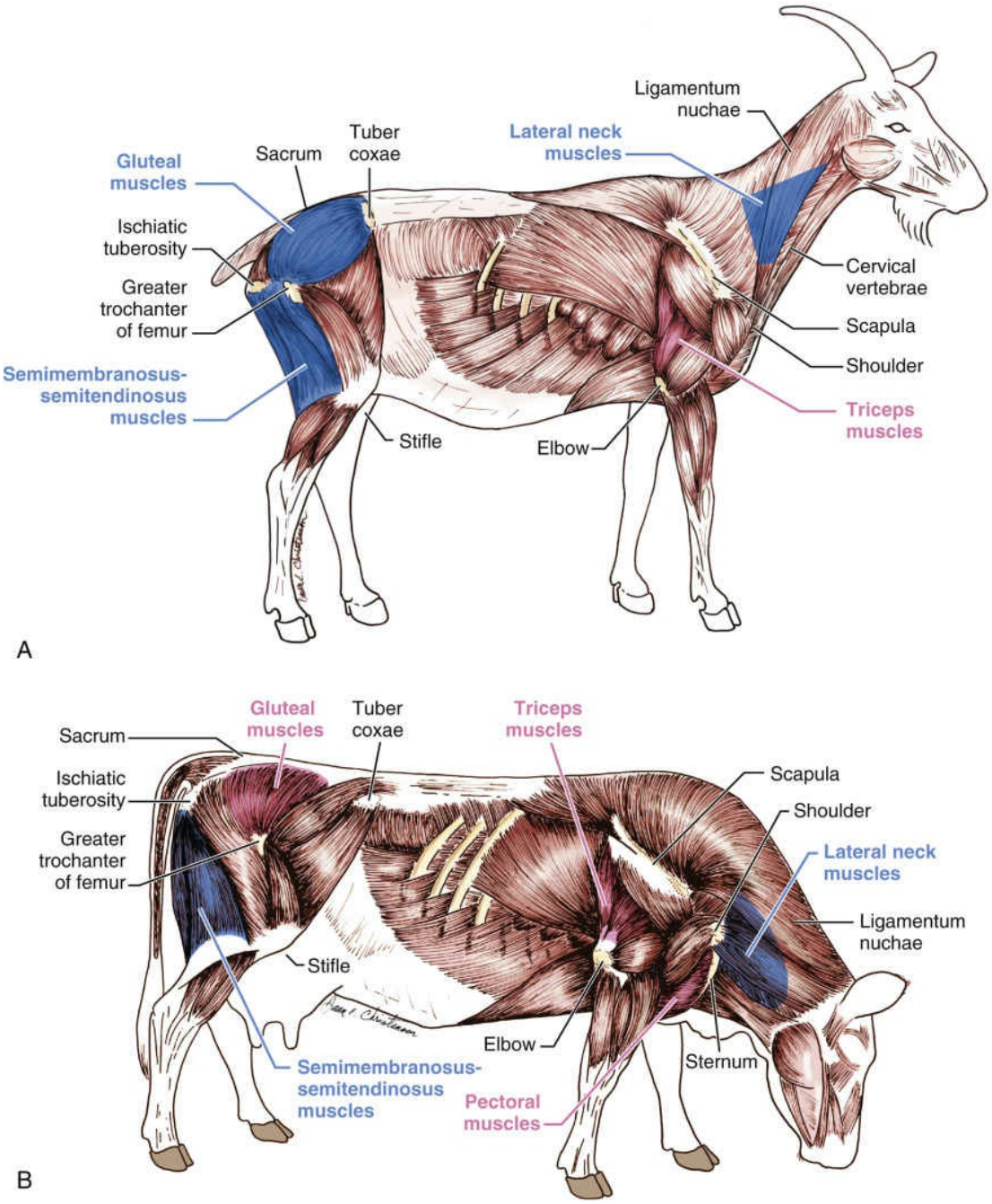

FIG. 4.14 (A) Common caprine intramuscular injection sites. (B) Common bovine intramuscular injection sites.

So, in this portion of the chapter, we will be lifting up magnificent bones and their ***articulations*** [*articul(o)-* joint + *-tion* state of]. We will begin with the basic anatomy of long bones.

Basic Bone Anatomy

Bones come in many shapes and sizes. They all have growth plates,

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different types of boney composition, and places for *articulation* with other bones. But some of these features are difficult to see on short, flat, and irregular bones. So we will use a long bone, like the femur (thigh bone) for this discussion (Fig. 4.15).

As you can see, the shaft, or ***diaphysis*** [*dia-* between, through + *physis* growth, growing], of the bone makes up most of its length. It is made very strong by thick, dense, compact ***cortical*** [*cortic(o)-* cortex + *-al* pertaining to] ***bone***. At the very center of the *diaphysis* we find a hollow, tube-like canal called the ***medullary*** [*medull(o)-* marrow, medulla + *-ary* pertaining to] ***cavity***. Of course, it's not truly just a hollow tube. It contains bone marrow. The cavity has an ***endosteal*** [*endo-* within + *oste(o)-* bone + *-al* pertaining to] connective tissue lining that surrounds the actual bone marrow within. As you may recall fromChapter 3, the bone marrow is the principal site of production of blood cells. The lion's share of that production takes place in long bones, like the femur we are currently looking at. As you may suspect, the medullary cavity has an excellent blood supply. In fact, the blood supply is so good that we can administer ***intraosseous*** [*intra-* within + *osse(o)-* bone + *-ous* pertaining to] fluid therapy. Can you believe that! No, it's not our first choice for fluid therapy. But when we run out of other options, *intraosseous* fluid administration provides a nice backup plan.

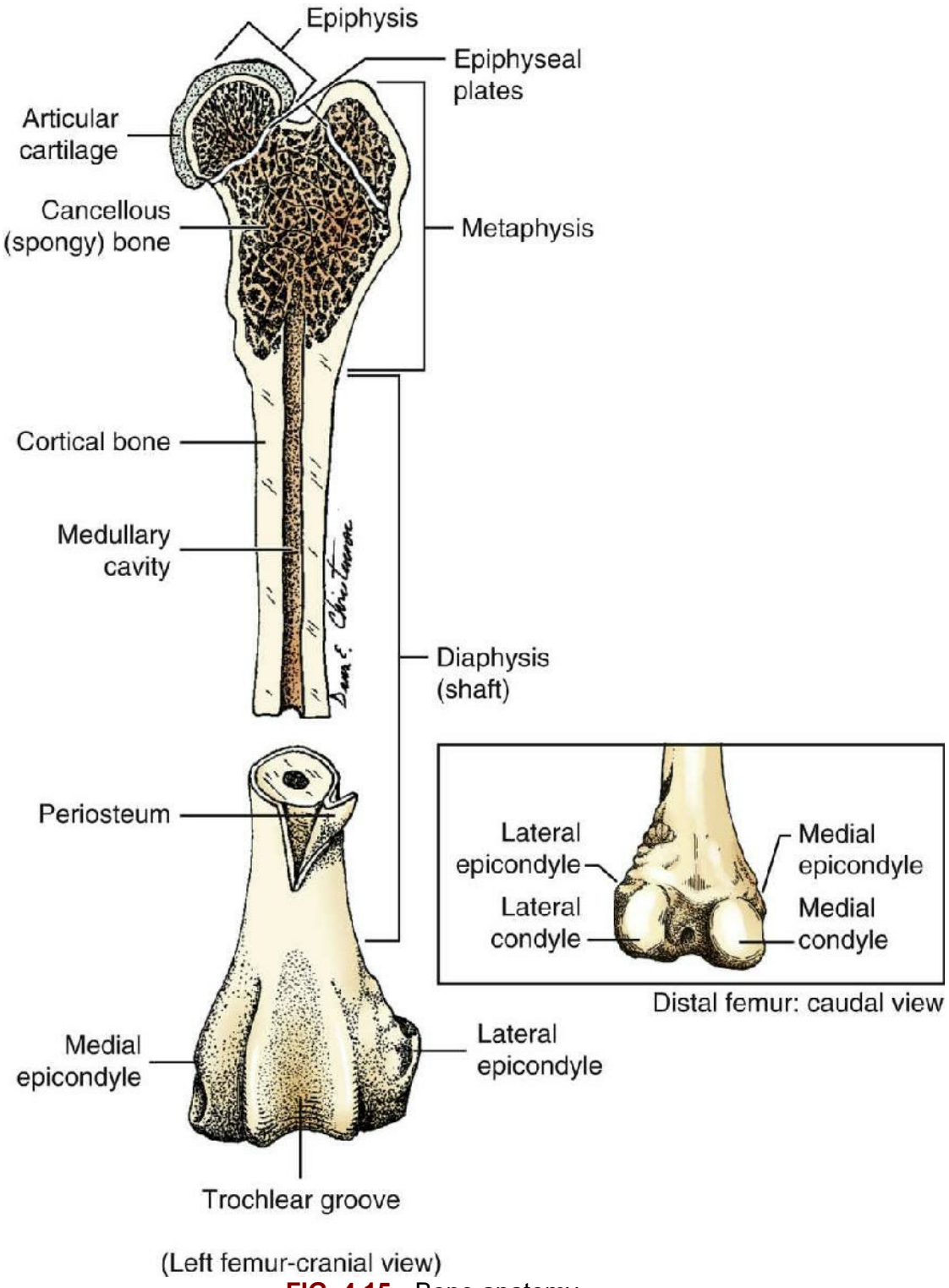

FIG. 4.15 Bone anatomy.

Vessels and nerves pass through the cortical bone of the diaphysis into the medullary cavity via small holes called ***foramina*** [fo-ra′min-uh; L. "hole", passage; singular = ***foramen***]. This blood supply not only provides nutrition for the bone itself, but also provides a means for blood cells to exit the marrow and enter

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circulation. We said a moment ago that endosteum lines the interior of the medullary cavity. Is there also connective tissue on the outside of the bone? Yes. ***Periosteum*** [*peri-* around, near + *oste(o)-* bone + *-um* the] is the connective tissue *around* (as the prefix implies) the bone. It covers all but the ***articular*** [*articul(o)-* joint + *-ar* pertaining to] surfaces. ***Periosteal*** [*peri-* around + *oste(o)-* bone + *-al* pertaining to] tissue is a connective tissue with numerous sensory nerve endings. If you're an athlete who's experienced "shin splints," you know about those sensory nerve endings. They "screamed" at you when the *periosteum* separated from the cortical bone and became inflamed. Ouch!

I said a moment ago that periosteum covered all but the *articular* surfaces of the bone. What covers articular surfaces? Cartilage. Cartilage is a very different type of connective tissue. It comes in a number of forms, including *fibrous* and ***hyaline*** [*hyal(o)-* glass + *-ine* pertaining to] cartilage. Articular surfaces have a thick layer of hyaline cartilage covering the bone. Like a thick layer of Teflon, the hyaline cartilage provides a smooth surface between the bones forming the joint, reducing friction in the joint. Hyaline cartilage is only slightly elastic. Like glass, it can break when subjected to extreme forces. If the cartilage is significantly damaged from an ***acute*** (sudden) injury or ***chronic*** [*chron(o)-* time + *-ic* pertaining to; i.e., long-term] wear and tear, it will not be replaced with a fresh layer. There are simply insufficient ***chondrocytes*** [*chondr(o)-* cartilage + *cyt(o)-* cell] to repair major damage. We'll talk about this later when we talk about ***hip dysplasia*** [*dys-* poor, bad + *plas(o)-* formation + *-ia* a condition of].

By the way, near the end of the long bone's articular surface is the ***epiphysis*** [*epi-* on, upon + *physis* growth, growing]. In young animals, ***epiphyseal*** [*epi-* on, upon *phys(o)-* growth + *-al* pertaining to] ***plates*** (Fig. 4.16) are ***chondral*** [*chondr(o)-* cartilage + *-al* pertaining to] regions containing many *chondrocytes*. This is where bone growth in young animals takes place. We'll talk about bone growth in the next section. Because cartilage is not dense mineralized tissue like cortical bone, we can see the *epiphyseal plates* easily on radiographs. They appear as dark areas, in contrast to the bright cortical bone.

Alright, so we have the main shaft of the long bone, the *diaphysis*,

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and the very end of the bone, the *epiphysis*. We need a region providing a gradual, broadening transition connecting the two. This is where the ***metaphysis*** [*meta-* after, beyond + *physis* growth, growing] comes in. As you can see, it's not solid cortical bone. That would make bones way too heavy. So, to lighten the load, under the outer cover of cortical bone we find a network of ***cancellous*** [*cancell(o)-* lattice + *-ous* pertaining to] or ***"spongy" bone***. The lattice-like network of bone is still quite durable. As a bonus, all of the little gaps between the lattice work provides more space for marrow.

We have all sorts of lumps, bumps, and depressions on bones. Naturally, early anatomists couldn't just let them go unnamed or give them seemingly simple names. At least, in our modern-day terms, they don't seem simple. But if we look at bones from the context of antiquity, many of the names given actually do make sense. For instance, looking at the femur in Fig. 4.15, we find a ***trochlear*** (tro'kle-ar [L. *trochlearis* "pulley"]) ***groove***. The patella or "knee cap" slides along in this groove, connected to the distal femur and proximal tibia by rope-like bands of tendons and ligaments, respectively. Well, if you've ever looked at how a pulley works, with the rope gliding over the groove of the pulley wheel, there is a striking resemblance between that and our *trochlear groove*. So the name really does make some sense. Then, as we think about lumps and bumps, they are often given names based on size. A *tuberosity* is usually a fairly large bump on a bone. Smaller bumps are given the name ***tubercle*** [*tuber(o)-* bump + *-cle* a small]. The suffix here makes all the difference in the size of the bump named. If you remember nothing else, remember that any term with the root word *tuber(o)-* in it is probably some sort of bump on a bone.

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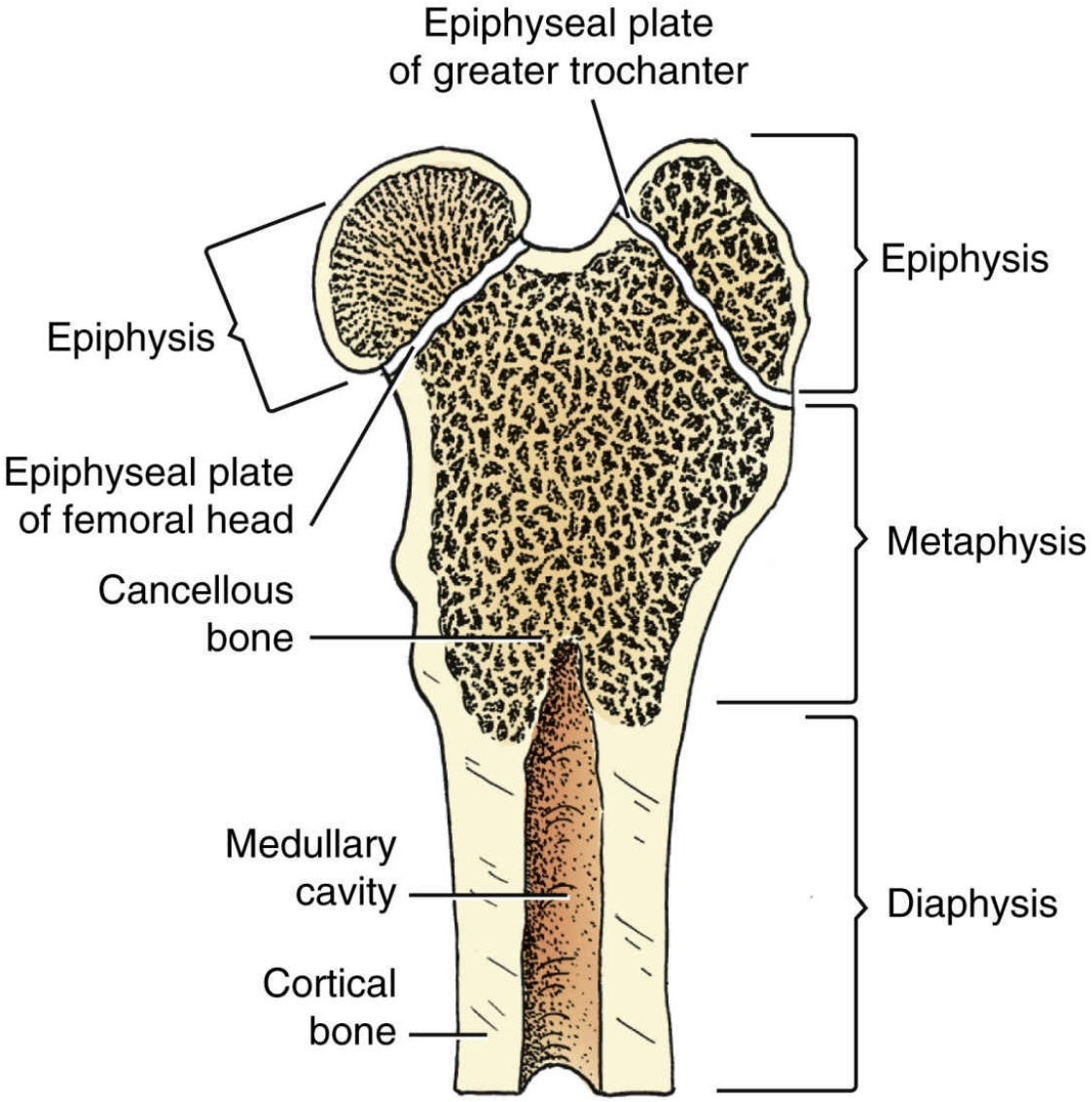

FIG. 4.16 Epiphyseal plates.

Why do we have so many and such prominent *osseous* bumps? Well, for one thing, we talked about leverage earlier. Most bumps serve as strong connection points for ***tendinous*** [*tendin(o)-* tendon + *-ous* pertaining to] and ***ligamentous*** [*ligament(o)-* ligament + *-ous* pertaining to] attachments. For leverage points, we connect tendons to key *tubercles*, *tuberosities*, and ***epicondyles*** [*epi-* upon + *condyl(o)-* knuckle; Fig. 4.15]. When the muscles connected to those tendons contract, leverage force is applied and moves the bones of the associated joint. For stabilization of joints, we use other *tubercles, tuberosities,* and *epicondyles* for ***ligamental*** [*ligament(o)-* ligament + *-al* pertaining to] attachment points. If you've ever torn your anterior cruciate ligament (ACL), you know how unstable your knee became without that important ligament. The cortical bone at all of

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these protuberances is a bit thicker than the rest, and its surface is very rough to provide for a strong attachment of the fibrous connective tissue. Compare the roughness of the *epicondyles* of the femur to the smooth, articular surface of the ***condyles*** [*condyl(o)-* knuckle] shown in the inset for Fig. 4.15. We need rough spots to securely anchor our ligaments and tendons.

Finally, before we move on to talk about bone growth, I should mention the various shapes of bones found in the body. We've already talked about ***long bones***, as the focus of our basic bone anatomy discussion. Most of these are found in the limbs; go figure. But long bones don't fit or suit all of the structural needs of the body. So in places like the carpus and tarsus, we find numerous ***short bones***. In Fig. 4.17A, you can see some of the short bones of the left carpus. Vertebrae, like those shown in Fig. 4.17B, that make up the spinal column have some pretty weird shapes, as you can see. Hence we refer to those as ***irregular bones***. Then there are ***flat bones***. You might think of the skull as a single bone, but it's really a bunch of flat bones connected at ***suture-lines*** [L. *sutura* "seam"] to make the unique cranial "container" for the brain (see Fig. 4.17C). Flat bones also make up the ribs, the pelvis, and the scapula. If you look at the scapula, in Fig. 4.17D, you'll see two very large depressions on either side of its "spine." Any depressed area like this is referred to as a ***fossa*** (fos'ah [L. "trench"]). Here again, anatomists of antiquity named these depressed areas logically, because they look like trenches. As you can see on the scapula, we have named each fossa in relation to the ***scapular*** [*scapul(o)-* scapula + *-ar* pertaining to] spine—the ***supraspinous*** [*supra-* above + *spin(o)-* spine + *-ous* pertaining to] ***fossa*** and ***infraspinous*** [*infra-* below + *spin(o)-* spine + *-ous* pertaining to] ***fossa***.

Finally, we have ***sesamoid*** [*sesam(o)-* sesame (seed) + *-oid* resembling] ***bones***. *Sesamoid bones* are generally small, rounded bones that are found around joints, or embedded in a tendon or joint capsule. The ***patella*** [L. "a small shallow dish"] is a sesamoid bone. It is a good example of how these bones help provide stability of a joint by helping to direct the movement and tension of the associated tendon. As you already know, the patella glides in the trochlear groove of the femur. This focuses the tracking of the patellar tendon midline over the cranial aspect of the stifle joint,

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helping to maintain the hinge-like action of the joint. Some animals, like some toy poodles with abnormally shallow trochlear grooves, fail to maintain midline tracking of the patella. In these dogs, the patella of one or both stifle joints either ***luxates*** [L. *luxatio* dislocate] or ***subluxates*** [*sub-* under, partial + *luxat(o)-* dislocate; i.e., partial dislocation]. When this happens, the joint becomes very unstable and the rear legs may simply "give way" in midstride.

Bone Growth

Believe it or not, bones do not begin as rock-hard, mineralized structures. Most bone actually begins as a cartilage matrix. This is true for a fetus in utero, for young growing animals at their epiphyseal plates, as well as in the initial stages of fracture repair. Most bone, in its "infancy," is a soft, flexible ***chondral*** [*chondr(o)-* cartilage + *-al* pertaining to] substance.

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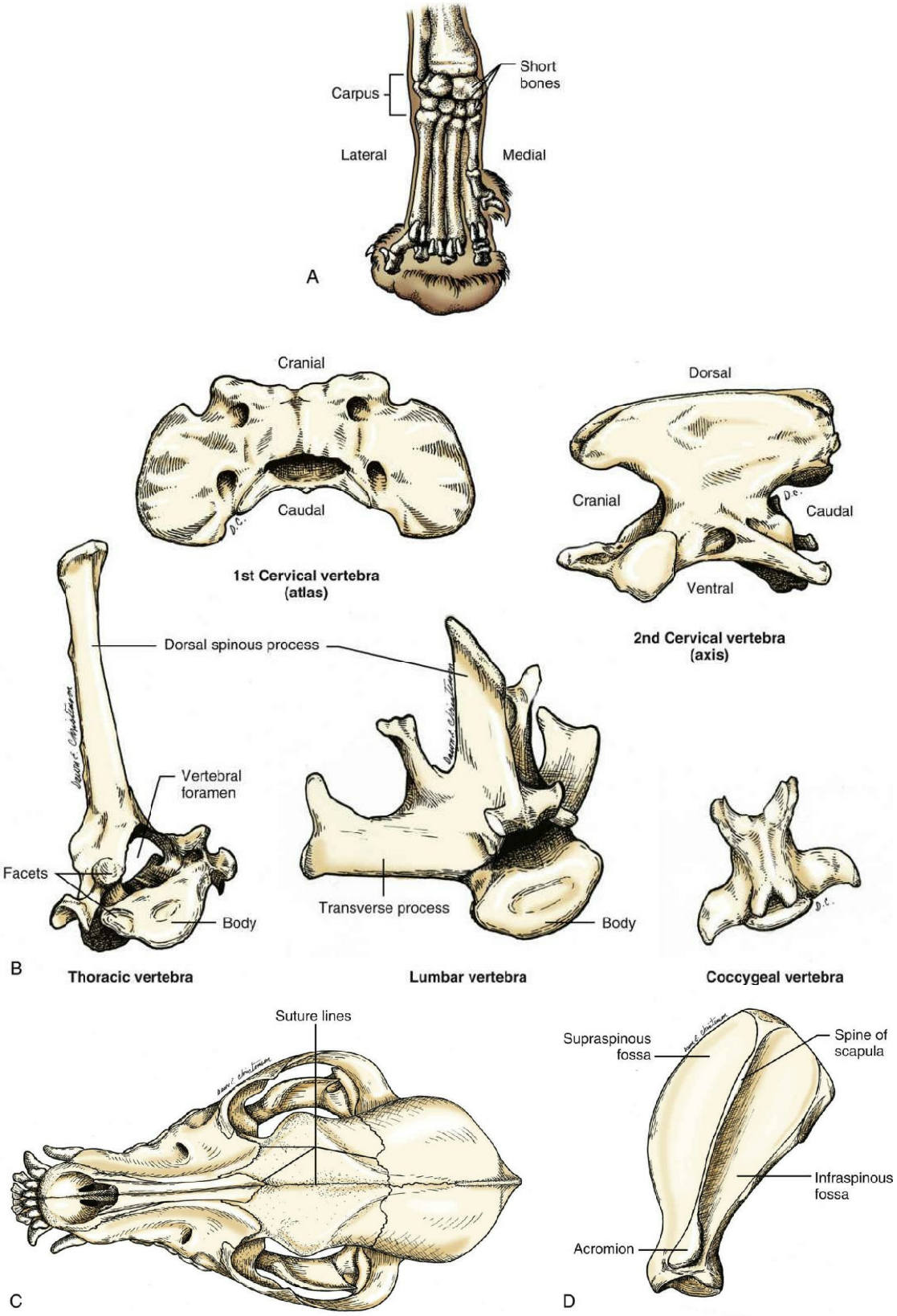

FIG. 4.17 (A) Left carpus, palmar view—short bones. (B) Vertebrae—irregular bones. (C) Skull—flat bones. (D) Scapula—flat bone.

How does this work? Well, in a fetus the basic bone shape is

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created as ***fibrocartilage*** [*fibr(o)-* fiber + *cartilage*]. This provides the "scaffolding" on which to build the bone. ***Osteoblasts*** [*oste(o)-* bone + *blast(o)-* germ, shoot], embedded in the cartilage matrix, begin to replace the cartilage by secreting the mineralized stuff of bone. They work tirelessly, replacing nearly all of the cartilage with mineralized bone. What I find rather humorous is the fact that the *osteoblasts* actually "hem" themselves in, so to speak. They literally encase themselves in the mineralized bone that they are creating. That would be like masons encasing themselves in brick, or like concrete workers encasing themselves in the cement that they're pouring. Crazy, right?! Once an osteoblast is fully encased in mineralized bone, we call it an ***osteocyte*** [*oste(o)-* bone + *cyt(o)-* cell]. Why the name change? Perhaps it's a functional distinction, because *osteoblasts* can engage in mitosis but *osteocytes* cannot (no room). I honestly don't know. It's really the same cell, in the same place, just different décor. Some things we just need to accept and move on, I guess.

In the fetus, it's easy to visualize the *osteoblasts* replacing the fibrocartilage of the diaphysis and metaphysis with mineralized bone. But what about newborns and young growing animals? Well, this is where the epiphyseal plates are so important. The epiphyseal plate is fibrocartilage, separating the epiphysis from the metaphysis (see Fig. 4.16). Like the fibrocartilage in fetal bone, we have osteoblasts in the epiphyseal plates, too. The difference here is the location. Because the epiphyseal plates are found at the proximal and distal ends of a bone, when mineralized bone is deposited the bone grows in length. This is why people often refer to epiphyseal plates as "growth plates." Eventually, when the animal (or person) has reached maturity, the epiphyseal plates themselves "close" and the fibrocartilage is replaced entirely with bone. In mature animals, we maintain a limited supply of osteoblasts in the periosteum and endosteum. These are great locations, especially if we want to strengthen and bulk up something like the diaphysis. They also come in handy in fracture repair.

By the way, the principal mineral of bone is calcium. Not only does bone give structural integrity to the body, it also provides a great storage area for calcium. If push comes to shove and we need to "dip into" that stored calcium, we have ***osteoclasts*** [*oste(o)-* bone

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+ *clast(o)-* break, breaker] to dissolve bone, releasing calcium for other bodily needs. We don't want to steal too much calcium or we lose the structural integrity and strength of the bones. That would leave them at risk for fractures.

Fractures and Fracture Repair

Fractures

Most of the time when we think of fractures (broken bones), we think of accidental trauma, perhaps from falling or being struck by an automobile. What about conditions like ***osteoporosis*** [*oste(o)-* bone + *por(o)-* pore, porous + *-sis* condition of], resulting in ***pathologic*** [*path(o)-* disease + *log(o)-* knowledge + *-ic* pertaining to] fractures? Well, ***osteolytic*** [*oste(o)-* bone + *-lytic* pertaining to destruction, dissolving of] syndromes, like *osteoporosis*, are far more common in people than in animals. In people with osteoporosis, hormonal changes cause *osteoclasts* to "run wild." That said, occasionally animals may develop *pathologic fractures* from generalized *osteolytic* disease. In my experience, this occurs most frequently in pet reptiles, due to poor nutritional intake of calcium and vitamin D. But generally in veterinary medicine, that kind of ***osteoclastic*** [*oste(o)-* bone + *clast(o)-* breaking + *-ic* pertaining to] activity in animals is most often due to another type of disease, like ***osteosarcoma*** [*oste(o)-* bone + *sarc(o)-* flesh + *-oma* a tumor of]. Tumors like that can leave bones very, very brittle, so that even normal activities of walking or running could result in fractures.

Some of the more common traumatic fractures are found in Fig. 4.18. Do such fractures only occur in the long bones shown? Of course not! Absolutely any bone can be broken, given the right set of circumstances and external forces. It's simply easier to show these common types of fractures on larger bones than smaller ones. Please note that these are not the only types of fractures. There are others. But I'll leave the more complicated types, especially those involving joints or epiphyseal plates, to your future study of *orthopedic* surgery and medicine.

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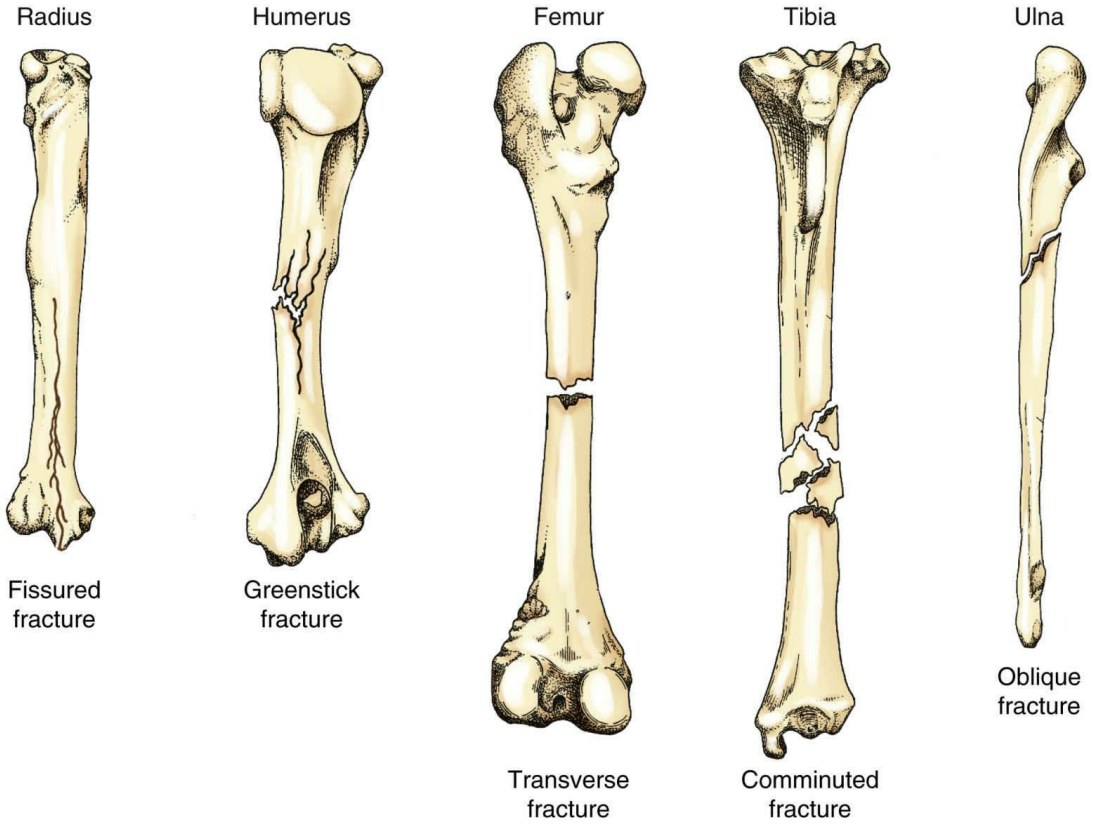

FIG. 4.18 Common traumatic fractures.

Notice the names of the fractures shown. For the most part, each name actually makes sense. For instance, look at the radius with the ***fissured fracture***. What is a fissure? Well, it's something like a small crack or groove. That makes sense for this fracture type, doesn't it? The bone simply appears to be cracked lengthwise. And those lengthwise *fissures* (cracks) may be only on this side of the bone. The other side is often completely intact. Provided there is no underlying ***osteopathy*** [*oste(o)-* bone + *-pathy* a disease of] or joint involvement, *fissured fractures* tend to heal quite well with temporary support and restricted activity.

Now look at the humerus with the ***greenstick fracture***. Again, this name makes perfect sense. Have you ever tried to break a fresh, green twig or tree branch? It's almost impossible to break it completely on the first attempt. The branch almost always remains intact (albeit bent) on one side. That is precisely what happens in a *greenstick fracture*. And like the young plant or tree whose branch we could not break completely, greenstick fractures almost exclusively occur in young animals. The bones of young animals are a bit more flexible than mature animals. Youngsters haven't

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bulked up their rock-hard cortical bone yet.

The fractured femur in Fig. 4.18 has probably the most logical name. If you think of the length of the bone as an axis, then we already know a transverse plane will be perpendicular (90 degree angle) to that axis. So then a ***transverse fracture*** follows suit. It is a break straight across [*trans-*] the axis of the bone. We happen to be showing a mid-shaft transverse fracture here. But it could be almost anywhere along the diaphysis or metaphysis.

The tibia shown has a whole bunch of loose fragments of bone at the fracture site. Okay, the name for this fracture is a bit more complicated. Have you ever heard someone talk about minutia? Minutia is a word meaning small [L. *minutiae* "trifles," Fr. *minutia* smallness]. The name of this fracture incorporates that. This is a ***comminuted*** [*com-* together, with + *minut(o)-* small + *-ed* to be] ***fracture***. Now does the name make sense? It truly means that the fracture has a bunch of small bits of bone sitting together at the fracture site. This kind of shattering usually involves extreme force to cause the fracture.

Finally, looking at the ulna in this figure, we see an ***oblique fracture***. As we learned inChapter 1, when talking about directional terminology related to radiographic views, the word *oblique* infers some sort of angle. So this type of fracture is at an angle. It is not parallel to the axis of the bone, and it's not perpendicular to it either. Oblique implies it's at some sort of angle to the axis of the bone. This type of fracture often results in a very sharp point of bone. If it's sharp and unstable, it can slice right through surrounding muscle, vessels, and skin.

Oblique and comminuted fractures, compared to any of the others shown, have the greatest potential to become "open" fractures. In an ***open fracture*** (Fig. 4.19), the sharp point of the broken bone pokes through all of the surrounding soft tissues and skin, opening the fracture site to the outside environment. Ouch! But the seriousness of an open fracture goes beyond the pain involved. Depending on the bone involved and the amount of muscular and vascular damage, we can have significant blood loss from an open fracture. And because it is open to the external (filthy) environment, the fracture site becomes contaminated. This puts the patient at serious risk of infection. But this is no ordinary infection.

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Remember, we've exposed raw bone and marrow. So an open fracture puts the patient at risk of ***osteomyelitis*** [*oste(o)-* bone + *myel(o)-* marrow + *-itis* inflammation of; i.e., inflammation of the bone and marrow caused by infection]. *Osteomyelitis* is an extremely painful and difficult to treat infection of the bone and marrow. This is why most fractures, but especially oblique and comminuted fractures, should be stabilized with some sort of supportive splint, cast, or bandage until they can be surgically repaired. We want to minimize any movement of the fractured ends of the bone to prevent it from becoming an open fracture. Worst case scenarios are hit-by-car patients with open fractures from the get-go. These open fractures are usually horribly contaminated with dirt and road grime, teaming with bacteria from the start. Because these types of contaminated injuries often develop profound infections and resultant *osteomyelitis*, in the best interest of the patient, the affected limb may be amputated. For the lucky ones who keep their legs and recover from osteomyelitis, the ***postoperative*** [*post-* after + *operative* pertaining to surgery] healing process tends to be very long.

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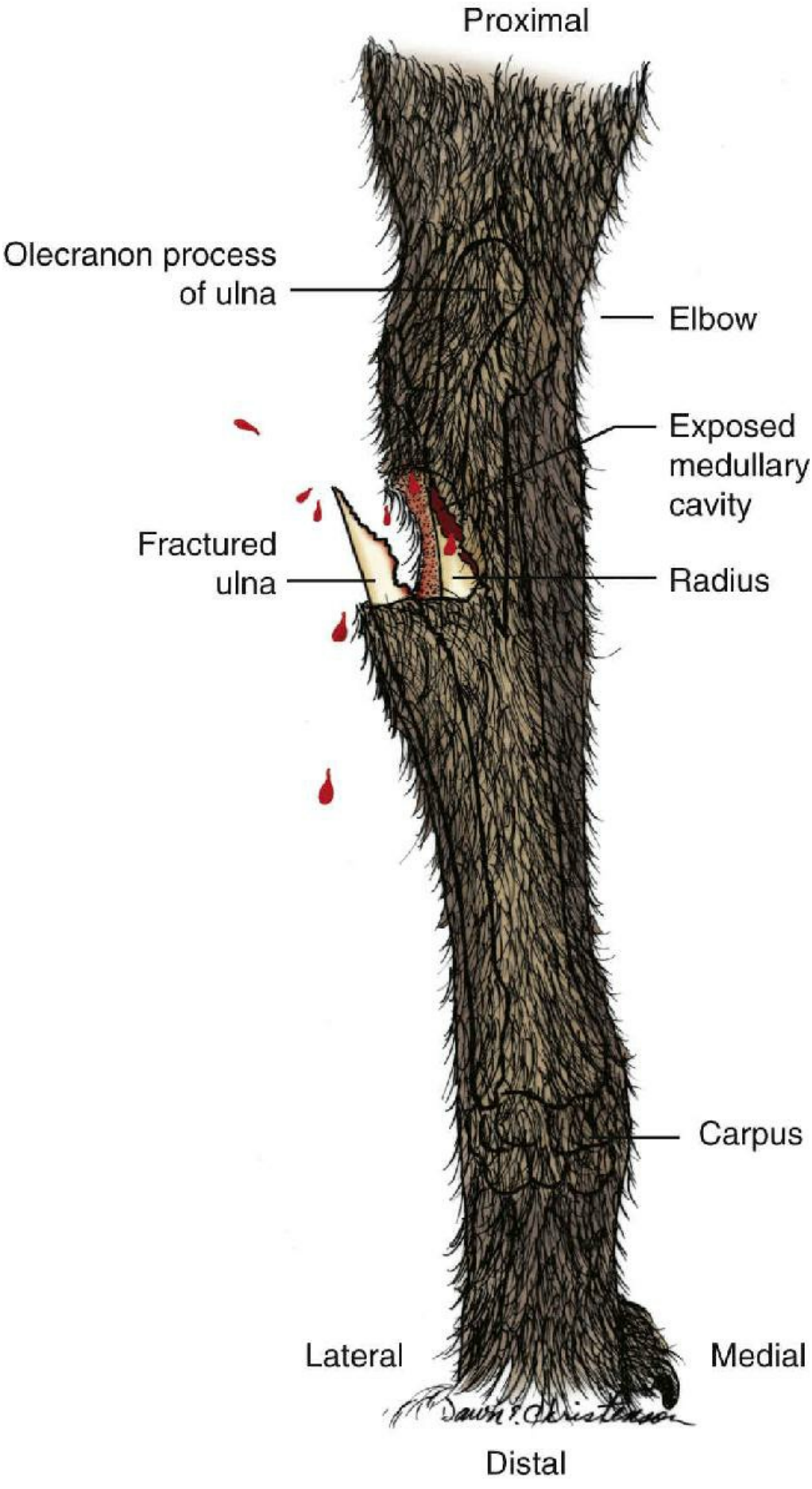
Proximal
Olecranon process of ulna
Elbow
Exposed medullary cavity
Fractured ulna
Radius
Carpus
Lateral
Medial
Distal

FIG. 4.19 Open fracture of a forelimb.

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Fracture Repair

Whatever type of fracture results, from trauma or surgery (yes, we create surgical fractures whenever we perform an ***osteotomy*** [*oste(o)-* bone + *-tomy* to cut (into)]), the site must be adequately aligned and stabilized to optimize repair. Then we simply need tincture of time for healing to take place. The older we are, the longer the process takes.

The repair process (Fig. 4.20) is very much like bone growth. From the initial injury, bleeding occurs. Once the ***hematoma*** [*hemat(o)-* blood + *-oma* swelling, accumulation] clots, the fibrin strands in the clot provide the basic scaffolding for cells infiltrating the area to lay down fibrocartilage. The fibrocartilage provides very weak structural support at the fracture site. Once osteoblasts arrive on the scene (principally from the periosteum and endosteum), they begin to build on and replace the fibrocartilage with mineralized bone. Here again, the cancellous bone deposited early in fracture repair offers weak security at the site. Teamwork between osteoblasts laying down bone and osteoclasts helping them shift and reorganize the new bone results in a bony callus. The callus bridging the broken ends of the bone is stronger than everything leading up to this point. But it is still weaker than the preexisting cortical bone, before the injury. So, we cannot afford for the patient to create too much physical stress on the site or have it subjected to any additional trauma. We need our construction crew to keep going, to strengthen the site. Our team of osteoblasts must work tirelessly, as they did in normal bone growth, boxing themselves in to the bone they deposit. Ultimately, the deposited bone will be filled in and reorganized into strong, compact cortical bone.

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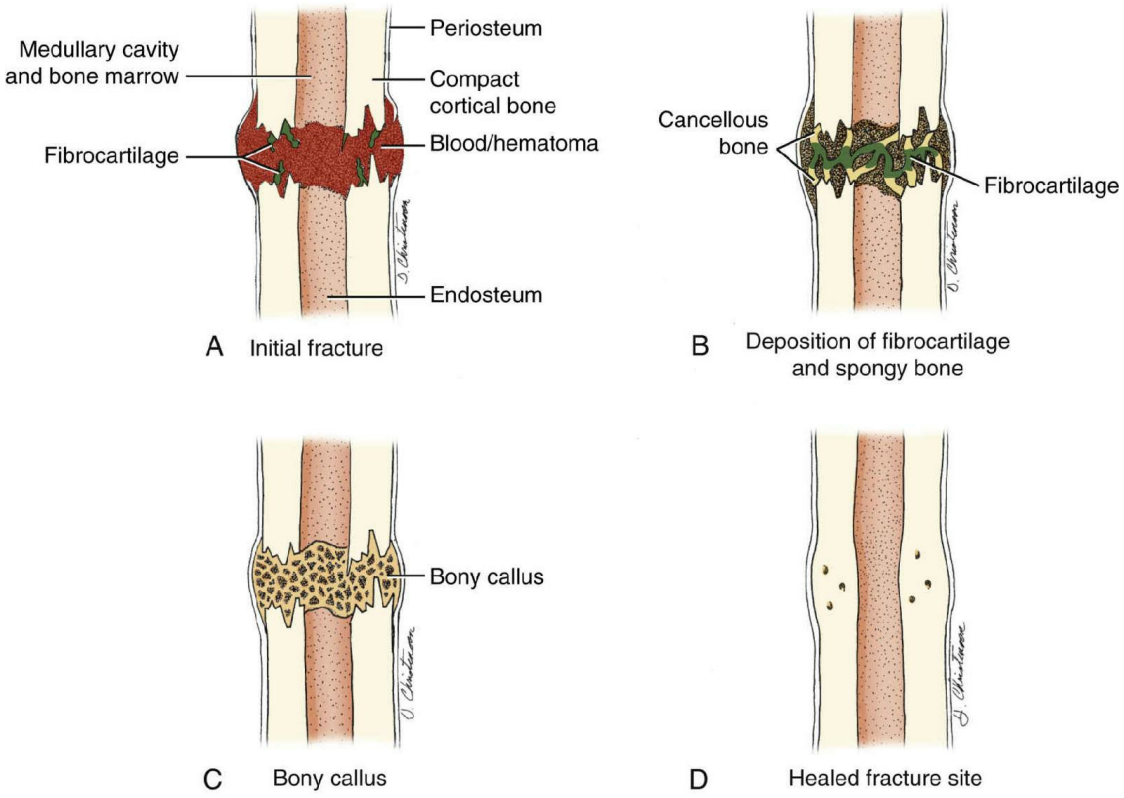

FIG. 4.20 (A to D) Fracture repair.

If the fracture site involves a limb and that limb has been immobilized in some fashion, our connective tissues (ligaments and tendons) of the immobilized joints will have experienced changes, like those discussed in an earlier section. But before we can attempt rehabilitation of the affected limb, we must understand the structure and function of the affected joints.

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Applied Joint Terminology

Joint Types

Joints are intersections, if you will, between bones. The flexibility and mobility of each joint, under normal conditions, depends on its type and structure.

Fibrous Joints

Fibrous joints are relatively immobile. These are joints, like the suture lines of the skull mentioned earlier (see Fig. 4.17C). We find similar joints between the bones of the pelvis, between bones like the radius and ulna or tibia and fibula, and between other limb bones in large animals (e.g., splint bones in the horse). In youngsters, these joints are connected with either fibrous ligamental tissue or perhaps fibrocartilage. Those connected with fibrocartilage (like the skull and pelvis) in young growing animals provide for growth of the bones themselves and a little flexibility. Flexibility? Yes. You see, for a young female guinea pig to give birth for the first time, she needs the *fibrous joints* at the midline of her pelvis (***pelvic symphysis*** [*sym-* together + *physis* growing]) to be able to separate a bit, expanding the size of the birth canal. In an older guinea pig pregnant for the first time, the *pelvic symphysis* may have already fused with bone. And that would probably create great difficulty giving birth, perhaps even requiring a C-section. In maturity, many of our *fibrous joints*, especially those between flat bones, experience ***arthrodesis*** [*arthr(o)-* joint + *-desis* fusion]. When I think of the *fibrous joints* of the skull, I think that's mighty important! I would like a secure container for my brain, thank you very much!

Cartilaginous Joints

Cartilaginous [*cartilagin(o)-* cartilage + *-ous* pertaining to] ***joints*** are those where the bones are separated exclusively by some sort of cartilage. Go figure. Some good examples of *cartilaginous joints* include ***intervertebral*** [*inter-* between + *vertebr(o)-* vertebrae + *-al* pertaining to] joints, separated by cartilaginous *intervertebral discs*

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(Fig. 4.21). Intervertebral discs are one of the more unique features of the body. On cross-section, in Fig. 4.22, you can see that there are two very distinct parts of each disc: the ***anulus*** [L. "ring"] ***fibrosus*** [*fibr(o)-* fiber + *-sus* the] and the ***nucleus pulposus*** [*pulp(o)-* flesh + *-sus* the]. The *anulus fibrosus* is a firm, fibrous outer ring, formed of fibrocartilage and fibrous connective tissue. It is like the dough surrounding the filling of a jelly doughnut. The jelly, then, is the *nucleus pulposus*. No, it's not jelly. It is a gelatinous collagen material. The difference between these collagen fibers, compared to those we discussed with muscles, is that the collagen fibers of the nucleus pulposus have a really, really loose arrangement. Mix that with a whole lot of water and you create a very gooey, gelatinous material. And that is the nucleus pulposus. This structure of the intervertebral discs provides excellent flexibility and shock absorption between the vertebrae.

Notice that the anulus fibrosus is not symmetrical. At its dorsal border, near the spinal cord, the anulus is thinner. This is an important thing to remember, especially in dog breeds predisposed to ***intervertebral disc disease*** (**IVDD**). You see, in breeds predisposed to IVDD, both the anulus fibrosus and the nucleus pulposus deteriorate at an accelerated rate compared to other animals. The anulus becomes more fragile. The nucleus becomes thick and mineralized—almost into a thick, chunky, toothpaste-like consistency. All this means less flexibility and less shock absorption. At this point, normal movements of the spine may begin breaking down the anulus fibrosus, bit by bit. Because it is thin at the dorsal border, if it completely breaks down there, the nucleus pulposus may rupture through it and into the spinal canal (vertebral foramen) that houses the spinal cord. This can have devastating consequences on the spinal cord. We'll talk about ***spondylopathies*** [*spondyl(o)-* vertebrae + *-pathy* a disease of] like that in Chapter 11. We'll refer back to this discussion when we get there. For now, let's try to keep focused on cartilaginous joints.

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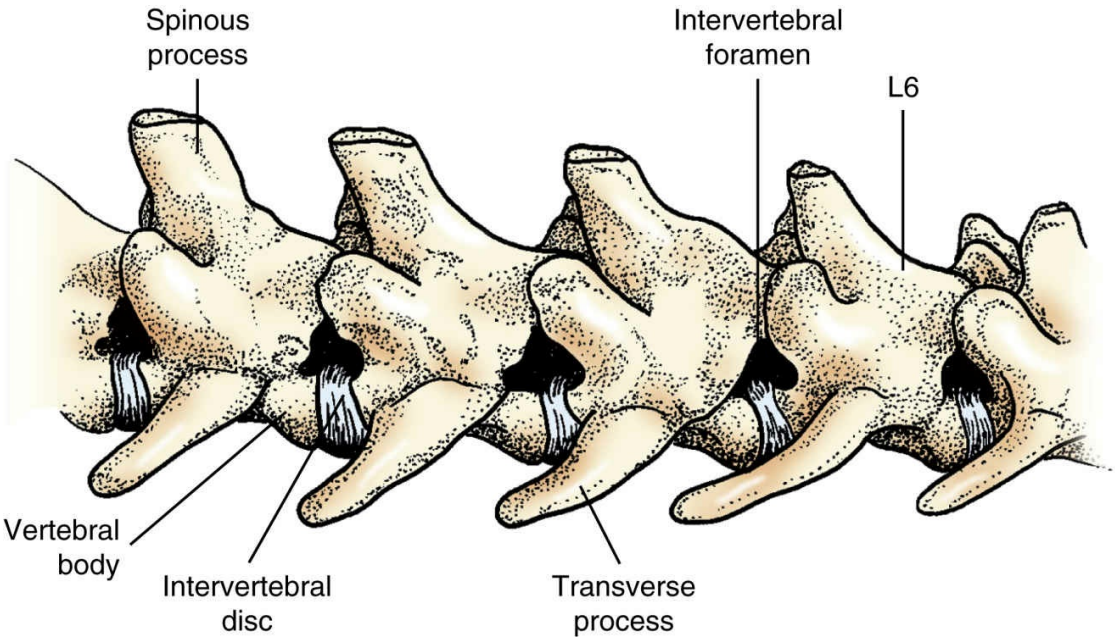

FIG. 4.21 Left lateral view of lumbar vertebrae—cartilaginous joints.

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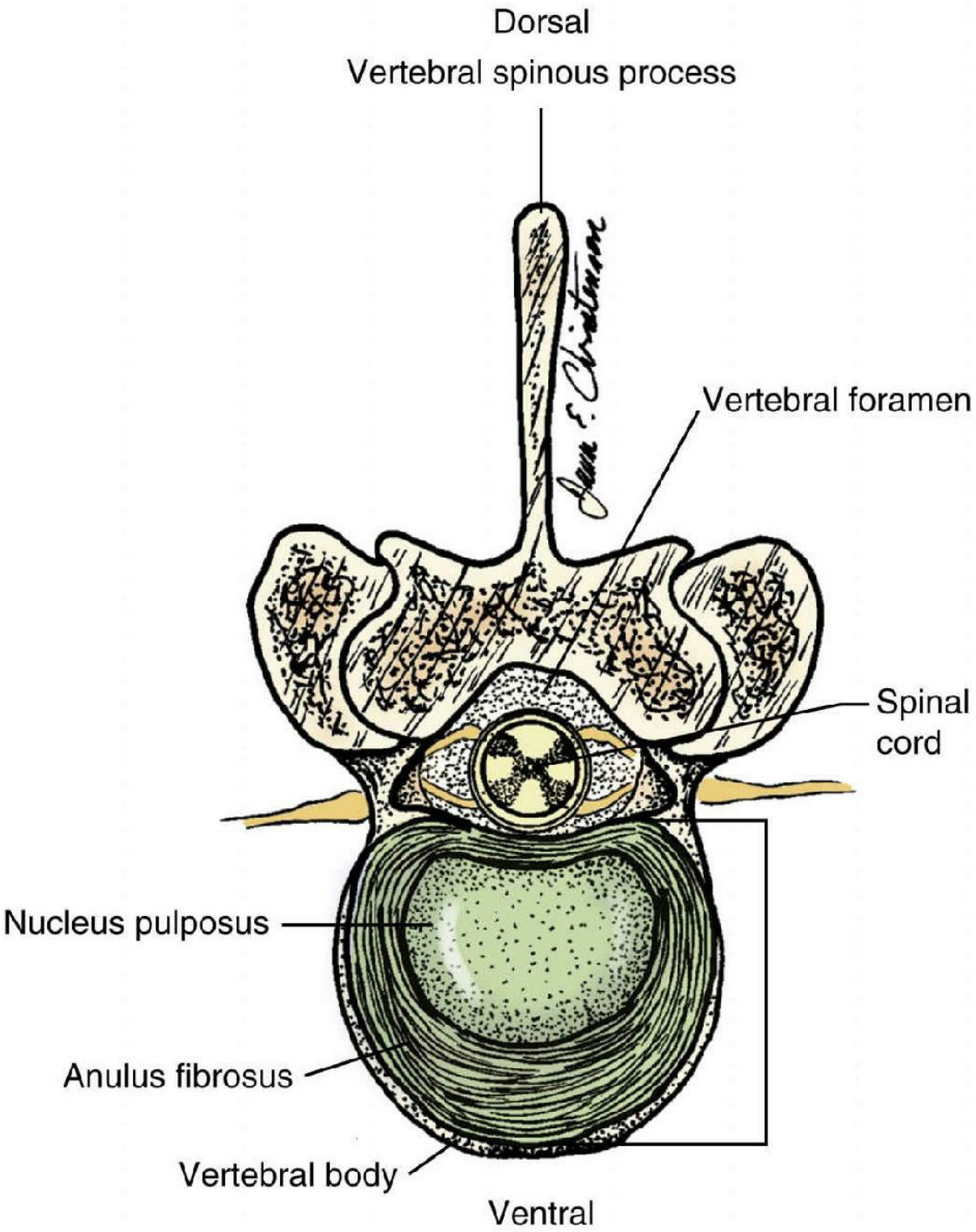

FIG. 4.22 Intervertebral disc anatomy—transverse spinal section.

The rib cage provides another great example of *cartilaginous joints*. Here, we find ***costal*** [*cost(o)-* rib + *-al* pertaining to] ***cartilage*** connecting the bony rib to the sternum (Fig. 4.23). This provides great flexibility for the rib cage. That's pretty important for breathing! Imagine if the rib cage was made of solid bone. We wouldn't be able to expand and move the rib cage when breathing. Okay, that would be fine if we did nothing but lay around or sit on

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the couch all day. For any kind of activity, especially strenuous activity (like trying to run away from an angry dog or bear), we need a flexible rib cage for deeper breathing. By the way, we'll mention the ***costochondral*** [*cost(o)-* rib + *chondr(o)-* cartilage + *-al* pertaining to] ***junction*** again in Chapter 5. The junction between the bony rib and the *costal* cartilage is a helpful landmark for things like listening to heart valves.

Synovial Joints

Ah, ***synovial*** [*syn-* together, with + *ov(o)-* egg + *-al* pertaining to] ***joints*** are probably *the* type of joints folks think of when thinking about joints. All right, you're probably wondering about the name, right? What the heck do eggs have to do with these joints? That's a fair question. It all has to do with the semi-liquid filling of the joints. ***Synovium*** [*syn-* together, with + *ov(o)-* egg + *-um* the] is a transparent, semi-liquid, proteinaceous material that looks very much like raw egg whites. Again, scientists of antiquity often named things based on how they resembled things from everyday life (*synovium*; it looks like egg whites.) *Synovial fluid* provides lubrication for synovial joints much like grease or oil lubricates moving parts on an automobile. That's important because all of the major synovial joints of our extremities can take a beating. So, they need good lubrication. Sometimes with various *arthropathies*, we need to evaluate and examine the synovium. Fortunately, it's liquid enough that we can collect diagnostic samples via ***arthrocentesis*** [*arthr(o)-* joint + *-centesis* to puncture; i.e., puncture of a joint for collection of samples or infusion of medications]. That's a discussion for another time. Let's take a closer look at a synovial joint to help you understand their structure.

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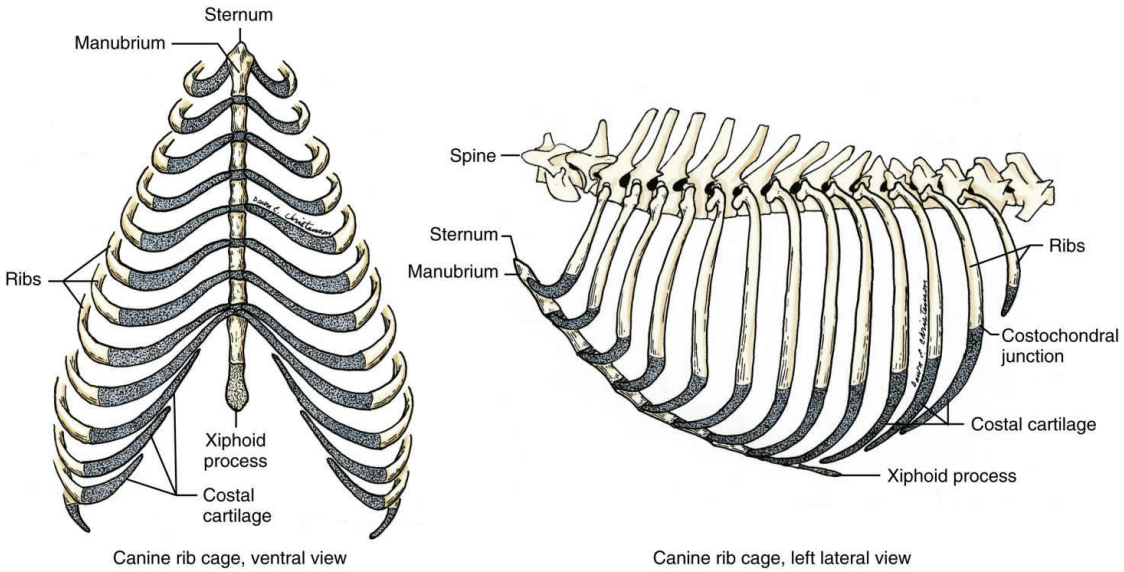

FIG. 4.23 Canine rib cage—ventral and left lateral views.

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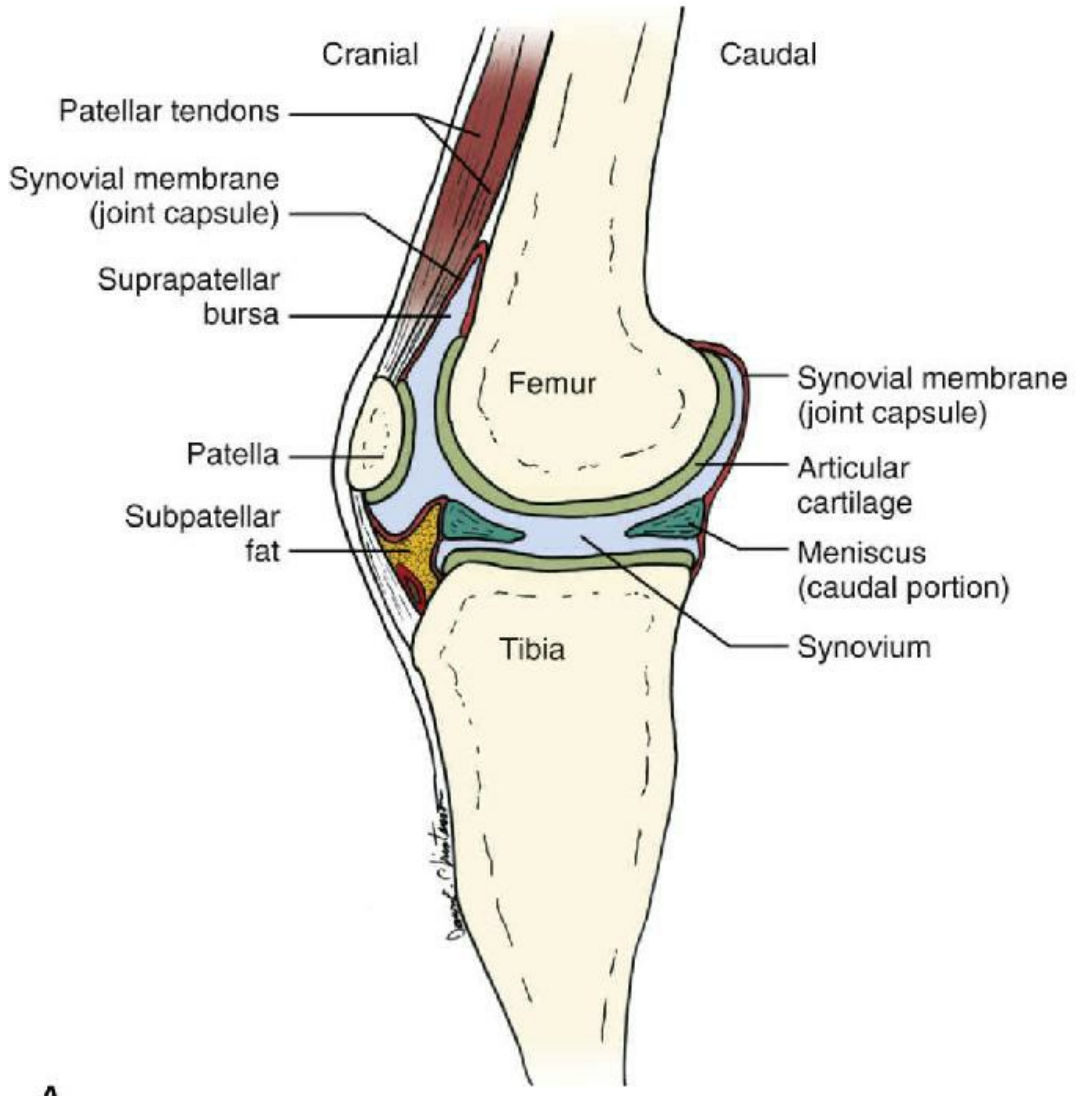

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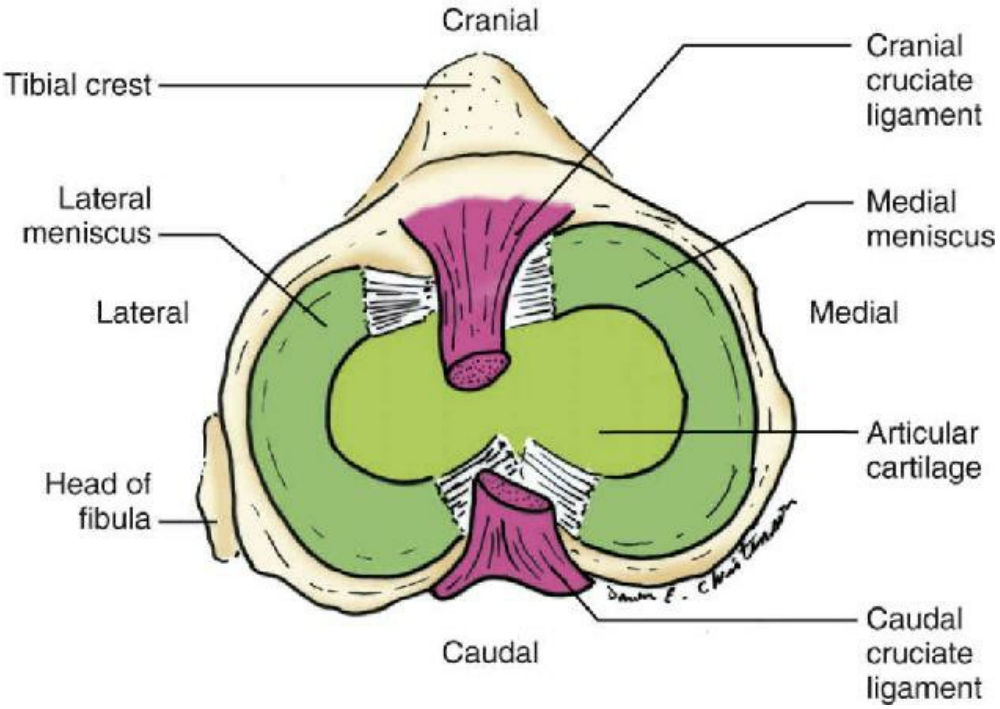

B

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FIG. 4.24 (A) Sagittal stifle joint—synovial joint schematic. (B) Proximal view of tibia.

Fig. 4.24A is a sagittal schematic of the stifle joint. Mind you, the stifle is but one example of many synovial joints of the body. As you can see, the articular surfaces of the femur, tibia, and patella are covered by smooth cartilage. Without the synovium, there would still be friction of the articular cartilage, wearing it away. Trust me, as one with bad knees and one knee already replaced, bone on bone pain without articular cartilage is no fun! The ***meniscus*** (men-is'kus [L. "crescent"]) you see here is only a small portion shown in cross-section. A *meniscus* is literally shaped as its name implies, as a crescent (see Fig. 4.24B). In the stifle joint, there are two *menisci* (men-is-ki', plural)—one for the medial condyle of the femur and one for the lateral condyle. They are made of fibrocartilage. The menisci kind of cradle the femoral condyles. Along with the ***cruciate*** (kroo'she-at [L. *crux* cross; the cruciate ligaments cross each other in the joint] ***ligaments***, the menisci help stabilize the stifle joint. The menisci also minimize wear and tear on the articular cartilage. (By the way, just like ACL tears in humans, dogs often tear [rupture] their cranial cruciate ligaments. We evaluate for this by checking for "drawer movement" in the stifle. Drawer movement is a cranial movement or "thrust" of the distal femur over the proximal tibia, sliding like a drawer. If the cranial cruciate ligament is intact, the joint is stable with no "drawer." With cruciate tears, drawer movement can be observed.) No, not all synovial joints have menisci. The ***joint capsule*** itself is composed of a continuous sheet of fibrous connective tissue. It envelopes the whole joint, kind of like a bubble, holding all of the synovial fluid within it. Do you notice how, for instance proximal to the patella, the joint capsule forms a sac or pouch of sorts? A pouch like that is called a ***bursa*** [Gr. *bursa* "a wine skin"]. See? Here again, ancient Greek anatomists named it like they saw it. These areas really do resemble wine skins! Have you ever had or known someone who had ***bursitis*** [*burs(o)-* bursa + *-itis* inflammation of]? Well, now you know what was inflamed.

So, that's the basic structure of a synovial joint. Now we need to consider the functional structure of synovial joints. A trip to the local hardware store may help you understand how each of these

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synovial joints actually work. Seriously, why not do what our scientific predecessors in antiquity did and look for examples in everyday life to help us understand? The other thing that might help your understanding is viewing the Evolve animation, Types of Joint Movement, to see those movements in action. (Note that there is no audio to this animation.)

Joint Form and Function

Hinged Joints

Alright, we have all passed through doors that open and close on hinges, right? Well, believe it or not, we have *hinged joints*. The elbow is probably the best example of a hinged joint. Like opening the door, we can open the angle of the elbow joint in extension. Like closing the door, we can narrow or close the angle of the elbow joint in flexion. (We looked at both of these movements earlier when we talked about extensors and flexors.) The stifle functions in a similar manner. Like the swinging door, we have only two movements of extension and flexion for hinged joints. And like the door, there are limits for how far we can safely flex and extend a hinged joint, without risking injury.

Ball and Socket Joints

I have heard that the *ball and socket joints* (ball and universal joints) of automobile steering mechanisms were actually designed based on the hip joint of animals and people. Okay, so most of us have never looked at, let alone thought about, the mechanical structure of steering mechanisms. (Call me weird, but I'm fascinated by that stuff.) We're simply thankful that the steering works, right? Well, perhaps you've seen a ball and socket mount on a camera tripod. No? What about a trailer hitch? All of these are variations on a theme of a ball and socket structure. In the body, the hip and shoulder joints are ball and socket joints. Structurally, there is a ball (the head of the femur and head of the humerus) that fits into a cup-like socket (acetabulum of the pelvis and glenoid cavity of the scapula, respectively). Whoa. We need to look more closely at those names: ***acetabulum*** (as"ĕ-tab'u-lum [L. *acetabulum* "vinegar-cruet"]) and ***glenoid*** (glen'oid [L. *glene* socket, pit + *-oid* resembling].

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Glenoid makes sense when we break it down like that, because it does resemble a socket or pit. But what the heck is a vinegar cruet? It is a little cup that was used in antiquity to hold vinegar. See? Ancient anatomists simply compared anatomy with everyday items. Speaking of anatomy, what's so great about ball and socket structure? It offers the fullest range of movement of any joint.

Like hinged joints, the hip and shoulder can flex and extend. They can also accommodate abduction and adduction. The limb can be rotated a little bit on its own axis, at the hip or shoulder. And, these joints provide for the unique movement of ***circumduction*** [*circum-* around + *duct(o)-* draw, lead + *-tion* the act of]. That's a challenging word. Let's see if we can help you visualize it. Reach your arm straight out to your side, perpendicular to your body. Now draw a really big, imaginary circle in the air with your arm. That movement of your shoulder is *circumduction*. Only ball and socket joints, like the hip and shoulder, can move that way.

Gliding Joints

The name of these joints is a bit misleading. The bones forming these joints may have somewhat flattened articular surfaces, but they don't glide across one another, like an iron gliding across the ironing board. The bones really rock against each other. But, unlike a rocking horse, the bones involved can rock in multiple directions. Perhaps that's why anatomists decided to call them *gliding joints*, rather than rocking joints. Carpal, tarsal, and intervertebral joints represent gliding joints of the body. For the most part, the carpus and tarsus engage in extension and flexion movements. The structure of these joints in animals is a bit more limiting than what we experience comparatively with our wrists and ankles. Still, there is a bit of mediolateral flexibility, especially in the carpus of dogs and cats. Most of the vertebral facets along the spine are good examples of gliding joints. This allows animals to bend their spines (not just their necks and tails, but their backs too) in dorsal, ventral, and lateral movements. Without the gliding joints formed by the vertebral facets, those stable spinal movements would not be possible.

Pivot Joints

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Pivot joints provide rotational movements. ***Rotation*** is a movement like that of an airplane propeller, an outboard motor propeller, or a lawn mower blade. Of course, these mechanical examples rotate a full 360 degrees. That doesn't happen in the animal world (although the rotation of an owl's head comes close!). The rotation of the head of any animal or person is made possible by the pivot joint formed between the first and second vertebrae (atlas and axis, respectively; refer back to Fig. 4.17B to see them). These are the only two cervical vertebrae with specific names. How do you keep them straight? In Greek mythology, the god Atlas held the world on his shoulders. So the atlas bone (C_1) holds the skull (like the world) on its "shoulders." The name of the joint formed by C_1 and C_2 also provides a clue as to which bone comes first: ***atlantoaxial*** [*atlant(o)-* atlas (1) + *axi(o)-* axis (2) + *-al*] ***joint***. The *atlantoaxial* joint is pretty much the only true pivot joint of the body. It gives us as humans the unique ability to rotate our heads from side to side and say "no," without uttering a word. Although, I swear one of my dogs did this when she refused to do something that I told her to do. Perhaps I read too much into her actions. At the very least, I know that all of my dogs have rotated their heads out of curiosity or to focus on a particular sound better.

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Comparative Skeletal Anatomy

We've talked about numerous, often disconnected, bits and pieces of skeletal anatomy. Let's finally put the whole package together, shall we? Each species is a variation on a theme. We'll begin by looking at canine and feline skeletal anatomy first. Of all domestic quadrupeds, dog and cat skeletons are most like ours. So, for the sake of familiarity, we'll begin with them. Then we'll take a comparative look at how the anatomy has been functionally modified in horses and ruminants.

Canine and Feline Skeletal Anatomy

Let's begin this adventure by naming the major joints of the body (Fig. 4.25, A and). Yes, we've already named some of them, but only in common terms. Now we need to name them scientifically. In order to do that, we generally need to know the names of the bones articulating in each joint. Consider the stifle joint, for example. Why do we call it the stifle joint? I have no idea. I've never been able to find the etymology for the word. Whatever you do, do **not** call this joint the knee. Yes, the comparable joint in us is our knee. But what we call the "knee" in animals (especially livestock) is a totally different joint. We'll talk about that in large animals a bit later. Now, you already know that the femur and the tibia articulate to make the stifle joint. (Note: the fibula lies along the lateral tibia but does not articulate at the stifle or tarsus.) So, it stands to reason that we would scientifically name the stifle the ***femorotibial*** [*femor(o)-* femur + *tibi(o)-* tibia + *-al* pertaining to] ***joint***. Simple, right? Yes, most of the time the naming of joints is this simple. But the hip is not quite that easy. One might expect to use the name of a portion of the pelvis with the femur to name it. After all, the *acetabulum* is the cup that holds the femoral head. So why not name it with that? Ancient anatomists simply used the Latin word that means hip—*coxa*. It seems rather redundant, but scientifically we call the hip the ***coxofemoral*** [*cox(o)-* hip + *femor(o)-* femur + *-al* pertaining to] ***joint***. For me, it helps knowing the Latin origins of the word, especially with odd terms like *coxofemoral*. I hope that it

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helps you too.

More distal in the rear limb, you are already familiar with the hock, scientifically the *tarsus*. (The tarsus in animals correlates to our ankle.) You also know that it is composed of a bunch of little short bones, forming a number of gliding joints that collectively create the whole. Well, they're all really short bones, except for that really big one at the caudal point of the hock. That's the ***calcaneus*** (kal-ka'ne-us [from L. *calx* heel]). It needs to be big because that's where the *gastrocnemius tendon* (aka *Achilles tendon*) attaches. Okay, here is a fun bit of mythological trivia for you. The Achilles tendon is named after the Greek warrior and hero, Achilles. According to mythology, Achilles was extremely strong and courageous. His only vulnerability was his heel. You see, according to legend, when he was an infant, his mother dipped him in the River Styx to make him invincible. And he was. The only place the waters did not touch him was on his heel, because that is where his mother held onto him when she dipped him in the river. That left his heel vulnerable. To this day, if anyone refers to their "Achilles heel," they are referring to a point of vulnerability, usually figuratively. There, wasn't that a nice diversion from anatomy and physiology?! Never thought you'd learn Greek mythology from a scientific text, did ya?

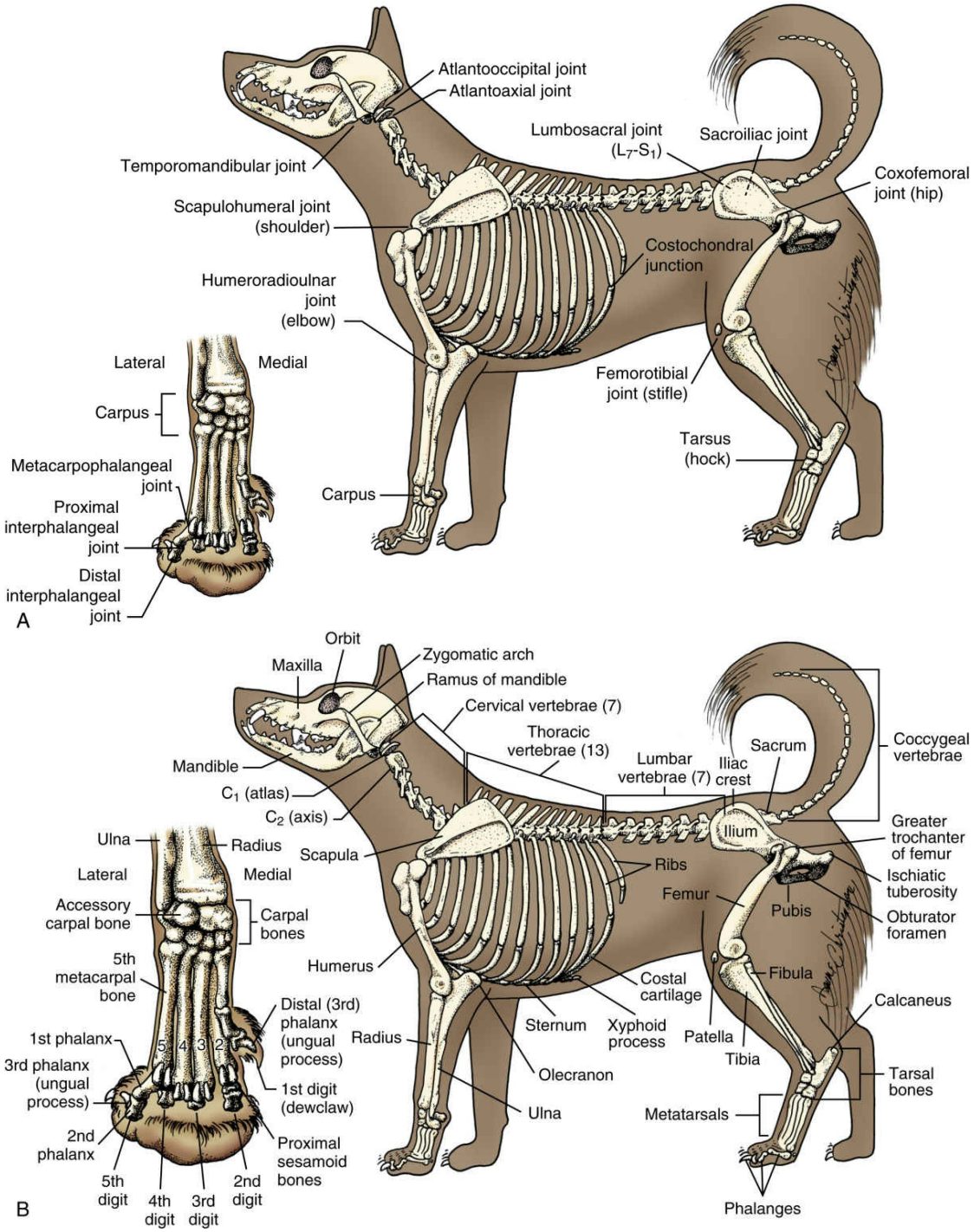

FIG. 4.25 Comparative skeletal anatomy: (A) canine joints. (B) canine bones.

Getting back to the joints of the rear limb, the bones distal to the tarsus come *after* or *beyond* the tarsus, right? The prefix meaning after or beyond is *meta-*. So, the bones immediately distal to the tarsus are the ***metatarsal*** [*meta-* after, beyond + *tars(o)-* tarsus + *-al*] ***bones***. If we want to talk about the articulation between the distal *tarsal* bones and the *metatarsal* bones, we would name them

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tarsometatarsal joints. The distal-most bones of the limb are the ***phalanges*** [from L. *phalanx* "a line of soldiers"]. (Well, I guess they are lined up kind of like little soldiers.) Each digit (toe) has three phalanges, with each ***phalanx*** numbered from proximal to distal (1, 2, 3). The distal (third) phalanx is the only one of the three in dogs and cats that has a special name. That name is the ***ungual*** (ung'gwul [L. *unguis* nail] ***process***. The nails of dogs and cats are attached to and supported by the *ungual process*. With regard to phalanges, this all holds true in the front limb. too.

The first phalanx of each toe articulates with a metatarsal bone. That joint is a ***metatarsophalangeal*** [*meta-* after, beyond + *tars(o)-* tarsus + *phalang(o)-* phalanx + *-al* pertaining to] ***joint***. If we were going to amputate a toe at the metatarsophalangeal joint, we would need to make sure that we were amputating the correct toe, and so we would need to be sure to indicate to which digital metatarsophalangeal joint we were referring. The digits (toes) are numbered, too, from medial to lateral. The ***dewclaw*** (front or rear limb) is always number one (our thumb). Even if no dewclaw is present, the next digit is numbered as the second (including the metatarsal bone; our index finger). The lateral-most digit is the fifth (our pinky finger). In all four limbs, our digits and phalanges are numbered the same. So we need to identify the foot, as well as the digit and parts of the digit to be amputated. Hypothetically, let's say that we're planning to amputate the lateral toe of the right rear limb. The surgeon might scientifically detail it this way: "amputation of the right rear fifth digit up to the metatarsophalangeal joint or mid-metatarsal if gross pathology of joint observed." By the way, when we ***declaw*** cats, we are literally amputating the ungual process of each digit. Remember, amputation in any way, shape, or form is a big deal.

We had a beloved Gordon setter (Serena, may she rest in peace), who required multiple digital amputations over a period of several years. She developed cancer, a type of ***carcinoma*** [*carcin(o)-* crab, cancer + *-oma* tumor; a malignant cancer made up of epithelial cells that infiltrates other tissues], in those toes. The disease appeared to begin in either the ungual process or in the vicinity of the distal ***interphalangeal*** [*inter-* between + *phalang(o)-* phalanx + *-al* pertaining to] ***joint***. The first amputation was of the right front first

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digit. It was roughly two years later that she developed similar lameness symptoms in her left front. This time, the cancer involved the second digit. It too was amputated, up to the ***metacarpophalangeal*** [*meta-* after, beyond + *carp(o)-* carpus + *phalang(o)-* phalanx + *-al* pertaining to] ***joint***. (Did you catch that? The carpus is in the front leg and the tarsus is in the rear. That means metacarpal bones are in the front limb, compared to metatarsal bones in the rear.) Finally, when she was nearly 14 years old, the cancer showed up in her right front fifth digit. As you can see in the right dorsopalmar radiograph (Fig. 4.26), she had *osteolytic* [*oste(o)-* bone + *-lytic* pertaining to destruction, dissolving of] changes of the ungual process and second phalanx (portions of those bones are absent, and there is a lack of brightness of the bone, compared to the other digits). Unfortunately, amputation was not an option this time. She was simply too old and weak to undergo anesthesia and surgery. When we could no longer adequately control her pain, we let her go.

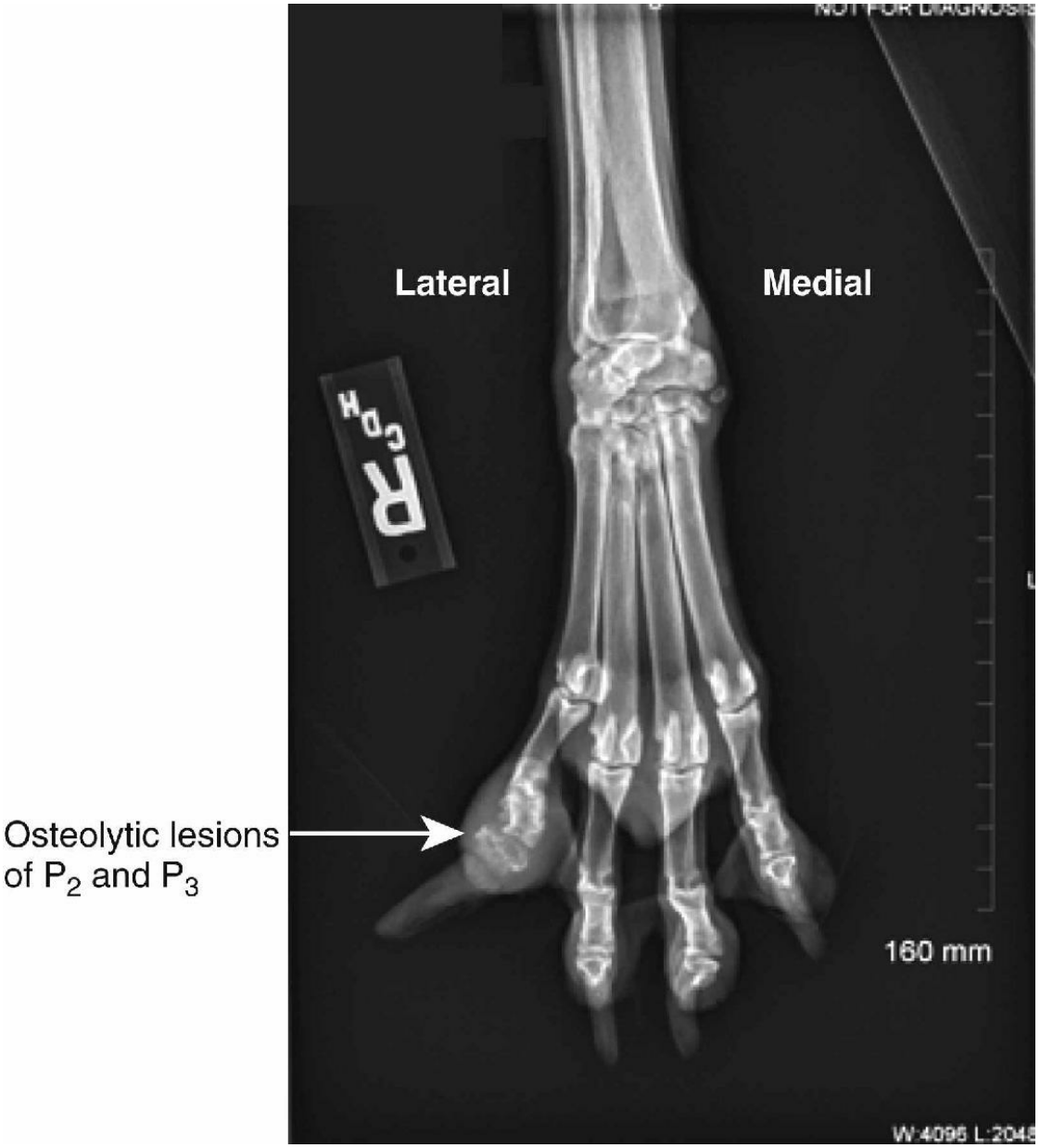

FIG. 4.26 DPa Rt paw of Serena.

Enough of that! Let's move proximally on the front limb. You are already familiar with the carpus. Articulating with the carpus are the bones of the *antebrachium*—the radius and ulna. Then there is the joint proximal to the carpus—the elbow, as you know it. Three bones articulate in the elbow. And if we name the joint, listing those bones beginning with the most proximal bone, we have the ***humeroradioulnar*** [*humer(o)-* humerus + *radi(o)-* radius + *uln(o)-* ulna + *-ar* pertaining to] ***joint***. See? If you simply think of the three bones forming the joint, that word doesn't seem so intimidating does it? By the way, the point of the elbow is created by the really large protuberance of the ulna. That part of the ulna is called the

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olecranon (o-lek'ruh-non [Gr. *olecranon* from *olene* elbow + *kranion* skull]). (Perhaps it was used in battle to crack the skulls of the enemy, giving it its weird name. Who knows?) At any rate, it's a great leverage point for the triceps muscle to extend the elbow. Finally, the most proximal joint of the forelimb is the shoulder. Because it is formed by the scapula and humerus, the scientific name of the shoulder is the ***scapulohumeral*** [*scapul(o)-* scapula + *humer(o)-* humerus + *-al* pertaining to] ***joint***. I love simplicity.

So that takes care of the extremities. What about ***axial*** [*ax(o)-* axis, axle + *-al* pertaining to] ***skeleton***? Well, we've already looked at the skull a little bit, with the suture lines between the flat bones. There are just a few more basic features to point out (Fig. 4.27). As we consider the muzzle of the dog, we generally refer to the upper jaw as the ***maxilla***. There are other bones there, too, but for our purposes we'll keep it simple with *maxilla*. The lower jaw is the ***mandible***. Because the ramus of the mandible articulates with temporal bone of the cranium, the joint formed is the ***temporomandibular*** [*tempor(o)-* temple + *mandibul(o)-* mandible + *-ar* pertaining to] ***joint***. Perhaps you or someone you know has TMJ (temporomandibular joint) syndrome. I do. I don't chew gum because of it; all of the resultant snapping and cracking is way too noisy in my head. Notice too that the ramus of the mandible lies medial to a boney arch. That is the ***zygomatic*** [*zygom(o)-* bolt, bar + *-tic* pertaining to] ***arch***. The zygomatic arch not only forms the "cheek," but also helps form the eye socket, or ***orbit***. Please note that the orbit of the dog and cat is incomplete. It is open on the lateral aspect. Remember this when we talk about the eye in Chapter 11. (This orbital structure is different from the large animals that we'll look at in a little bit.) That lateral opening makes it easier for the eye to pop out. Our final point of interest on the skull is the caudal bone of the cranium: the ***occipital*** [from Gr. *caput* head] ***bone***. The ***atlanto-occipital joint*** is the articulation of the occipital condyles with the first cervical vertebra, the atlas.

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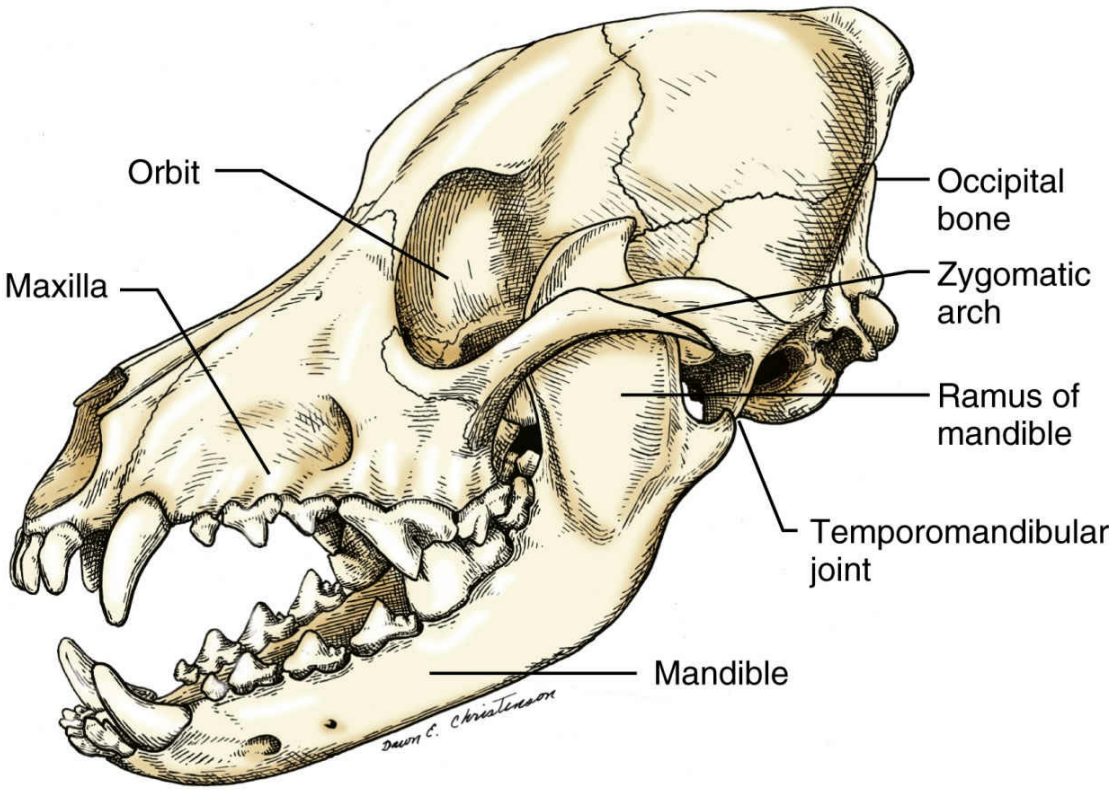

FIG. 4.27 Canine skull.

That leads us to the spine. We've already touched on bits and pieces of the spine, from the gliding joints formed by vertebral facets to intervertebral discs that separate the vertebral bodies. Each vertebra has a hole through it, where the spinal cord actually passes through (see Figs. 4.17B and 4.22). This is the ***vertebral foramen***. There are also small holes bilaterally where the vertebrae articulate, allowing spinal nerves to pass from the spinal cord out to other parts of the body. These are ***intervertebral foramina***, because they are between the vertebrae Fig. 4.21. Makes sense, right? (As a reminder, the plural of foramen is foramina.) Then we have some prominent protuberances that stick out more on some vertebrae than on others. ***Cervical vertebrae*** don't have any extraordinarily large protuberances. But the thoracic vertebrae have huge dorsal spinous processes.

Look at the thoracic vertebra in Fig. 4.17B. Isn't that ***dorsal spinous process*** huge compared to the rest of the vertebrae? Obviously, those processes are named for where they are located. They are not all the same size either. The more caudal we get along the thoracic spine, the smaller the dorsal spinous processes become,

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until the one on the last thoracic vertebra is roughly the same height as the first lumbar vertebra. The dorsal spinous processes of the lumbar vertebrae are pretty close to the same height. If you look at the lumbar vertebra in Fig. 4.17B, you'll notice that it has relatively large ***transverse processes***. Yes, they line up with transverse planes, hence the name. The transverse processes are there to help support the epaxial muscles. No, we don't have transverse processes along the thoracic spine, because they would be in the way of the ribs. Plus, we don't need transverse processes on the thoracic vertebrae to support the epaxial muscles, because the ribs do that. And a fine support the ribs make, too. They're not free-floating, remember. They articulate with the thoracic vertebrae and the bones of the sternum. While we're on the subject, you should know that we give specific names to the most cranial and most caudal sternal bones (see Fig. 4.23). The most cranial bone of the sternum is the ***manubrium*** [L. "handle"], and the most caudal bone of the sternum is the ***xiphoid*** [*xiph(o)-* sword + *-oid* resembling]. As its name implies, the ***xiphoid process*** sticks out like a sword. Its tip is made of xiphoid cartilage that is supported by the xiphoid bone. By the way, xiphoid may be spelled two ways: *xiphoid* and *xyphoid*. Both are correct and mean the same thing. My advice to you is to pick one spelling and stick with it. But I digress. Let's get back to the spine.

The ***lumbosacral*** [*lumb(o)-* lumbus, loin + *sacr(o)-* sacrum + *-al* pertaining to] ***joint*** is formed between the last lumbar vertebra and the sacrum. The ***sacrum*** is made up of a number of fused vertebrae. Why fuse them and form just one bone, you ask? Well, we need a strong point of connection for the pelvis. The ***sacroiliac*** [*sacr(o)-* sacrum + *ili(o)-* ilium + *-ac* pertaining to] ***joints*** connect the sacrum bilaterally with the "wings" of the ilium of the pelvis. Don't be misled by the term "joint" here. There is extremely limited movement at each sacroiliac joint, compared to other joints of the body. The ligaments surrounding the sacroiliac joints hold the bones very firmly together, allowing for just a little bit of wiggle, if you will. That minimal flexibility is important, especially when walking or running. I'll come back to the pelvis in a moment. First, let's complete the tail end of this beast with the ***coccygeal*** [*coccyg(o)-* tail + *-al* pertaining to] ***vertebrae***. Wait. Doesn't caudal also mean

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"tail?" Yes, it does. So why not call them caudal vertebrae? Well, technically you could. However, will everyone else know what you mean when you do that? Perhaps not. You see, they may wonder if you're referring to the actual anatomic tail or if you're directionally referring to the caudal-aspect of another structure. We cannot afford confusion, especially for patient care. Confusion often leads to mistakes and medical errors. Our patients deserve better. So, to minimize confusion, we tend to use "caudal" as a directional term and "coccygeal" as an anatomic term.

Getting back to the pelvis now, let's look at a few more of its features. The ilium is the largest portion of the pelvis. The ischium is the caudal part. You already know that the acetabulum forms the socket for the coxofemoral joint. That leaves us with the pubis, forming a large portion of the pelvic floor to support the rectum, as well as caudal portions of the urinary and reproductive structures. The *pelvic symphysis* could also be called the *pubic symphysis*, because it is formed by the pubic bones. Lightening the weight of the pelvis and providing a passage for nerves and vessels, we have the ***obturator*** [ob"ter-a'ter L. *obturatus* "one that closes"] ***foramen***. Okay, it seems counterintuitive to name an opening with a word that means closed or obstructed. I won't even attempt to figure out the ancient anatomists' thinking here. Perhaps they simply emptied one too many wineskins. The *obturator foramen* is a large hole in the pelvis. That's all you need to know.

Believe it or not, in young dogs with an anatomic predisposition (i.e., shallow acetabulum) for the development of ***hip dysplasia*** [*dys*-bad, poor + *plas(o)*- formation + *-ia* condition of], we can surgically alter the pelvis to minimize or prevent hip dysplasia. We use a *triple pelvic osteotomy* [*oste(o)*- bone + *-tomy* to cut] (TPO) for this. By cutting through the ilium, ischium, and pubis (the obturator foramen makes it a bit easier to cut through the ischium and pubis), we can actually rotate the surgically-freed central portion of the pelvis. Once repositioned, we secure the bones with orthopedic plates and screws. Our goal with this *osteotomy* is to make the acetabulum sit more dorsal over the femoral head. This makes the hip more secure, preventing excess wear and tear and the resultant debilitating ***osteoarthritis*** [*oste(o)*- bone + *arthr(o)*- joint + *-itis* inflammation of]. Please note that this procedure is an *osteotomy*

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NOT an ***ostectomy*** [*oste(o)-* bone + *-ectomy* to cut out]. In a TPO, we are only cutting [*-tomy* to cut] the bone, creating a surgical fracture. We are not removing bone, as the suffix *-ectomy* indicates. Just a two-letter difference between those suffixes makes a world of difference in the actual procedure. It is very important that you remember the difference. Would it be appropriate to use the term ***osteoplasty*** [*oste(o)-* bone + *-plasty* a reforming of] for a procedure, like a TPO? Yes. We are literally reforming or reshaping [*-plasty*] the pelvis. Still, the procedure is most often referred to as a triple pelvic osteotomy.

You know there are a number of arthropathies, like hip dysplasia, that have breed predisposition. Hip dysplasia is common in many large canine breeds, like Labrador retrievers and German Shepherds. And while certain breeds may be predisposed to certain diseases, that does not mean that every dog within the breed will develop the problem. It also does not mean that dogs outside the affected breed will not develop the same problem. I have seen small dogs develop hip dysplasia. No, that's not common. But it happens. Just because my Cardigan Welsh Corgi, Ellie, had a long back, did not mean that she was destined to develop IVDD. She never did. She was a ***chondrodystrophic*** [*chondr(o)-* cartilage + *dys-* bad + *troph(o)-* development + *-ic* pertaining to; *dystrophy* is poor development] breed, with her dwarfed, short, stumpy legs. And because of that she did develop ***arthritis*** [*arthr(o)-* joint + *-itis* inflammation of] in some of her joints (e.g., carpus, interphalangeal, shoulder). My Basset Hound, Sadie, had similar issues with ***arthritic*** [*arthr(o)-* joint + *-tic* pertaining to; i.e., pertaining to inflamed joints] joints. I know one of her elbows had a loose fragment floating around in it. Most of the time it didn't bother her. But when it did, she couldn't bear weight on the leg. I wish that ***arthroscopic*** [*arthr(o)-* joint + *scop(o)-* view + *-ic* pertaining to] surgery had been perfected while she was alive. I probably would have opted for the ***arthroscopy*** [*arthr(o)-* joint + *-scopy* process of viewing; i.e., joint surgery using an instrument called an *arthroscope*] to remove that annoying floater. *Arthroscopic* surgery in veterinary medicine was not as common then as it is today. Recovery from these surgeries is much faster than from conventional ***arthrotomies*** [*arthr(o)-* joint + *-tomy* to cut; i.e., cutting into a joint, opening it up

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for gross visualization of structures].

Sadie also developed some painful *spondylopathies* [*spondyl(o)-* vertebra + *-pathy* a disease of]. You see, *chondrodystrophic* breeds like hers not only have structural abnormalities putting unusual stresses on various joints, but they also may have predisposition to weak cartilage. For Sadie, these factors affected her intervertebral joints, between vertebral facets as well as vertebral bodies. As the joints deteriorated over time, ***spondylitis*** [*spondyl(o)-* vertebra + *-itis* inflammation of] developed. Along the spine, this kind of *osteoarthritis* usually results in the development of large bone spurs. It can be so significant, as it was for Sadie, that the extra bone deposited actually bridges and fuses adjoining vertebrae. ***Ankylosis*** [*ankyl(o)-* bent, fused + *-sis* condition of] of the spine like this is called ***spondylosis*** [*spondyl(o)-* vertebra + *-sis* condition of]. You see, any joint can ***ankylose*** [fuse] from injury, disease, or surgery. You might think that once the vertebrae *ankylose*, they become stable resulting in less pain. This may be true to a point. At least we would no longer have bone spurs crunching against one another. Unfortunately, the excess bone from the *spondylosis* may press on spinal nerves exiting through the intervertebral foramina. And that is very painful. Later in life, Sadie's *arthritis* and *spondylosis* slowed her down quite a bit. When going on walks was simply too painful, I did the walking while pulling her in a wagon. She loved it, especially with all of the attention from the neighbors as we passed by.

Mind you, problems like arthritis and such are not limited to small animals. In fact, arthroscopic surgery in horses is probably more common than it is in small animal orthopedic surgery. That said, it sounds like a good time to make some comparisons to large animals now.

Equine Skeletal Anatomy

Do horses really differ that much from dogs and cats? Yep. (See Figs. 4.28A and B.) For instance, the ilium is rotated laterally in the horse. Rather than having the nice rounded iliac crest, as we have in the dog and cat, the cranial part of the ilium is somewhat squared off. This provides for a large craniolateral border, extending out to

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the *tuber coxae* (mentioned with equine IM injection sites) laterally, to attach numerous large muscles (e.g., gluteals). Another difference: the orbit of the skull completely encircles the eye of the horse. Unlike dogs and cats, the orbit of a horse would need to be fractured in order for the eye to pop out. The *ligamentum nuchae* along the dorsal border of the neck is huge, to support the horse's large, heavy head. We can point out numerous subtle differences between horses and dogs and cats. Yet limb anatomy is where we find the most pronounced differences. Yes, the bones are more massive, as you would expect for an animal that is so much larger than a dog or cat. But it goes beyond that. For instance, the radius and ulna are not two separate bones in the horse. They are fused in the horse, with the olecranon process being the largest recognizable part of the ulna remaining. (The same is true for the fibula in the rear leg. There is only a proximal remnant of the fibula fused to the lateral aspect of the proximal tibia.) Of course, at the distal end of the radius and ulna is the carpus, just like in dogs, only much bigger. Plus, the carpus in the horse is given what many non-horse people consider an odd name—the knee. Do you see why I told you not to refer to the femorotibial joint as the knee? While all of this is different from the dog and cat, it's really in the distal extremities that we find the most dramatic differences. I recommend looking closely at the enlarged views of the distal limbs in Figs. 4.28A and B for this discussion.

As you'll recall from the previous section, dogs and cats have four digits on which they bear weight. They are weight-bearing on digits two, three, four, and five. The horse bears weight exclusively on its third digit. Yes, that is comparable to our middle finger. What happened to the rest of the equine digits? Well, we kept remnants of the second and fourth metacarpal bones, in the front limb. The same is true with the metatarsals in the rear limb. Among horse folks, you'll hear them talk about these bones as the ***splint bones*** (fourth metacarpal and fourth metatarsal bones = lateral splint bones; second metacarpal and second metatarsal bones = medial splint bones). Obviously, the third metacarpal and metatarsal bones, commonly called ***cannon bones***, have been really "beefed up" to support the weight of the horse. What do the splint bones do? Not much, other than cause painful problems for some horses.

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Then there are the phalanges and associated sesamoid bones. As with the other bones, they are large, "beefed-up" bones. The metacarpophalangeal joint has two large sesamoid bones on the palmar aspect of the joint. The same is true on the plantar aspect of the metatarsophalangeal joint. These sesamoid bones help support and guide some of the digital flexor tendons. By the way, the common name for these joints is the ***fetlock***, in both front and rear limbs. If you use common names for these joints, you'd better be sure to specify front versus rear, as well as right versus left, because horses have four fetlocks. The first phalanx, just distal to the fetlock, is commonly called the ***long pastern***. This distinguishes it from the second phalanx, which is commonly called the ***short pastern***. Yes, they have been named based on their size. The proximal interphalangeal joint formed by these two bones is commonly called the ***pastern***. Again, these names of the interphalangeal joints (scientific and common) are the same in both front and rear legs. So, be specific if you want to talk about the pastern. Otherwise, people won't have a clue of which leg you're talking about, or if you're talking about the joint or one of the bones. This is the value that I find with medical terminology–it provides clarity all by itself. If I say, "left front proximal interphalangeal joint," it is quite clear which anatomic feature I am referring to. If I want to talk about one of the pastern bones, I can be very clear by specifying either first (or proximal) or second phalanx. Clarity and precision are valuable, intrinsic characteristics provided by medical terminology.

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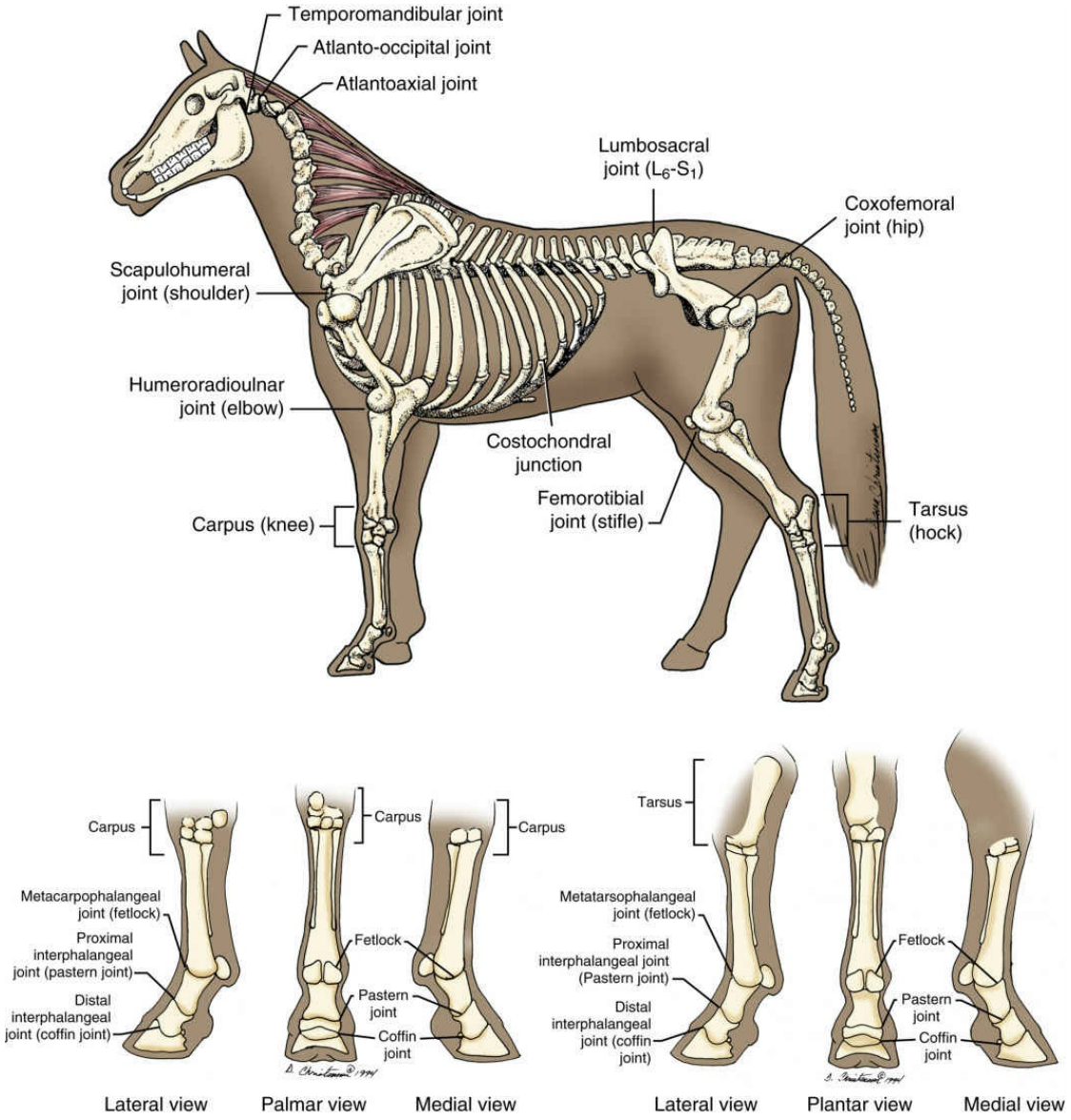
Temporomandibular joint
Atlanto-occipital joint
Atlantoaxial joint
Lumbosacral joint (L_6-S_1)
Coxofemoral joint (hip)
Scapulohumeral joint (shoulder)
Humeroradioulnar joint (elbow)
Costochondral junction
Femorotibial joint (stifle)
Carpus (knee)
Tarsus (hock)
Carpus
Metacarpophalangeal joint (fetlock)
Proximal interphalangeal joint (pastern joint)
Distal interphalangeal joint (coffin joint)
Carpus
Fetlock
Pastern joint
Coffin joint
Carpus
Tarsus
Metatarsophalangeal joint (fetlock)
Proximal interphalangeal joint (Pastern joint)
Distal interphalangeal joint (coffin joint)
Fetlock
Pastern joint
Coffin joint
Lateral view
Palmar view
Medial view
Lateral view
Plantar view
Medial view

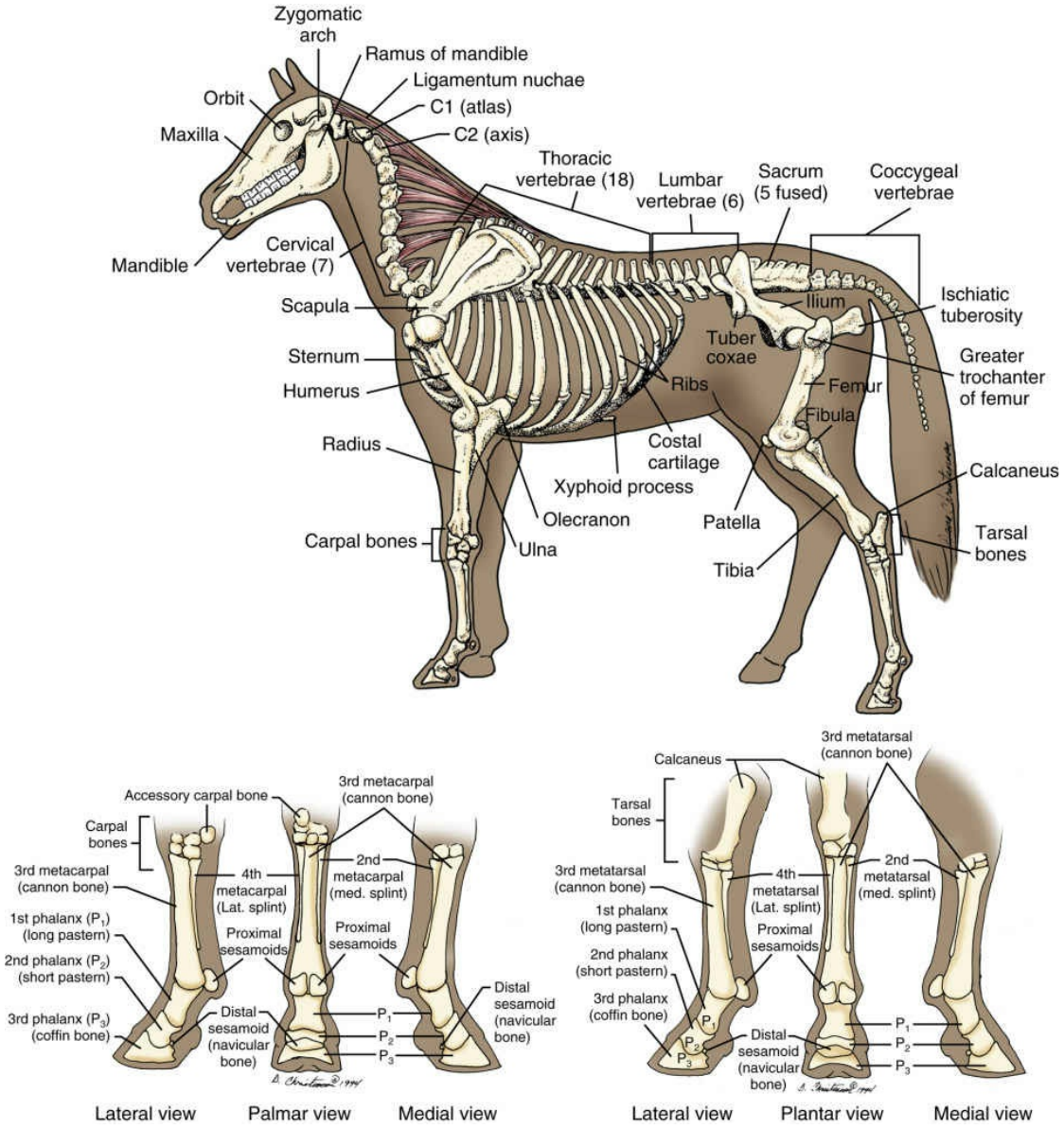

FIG. 4.28 (A) Comparative skeletal anatomy: equine joints. (B) Comparative skeletal anatomy: equine bones.

The third phalanx is commonly called the ***coffin bone*** (perhaps because it is "buried" in the hoof). They decided to use the same common name for the distal interphalangeal joint, too. Here again, if you're going to use these common names, you need to specify front versus rear, right versus left, AND, in the case of the coffin, bone versus joint. By the way, the coffin bone in horses is much like the ungual process in dogs and cats. We said in dogs and cats that the nail was attached to the ungual process by connective tissue (the "quick"). In horses, the coffin bone is attached to the hoof wall by connective tissues (lamina). We'll talk more about this in Chapter 8, when we discuss laminitis [*lamin(o)-* lamina + *-itis*

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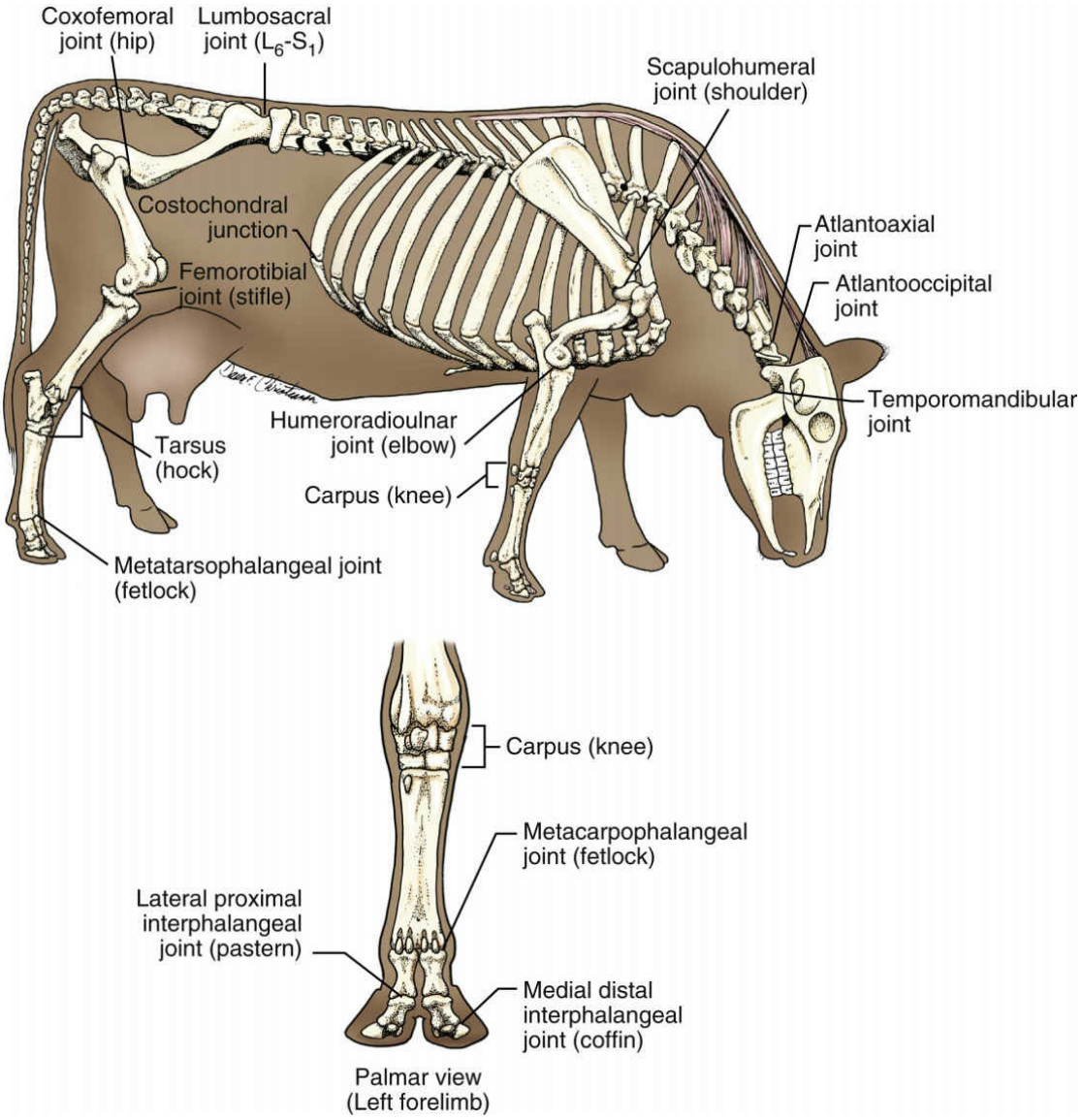
inflammation of]. ***Laminitis*** is a common and serious problem for many horses.

Finally, we have the *distal sesamoid bone*. This is commonly called the ***navicular*** (nuh-vik'u-ler [L. *navicular* "boat"]) ***bone***. I guess, if I use my imagination, it seems to be shaped kind of like a small boat, like a canoe or kayak. This bone is very important for gliding and guiding the digital flexor tendons down into the foot.
Unfortunately, not all horses are built the same. Those with smaller feet and straighter angulation from the fetlock to the foot, like Quarter Horses, are more likely to have problems with *navicular disease*. The front feet are most likely to develop problems, with navicular disease (and laminitis). This is because roughly 60 percent of a quadruped's body weight, at rest, is distributed over the front feet. Put the animal in motion and the forces exerted on the front feet may be extreme. These forces, especially in anatomically predisposed horses, can damage the navicular bone, vessels in the foot, ligaments, tendons, joint bursa, and so forth. All of these factors ultimately result in the chronic, painful degeneration of the navicular bone. Navicular disease can be a painful, debilitating condition. It can be managed, with medications, corrective hoof trimming and shoeing, and realistic activity restrictions. It cannot be cured. Our goal is to simply control pain and inflammation, as well as minimize further damage.


Coxofemoral joint (hip)
Lumbosacral joint (L_6-S_1)
Scapulohumeral joint (shoulder)
Costochondral junction
Femorotibial joint (stifle)
Atlantoaxial joint
Atlantooccipital joint
Temporomandibular joint
Humeroradioulnar joint (elbow)
Tarsus (hock)
Carpus (knee)
Metatarsophalangeal joint (fetlock)
Carpus (knee)
Metacarpophalangeal joint (fetlock)
Lateral proximal interphalangeal joint (pastern)
Medial distal interphalangeal joint (coffin)
Palmar view (Left forelimb)

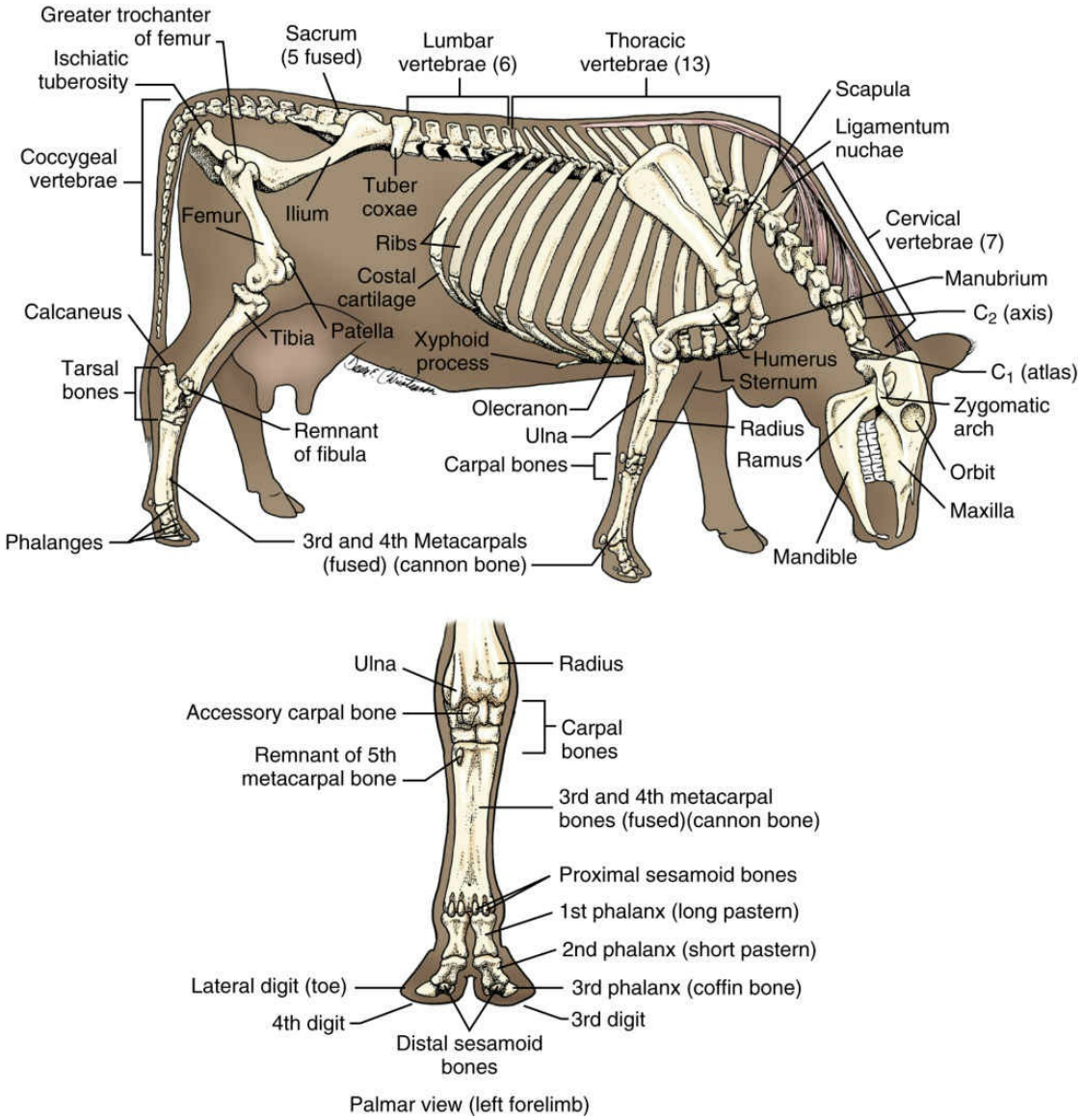


FIG. 4.29 (A) Comparative skeletal anatomy: bovine (ruminant) joints. (B) Comparative skeletal anatomy: bovine (ruminant) bones.

In spite of their size, horses can be such delicate and fragile creatures. In my experience, the same cannot be said for ruminants, especially cattle. For the most part, ruminants tend to be pretty rugged.

Ruminant Skeletal Anatomy

There are many similarities between equine and bovine (other ruminants, too) skeletal anatomy (Figs. 4.29A and B). For instance, the radius and ulna are fused. The carpus is commonly called the "knee." A large, strong ligamentum nuchae supports the head. The orbit completely encircles the eye. Truly, our biggest differences

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between horses and ruminants are found in the distal extremities (i.e., distal to the carpus and tarsus). We said that the horse bears weight exclusively on its third digit. Cattle (and other ruminants) have an even more "beefy" (pun intended) cannon bone. This is because third and fourth metacarpal bones (metatarsals in the rear) are fused. Beyond that distally, ruminants have two distinct digits (toes). This is why they are sometimes called "cloven hoofed" animals. The foot appears to be "cleaved" or split. This is because, anatomically, ruminants are simply bearing weight on the phalanges of the third and fourth digits. All of the same terminology (scientific and common) can be applied to the phalanges and the joints associated with them. There's one catch—you need to specify which digit (third or fourth) or toe (medial or lateral) of which foot you are referring to. Remember, you need to be precise to minimize confusion and mistakes.

Can ruminants ever develop arthropathies and other skeletal problems, like dogs and horses do? Sure, they can. However, if you consider the activity level of most ruminants (cattle, sheep, and goats), compared to that of many dogs and horses, they probably don't stress their joints as much. Have you ever seen a racing goat or a cow doing agility? I didn't think so. Most of their time is simply spent grazing—no athleticism there! So aside from a traumatic incident, most ruminants will probably never develop problems like arthritis.

Well, there you have it. You've waded through a great deal of musculoskeletal information. Congratulations! You did it! Now, take a deep breath before putting your newly-acquired knowledge to the test. Ready? Let's go.

Case Study

Our patient is Hank, a 6-year-old, neutered male, German Shorthaired Pointer. He has always been very active and quite athletic. The owners have brought him in because of a progressive lameness in his right rear. Per the owners' historical account, 2 weeks ago Hank was playing with his Frisbee with the owner. On one occasion, when he made a running leap to catch the Frisbee, he

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yelped. From that moment on, he has been lame on his right rear. Some days are worse than others for him. Occasionally, he either barely toe touches or refuses to put that foot down at all. He occasionally cries out, especially when turning abruptly on the right rear. The owner's perception is that Hank sprained his hock.

On physical examination, Hank was 60 pounds and in perfect body condition. He was lightly weight-bearing on his right rear. His gait demonstrated a limp, favoring the right rear. The veterinarian performed a bilateral comparative musculoskeletal examination on the rear limbs, palpating each joint. The right tarsus appeared normal. Mild swelling of the right femorotibial joint was noted. PROM was evaluated with no significant abnormalities noted, other than mild stiffness and muscular guarding of the right stifle. On evaluating both femorotibial joints for "drawer movement" (i.e., excessive cranial thrust of the distal femur over the proximal tibia), significant "drawer" was noted in the right stifle. With this information, the clinician diagnosed a torn right cranial cruciate ligament. Radiographs of the limbs appeared normal. Concerned that the current instability of the joint would eventually lead to the development of osteoarthritis in the affected stifle, the doctor recommended a tibial plateau leveling osteotomy (TPLO). She explained that this orthopedic procedure changes the angulation of the proximal tibia in the joint, reducing excessive cranial thrust of the femoral condyles. Consequently, abnormal wear of the articular cartilage is minimized, reducing the potential for arthritis. The owners agreed to the surgery and scheduled it for 4 weeks later. In the meantime, the owners were instructed to keep Hank quiet. He was restricted to leash walks only, with no running or jumping.

A month later, Hank returned for his osteotomy. On the day of surgery, he appeared healthy, aside from his persistent right rear lameness. All of his blood work was normal, permitting him to undergo the procedure. The surgery went very well, with no complications. After 24 hours postoperatively, Hank was discharged on an oral pain medication. His activity was restricted to leash walks on a 6-foot leash for urination and defecation only for 2 weeks. After his 2-week postoperative recheck, the surgical site was stable and healing well. The owners were now permitted

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to take Hank on short to moderate leash walks, no greater than one mile, using a six-foot leash. Hank loves swimming, so the surgeon also approved and even recommended swimming, provided Hank was not permitted to run and jump into the water. After Hank's 1-month postoperative recheck, the surgeon approved longer leash walks, with periods of "gentle" running/trotting. Hank was still restricted from full-on running and jumping. Swimming was again encouraged. Because his recovery was going so well, the surgeon anticipated that Hank would be able to gradually increase his activity, resuming all normal activities by 3 to 4 months postop. However, the surgeon stated that Hank may be anatomically predisposed to future problems. He has the potential to rupture the cranial cruciate ligament in his left stifle.

Case Study

Bovine Musculoskeletal Challenge

Set a timer for 15 minutes. Looking at Fig. 4.30, respond to each of the numbered corresponding questions by providing the scientific or common name as requested. Remember, be specific. Try to respond to as many as possible in the allotted time. Challenge yourself further by reducing time allowed and repeating the challenge.

1. Scientific name of joints

   _____

2. Scientific name of joints

   _____

3. Common name of bone

   _____

4. Scientific name of joint

   _____

5. Scientific name of bone

   _____

6. Scientific name of bone

   _____

7. Scientific name of joint

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_____

8. Scientific name of bone

_____

9. Scientific name of joint

_____

10. Scientific name of boney protuberance

_____

11. Scientific name of boney protuberance

_____

12. Scientific name of bone

_____

13. Scientific name of boney protuberance

_____

14. Scientific name of bones

_____

15. Name of bone

_____

16. Scientific name of junction

_____

17. Scientific name of process

_____

18. Scientific name of boney protuberance

_____

19. Common name of joint

_____

20. Scientific name of bones

_____

21. Scientific name of bones

_____

22. Scientific name of joints

_____

23. Common name of joints

_____

24. Scientific name of bone

_____

25. Scientific name of bone

_____

26. Scientific name

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27. Scientific name of joint

_____

28. Scientific name of bones

_____

29. Scientific name of band of connective tissue

_____

30. Scientific name of bone

_____

31. Scientific name of joint

_____

32. Scientific name of bone

_____

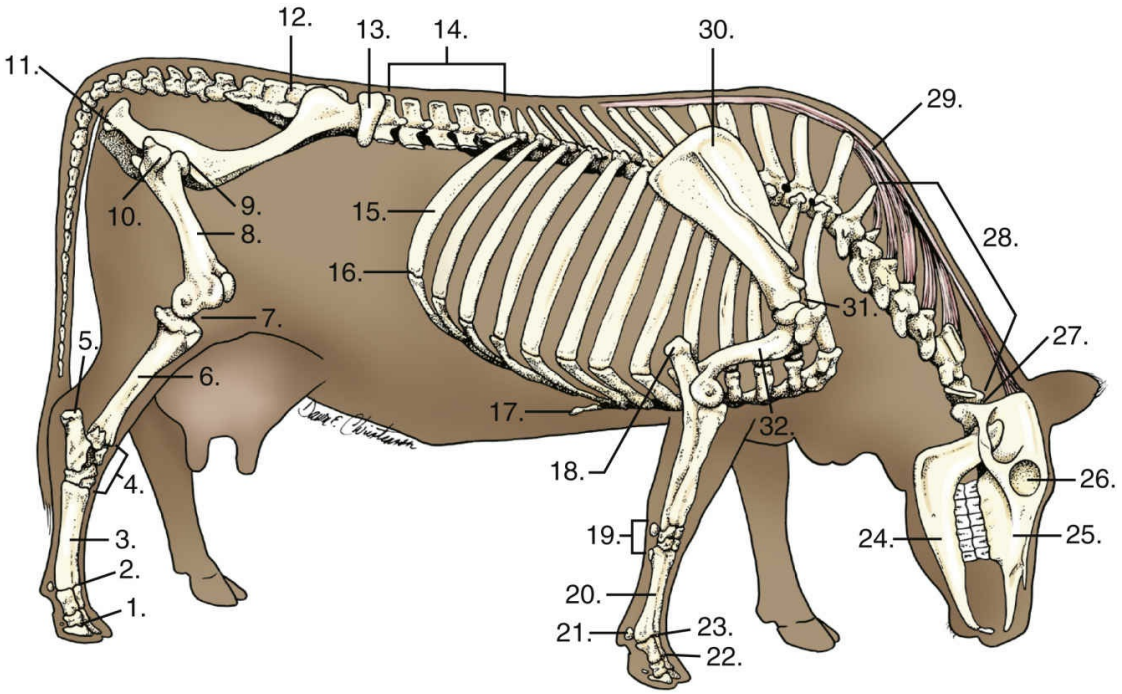

FIG. 4.30 Bovine musculoskeletal challenge.

Case Study

Canine Musculoskeletal Challenge

Set a timer for 15 minutes. Looking at Fig. 4.31, respond to each of

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the numbered corresponding questions by providing the ***scientific name*** of the bone, joint, protuberance, or muscle. Remember, be specific. Try to respond to as many as possible in the allotted time. Challenge yourself further by reducing time allowed and repeating the challenge.

1. Bone _____
2. Bone _____
3. Bones
   _____
4. Bones
   _____
5. Bones
   _____
6. Bone _____
7. Bones
   _____
8. Bone _____
9. Bones
   _____
10. Junction
    _____
11. Point/process
    _____
12. Bony structure
    _____
13. Bone
    _____
14. Joint _____
15. Bone
    _____
16. Joint _____
17. Bone
    _____
18. Bone
    _____
19. Joint _____
20. Bones

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_____

21. Joints

_____

22. Joints

_____

23. Bone

_____

24. Joint _____

25. Bone

_____

26. Protuberance

_____

27. Joint _____

28. Bone

_____

29. Bone

_____

30. Bone

_____

31. Bones

_____

32. Bones

_____

33. Scientifically name the IM injection site found in the region of 5. _____

34. Scientifically name the IM injection site found between landmarks 24 and 27, caudal to 25.

_____

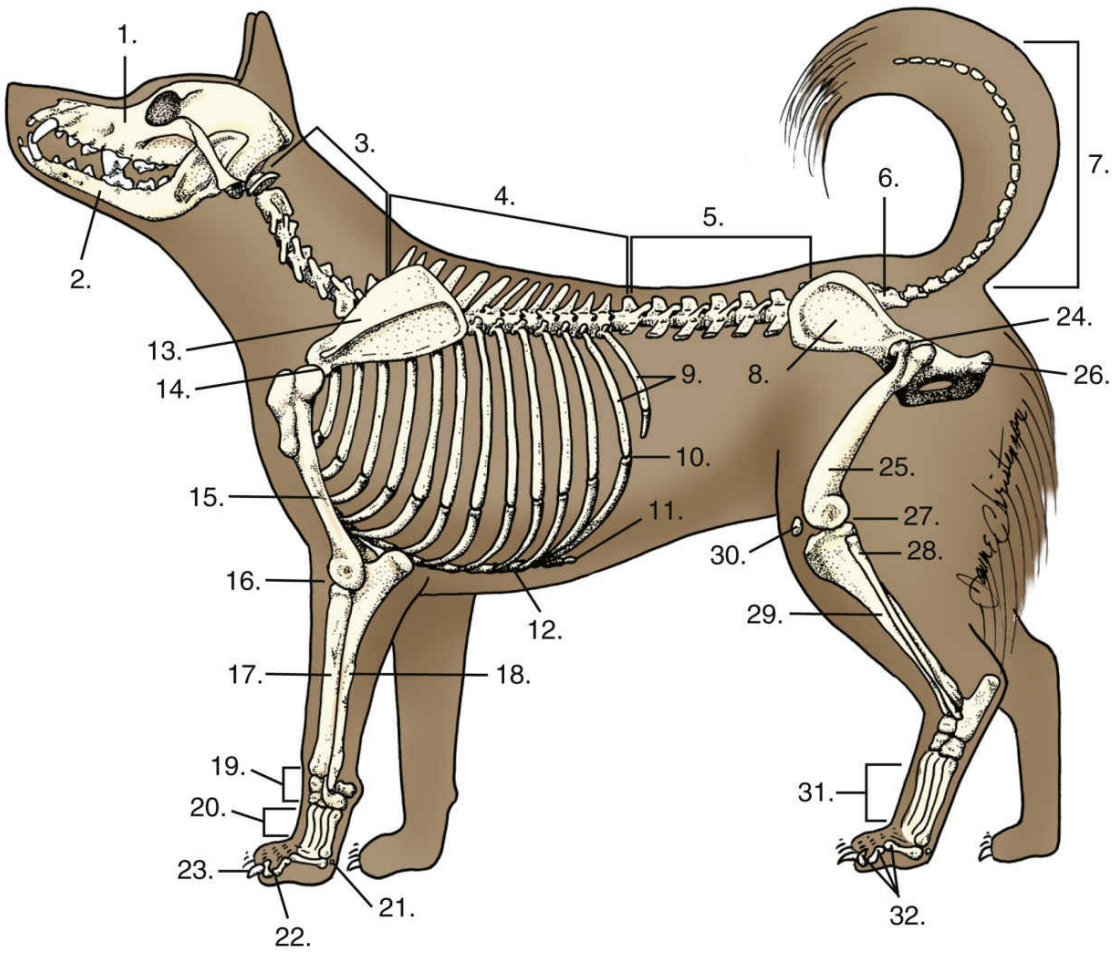

FIG. 4.31 Canine musculoskeletal challenge.

Case Study

Equine Musculoskeletal Challenge

Set a timer for 15 minutes. Looking at Fig. 4.32, respond to each of the numbered corresponding questions by providing the scientific or common name as requested. Remember, be specific. Try to respond to as many as possible in the allotted time. Challenge yourself further by reducing time allowed and repeating the challenge.

1. Scientific name of connective tissue band

   _____

2. Scientific name of bones

   _____

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3. Scientific name of bones
_____
4. Scientific name of bones
_____
5. Scientific name of bone
_____
6. Scientific name of bone
_____
7. Common name of joint
_____
8. Scientific name of bone
_____
9. Common name of joint
_____
10. Scientific name of bone
_____
11. Common name of joint
_____
12. Scientific name of bone
_____
13. Scientific name of joint
_____
14. Scientific name of bone
_____
15. Common name of bone
_____
16. Common name of joint
_____
17. Scientific name of bone
_____
18. Scientific name of bone
_____
19. Scientific name of joint
_____
20. Scientific name of bones
_____
21. Scientific name of protuberance
_____

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22. Common name of joint
_____
23. Scientific name of protuberance
_____
24. Scientific name of protuberance
_____
25. Scientific name of bone
_____
26. Common name of joint
_____
27. Scientific name of bone
_____
28. Common name of bone
_____
29. Common name of joint
_____
30. Common name of joint
_____
31. Scientific name of joint
_____
32. Common name of bone
_____
33. Scientific name of bone
_____
34. Common name of bone
_____
35. IM injection site found between landmarks 1, 2, and 6
_____
36. IM injection site found between landmarks 5, 21, 23, and 24
_____
37. IM injection site found between landmarks 23 and 26, caudal to 25 _____
38. IM injection site found between landmarks 7 and 9, caudal to 8 ____

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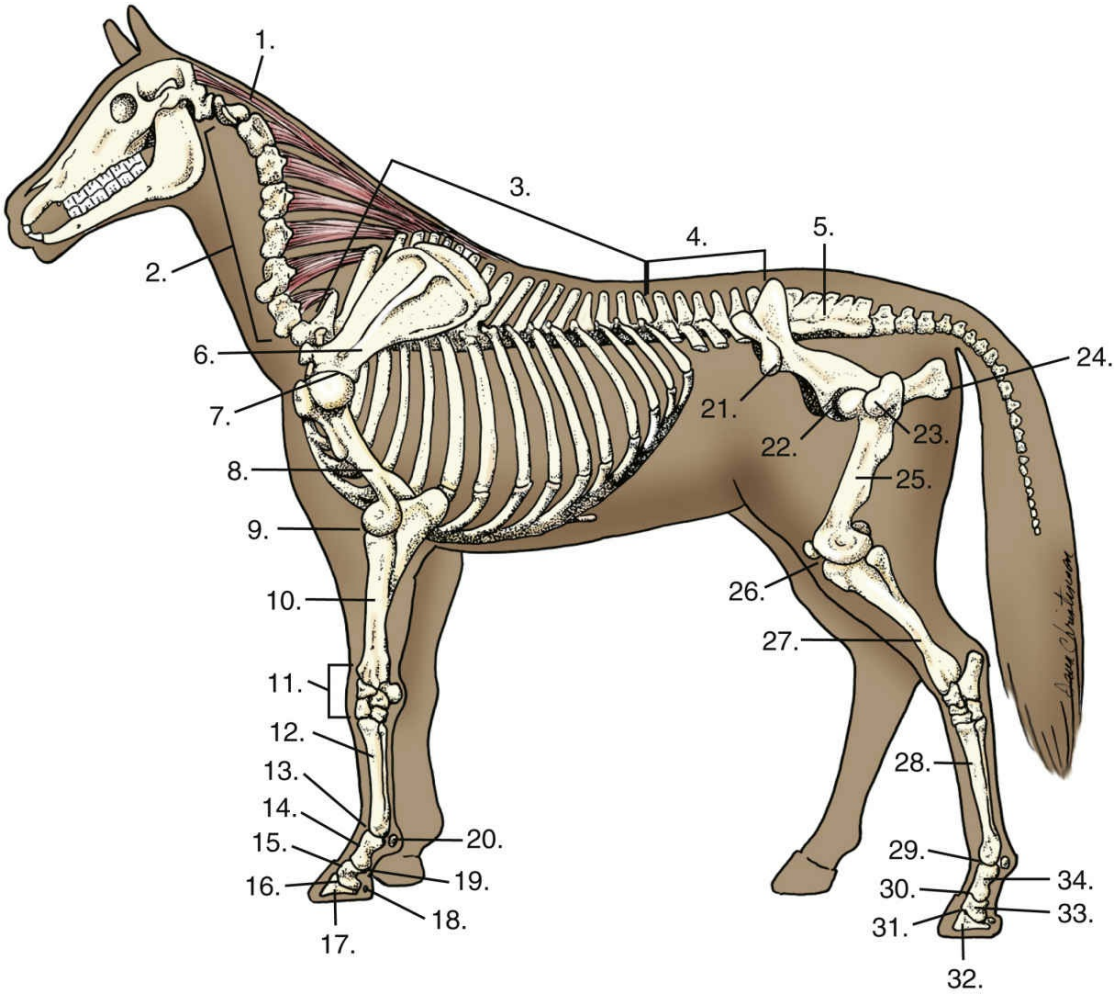

FIG. 4.32 Equine musculoskeletal challenge.

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Case Study Questions

1. When Hank was first presented, the veterinarian performed a _____ examination, to evaluate Hank's muscles, joints, and bones.
2. The examination of Hank's rear legs was performed in a _____ (both sides) comparative manner.
3. The scientific name for the stifle is the _____ joint.
4. Hank's owners thought that he had sprained his right hock or _____.
5. The veterinarian determined that Hank tore his right cranial _____ _____ (2 words). This is a tough band of connective tissue that "crosses" the stifle.
6. Radiographically, Hank's stifle showed no signs of _____ (i.e., inflammation of the bone and joint).
7. Hank's surgery was a tibial plateau leveling _____, meaning the surgeon would cut into the tibia.
8. The surgical procedure is designed to reduce cranial thrust of the knuckles of the femur, scientifically called the femoral _____.
9. By reducing thrust, the _____ (i.e., joint) cartilage would be protected from abnormal wear.
10. By minimizing or preventing abnormal wear, Hank's risk of developing _____ or inflammation of the joint is greatly reduced.

The Answer Key to these case study questions may be found in Appendix B.

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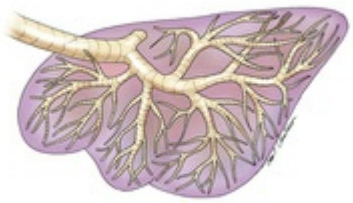

Applied Cardiovascular and Respiratory Terminology

Cardiovascular System,
- Heart,
  - Cardiac Layers and Pleura,
  - Chambers,
  - Great Vessels,
  - Valves,
  - Cardiac Conduction System,
- Vessels,
  - Arteries,
  - Veins,
  - Capillaries,
- Blood Flow,
- Blood Pressure,

Respiratory Tract,
- Nose and Nasal Passages,
- Sinuses,

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- Upper Airway,
  - Larynx,
  - Trachea,
- Lower Airways,
  - Bronchi,
  - Bronchioles,
  - Alveoli,
- Ins and Outs of Breathing,
  - Respiratory Volumes,

Integration in Health and Disease,

- Cardiomyopathy,
  - Echocardiography,
  - Dilatory Cardiomyopathy,
  - Hypertrophic Cardiomyopathy,
  - Saddle Thrombus,
- Heart Failure,
  - Pulmonary Edema,
  - Pleural Effusion,
  - Ascites,
- Heartworm Disease,
  - Pulmonary Embolus,
- Infectious Respiratory Diseases,
  - Pneumonia,
- Heat Stroke,

Case Study,

GOALS AND OBJECTIVES

By the conclusion of this chapter, the student will be able to:

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1. Recognize common root words, prefixes, and suffixes related to the heart, vessels, and respiratory system.
2. Divide simple and compound words into their respective parts.
3. Recognize, correctly pronounce, and appropriately use common medical terms related to the cardiovascular and respiratory systems.
4. Demonstrate familiarity with common pulse points and blood collection sites in domestic animals.
5. Demonstrate familiarity with blood flow.
6. Demonstrate familiarity with breathing and gas exchange.
7. Demonstrate an understanding of the integral relationship of the cardiovascular and respiratory systems.
8. Demonstrate an understanding of chest, heart and vessel, and respiratory anatomy.
9. Demonstrate an understanding of the cardiac cycle, including heart sounds and electrical conduction.
10. Demonstrate an understanding of blood pressure in health and disease.
11. Demonstrate an understanding of the relationship between the cardiovascular system and edema formation.
12. Demonstrate an understanding of the cardiovascular and respiratory systems with regard to health, homeostasis, and common diseases.

Before we begin looking at specifics of the ***cardiovascular*** [*cardi(o)-* heart + *vascul(o)-* vessels + *-ar* pertaining to] and ***respiratory*** [*re-* again + *spira(o)-* breathing + *-tory* pertaining to] systems, I need to clarify something. Technically, these are two very separate body systems. However, they are also two of the most closely intertwined systems of the body. Each depends upon and augments the work of the other. That is why we so often speak of them in the same breath, so to speak, with terms such as ***cardiopulmonary*** [*cardi(o)-* heart + *pulmon(o)-* lungs + *-ary* pertaining to]. All-important *cardiopulmonary* teamwork places both the heart and the lungs close together in the ***thoracic*** [*thorac(o)-* chest + *-ic* pertaining to] ***cavity,*** carefully

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protected by the rib cage (Fig. 5.1). The ribs protect the *thoracic* ***viscera*** [L. *viscera* organs] and also provide leverage for breathing, as we'll discuss later. But that's not all there is. There is so much more to these systems than just the heart and the lungs. Yet trying to functionally separate these systems is like trying to separate all of the ingredients of a cake once it's baked. Impossible. As a former colleague of mine was so fond of saying, the only time we can truly separate body systems like these is on the ***necropsy*** [*necr(o)-* death + *-opsy* to view; cf. "*autopsy*" in people] table. So, I will do my dead-level best (pun intended) to separate them, providing information specific to each system AND also showing their remarkable integration. Because the heart begins functioning first (before birth), let's start with the *cardiovascular* system.

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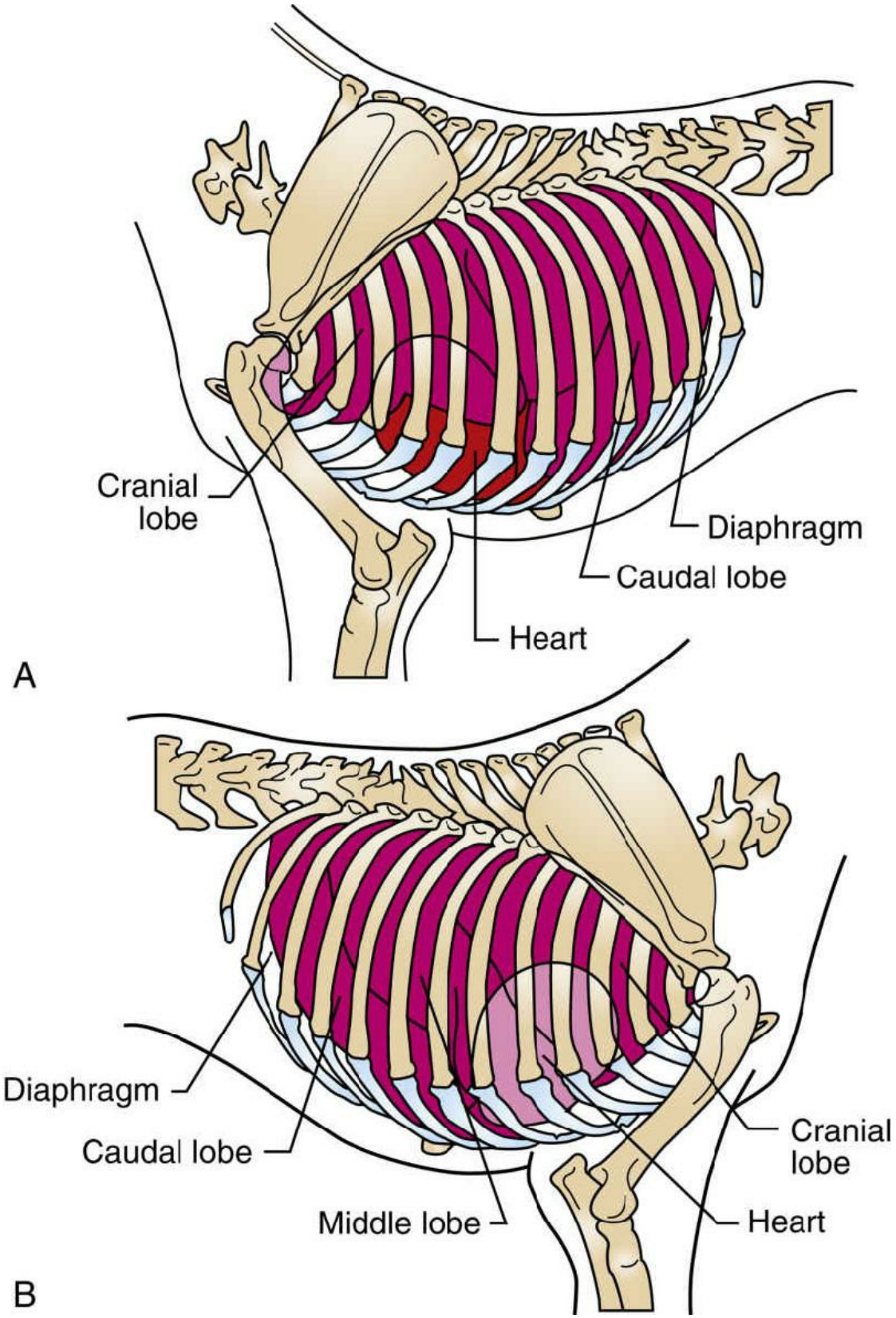

FIG. 5.1 Thoracic organs of the dog
From Colville T, Bassert JM. *Clinical Anatomy and Physiology for Veterinary Technicians*, 2nd ed, St Louis: Mosby; 2008.

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Cardiovascular System

The *cardiovascular* system is a simple concept of a pump and plumbing, right? Well, that is the concept, but how it functions is anything but simple. The heart is the pump—a two-sided pump—that simultaneously pumps blood to the lungs and body tissues, as shown in the Evolve animation *General Body Circulation.* As to the plumbing, it's the vasculature that provides the transit routes for the blood to and from the heart. ***Arteries*** carry blood away from the heart, and blood returns to the heart through a series of ***veins***. The primary goal, as you will learn, is the delivery of oxygen and removal of carbon dioxide. But I'm getting way ahead of myself. Let's take a close look at the all-important heart.

Heart

The heart is, for me, the most remarkable organ of the body. Think about it. It never rests. It can't rest because if it stops pumping, we die. Under normal circumstances, it begins pumping blood in the embryo long before birth and stops only once, when the individual dies. And there are usually many years between birth and death. That's amazing endurance! And it's the unique structure of ***cardiac*** [*cardi(o)-* heart + *-ac* pertaining to] muscle, as you read inChapter 4, that provides such amazing strength and endurance.

Myocardium [*my(o)-* muscle + *cardi(o)-* heart + *-um* the] is striated (striped) muscle tissue (Fig. 5.2), similar to skeletal muscle. But there is one unique feature that sets *myocardium* apart from its striated cousin. That feature is something called ***intercalated*** [*inter-* between + *calat(o)-* to call + *-ed* to be, being; i.e., to be "called," inserted between] ***discs***. These unique structures don't simply give ***myocardial*** tissue unusual stripes. *Intercalated discs* are the roughest, toughest connections found between muscle cells. There is absolutely nothing else like them anywhere in the body. And it's these *intercalated discs* that give *cardiac* muscle outstanding strength. They also provide seamless delivery of nerve impulses, from one *myocardial* cell to another. This makes *myocardial* contraction extremely well-coordinated. And coordination is a necessity for

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efficient pumping of blood from the heart.

Cardiac Layers and Pleura

Of course, there's more to the heart than just muscle, right? As in most organs of the body, there are multiple tissue layers (Fig. 5.3), each with its own purpose. For example, the interior of all the *cardiac* chambers is lined with ***endocardium*** [*endo-* within + *cardi(o)-* heart + *-um* the]. *Endocardium*, like the ***endothelial*** lining of the interior of all blood vessels, is a thin, single layer of ***endothelium*** (Fig. 5.4). Notice in the diagram that the cellular lining is labeled as *"epithelium/endothelium."* You see, if these cells are lining the interior of a completely closed system (like the heart and blood vessels), it is called *endothelium* because the prefix *endo-* means "within" or "inside." *Epithelium* lines other surfaces that may be exposed to the outside environment, because the prefix *epi-* means "on" or "upon." We'll talk about simple epithelium lining the walls of the ***alveoli*** [from L. *alveus* hollow; i.e., the air sacs in the lungs; singular alveolus; plural alveoli]. Obviously, that ***alveolar*** *[alveol(o)-* alveolus + *-ar* pertaining to] epithelium is exposed to outside air, during breathing. But whether it's epithelium or endothelium, it's pretty much the same tissue, just in a different location.

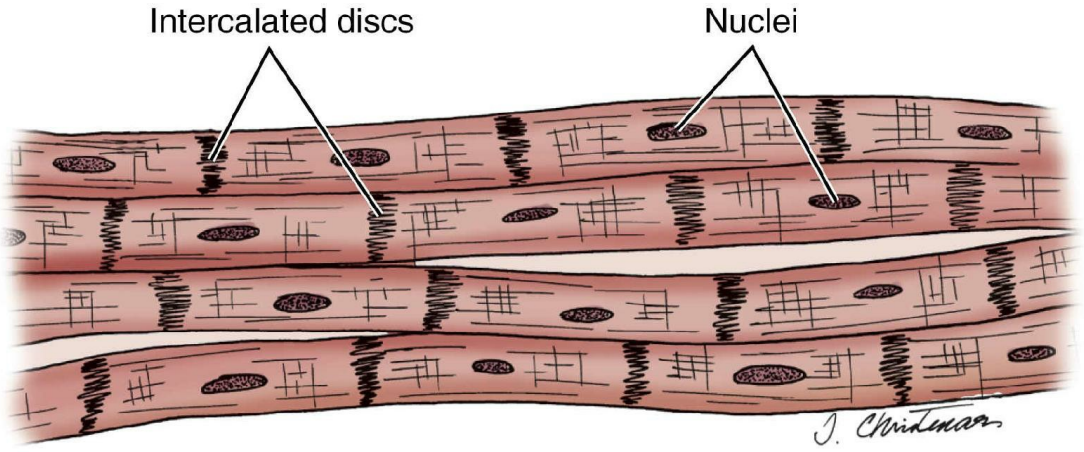

FIG. 5.2 Myocardium.

Anyway, the *endocardium* covers the interior of the *cardiac* chamber walls and valve leaflets. *Endocardium* (and *endothelium*) provides a smooth, slippery surface—important to prevent

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activation of the clotting cascade. You may recall fromChapter 3 that exposure of blood to the ***collagen*** [Gr. *kolla* glue + *gen(o)-* produce, producer] of underlying connective tissues activates platelets and clotting. So the margins of *endothelial* cells overlap to prevent exposure of *collagen*. Plus, the endothelium itself has ***anticoagulant*** [*anti-* against + *coagul(o)-* clotting + *-ant* pertaining to] properties. Obviously, this is important within the heart and all of the ***vasculature*** [*vascul(o)-* vessels + *-ture* the].

On the flipside of the *myocardium* on the heart's exterior is a thin tissue layer of ***epicardium*** [*epi-* on, upon + *cardi(o)-* heart + *-um* the]. This too has a connective tissue base covered in simple ***cuboidal*** [*cuboid* resembling a cube + *-al* pertaining to] epithelium (Fig. 5.24). The purpose of the *cuboidal epithelial* cells is to produce a thin, ***proteinaceous*** [*protein* + *-ceous* pertaining to] coating of lubricating liquid that prevents friction between the ***epicardial*** [*epi-* upon + *cardi(o)-* heart + *-al* pertaining to] surface and the surrounding ***pericardial*** [*peri-* around + *cardi(o)-* heart + *-al* pertaining to] ***sac***. Now, the *pericardial sac* is named as such because of its location. But the *pericardium* is actually ***visceral*** [*viscer(o)-* organ + *-al* pertaining to] ***pleural*** [Gr. *pleura* rib, side + *-al* pertaining to] connective tissue. Wait, what the heck is ***pleura***?

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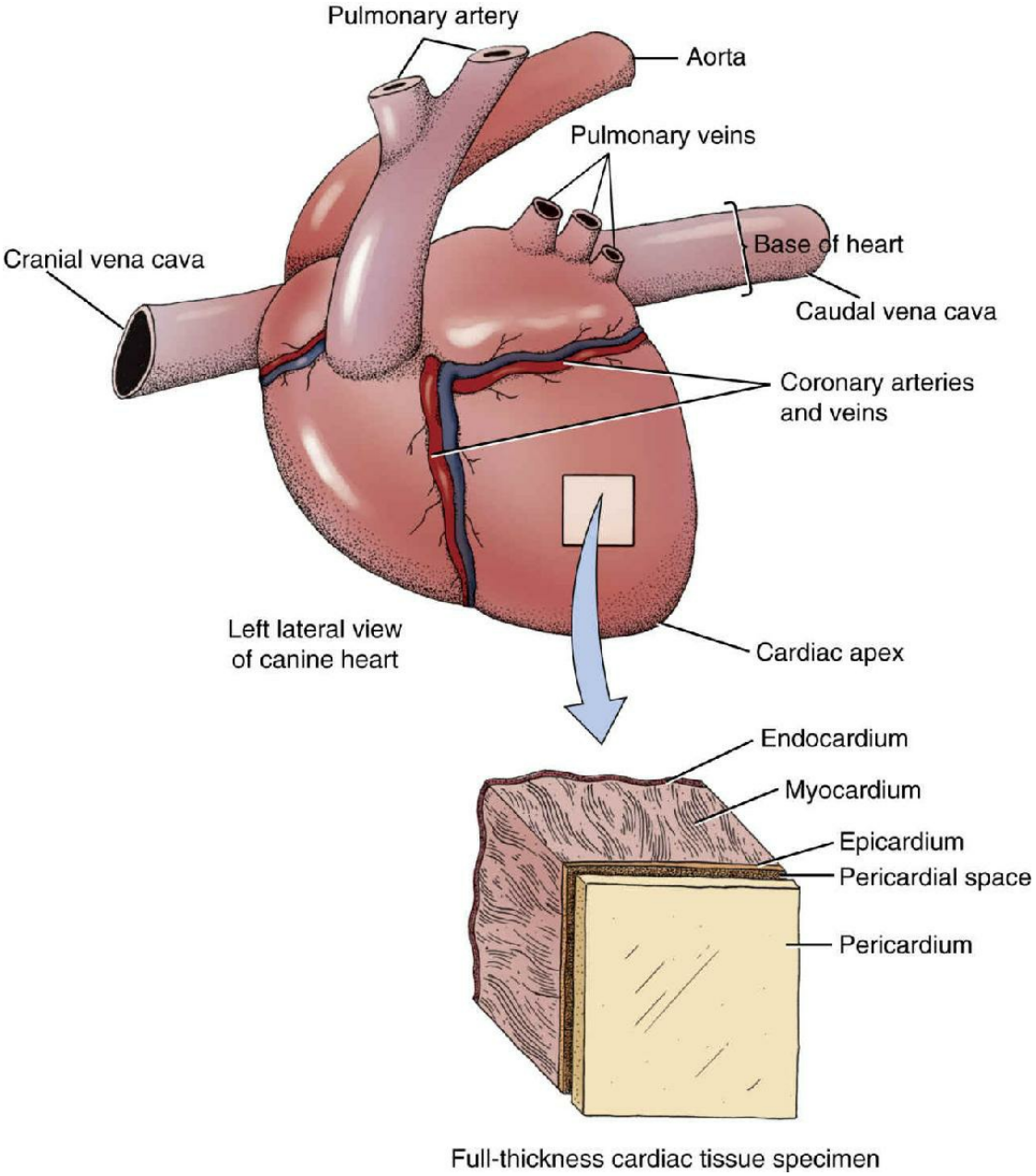

FIG. 5.3 The heart and cardiac tissue layers.

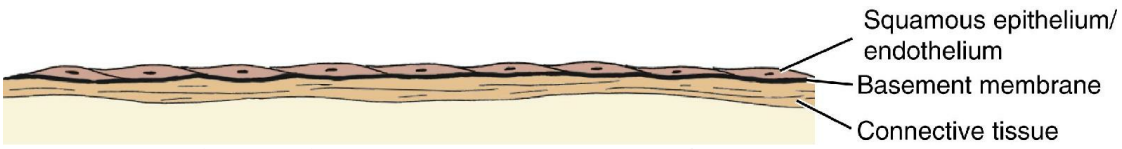

FIG. 5.4 Simple squamous epithelium/endothelium.

The ***pleura*** is a continuous sheet of connective tissue that lines the whole *thoracic* cavity and thoracic viscera. We simply name various portions of that *pleural* sheet based on its location. So, the portion

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that's "around the heart" is the *pericardium* or *pericardial sac*. The portion that lines the chest wall itself, including the diaphragm, is the ***parietal*** [*pariet(o)-* wall + *-al* pertaining to] ***pleura***. The portion covering the organs (lungs, thymus, heart, etc.) is ***visceral*** [*viscer(o)-* organ + *-al* pertaining to] ***pleura***. And that is why the *pericardial sac* is technically *visceral pleura*, because the heart is an organ. But if the pleura is one continuous sheet, how is the *visceral* and *parietal pleura* connected? Glad you asked! The ***mediastinum*** [L. *mediastinum* the middle partition] is that portion of the *pleural* sheet that extends through the midline of the chest, dividing the ***thorax*** (chest) in equal halves. It is connected at the sternum ventrally and the vertebrae dorsally. There are numerous ***mediastinal*** [*mediastin(o)-* mediastinum + *-al* pertaining to] ***lymph nodes*** throughout this sheet of connective tissue. By the way, pleural epithelium secretes a thin coating of lubricating liquid that does two important things. First, the fluid allows freedom of movement of the heart, lungs, and *thoracic* wall, allowing them to slide over one another without friction. Second, it creates surface tension between the *pleural* surfaces of the lungs and chest wall. We'll talk about the importance of that surface tension when we discuss breathing later on. Anyway, a band of *mediastinum* along the ventral border of the chest lies between the sternum and the heart, near the ***cardiac apex*** (see Fig. 5.3). The dorsal band of mediastinum lies between the base of the heart and the thoracic vertebrae. And everything that lies in between is enveloped in pleural tissue.

Okay, enough about layers. Let's get down to the nitty-gritty about this amazing pump.

Chambers

Imagine slicing through the heart, base to apex, so that we can see the interior. That's what we're looking at in Fig. 5.5. Before you panic looking at all of those pieces and parts, let me orient you. The ***cardiac base*** is at the top of the diagram, where all of the great vessels are sticking out. And the ***cardiac apex*** is at the bottom of the diagram. This is the way the heart lies in the thoracic or pleural cavity. That means the *cardiac base* is dorsal and the *apex* is ventral in the chest.

You'll notice that there are four chambers—two are dorsal and

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two are ventral. They are connected and organized to optimize pumping of blood. The two dorsal chambers are the ***atria*** (a'tre-uh [from Gr. *atrion* "hall"]). Have you ever been in a home or corporate building where you entered the building through the front door and stepped into a large room or "great hall"? Well, that's how these chambers got their name. In fact, a "great hall," that extends multiple stories in the building, is often referred to as the building's ***atrium*** (a'tre-um [L. *atrium* from Gr. *atrion* "hall"]). Of course, the cardiac atria (plural) don't receive visitors or guests. The cardiac *atria* are small muscular chambers that receive blood returning to the heart. And each *atrium* is very specific as to where the blood is received from. The ***right atrium*** receives ***venous*** [*ven(o)-* vein + *-ous* pertaining to] blood from most of the body. The ***left atrium*** receives *venous* blood from the lungs alone. The ***interatrial*** [*inter-* between + *atri(o)-* atrium + *-al* pertaining to] ***septum*** [L. *septum* "a wall"] creates separation of the atria. (Sorry, you can't see it in Fig. 5.5 because it's obscured by those big vessels. But the *septum* is there. Trust me.) Each atrium squeezes the received blood into their corresponding ventral chambers of the heart.

The ventral chambers of the heart are the ***ventricles*** [from L. *ventriculus,* dim. of *venter* "belly"; i.e., "little belly"]. Notice how much more muscular the *ventricles* are, compared to the atria. They need to be because the ventricles are responsible for pumping blood out of the heart. And like the atria, each ventricle is very specific regarding the destination of the blood it pumps out. The ***right ventricle*** receives blood from the right atrium and then pumps the blood to the lungs. That's why it's less muscular. It doesn't have to pump blood very far at all. The ***left ventricle***, on the other hand, receives blood from the left atrium and then has to pump the blood to the rest of the body. That takes tremendous force. So, the left ventricle is very muscular. In fact, it's the massive *myocardium* of the left ventricle that actually forms the *cardiac apex*. Obviously, with such different pumping destinations, the ventricles need good separation. So, the ***interventricular*** [*inter-* between + *ventricul(o)-* ventricles + *-ar* pertaining to] ***septum*** [L. *septum* "a wall"] provides that separation.

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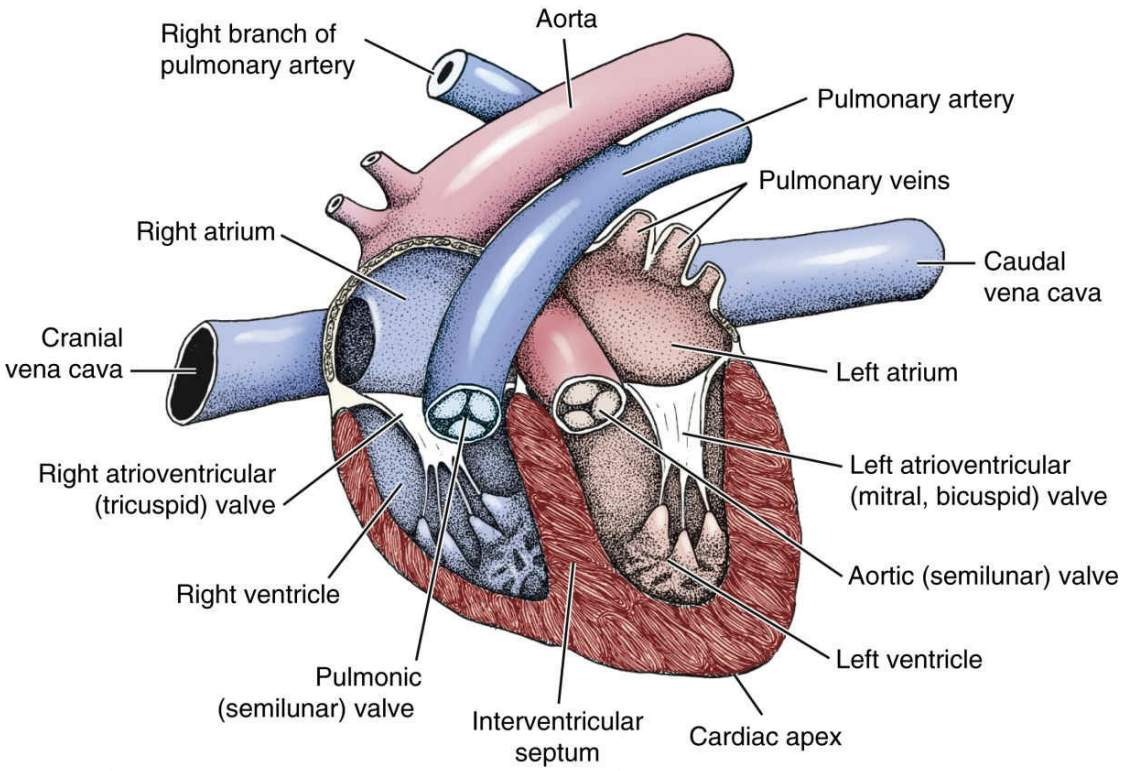

FIG. 5.5 Cardiac chambers and valves (schematic sagittal view of canine heart).

Great Vessels

Of course, we need plumbing to get the blood to and from the heart, right? So, we have vessels—***great vessels***—serving that purpose. These are "great vessels" because they are directly connected to one of the greatest, important, and amazing organs of the body—the heart. We'll talk about vessels in general a little later. But in general, remember that ***veins*** return blood to the heart and ***arteries*** carry blood away from the heart.

With that in mind, we have very specific veins returning blood to the atria. The ***vena*** [L. *vena* "vein"] ***cava*** [L. *cava* "hollow"] is the largest vein of the body. Nearly all of the venous blood returning to the heart passes through this huge vein. Venous blood from the head, neck, and forelimbs passes through the ***cranial vena cava***. Venous blood from caudal portions of the body (abdomen, rear limbs, etc.) passes through the ***caudal vena cava***. All of the blood from the vena cava (both cranial and caudal) pours into the right atrium. On the opposite side of the heart, we have the left atrium. We said that the left atrium receives venous blood from the lungs.

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So logically, the ***pulmonary*** [*pulmon(o)-* lung + *-ary* pertaining to] ***veins*** carry venous blood from the lungs to the left atrium.

Arterial [*arter(o)-*, *arteri(o)-* artery + *-al* pertaining to] great vessels include the ***pulmonary artery*** that carries blood from the heart to the lungs. Don't you love it when they name things logically? Notice how the *pulmonary artery* quickly subdivides into right and left branches. This facilitates carrying blood to right and left lung lobes, respectively. The ***aorta*** [Gr. *aorte* "to lift"] is the largest artery of the body. It needs to be large. This artery "lifts" blood from the heart to the rest of the body.

But wait, those vessels only carry blood to and from the lungs and the rest of the body. What about the heart? Doesn't it need its own vessels? It sure does. And that's what the ***coronary*** [from L. *corona* "crown" + *-ary* pertaining to] arteries and veins are for. As you can see in Fig. 5.3, some of the coronary vessels encircle the heart, kind of like a crown encircles the head of a queen or king. That's how they got their name: *coronary*.

Valves

Think about your home. Whether it's an apartment, a house, or a dorm room, it has doors, right? And those doors provide access to the various rooms when they open. And when closed, the doors provide needed seclusion and privacy. Most doors are hinged to swing open only one way. Valves in the heart are similar. When open, valves allow access to wherever the valve opens. When closed, the valves seclude the area behind the door. And because valves swing open only one way, when they close they provide a "weather-tight" seal. Of course, in this case, the seal prevents backwash of blood. Fortunately, most cardiac valves are named logically, based on their location or where the blood is being sent.

So, ***atrioventricular*** [*atri(o)-* atrium + *ventricul(o)-* ventricle + *-ar* pertaining to] ***valves*** are found, wait for it...between the atria and the ventricles. The ***right atrioventricular*** (***AV***) ***valve*** is the "door" between the right atrium and the right ventricle. The *right AV valve* is also called the ***tricuspid*** [*tri-* three + L. *cuspis* point; i.e., three cusps or points] ***valve***, because it has three valve leaflets. Those leaflets swing open into the right ventricle. The ***left AV valve*** is also called the ***bicuspid*** [*bi-* two + L. *cuspis* point; i.e., two cusps or

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points] ***valve***, because it has two valve leaflets. And because those leaflets are shaped like a bishop's hat, which is called a miter [L. *miter* headband; Bishop's hat], the *left AV valve* is also referred to as the ***mitral valve***. The leaflets of the left AV valve swing open into the left ventricle.

Okay, so the AV valves separate the atria from the ventricles when closed. But we also need "doors" between each ventricle and their respective great arteries (i.e., pulmonary artery and aorta). Fortunately, we name these valves based on the artery each opens into. So, the ***pulmonic*** [*pulmon(o)-* lung + *-ic* pertaining to] ***valve*** opens into the pulmonary artery. Likewise, the ***aortic*** [*aort(o)-* aorta + *-ic* pertaining to] ***valve*** opens into the aorta. Because the ***valvular*** [*valvul(o)-* valve + *-ar* pertaining to] leaflets of the pulmonic and aortic valves are shaped like partial phases of the moon, they are also referred to as ***semilunar*** [*semi-* partial + *luna* moon + *-ar* pertaining to] ***valves***.

Heart sounds

When we close a door, it makes noise, right? In fact, the harder we close the door, the louder the noise. Slam it closed and it makes a big bang. So, it stands to reason that when the cardiac valves close they too make noise.

Normal heart sounds

The heart pumps blood in a very orderly fashion. In fact, as noted in the Evolve animation entitled the *Cardiac Cycle*, we could really consider the heart as two pumps in one. The right side of the heart is one pump and the left side of the heart is the other pump. Both sides are synchronized in their activity. So, the "doors" slam shut in a very synchronous manner. And this synchronous slamming shut of the valves creates the normal heart sounds we hear as "lub dub" or "lub dup."

Which valves are associated with which sound? Well, the ***first heart sound*** (lub) is created when the *AV valves* close. They are slammed shut hard because of increasing pressure on the ***ventricular*** [*ventricul(o)-* ventricle + *-ar* pertaining to] side of the valves. Yes, they close when the ventricles pump blood out of the heart. So, the first heart sound marks the beginning of the ***systolic***

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[from L. *systole* contraction + -ic pertaining to] ***phase*** of the ***cardiac cycle***. The ***second heart sound*** (dub or dup) is created by the simultaneous closing of the *pulmonic* and *aortic valves*. In general, arteries are under higher pressure than veins. That's why arterial walls are so much thicker. So, the pressure in the aorta and pulmonary artery help close those valves at the beginning of the ***diastolic*** [from L. *diastole* expansion + *-ic* pertaining to; i.e., resting phase] ***phase*** of the *cardiac cycle.*

Can a heart ever have asynchronous closure of the valves? Yes, that is possible. That would create what we call ***split heart sounds*** —sort of a stuttering, like lub-d-dup. But this doesn't occur as often as ***murmurs***.

Abnormal heart sounds—murmurs

Murmurs [L. *murmur* "roar," "grumble"] are sounds created by turbulent blood flow and forceful movement of blood through a tiny space. Blood is intended to flow in one direction. Valves are intended to open in one direction, allowing free, unobstructed flow of blood through the wide-open "doorway." Only valvular closing should create a sound. But sometimes the doorways themselves are narrow. Both ***pulmonic stenosis*** [*sten(o)-* narrow, narrowing + *-sis* condition of] and ***aortic stenosis*** create a "roar" or whooshing sound, when there should be silence. That whooshing noise is a *murmur*. So, in the case of aortic or pulmonic stenosis, we'll hear the *murmur* when the blood is being ejected (pumped) through those valvular openings, right? The ventricles are contracting at this point in the cardiac cycle, forcing the blood into the pulmonary artery and aorta respectively. The sound of the murmur will be heard between the first and second heart sound. So, instead of the normal "lub-dub," we hear "lub-WHOOSH-dub."

We also classify this as a ***systolic murmur***, because it occurs during the *systolic phase* of the cardiac cycle (i.e., when the ventricles are contracting). And because the blood is under so much force, the whooshing sound (especially with aortic stenosis) is pretty loud and harsh. That harshness is typical for ***ejection murmurs***. In fact, because the sound is generated throughout ***systole*** [L. *systole* contraction], we often classify these as ***holosystolic*** [*hol(o)-* entire + *systol(o)-* contraction + *-ic* pertaining to] murmurs. Valvular stenosis

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like this is often a ***congenital*** [*con-* with + L. *genitus* to bring forth, birth + *-al* pertaining to; i.e., born with] abnormality. So we often hear murmurs from *pulmonic stenosis* or *aortic stenosis* in young animals. That's why we should always listen to the heart of every animal. We cannot assume, just because an animal is young, that it is completely healthy.

More frequently, murmurs are caused by leaky valves. We said earlier that valves when closed should create a very tight seal. If they leak, they are insufficient in their function. That's why we often refer to leaky valves as ***valvular insufficiencies***. And because the blood floods backward through the leaky valve, we also refer to it as ***valvular regurgitation*** [*re-* back + *gurgit(o)-* flooding + *-tion* the act of]. Any valve can leak. The most common valvular insufficiency is ***mitral insufficiency***.

A *mitral insufficiency*, as shown and discussed in the Evolve animation entitled *Leaky Valve*, is created when the valve does not close properly. Without an effective seal, blood leaks or regurgitates backward from the left ventricle into the left atrium. That regurgitation creates a swishing sound between heart sounds: lub-swish-dub. Because of the timing of the abnormal swishing sound, mitral regurgitation is also classified as a *systolic murmur*. These murmurs don't tend to be *holosystolic*, like aortic stenosis. ***Mitral regurgitation*** also doesn't tend to create the harsh sound of ejection murmurs, like pulmonic and aortic stenosis. When the regurgitation is severe, these murmurs can get loud. But they are never as harsh as ejection murmurs. The same is true for ***tricuspid insufficiency*** (i.e., leakage of the right AV valve). It too creates a systolic murmur.

If we consider which valves are closed during which phase of the cardiac cycle, it's pretty easy to figure out which valvular insufficiencies will create ***diastolic murmurs***. That's right, both ***pulmonic*** and ***aortic valve insufficiency*** cause *diastolic murmurs*. Note that both mitral and aortic insufficiency are shown and discussed in the Evolve animation entitled *Mitral and Aortic Valve Regurgitation.* I strongly advise watching this animation multiple times. Not only does it explain and show these valvular insufficiencies well, but it also provides an excellent review of systemic and pulmonary circulation. Plus, it discusses some of the consequences associated with these particular valvular

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insufficiencies. We'll address those consequences later on, especially when we discuss heart failure.

Still other murmurs are created by ***septal*** [from L. *septum* wall + *-al* pertaining to] ***defects*** and other *congenital* abnormalities. Just imagine the turbulent blood flow created by a hole in either the atrial septum or the ventricular septum. That turbulence results in a murmur. And defects like these really mess up the efficiency of our pump. Fortunately, *septal* defects do not happen that frequently.

A much more common *congenital* defect is something called a ***patent*** [from L. *patens* open] ***ductus*** [L. *ductus* a duct] ***arteriosus*** [L. *arteriosus* an artery] (***PDA***). The *ductus arteriosus* is a vessel that connects the aorta to the pulmonary artery in the fetus. Obviously, the fetus has no use of its lungs yet; so, blood is detoured away from the lungs to the aorta. The *ductus arteriosus* should close at or shortly after birth. If it remains open (*patent*), with large amounts of blood flowing through both the pulmonary artery and aorta, tremendous turbulence of blood and vibration of the ductus wall are created. If you ever place your stethoscope on the chest of a youngster (usually a puppy) with a *PDA*, it will literally sound like a washing machine agitating ("whoom-vroom-whoom-vroom"). In fact, it sounds so much like this that we often call it a ***machinery murmur***. The sound is *holosystolic* and ***holodiastolic*** [*hol(o)-* entire + *diastole* expansion + *-ic* pertaining to; i.e., during the whole resting phase of the cardiac cycle]. It's loud too—so loud that you most likely can't even hear the normal "lub-dub." In fact, there is so much vibration created by a *PDA* that you can literally feel the vibration through the chest wall, without even trying. Fortunately, surgical correction of a *PDA* is relatively easy. Yes, it involves a ***thoracotomy*** [*thorac(o)-* chest + *-tomy* to cut] and that has its own risks. But the surgical correction of the PDA itself simply involves ***ligating*** [from L. *ligatio* to tie or bind] the ductus arteriosus with suture material or a ***vascular*** [*vascul(o)-* vessel + *-ar* pertaining to] clamp. Once the ductus arteriosis is closed with a ligature [L. *ligatio* to tie or bind + *-ture* a, the], there is no longer any turbulence—no more murmur! And because blood is being pumped efficiently and oxygenating optimally, these patients have improved (pink) mucous membrane and color and have much more energy (as a youngster should).

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Cardiac Conduction System

This magnificent pump needs a mechanism to regulate all of its coordinated activity. That regulating mechanism is the ***cardiac conduction system***. It's a system of electrical wiring, if you will. The components of the *cardiac conduction system* are shown in Fig. 5.6 and demonstrated in the Evolve animation entitled *Electrical Conduction System of the Heart.* Please note that the heart rate discussed in this animation is a human normal. There is a much wider range among domestic animals. But I'm getting ahead of myself. Let's begin this discussion by looking at each component of our "wiring."

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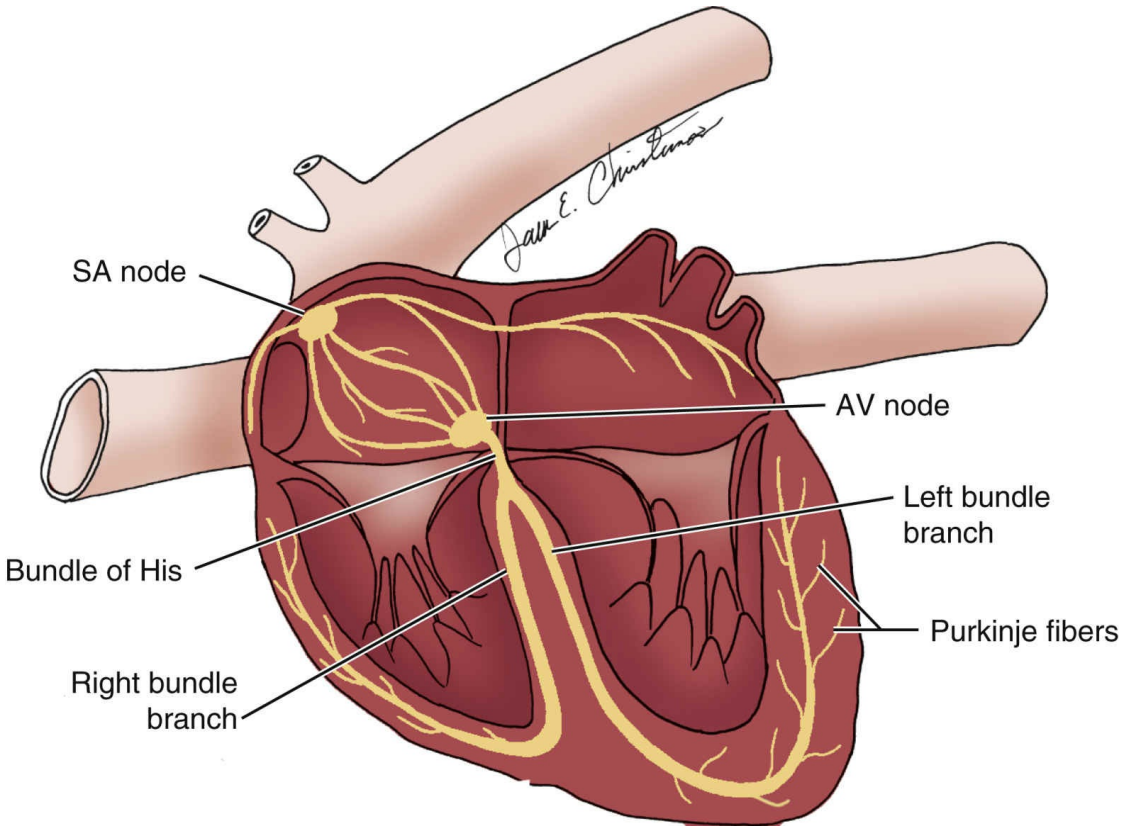

FIG. 5.6 Cardiac conduction system anatomy. *AV*, atrioventricular; *SA*, sinoatrial.

You can see in Fig. 5.6 that the ***sinoatrial*** [*sin(o)-* sinus, L. "a hollow" + *atri(o)-* atrium + *-al* pertaining to] (***SA***) ***node*** lies in the wall of the right atrium. The name "*sinoatrial*" is because of the node's close proximity to the "sinus" or "hollow" formed by the vena cava at its entrance into the right atrium. The ***SA node*** is the pacemaker of the heart. It controls the pace of pumping. I like to think of the SA node as a rechargeable battery that discharges its electrical current at a regular rate (zap—zap—zap). After discharging its electrical impulse, it quickly recharges. Technically, we call the discharging ***depolarization*** and the recharging ***repolarization***. Notice that attached to the SA node are "wires," actually specialized nerve fibers, that extend throughout the atria. These are important for coordinated muscular contraction of the atria, to squeeze the final volume of blood into the ventricles. That maximizes the volume of blood that will eventually be pumped out of the ventricles.

Ah, but we need a slight pause along this electrical pathway, to allow the atria to squeeze that last little drop of blood into the ventricles. That's where the ***atrioventricular*** [*atri(o)-* atrium +

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ventricul(o)- ventricle + *-ar* pertaining to] (***AV***) ***node*** comes into play. The ***AV node*** is positioned in the perfect location to pause the electrical impulse – right in the middle of the heart, near the *atrial septum* and the atrioventricular border. It's all in a name, right? The tiny pause created by the AV node is really important, because beyond this point things happen very rapidly.

The next segment of the conduction system is the ***bundle of His*** (pronounced "hiss"). The *bundle of His* provides a short electrical connection from the AV node in the atrium to the ***right and left bundle branches*** in the *ventricular septum*. We need *right and left bundle branches* because we need both the right and left ventricles to contract simultaneously. Electrical impulses pass very quickly through these large "wires" of the bundle branches. Those impulses travel to the ***Purkinje*** (pur-kin'je) ***fibers***, where the *ventricular myocardium* is stimulated to contract. And the contraction follows the *Purkinje fibers*, beginning near the cardiac apex and moving dorsally toward the base of the heart. This facilitates forcing blood from the "bottom" of the ventricles first, so that all of the blood is fully ejected from those chambers. By the way, in case you're wondering about those odd names, His and Purkinje: they are named after people. The *bundle of His* was named after the Swiss physician Wilhelm His, Jr. (1863–1934). The *Purkinje fibers* were named after the Czech physiologist Jan Evangelista Purkinje (1787–1869). Make important discoveries and those discoveries are named after you.

At this point in our discussion, I strongly recommend that you watch the Evolve animation *Conduction of Heart Impulses: Electrical Activity of the Heart.* It provides a good review of the anatomy and shows the coordinated muscle contraction very well—first through the atria and then through the ventricles. Notice how it actually shows the electrical movement across the atria. Before moving on, let's take a moment to review the cardiac cycle. Remember, blood is freely flowing through venous return into the heart, during the *diastolic phase* (resting phase) of the cardiac cycle. The SA node discharges, causing depolarization and contraction of the atria. This forces a greater volume of blood into the ventricles. The electrical impulse is paused briefly at the AV node to allow the atria to empty fully into the ventricles. As the impulse travels quickly,

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depolarizing through the bundle of His, left and right bundle branches, and Purkinje fibers, ventricular contraction (systole) is initiated. The AV valves slam shut ("lub"). Blood is forced from the ventricles through the pulmonary artery and aorta. This is when peripheral pulses can be felt. Once this systolic phase of the cycle is complete and blood has been emptied from the ventricles, the pulmonic and aortic valves slam shut ("dub"). Then there is a brief diastolic pause while everything repolarizes to be ready for the next firing of the SA node. The length of the diastolic pause depends on the species and the individual within that species. And that leads us to a brief discussion of heart rate.

Heart rate

Of course, the actual ***heart rate*** is regulated by the pacemaker—the SA node. But the SA node is not a free agent; its activity is controlled by the brain stem. The pace of the SA node's firing to set off the systolic phase of the cardiac cycle needs to be reasonable for the individual. If the rate is too fast, the diastolic filling phase will be cut short. Adequate filling is important for the ventricles to pump out adequate volumes of blood.

Everyone has a normal pace for their heart rate. As you heard in one of the animations, the average human heart rate (i.e., resting heart rate—when the person is inactive) is between 60 and 100 bpm (beats per minute). For an adult draft horse, like a Clydesdale, the rate is much slower, averaging around 30 bpm or less for a resting heart rate. Wow, that means diastole lasts roughly 2 seconds. Compare that to a cat, who may have a resting heart rate of 180 to 200 bpm. And that's a pretty "chill" cat. Still, there's not much time for diastole there! There's even less filling time for a mouse, with a heart rate that could easily exceed 500 bpm. But as we consider the normal resting heart rate and systole versus diastole, we need to consider both volume and distance for the blood being pumped.

Typically, the larger the animal, the slower the heart rate. Why? Because it takes longer for the blood to actually return to the heart; it has further to go. In a tiny mouse, it doesn't take long at all to get blood to and from its destinations. Size variation among dog breeds makes for tremendous variation in heart rates. A Great Dane could easily have a resting heart rate of 60 bpm. For a Chihuahua, that

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would be considered ***bradycardia*** [*brady-* slow + *cardi(o)-* heart + *-ia* state or condition of]. By the same token, a Chihuahua's resting heart rate, of perhaps 120 bpm, would be considered ***tachycardia*** [*tachy-* fast + *cardi(o)-* heart + *-ia* state or condition of] for a Great Dane, and most certainly for a Clydesdale. Everything is relative. So, as you consider normal resting heart rates of various animals, you need to take a number of things into consideration, including body size.

Of course, there are many things that may influence heart rate. We'll address many of these factors as we wade through the cardiopulmonary information throughout this chapter. Let's just touch on a few here. First, if we engage in active exercise, our muscles require more oxygen to sustain their activity, right? And you learned in the *General Body Circulation animation* and inChapter 3 that oxygen is transported by red blood cells. So, if the tissues need more oxygen for increased activity, how do we get it there? We speed up the delivery process. Move RBCs faster and we have greater delivery of oxygen to tissues. The brain stem responds to oxygen needs in the body and dictates to the SA node to pick up the pace, increasing the heart rate. The same is true for temperature regulation in the body. Core body temperature needs to remain close to the set-point in the brain. When thermal sensors perceive increased body heat, the brain stem will dictate to the SA node to increase heart rate. By increasing the heart rate, blood is taken quickly from the core to the periphery to dissipate heat. These are just a couple of simple examples, associated with everyday life, that can influence heart rate. We'll talk about others, in health and disease, as we move along.

Before we complete our discussion of the cardiac conduction pathway, let's take a look at how the pathway and myocardial activity relate to the *electrocardiogram* [*electr(o)-* electricity + + *cardi(o)-* heart + *-gram* a measurement or record] *(ECG*; sometimes referred to as EKG; the "K" is from the German translation: elektroKardiographie).

Electrocardiography

We've already said that there are electrical impulses traveling along the conduction pathway. There is both depolarization and

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repolarization of the pathway and the muscle tissue during the *cardiac cycle*. And those electrically charged events can be recorded through ***electrocardiography*** [*electr(o)-* electricity + *cardi(o)-* heart + *-graphy* recording of]. Let's see if we can correlate each step along the conduction pathway with the waves we see on an ***electrocardiogram*** (***ECG***). See Fig. 5.7 and the Evolve animation entitled *Normal Sinus Rhythm* to help with your understanding.

So, we know during the beginning of the cardiac cycle, the *SA node* fires and the *atria* rapidly depolarize and contract. This results in a positive deflection on the ECG called the ***P wave***. But we said that before the ventricles contract, we want a slight pause along the conduction system, right? This allows the atria to squeeze as much blood as possible into the ventricles. That pause occurs at the *AV node* and is seen on the ECG as the ***P-R segment***. Once we get past the AV node, depolarization progresses rapidly through the *bundle of His, left and right bundle branches, and Purkinje fibers*, resulting in depolarization and contraction of the *ventricles*. That's a lot of activity. So, it produces a huge positive deflection on the ECG called the ***QRS complex***. The SA node and the atria are actually repolarizing (recharging) during this time. But that activity is completely obscured by the huge QRS complex. With the *systolic* phase of the cardiac cycle complete, everything from the *bundle of His* through the *ventricular myocardium* repolarizes (recharges). This activity produces the ***T wave*** and marks the beginning of *diastole* (rest and recharge).

When looking at an ECG, we always look for these basic components: P wave, P-R segment, QRS complex, and T wave. Each heartbeat should, under normal circumstances, be accompanied by all of these waves on an ECG. Abnormal size, structure, and/or absence of some of these ECG components is what we refer to as an ***arrhythmia*** [*a-* without + *rhythm* + *-ia* state or condition of] or ***dysrhythmia*** [*dys-* bad + *rhythm* + *-ia* state or condition of]. Those words, in essence, mean the same thing and can be used interchangeably.

Common arrhythmias

As mentioned in the Evolve animation *Normal Sinus Rhythm,* there are things like atrial enlargement and damage to myocardium that

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can affect the appearance of wave forms on an ECG. In the case of the atrial enlargement mentioned, the P wave produced is wider (longer) because it's taking longer for the electrical wave to traverse the larger atrial wall. The same is true with ***cardiomegaly*** [*cardi(o)-* heart + *-megaly* enlargement of] involving the ventricles. (Note: the whole heart is enlarged with cardiomegaly. I am merely trying to focus our attention on the ventricular effects here.) With ventricular enlargement, depolarization takes longer so the QRS complex is wider on the ECG. How wide is too wide for these waves? For most of us, it's obvious when they're super wide, compared to the normal ECG you've been shown in Fig. 5.7 and the Evolve animation. Subtle differences in width and height will be left to a ***cardiologist*** [*cardi(o)-* heart + *log(o)-* study, knowledge + *-ist* a specialist of] to discern. It is not the purpose of this text or our discussion here to make you a budding *cardiologist*. My goal is to merely demystify *electrocardiography* and guide you toward recognition of normal versus abnormal. The first step is familiarity with normal. Even if you don't know the ***etiology*** [*eti(o)-* cause + *-logy* study of; i.e., the cause] of an *arrhythmia*, recognizing it as a departure from normal empowers you to draw it to someone's attention who has more experience and knowledge.

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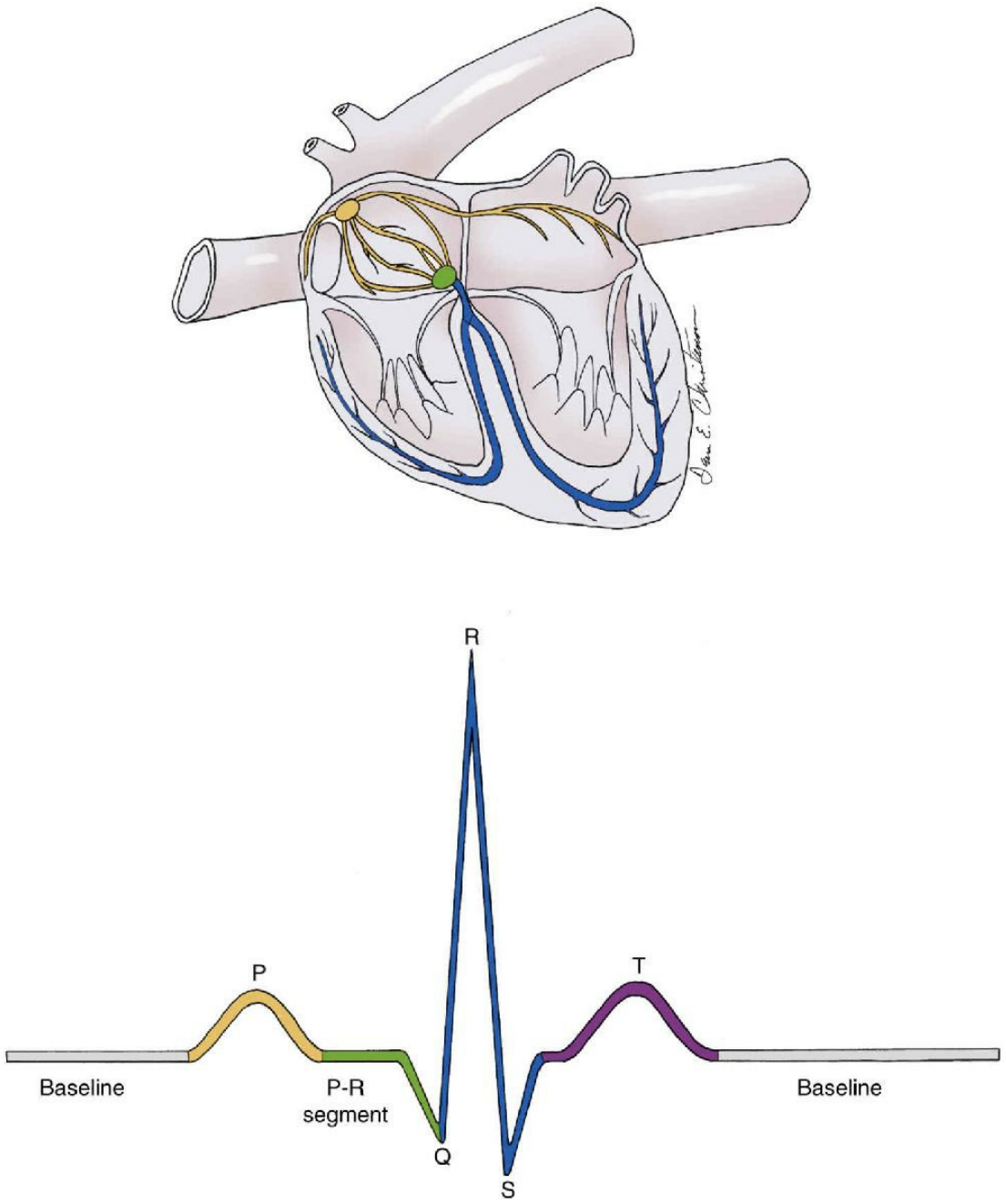

FIG. 5.7 Electrocardiogram.

Let's look at common *dysrhythmias* based on what you know about the cardiac conduction system. Abnormalities of the ***P wave*** point to abnormalities of the ***SA node and atria***. What if we don't see P waves on an ECG? Well, that would tell us that the SA node is not firing and the atria are not depolarizing. Is that a problem? Yes. Remember, the SA node is the pacemaker of the heart. If it doesn't fire, the heart rate and cardiac output will be negatively affected. The atria need to contract to force an optimal amount of blood into the ventricles. And a regular, adequate heart rate helps ensure the

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heart's pumping efficiency to meet the needs of the body.

What about the ***P-R segment***? How might that point to a problem with the ***AV node***? Well, the AV node could delay the impulse too long. In this case, we would have a P wave followed by a really long P-R segment. Because the impulse is temporarily blocked, this is an *arrhythmia* called ***first degree heart block***. Oh. So, if this is first degree heart block, then there must be other variations of heart block that are much worse. Yep. In ***second degree heart block***, we see P waves, progressively longer P-R segments, and then periodically the QRS complex is missing. That's not good. In ***third degree heart block***, nothing gets through the AV node, so we have nothing but P waves. Uh, that's really bad. Wait, does that mean the heart doesn't pump any blood? Well, if the animal is alive it is—not well, but it's still pumping. You see, the AV node itself or the ventricles may randomly depolarize. This creates a pretty weird-looking wave, similar to the ventricular premature complexes (VPC) shown in Fig. 5.8. The abnormal waves occur independent from any SA node and atrial activity. As you might expect, heart block (especially second and third degree heart block) can produce a significant *bradycardia*. This usually requires surgical placement of a pacemaker. Yes, we use those in animals too, generally dogs.

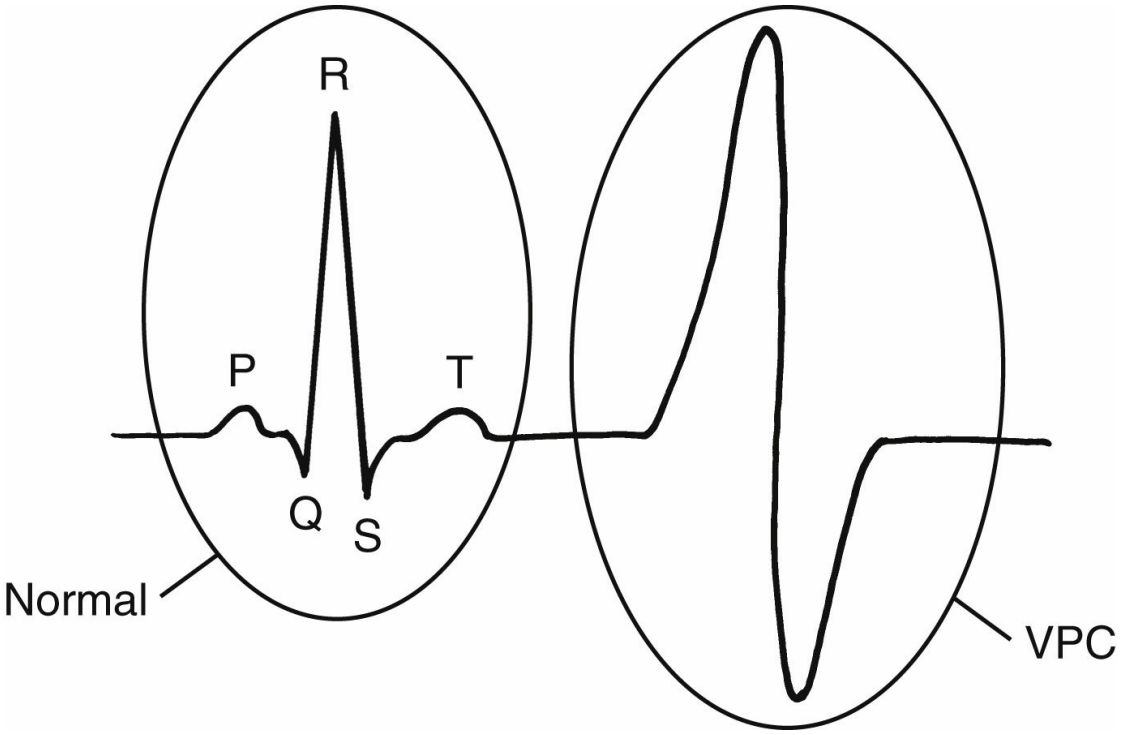

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FIG. 5.8 Ventricular premature complex (VPC).

Since we mentioned ***VPCs*** (***ventricular premature complexes***), let's discuss those briefly. VPCs are one of the most frequently seen *arrhythmias* on ECG. Notice that the VPC shown in Fig. 5.8 is not preceded by a P wave. This is what's making it *premature*. The ventricles are not waiting their turn, as they should. The ventricles should not contract until the atria have completed their contraction. That means a VPC is actually occurring during diastole—the filling/resting phase of the cardiac cycle. So if the ventricles contract prematurely, will they have sufficient blood to pump? Nope. Because of that most VPCs do not produce a peripheral pulse. If they do, it's a really weak pulse. Now, a single, rare VPC is no big deal. Many people and animals "throw" a rare VPC now and again without consequence. But frequent VPCs are a concern. In my experience, one of the most common causes of VPCs in a normal animal is ***hypoxia*** [*hypo-* low + *ox(o)-* oxygen + *-ia* condition of]. Wait, why would a normal animal be ***hypoxic*** [*hypo-* low + *ox(o)-* oxygen + *-ic* pertaining to]? Anesthesia and heavy sedation for routine procedures can adversely affect breathing rates and depths. And that can affect oxygenation. The myocardium gets pretty irritated by *hypoxia*. And if it's irritated, it often reacts with VPCs. So, if you're monitoring a patient during a routine procedure and you begin seeing VPCs, do something to improve the patient's breathing and oxygenation. We'll talk about breathing and oxygenation a little later, with the respiratory information.

Of course, VPCs can and are associated with underlying cardiac conditions. The story I'm about to share is a powerful one that proves the need for regular patient assessments. I will never forget a German Shepherd who was in my care in ICU. His owners brought him in because he wasn't acting right and he had diarrhea. He was hospitalized for observation and diagnostic testing. For any animal in my care, I always do a quick physical assessment, especially at the beginning of my shift. My initial assessments familiarize me with each individual patient. I repeat those assessments throughout my shift to pick up on changes in a patient's condition. The greater a patient's problems, the more frequently I assess—often more frequently than requested by the attending veterinarian.

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With this particular patient, I did my usual quick assessment, including attitude, mucous membrane color, capillary refill time, respiratory rate, and heart rate. Did any portion of that assessment have anything to do with diarrhea? No. But I did it anyway. You never know what you might find. As I felt for his pulses to get a heart rate, I had difficulty. This should not have been a problem. He was a big dog. Pulses should be easy to feel, especially using the ***femoral artery***. Those that I was able to feel seemed weak and slow. I needed to be sure. So I placed my stethoscope on his chest to see if I could get an accurate heart rate through listening. He was *tachycardic,* according to what I heard. And if anything, his weak pulses were *bradycardic*. The heart rate through auscultation (listening) and palpation of peripheral pulses should always match. The difference I found was very concerning. I was determined to gather more information before calling the attending doctor. So, I decided to check a quick ECG. I hooked him up to the leads. What I saw scared the crap out of me.

He was in ***ventricular tachycardia*** (***V-Tach***)—nothing but VPCs in rapid succession on that ECG machine. No wonder the dog had poor pulses and a ***pulse deficit*** (fewer pulses compared to audible heart beats)! Technically, strings of 6 or more VPCs constitutes V-Tach. This dog had a VERY serious problem: he had nothing but VPCs. I rushed to get the attending veterinarian. He too was shocked. And he quickly initiated ***intravenous*** [*intra-* within + *ven(o)-* vein + *-ous* pertaining to] (***IV***) medication orders to get the problem under control while we waited for the *cardiologist* to consult with us. We had to reduce the VPCs and get the dog out of V-Tach. Fortunately, the ***constant rate infusion*** (***CRI***) of the medication did eliminate many of the VPCs. He still had some, but at least he was no longer in V-Tach. If I hadn't done my routine assessment, despite the fact that it had nothing to do with a primary complaint of diarrhea, that German Shepherd could have died on my watch. It was a powerful reminder for me to always fully assess my patients, no matter what. I am so glad that I did in this case.

One final thought on *arrhythmias*, before we finish up here. In the *Normal Sinus Rhythm* animation, it was mentioned that changes to the T wave may indicate problems with the ventricles, including myocardial damage. That is true. And, in my experience, T wave

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abnormalities (including changes in size and direction of deflection) are frequently associated with electrolyte disturbances. Potassium is one electrolyte that can have a dramatic impact on cardiac function and the appearance of T waves. And because ***hyperkalemia*** [*hyper-* excess + *kal(o)-* potassium + *-emia* a blood condition of] creates significant *bradycardia* and may even stop the heart, recognizing changes in the T wave is important. That's the key in electrocardiography, both for the cardiologist and the novice who's still learning: recognition of ECG changes and deviations from normal.

Vessels

Since I just shared the story about the German Shepherd in V-Tach, with weak pulses, and who received IV medication, this might be a good time to talk about vessels. This is invaluable information for you, as a future veterinary professional. At the very least, you need to know the most accessible ***pulse points*** and ***phlebotomy*** [*phleb(o)-* vein + *-tomy* to cut; i.e., blood collection from a vein] sites for *venous blood collection* and sites for the administration of *intravenous* fluids or medication. You also need to know sites for *arterial blood collection* for *arterial blood gas* analysis. (Arterial blood is preferred when we want to know levels of oxygen actually being delivered to the tissues.) You'll learn about these things in this section. You'll also gain a better understanding of circulation—important before we take a closer look at gas exchange and blood pressure later on.

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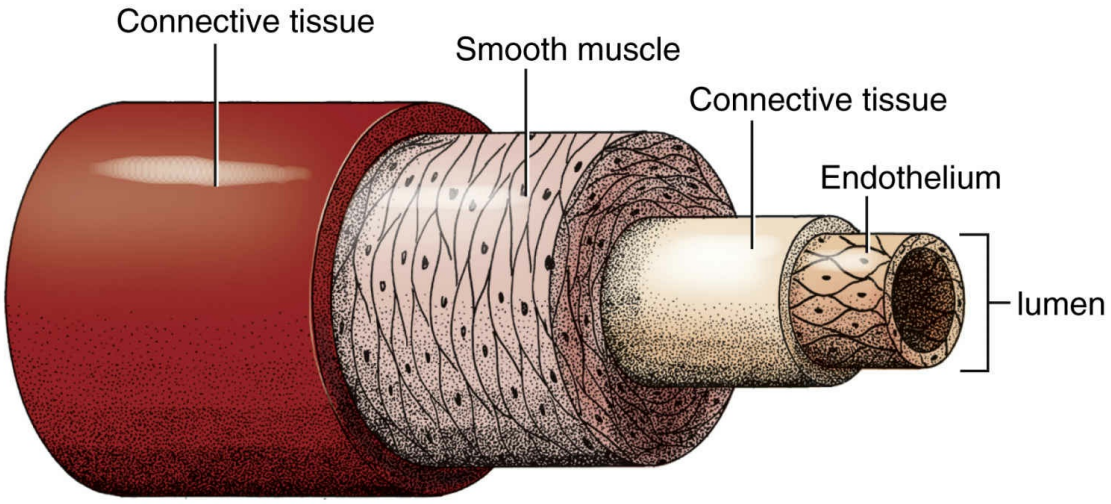

FIG 5.9 Arterial wall schematic.

Arteries

You already know that ***arteries*** carry blood away from the heart. We've already mentioned a couple of really large arteries, namely the *aorta* and *pulmonary artery*. Obviously, these two arteries, receiving blood directly from the ventricles, must experience profound pressure with each ventricular contraction. Truth be told, all arteries are exposed to higher pressures than any other vessels in the body. So structurally, arteries need really thick walls to withstand those pressures. In Fig. 5.9, you can see all of the layers that make up an arterial wall. The thickest layer is the smooth muscle layer. Why do arteries need smooth muscle? Before answering that question, I remind you of the discussion of smooth muscle inChapter 4. There you learned that smooth muscle is involved with involuntary activity along the respiratory, digestive, and urinary tracts, as well as blood vessels. In vessels, like the arteries we're currently talking about, their smooth muscle is an important contributor to the maintenance of blood pressure. That's right, maintenance of blood pressure relies on more than blood volume and the heart alone. By contracting smooth muscle along arteries and other vessels, we effectively decrease the ***lumen*** size (i.e., the interior space) of the vessels. And that can help us raise blood pressure. We'll cover more on blood pressure later.

It's important to note that smallest of arteries are called ***arterioles*** [*arteri(o)-* artery + *-ole* a small]. Remember, the whole purpose of

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arteries is to deliver oxygen-rich blood to the tissues. Actual delivery of the oxygen takes place at the smallest, thinnest vessels of the body—***capillaries***. *Arterioles* help us make the transition from larger arteries to tiny capillaries. And it's the arterioles that control blood flow to the capillaries. Even arterioles have a layer of smooth muscle. They are so small that if the smooth muscle contracts, the ***vasoconstriction*** [*vas(o)-* vessel + *constrict* + *-ion* act of] that results in the *arterioles* can significantly reduce if not stop blood flow to capillaries in the area. Think about the *arterioles* in the skin for a moment. When we are cold, to conserve heat for the body, ***dermal*** [*derm(o)-* skin + *-al* pertaining to] arterioles ***vasoconstrict***. This minimizes heat loss by reducing blood flow to superficial capillaries. On the flipside, when we're hot, *dermal* arterioles experience ***vasodilation*** [*vas(o)-* vessel + *dilat(o)-* dilating, expansion + *-ion* act of]. This increases blood flow to superficial capillaries, making it easy to dissipate heat from the body. There are other very important areas of the body where arterioles play key roles. For example, inChapter 6, we'll focus on the importance of the arterioles that lead to and from the ***glomerulus*** (the filtration unit) in the kidney. *Vasoconstriction* and *vasodilation* of these arterioles control filtration of the blood by the kidneys. So, while *vascular smooth muscle* contraction and relaxation occurs along numerous vessels of the body, it's really along the *arterioles* that this muscle activity can have the greatest impact.

Major arteries and arterial pulse points

Under normal circumstances, for each heartbeat (i.e., ventricular contraction) we should feel a *peripheral pulse*. Physical species variations, accessibility, and animal behavior determine which arteries can be used as pulse points in a given animal. Please note that the arterial schematics shown in Figs. 5.10 and 5.11 include commonly used pulse points, as well as some other major arteries of importance. As I discuss many of those arteries in this section, I will try to share the relevance of each. Please note that I will be referencing various bones and skeletal points to guide you in locating these arteries. If you need to, please refer toChapter 4 to review skeletal information. Let's begin at the head and work our way caudally.

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Sublingual artery

The ***sublingual*** [*sub-* under + *lingu(o)-* tongue + *-al* pertaining to] ***artery*** is located along the ventral midline of the tongue of all animals. Obviously, we can't use this artery as an everyday pulse point. We'd lose fingers in the process! However, in an anesthetized animal, it becomes a valuable site to palpate pulses and collect blood for arterial blood gas analysis. It is most commonly used as a pulse point in anesthetized dogs and cats. However, it could be used for this purpose in almost any anesthetized animal. You still need to be careful, especially if the animal is a little too "light" under anesthesia (i.e., not sleeping deeply). If the animal bites down, either your fingers or the animal's tongue may be injured. The *sublingual artery* is small, so use a gentle touch to avoid occluding the vessel. If you can't feel a *sublingual* pulse, you're likely pressing too hard.

Facial artery branches

The branches of the ***facial artery*** shown in Fig. 5.11 are most frequently used as pulse points in livestock (horses, cattle, sheep, and goats). Some of these arterial branches are easier to use than others. The ***transverse facial artery*** branch lies along the zygomatic arch on the face. In most livestock this is one of the easiest branches for us to palpate pulses, provided the animal is not head-shy. I have even used this periodically in some larger anesthetized dogs, when my access to other pulse points was limited. In anesthetized large animals, this artery is also valuable for arterial blood collection for blood gas analysis. The ***lateral nasal artery*** is most easily palpated where it passes over the maxilla, at approximately a midpoint on the face. This is also easily palpated and may be used for arterial blood collection. The ***facial artery*** itself is a relatively large artery that is most easily palpated where it passes over the ventral mandible. If you feel for a notch along the ventral border of the mandible near the ramus, you'll find it. The biggest difficulty in using this as a pulse point is mandibular movement. This is especially true in ruminants because they're always chewing their cud. But even horses move their jaws, usually right when you've started counting their pulse. Then you have to start over again. That's why I tend to prefer using the other branches. Regardless of

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the branch used, be patient. Remember, the larger the animal the slower the pulse (sometimes 30 bpm or less). So, locate the artery, gently place your fingers over it, and patiently wait. If possible, it's also wise to count the pulses over a full minute. That will provide the greatest accuracy and help you detect certain *arrhythmias*.

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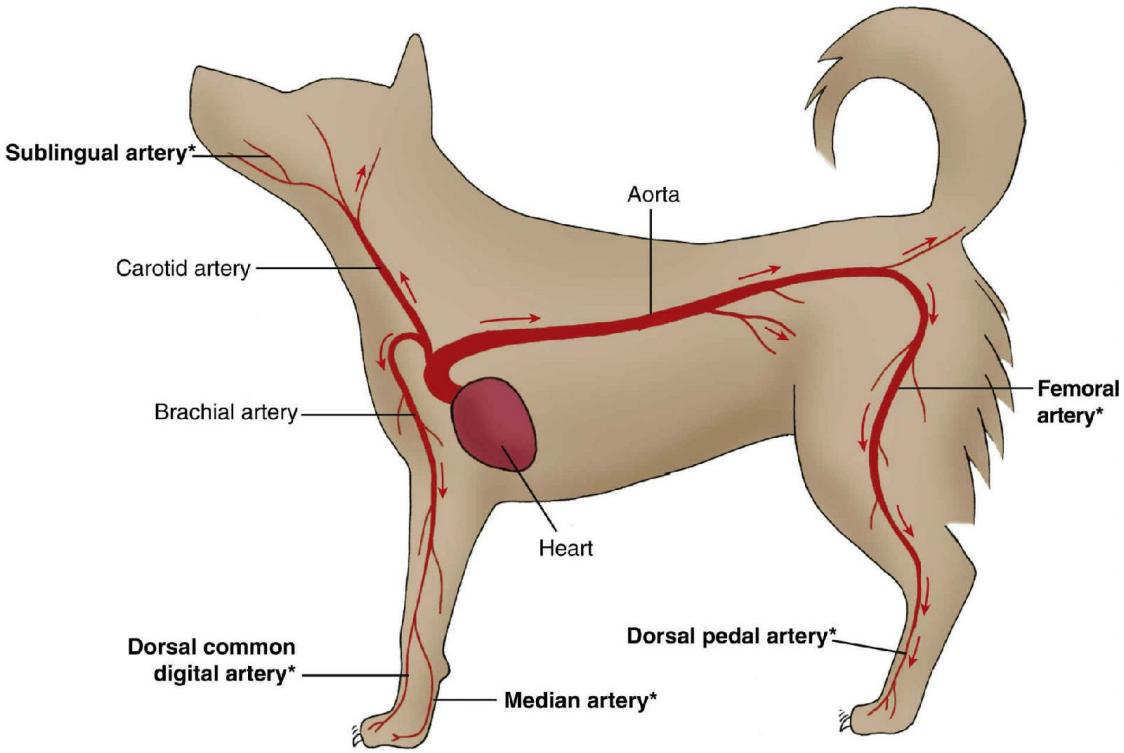

FIG. 5.10 Canine arteries and arterial pulse point schematic. (The *bold text* and *asterisk [*]* indicate common pulse points for dogs and cats. *Arrows* indicate direction of blood flow.)

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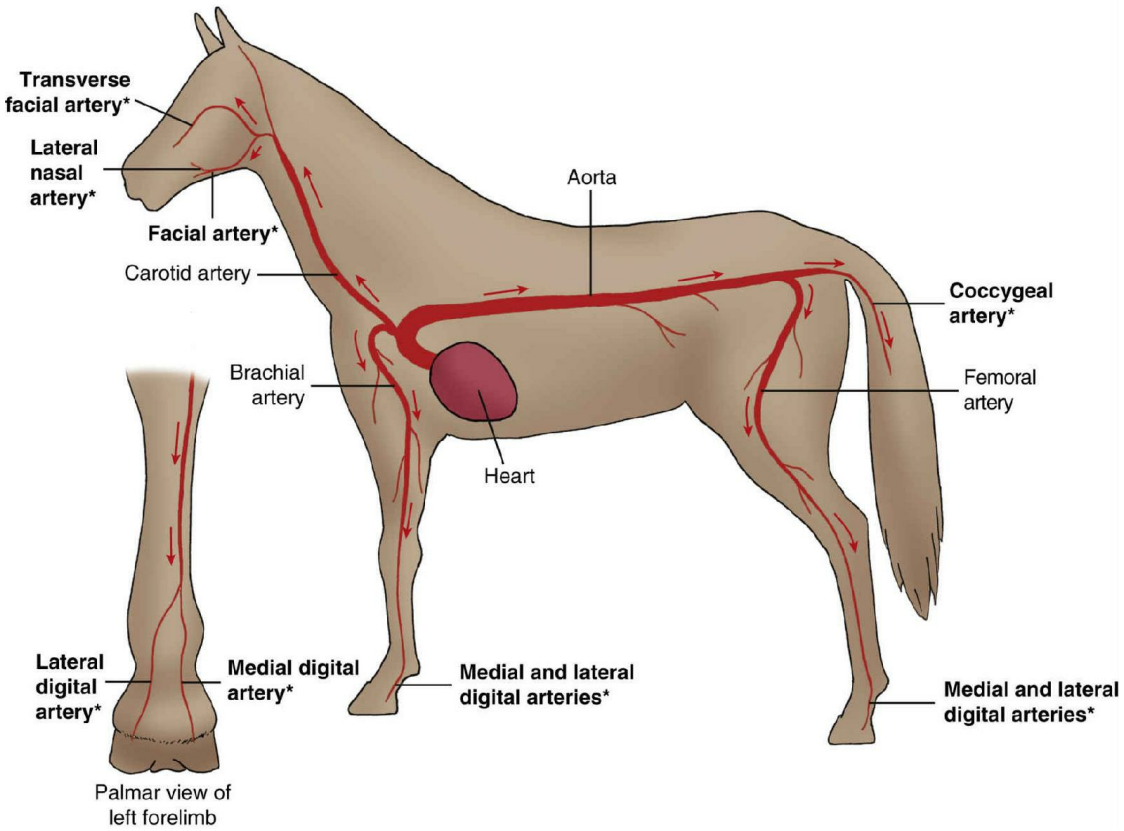

FIG. 5.11 Equine arteries and arterial pulse point schematic. (The *bold text* and *asterisk [*]* indicate common pulse points for horses. *Arrows* indicate direction of blood flow.)

Carotid artery

The ***carotid artery*** is *NOT* used as a pulse point, because it typically lies too deep in the muscles of the lateral neck. (Most animal necks are built like linebackers.) Still, I have included the *carotid artery* here because it is relatively close to the jugular vein, especially near the head and the shoulder. Why is this important? We often use the jugular vein for blood collection. And in livestock we often use the jugular vein for *IV medication administration*. ***Intravenous*** [*intra-* within + *ven(o)-* vein + *-ous* pertaining to] (***IV***) medications are intended to be carried to the heart for distribution throughout the body. In the process, the drug is diluted. In the liver, it may be altered. Where does blood in the carotid artery go? That's right, directly to the head. And what's in the head?—the brain. If we are not careful and mistakenly administer a medication into the carotid artery, we essentially send a bolus of medication directly to the brain. Depending on the drug, this could precipitate a seizure. And if you're giving that drug to a horse or a cow, that places you in a

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very dangerous position standing next to that animal when it abruptly goes down and begins to convulse. I've seen animals die because of this error too. So, always aspirate before giving IV medication, especially if you're using the jugular vein. If the blood you aspirate is bright red, it's arterial. Abort! Don't give that drug.

Brachial artery

The ***brachial*** [*brachi(o)-* arm + *-al* pertaining to] ***artery*** is another artery that is typically *not* used as a pulse point. Generally, it's simply too deep in the muscles along the medial aspect of the humerus. On rare occasions, I've been able to palpate it in a skinny cat or small dog, but that's rare. So why point out this artery here? It is a valuable ***pressure point*** (especially in dogs and cats), when we have excessive bleeding from the distal forelimb. Direct pressure over the medial humerus may help control the bleeding. Tourniquet placement over the distal humerus may effectively stop the bleeding by completely occluding the brachial artery. Just remember, if you completely stop blood flow by occluding this artery, you've got 30 minutes before major tissue death results distal to the tourniquet. Mark the time on the tourniquet when it is placed and set a timer for 30 minutes. If we haven't repaired the injury within that time, we will have to release the tourniquet momentarily to restore blood flow to the tissues. Yeah, that's a messy necessity. If the tourniquet is applied again, mark the new time on it and reset the timer. Let's face it: our repairs to stop the bleeding are pointless if we lose the leg because of tissue death from the tourniquet.

Dorsal common digital artery and median artery

The ***dorsal common digital artery*** is a pulse point used in dogs and cats. Based on its name, you know that it must be in the vicinity of the digits (toes). You know, then, it's somewhere near the paw. It lies on the dorsal surface of the metacarpus. If you remember that it passes over the metacarpus at a mediolateral oblique angle, you can find it more easily. Remember, too, that it's a digital artery, so it's relatively small. Use a light touch or you may easily occlude it. In smaller dogs and cats, you'll probably have difficulty feeling pulses here, especially in a wiggly patient who doesn't like its feet messed

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with.

The ***median artery*** is found on the palmar aspect of the metacarpus, pretty close to midline (as its name implies). It's another small artery. However, even in smaller dogs and cats, I've had an easier time palpating pulses here, compared to the dorsal common digital artery. If you can't feel either of these arteries, try for a larger arterial pulse point.

Digital arteries of the horse

In the horse, there are two prominent digital artery branches in the distal limb. As you can see in the palmar view of the forelimb (see Fig. 5.11), there is a ***lateral digital artery*** branch and a ***medial digital artery*** branch. These are found in both front and rear limbs, and most easily palpated over the palmar or plantar aspect of the pastern bones (1st and 2nd phalanges). Horses are quite used to having their feet and distal limbs handled, by farriers and their owners, so most horses are quite tolerant of you palpating pulses here. Other livestock have these digital arteries too. But because they're not used to having their feet handled, you probably won't be able to palpate pulses here, not safely anyway. In horses, if we're concerned about ***laminitis*** [*lamina* + *-itis* inflammation of], as discussed inChapter 8, we often assess the quality of digital pulses here. Bounding digital pulses are often indicative of inflammation in the foot.

Dorsal pedal artery

The ***dorsal pedal artery*** is a valuable pulse point in dogs and cats. To help you remember that it's located over the dorsal metatarsus, think about pedaling a bike. We use our feet for that, right? If a dog or cat could be trained to ride a bicycle (I've seen it), it would pedal using its rear feet. So, the dorsal pedal artery is found in the rear limb. Compared to the dorsal common digital and median arteries, the dorsal pedal artery is larger. So, it is more easily palpated for pulses. I've also used this artery frequently in dogs and cats, when I've needed to collect arterial blood for blood gas analysis, for placement of an arterial catheter for direct blood pressure assessment, and for noninvasive Doppler blood pressures. I love this artery. (If you're wondering why Doppler is capitalized, it's the

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name of the Austrian physicist, Christian Doppler, who lived in the 19th century.)

Femoral artery

The ***femoral*** [*femor(o)-* femur + *-al* pertaining to] ***artery*** lies along the medial femur (hence the name) in all animals. Clinically, for our purposes, this artery is only of value as a pulse point in dogs and cats. Horses and other livestock are simply too heavily muscled in the medial thigh for us to palpate the femoral artery. Plus, it's probably not a good idea to endanger yourself by attempting to palpate it in a horse or a cow. Don't do it. But in dogs and cats, this is *the* go-to artery for pulses. It's big and easily palpated. Well, except in animals like that German Shepherd I dealt with who was in V-Tach. If you have trouble palpating *femoral pulses*, you need to be concerned. When push comes to shove and we can't collect an arterial blood sample for blood gas analysis from any other artery in a dog or a cat, we may turn to the femoral artery. It's not preferred, because it is difficult to apply pressure over the site after collection to prevent a large ***hematoma*** [*hemat(o)-* blood + *-oma* swelling; i.e. a collection of blood outside a vessel] formation. By the way, like the brachial artery, the femoral artery is also useful as a *pressure point* to control bleeding in the distal rear limb.

Coccygeal artery

Finally, the ***coccygeal*** [*coccyg(o)-* tail + *-al* pertaining to] ***artery*** is most useful in livestock. This is my go-to artery for pulses in cattle. As you can see in Fig. 5.12, the *coccygeal artery* is found along the ventral midline of the tail. I've used the pulse point frequently in horses too, while I'm hanging out in the rear taking a rectal temperature. Might as well multitask, right? Just remember that if you're going to palpate coccygeal pulses, you need to select a ventral point along the tail that doesn't have too much tissue covering the artery. Tissues are most thick closest to the body. So if you slide your hand one to three vertebrae distal from the body, you'll have an easier time palpating the pulses. Don't go too far distal. The artery gets smaller the more distal along the tail it gets. Plus, the more distal on the tail you go, the more that tail tends to move. There is less movement closer to the body. By the way,

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please stand close alongside the rear leg and hip when you use this pulse point. Standing directly behind a horse or a cow is probably not wise. You could be seriously injured if the animal kicks. One final note: while the coccygeal artery is used as a pulse point most frequently in horses and cattle, I've also found it useful in medium- to large-sized dogs. In fact, we've even shaved the ventral tail of dogs and taped the probe of the pulse ***oximeter*** [*oxi(o)-, oxy(o)-* oxygen+ *meter* measurer] there for continuous assessment of oxygen saturation. Cool, huh?

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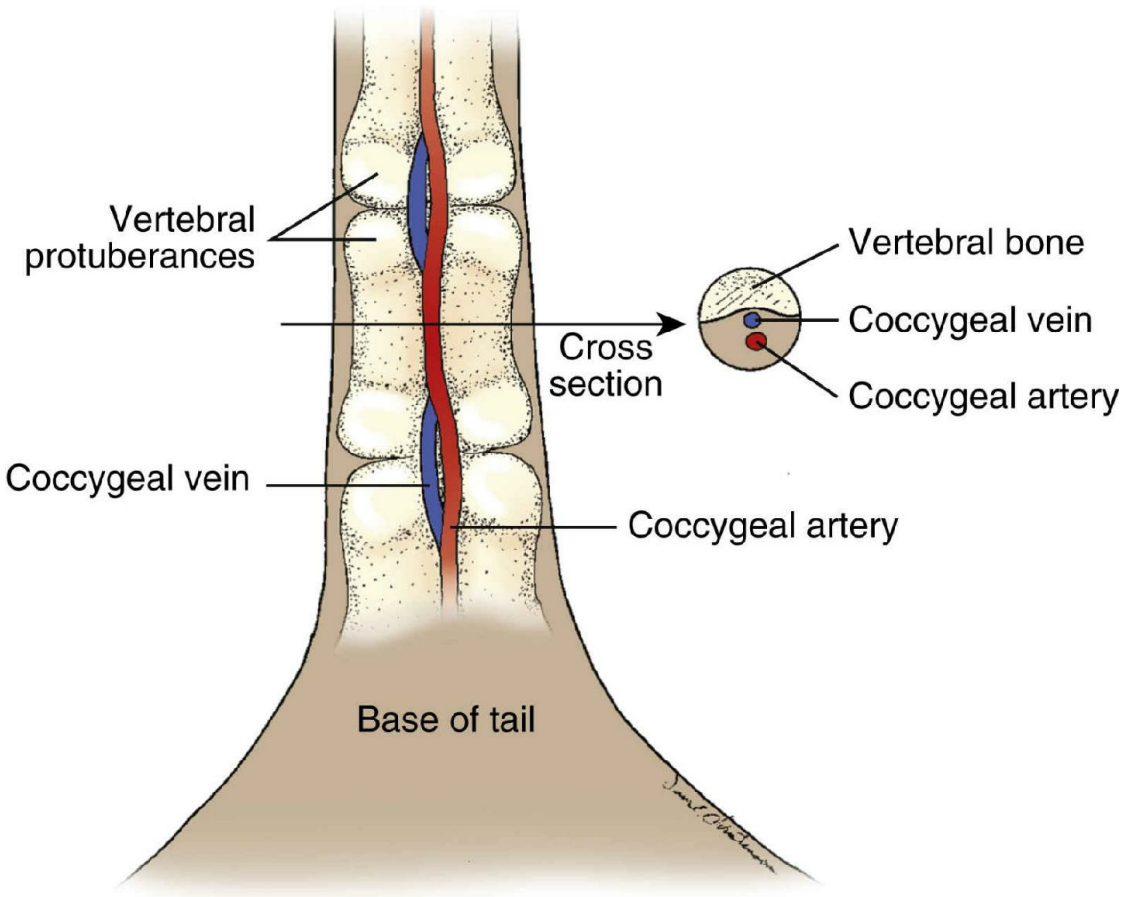

FIG. 5.12 Bovine coccygeal artery and vein schematic.

Veins

Alrighty, you know that arteries carry blood away from the heart. ***Veins*** return blood to the heart. Comparatively, veins have much thinner, less muscular walls than arteries. There is very little pressure in veins. There's no need for a bulky wall, so ***venous*** [*ven(o)-* vein + *-ous* pertaining to] walls have thinner layers, as you can see in Fig. 5.13. Probably the most unique feature of veins is the ***unidirectional*** [*uni-* one + *directional*; i.e., one-way] ***valves***. You see, pressure is so low in veins, if we didn't have the *unidirectional valves* at regular points along the *lumen*, blood might easily back up and pool. And if venous blood were to back up all the way to the capillary level, ***edema*** (swelling) in the surrounding tissues would likely develop. By the way, the tiny veins that connect larger veins with capillaries are called ***venules*** [*ven(o)-* vein + *-ule* a small].

Okay, so if the pressure is so low in veins, what makes blood move through them? Good question. Two factors largely affect

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blood flow through veins. First, muscular movement of the body helps to squeeze veins, pushing blood along. And because of the *unidirectional valves*, venous blood can only flow in a ***centripetal*** [*centr(o)-* center + *-petal* directed, from L. *petere* "to seek"; i.e., moving toward the center] direction toward the heart when the veins are squeezed. This is why, for people on long flights or car rides, it is important to take time to get up and walk around and wiggle their feet. That movement facilitates *centripetal venous return* from our legs. And that is important for the prevention of ***deep vein thrombosis*** [*thromb(o)-* clot, clotting + *-sis* condition of] (***DVT***). As you may recall from our discussions on clotting inChapter 3, we said that pooling of blood may activate the clotting cascade. This is important for us to remember for any of our patients who, due to paralysis, for example, are recumbent and unable to move their limbs. This brings to mind the second important factor affecting venous blood flow—gravity. Generally, muscle activity/body movement is the primary factor affecting venous return. Gravity has only minor influence on blood flow, unless an animal is experiencing immobility and prolonged recumbency. In a recumbent animal, gravity will dictate where venous blood drains from (i.e., the upside of the animal) or pools (i.e., on the downside of the animal). This is, in part, why it is so important to turn recumbent animals every 2 to 4 hours. If an individual limb is immobilized, it's important to elevate the limb, if possible. By elevating the limb above the level of the heart, gravity will promote *centripetal* venous blood flow from the limb.

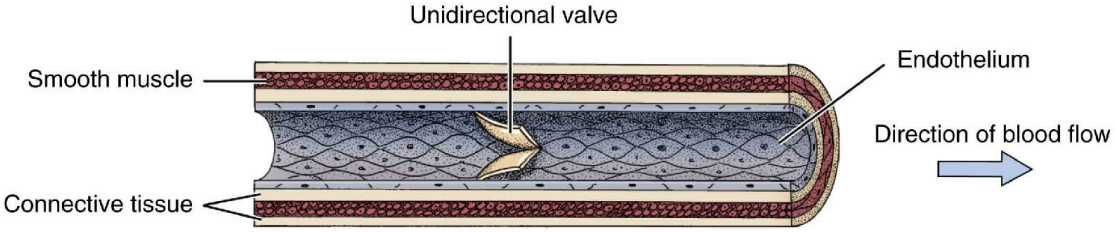

FIG. 5.13 Midsagittal section of vein/venous wall schematic.

Major veins and phlebotomy sites

We already mentioned some major veins when we discussed the

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heart and great vessels earlier. In that discussion, we made note of the *pulmonary veins* returning blood to the left atrium from the lungs. We also mentioned the *cranial vena cava*, carrying venous blood from the head, neck, and forelimbs, and the *caudal vena cava*, a much longer vessel, carrying blood from most of the caudal parts of body to the heart. Both segments of the vena cava flow into the right atrium. In other chapters, we'll mention other veins of importance to the given body system. For instance, in Chapter 7, we'll talk about the importance of ***mesenteric*** [*mesenter(o)-* mesentery + *-ic* pertaining to] ***veins*** from the digestive tract and the ***portal vein*** of the liver.

For our purposes in this chapter, we'll focus on those peripheral veins that are most frequently used for *phlebotomy* (for venous blood collection and medication administration). Species variations, accessibility, and animal behavior determine which veins can be used as phlebotomy sites in a given animal. Please note that the venous schematics shown in Figs. 5.14 and 5.15 include commonly used phlebotomy sites, as well as other major veins, like the vena cava. As I discuss many of the veins in this section, I will try to share the relevance of each for domestic animals. As with arteries, I will be referencing various bones and skeletal points to guide you in locating these veins. Refer to Chapter 4 if you need to review that skeletal anatomy.

By the way, remember that there is little pressure in veins. At least with arteries we could feel for pulses to find the vessel. That's not possible in veins. So what we need to do is temporarily obstruct venous return at a proximal point along the vessel. By applying pressure at a proximal point, blood will pool in the distal portion of the vein, making it bulge out. Once the *occluded vein* is distended with pooled blood, we can feel it and sometimes see it. If you're uncertain that you've actually isolated the vein, release your pressure. The vein should "disappear" when no longer occluded. If it doesn't "disappear," you're probably feeling or seeing something else. Once you've isolated the vein, occlude it long enough to pool enough blood. If we're trying to collect blood, the pooled venous blood needs to provide enough volume to fill our blood collection tube(s) without completely collapsing the vessel. This is something to bear in mind when selecting a phlebotomy site. You should ask

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yourself: How much blood do I need? Which vein will best accommodate that volume?

One final comment before we look at specific veins—please use ***aseptic*** [*a-* without + *sep(o)-* infection + *-tic* pertaining to] ***technique*** when performing collection of venous blood and/or administering IV medications. This is an invasive procedure. You're penetrating the vein with a needle. So you need to minimize any risk of dragging contaminants into the lumen of the vessel. At the very least, contaminants and trauma could create localized ***phlebitis*** [*phleb(o)- vein* + *-itis* inflammation of] at the puncture site. At the very worst, because all venous blood goes to the heart, contaminants could precipitate ***endocarditis*** [*endo-* within + *card(o)-* heart + *-itis* inflammation of]. And that could be lethal. Protect your patients. Always use *aseptic technique*.

Marginal ear vein

The ***marginal ear vein*** is found along the margins of the ear pinna (flap). It has limited use in domestic animals. The animal has to have pretty big ears to have marginal ear veins large enough to be able to collect blood or administer medications. Rabbits and certain dog breeds, like Basset Hounds and Bloodhounds, have very nice marginal ear veins. In fact, when faced with the crooked leg veins of a Basset Hound, I would much rather use the marginal ear vein. I've even placed IV catheters in the marginal ear vein of rabbits and Basset Hounds. A roll of gauze is quite useful to wrap around the ear, to stabilize and secure the catheter. That said, this doesn't work well for a long-term indwelling catheter. Animals inevitably shake their heads and shake out the catheter. (That can become a bloody mess!) But for anesthesia, the marginal ear vein can be perfect for catheter placement, because the animal doesn't move. Whether collecting blood or placing an IV catheter, occlude the vein near the base of the ear.

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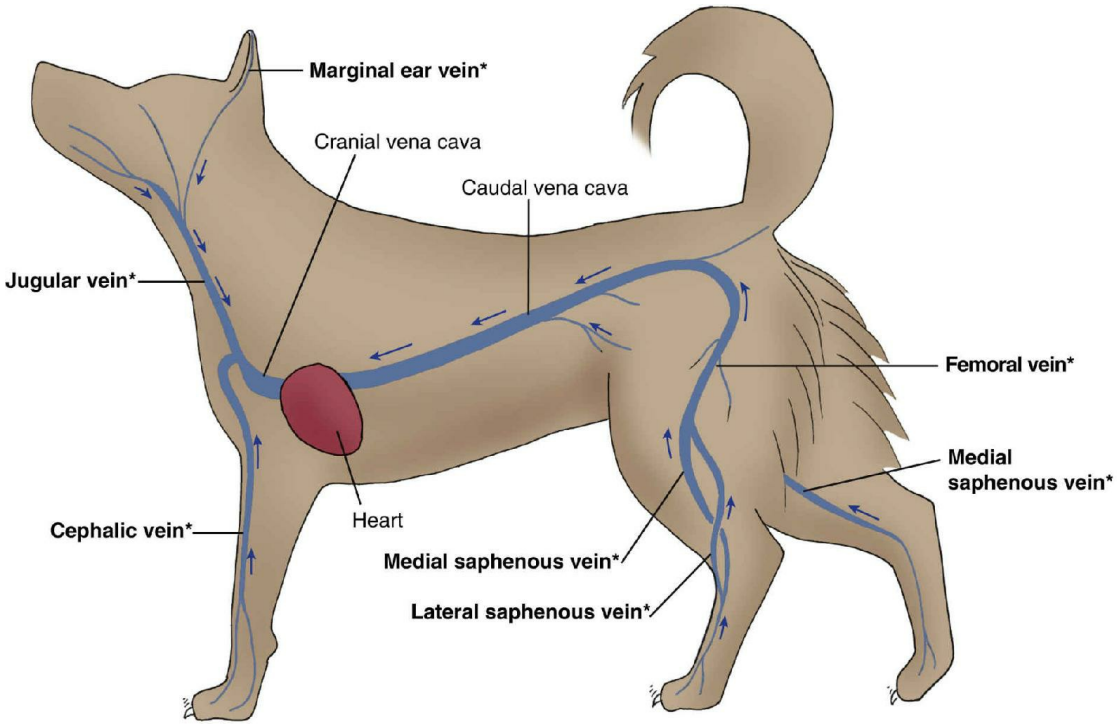

FIG. 5.14 Major canine and feline veins and phlebotomy sites. (The *asterisk [*]* and *bold text* indicate common phlebotomy sites for dogs and cats. *Arrows* indicate direction of blood flow.)

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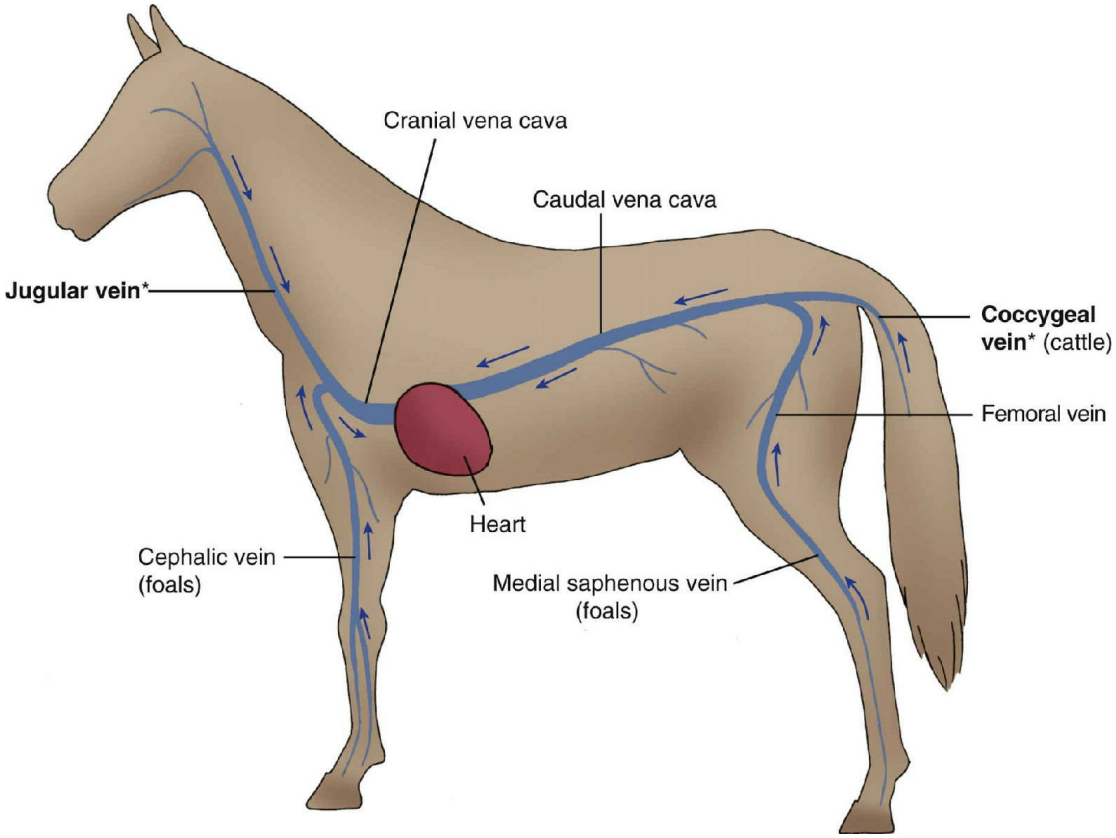

FIG. 5.15 Major large animal veins and phlebotomy sites. (The *asterisk [*]* and *bold text* indicate most commonly used phlebotomy sites. *Arrows* indicate the direction of blood flow.)

Jugular vein

The ***jugular*** [from L. *jugularis* "neck"] ***vein*** is the largest, most accessible, peripheral vein in most domestic animals. Of course, the animal has to have a reasonable neck to allow easy access to the jugular vein. And, well, pigs and guinea pigs just don't have much of a neck. It's like their heads are attached to their shoulders. So in these animals, collecting blood from the jugular vein is challenging to say the least. For everyone else, the jugular vein is easily accessed, provided the animal is cooperative and/or able to be restrained adequately. We certainly don't want the animal thrashing around with a needle in its neck. Remember, the *carotid artery* and the *vagus nerve* are in the neighborhood. We cannot risk damaging those. We'll find out more on the consequences of that in a bit.

There are some features of the lateral neck that may help guide you to the jugular vein. First, the vein lies in the ***jugular furrow***. That's a shallow trench formed by some of the neck muscles and

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cervical vertebrae. If you can't see the jugular furrow, you may be able to feel it. In many dogs and cats, the fur often forms a cowlick in the vicinity of the jugular furrow. If neither of these are evident, you know that the jugular vein is in the lateral neck. So, press your hand against the *ventrolateral neck* at the base of the neck and wait for the vein to distend. Remember, the larger the animal, the larger the jugular vein, and the more blood is needed to fill it. So, you need to be patient—especially with a horse or a cow. Their jugular veins are the size of a garden hose!

Regardless of whose jugular vein you're collecting blood from, you should use the middle third of the vein. The closer you get to either end of that vein (i.e., near the head or shoulder), the closer you get to the *carotid artery* and *vagus nerve*. Does it matter if you collect blood from the carotid artery for routine laboratory testing? No. But that artery is under extreme pressure. So, after withdrawing your needle, it's going to be difficult to prevent bleeding into the ***perivascular*** [*peri-* around + *vascul(o)-* vessel + *-ar* pertaining to] tissues of the neck. In fact, this could result in a huge *hematoma* in the neck. If you traumatize the vagus nerve, you have bigger things to worry about. Trauma to the vagus can create a significant ***acute*** (sudden) *bradycardia* and send the blood pressure plummeting. Those events could become life-threatening. So play it safe: use only the middle third of the jugular vein for blood collection to avoid serious consequences. And never, ever use the jugular vein for medication administration (unless it's through a central line—a large, long catheter that is carefully placed and sutured in place, to guarantee it's in the jugular vein). Remember, the carotid artery is nearby. We already talked about the dangers of inadvertently injecting medication into that artery.

Cephalic vein

The ***cephalic*** [*cephal(o)-* head + *-ic* pertaining to] ***vein*** is found in the *cephalic* or cranial limbs (i.e., forelimbs). You'll find this vein along the cranial aspect of the forelimb, between the carpus and the elbow. We use this vein most often in dogs and cats. However, it also provides a reasonable venous site in foals, especially for IV medication administration. It has to be a pretty sick foal. Unlike dogs and cats, healthy foals and adult horses are nearly impossible

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to restrain for safe use of veins like the cephalic. They simply won't hold those legs still, especially when we start poking them with a needle. And they can do significant harm to us or to the handler if they strike with their hooves.

Remember, blood flows through the *cephalic vein* from the carpus to the elbow and beyond. So, you need to occlude it near the elbow, to make it distend with blood. And always make your initial attempt at cephalic phlebotomy as distal as possible (i.e., near the carpus). If the dog or cat struggles, resulting in a hematoma, you can always make further attempts more proximally along the vessel, without the hematoma obscuring the vein. If you're using the cephalic vein for delivery of IV medication, you have to start distal on the vessel. If your first unsuccessful attempt is proximal on the vein, giving the medication from a point distal to that initial puncture will result in *perivascular* infusion of the drug (i.e., it will leak from the initial puncture). Some medications, if accidentally given ***extravascularly*** [*extra-* outside + *vascul(o)-* vessel + *-ar* pertaining to], can cause significant pain, inflammation, and even ***necrosis*** [*necr(o)-* death + *-sis* condition of] of *perivascular* tissues.

Saphenous veins

The ***saphenous*** [from Gr. *saphena* "manifest" (obvious, visible) + *-ous* pertaining to] veins are found in the rear limbs, just proximal to the tarsus. There is typically very little in the way of bulky tissue covering this region of the limb, so the *saphenous veins* are very visible. Perhaps that's why ancient Greeks named them *saphenous*? One has to wonder. At any rate, there are actually two prominent branches. There is a *medial saphenous vein* and a *lateral saphenous vein*.

In foals and cats, the ***medial saphenous vein*** is the larger of the two branches. As with the cephalic vein in foals, you'll most likely only be able to access the medial saphenous vein if the foal is quite ill. Otherwise, it will be too difficult and dangerous to use. (It's on the medial side of the leg, remember. Think about it. Do you want to put yourself under the belly of a rambunctious foal or horse to collect a blood sample? You might not live to tell about it.) The *medial saphenous vein* is most frequently used in cats. It's relatively straight, running along the medial aspect of the tibia. And it's

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pretty easy to have someone restrain the cat in lateral recumbency, putting pressure over the medial thigh with the side of their hand. I've used the medial saphenous vein only occasionally in dogs. In many dogs, it's just not a fabulous vein—for phlebotomy, that is.

In dogs, the ***lateral saphenous vein*** is most prominent. It passes over the distal lateral tibia at an oblique angle. In a dog with a short hair-coat, the lateral saphenous is easy to see, even when it's not occluded. The biggest problem I've found with the lateral saphenous vein in dogs is the way it rolls. There is very little surrounding tissue along the lateral, distal tibia. So there is very little to stabilize the vein, to keep it from rolling side to side. Another thing that makes it challenging for phlebotomy or IV catheter placement is its uphill-downhill nature, as it courses over the tibia and then falls into the gap between the tibia and the Achilles tendon. But if you have your restrainer straighten the leg well and you put a little gentle tension over the distal part of the vein, it's actually a very nice phlebotomy site. In fact, in most dogs, I prefer it for blood collection. I've used it frequently for medication administration too. I like it. It's away from the biting end of the dog, and most dogs don't seem to mind being poked by a needle there, as much as other sites.

Femoral vein

The ***femoral vein*** lies along the medial thigh, right next to the femoral artery and femur (go figure). Generally, we don't use this vein often for phlebotomy. If we do, it's probably in a small dog or a cat. And we've probably exhausted other possible sites. Sometimes it's a matter of convenience. If we've tried and failed to collect our blood sample from the medial saphenous vein of a cat and/or we need a larger sample than the medial saphenous vein can provide, we may move proximal on the limb to the femoral vein. That's convenient, if our cat is well restrained at the moment but losing patience with us. The cat may not grant us another opportunity. It's also convenient because the medial saphenous vein leads us directly to the femoral vein. All we need to do is follow the saphenous vein proximal past the stifle and we've found the femoral vein. I've only ever used the distal third of the femoral vein. Frankly, I prefer not to use it if I have other options. It's

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simply too hard to apply enough pressure over the puncture site long enough to prevent hematoma formation. But this vein is a valuable option when we're "backed into a corner," so to speak.

Coccygeal vein

You've already seen the ***coccygeal vein*** earlier, when you looked at the *coccygeal artery* in Fig. 5.12. The *coccygeal vein* is used almost exclusively in cattle, especially dairy cows. Using this site for blood collection is a solo procedure. You have to restrain and collect the blood all by yourself. Your restraint involves "jacking" the tail dorsally, holding it as vertical as possible. This also helps occlude the vein. Then your free hand is used to cleanse the area (always necessary; the ventral tail is always covered in manure) and perform the phlebotomy. (I'm right-handed. So, I apply the tail-jack with my left and do the phlebotomy with my dominant, right hand.) Yes, this means you have to stand directly behind the cow. Just stand "skinny" with your (restraint) side toward the cow. Then if she kicks, she'll probably miss. And if one of those girls is taking a swing at me, I'd rather she hit my hip than my belly. You think I'm kidding? Oh, trust me—I'm not.

Seriously, this is an excellent phlebotomy site. Believe it or not, we can actually collect large volumes of blood from the *coccygeal vein* (easily 3 or 4 10-mL collection tubes). And it is easier to collect blood from the coccygeal vein than the jugular vein of a cow. The skin of the ventral tail is relatively thin and easily penetrated with the needle. The skin over the jugular vein is thick and tough like boot-leather. Plus, most cattle don't like us messing with them around their heads. I've been body-slammed by cows who didn't want me near their heads. So I'll use the tail, thank you very much.

Capillaries

A ***capillary*** [from L. *capillaris* "hair-like"] is the smallest vessel of the body. *Capillaries* are so small that red blood cells have to pass through them in single file. These are the tiny, vascular connections between *arterioles* and *venules*. Their walls are nothing like arteries or veins. Capillaries have only a single layer of *endothelium* on a thin *basement membrane*. And within their walls, blood transitions from arterial to venous, from highly oxygenated to deoxygenated [*de-*

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reduced]. Both their minute size and thin vascular wall structure make capillaries absolutely perfect for diffusion of blood gases, osmosis of water, and diffusion of various electrolytes and other substances. As you may recall from Chapter 2 and the edema discussion inChapter 3, *osmosis* is the movement of water across a semipermeable membrane. And *diffusion* is the movement of particles across a semipermeable membrane, from an area of high concentration to low concentration. The capillary wall is a thin, *semipermeable membrane*. When it comes to delivery of goods and removal of wastes, it's within capillaries that the "rubber meets the road." And for movement of white blood cells out into the tissues, many capillary walls have itsy-bitsy fenestrations (holes) and gaps in the endothelium, making it easier for WBCs to get where they need to go.

But these delicate, diminutive vessels are at the mercy of larger vessels to do their part in maintaining adequate blood flow and just the right amount of ***intravascular*** [*intra-* within + *vascul(o)-* vessel + *-ar* pertaining to] pressure. We said earlier that arterioles were important for controlling blood flow and pressure in the capillaries. That is true. However, even venules can play a role too, especially if venous return is impaired. Too much pressure in capillaries can easily force water through their walls into the tissues, creating excess *interstitial fluid* (i.e., *edema*). Excessive pressure could cause their fragile walls to literally break. Since capillaries rely so heavily on the rest of the cardiovascular system, this is probably a really good time to review ***blood flow***. Then we'll move into a detailed discussion of blood pressure.

Blood Flow

You may want to watch the Evolve animation called *General Body Circulation* again. In fact, even the animation *Mitral and Aortic Valve Regurgitation* provides an excellent review of both pulmonic and systemic circulation. Let's review what you already know about circulation and blood flow, in parts. Let's think about cardiac blood flow, to review the sequence of chambers, valves, and even the electrical activity that produces *myocardial* contraction. Then let's talk about systemic circulation, taking a closer look at tissue gas

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exchange. Finally, let's review pulmonary circulation.

To begin with ***blood flow through the heart***, venous blood returns to the atria. When the SA node fires, the atria quickly depolarize and contract (P wave), pushing blood through the AV valves. There is a slight hesitation of the electrical impulse at the AV node (P-R segment) so that the atria can force as much blood into the ventricles as possible. The AV valves close (lub), and there is rapid depolarization of the bundle of His, right and left bundle branches, Purkinje fibers, and ventricular myocardium (QRS complex). Contraction of the ventricles forces blood through the aortic and pulmonic valves into the aorta and pulmonary artery respectively (systole). When empty, the ventricles relax and repolarize (T wave), while the aortic and pulmonic valves close (dub; diastole).

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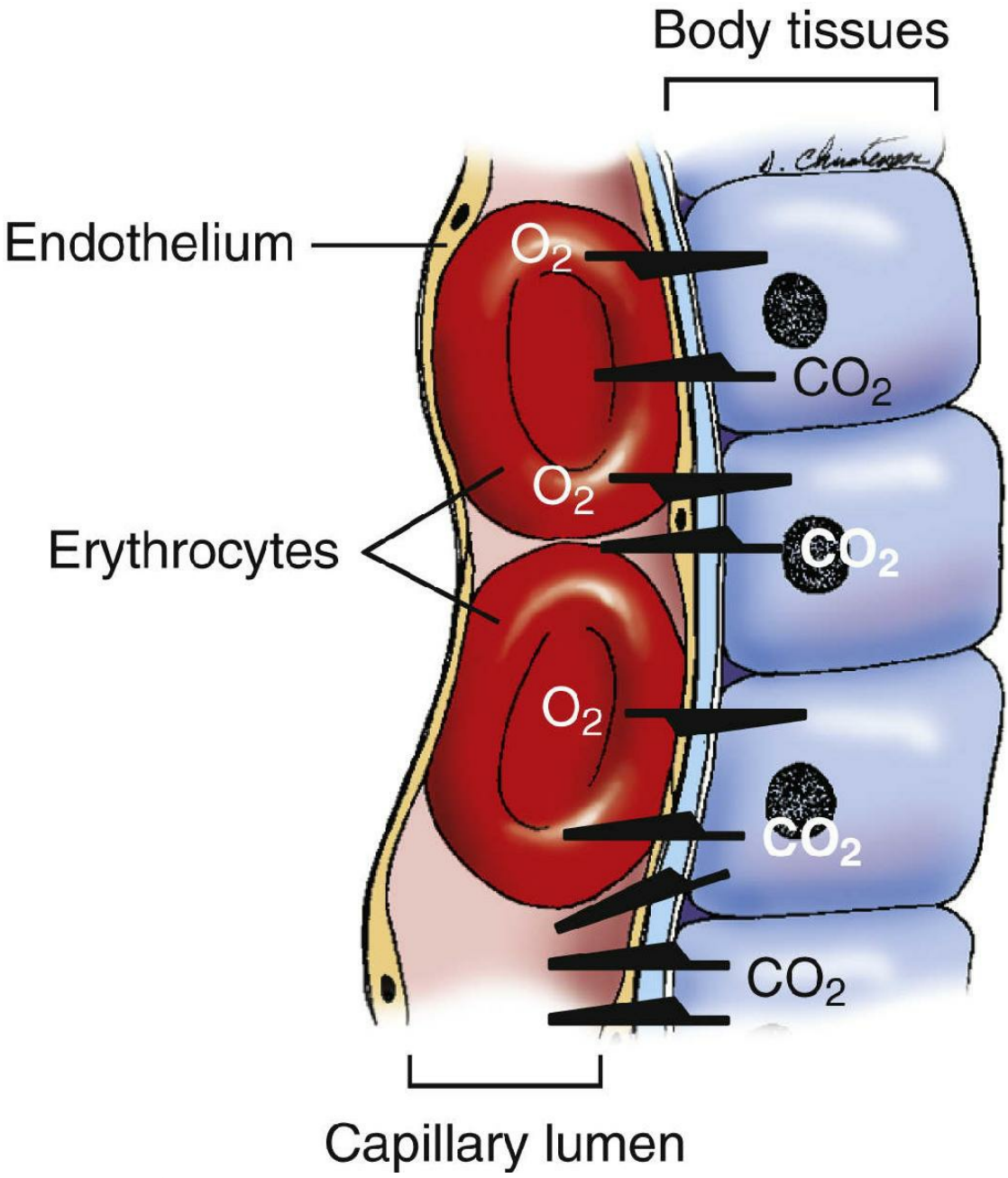

FIG. 5.16 Tissue capillary and systemic gas exchange.

Let's pick up ***systemic circulation*** with the blood entering the aorta from the left ventricle. The highly oxygenated aortic blood is diverted to smaller arteries that carry it throughout the body to most organs and tissues. The arteries continue to branch, becoming progressively smaller as they approach the tissue level. Arterioles are the smallest of all arteries that carry blood into the capillary beds of the systemic tissues. As the blood passes through the capillaries, oxygen diffuses out of the blood into the tissues and carbon dioxide diffuses from the tissues into the blood (Fig. 5.16).

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That deoxygenated blood flows from the capillaries into tiny venules that lead to veins. The small venous branches progressively become larger and converge on larger veins. Ultimately all systemic venous blood flows into the vena cava. The vena cava pours its blood into the right atrium. And per the previous review, you know that this blood flows through the right AV valve (tricuspid), into the right ventricle, and then is pumped through the pulmonic valve, into the pulmonary artery.

Let's pick up ***pulmonary circulation*** in the pulmonary artery. The pulmonary artery quickly bifurcates (divides) into right and left branches carrying blood to the lungs. Okay, let's pause. This is a place that often trips folks up. Arteries are supposed to carry oxygenated blood. But the blood at this point in circulation (i.e., in the pulmonary artery) has not picked up oxygen yet. It's on its way there but hasn't done it yet. The pulmonary artery is the *only* artery, under normal circumstances, that carries deoxygenated blood. This ***deoxygenated blood*** is diverted to numerous smaller arteries that disburse the blood through the lungs. When nearly at the alveoli (air sacs) the blood passes through arterioles. The arterioles direct blood to capillary networks that surround each alveolus (air sac). As blood passes through these pulmonary capillaries, CO_2 rapidly diffuses out of the blood and into the alveoli, while O_2 diffuses into the blood (Fig. 5.17). The now highly oxygenated blood leaves the capillary beds through pulmonary venules and progressively to larger and larger pulmonary veins. The largest of the pulmonary veins carry the oxygenated blood to the left atrium. From there it passes through the left AV valve (mitral, bicuspid), into the left ventricle, and is pumped out through the aortic valve, into the aorta.

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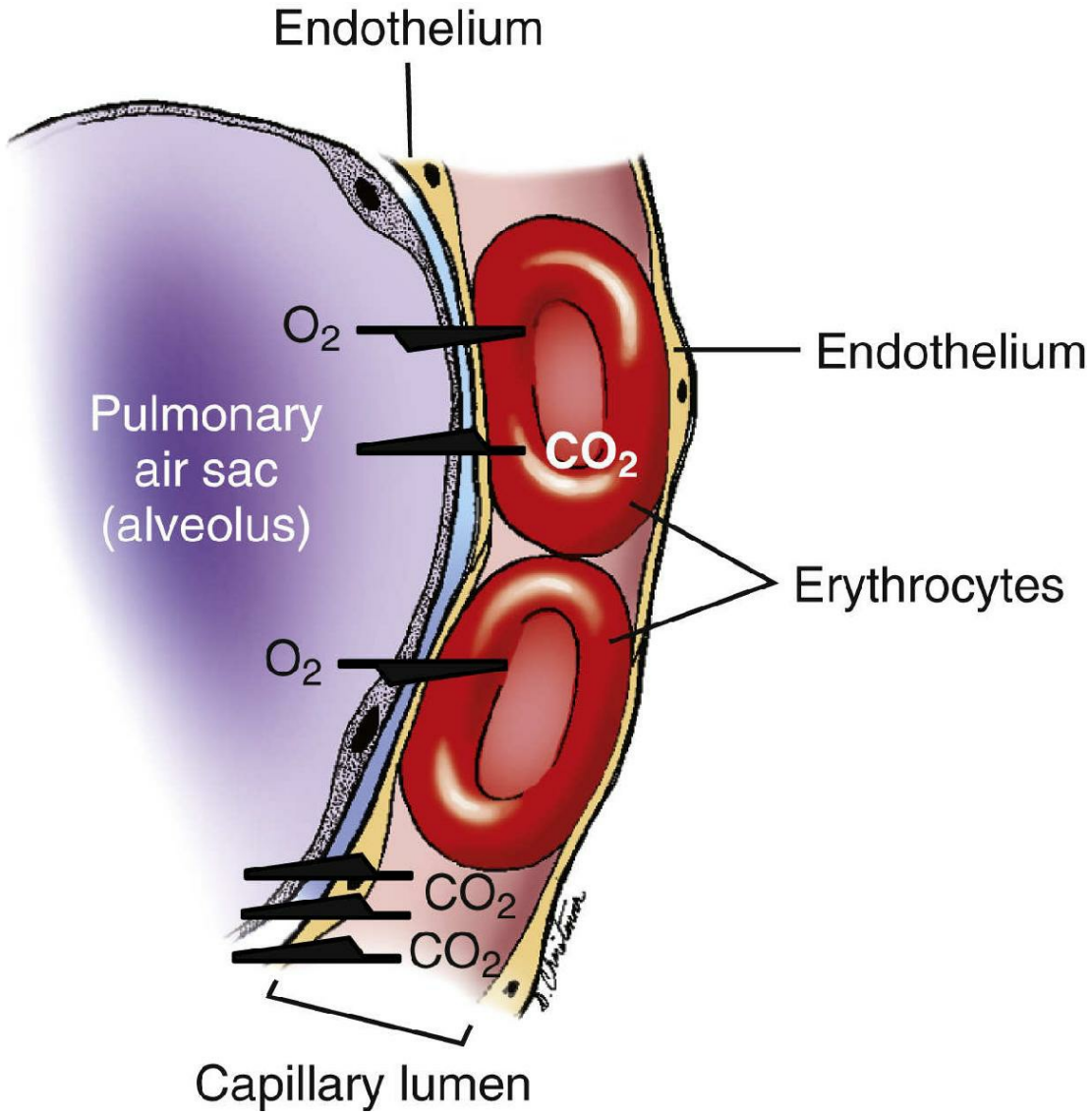

FIG. 5.17 Pulmonary capillary and gas exchange.

Think of the number of times these circuits and activities take place, minute to minute, hour to hour, day to day, and over the course of a lifetime. This brief review alone should help you understand how interdependent the heart, lungs, and body tissues are—all intimately connected by a series of arteries, capillaries, and veins. And the flow of blood on its regular circuit through the vasculature depends upon maintenance of blood pressure.

Blood Pressure

Blood pressure, *adequate* ***blood pressure***, is absolutely essential for life. That is why so many systems and ***homeostatic*** [*home(o)-* unchanged + *stat(o)-* standing + *-ic* pertaining to] mechanisms

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integrally involved in maintaining adequate blood pressure. That is also why blood pressure is being discussed not just once in this text. It is discussed in detail in multiple chapters of this book. It is being discussed in detail here, and in Chapters 6 and 10. It is also mentioned in Chapters 7 and 11. Each chapter discussion focuses on blood pressure from the perspective of the body system at hand, within the context of the whole. No matter the vantage point, the goal is to preserve the whole—life itself. I hope that the repetition provides you with a thorough understanding of, as well as a deep appreciation for, maintenance of blood pressure.

Blood pressure is complex. There are so many events and factors that influence it, like hydration status and blood volume, heart rate, cardiac stroke volume and strength of contraction, nerve input, vessels, and numerous hormonal controls. And nearly all of the events and mechanisms involved in blood pressure ***homeostasis*** [*home(o)-* same, unchanged + *-stasis* state of standing; i.e., equilibrium] are directed at the *cardiovascular system*. So let's make the heart and vasculature our starting point. How do the heart and vessels affect blood pressure? We'll answer that step by step. Once we gain an understanding of cardiovascular effects, then we can consider how other body-system influences can contribute to those effects.

Let's begin by looking at the heart and ***cardiac output***. You are already familiar with *systole*, when the ventricles contract to pump blood to the lungs and the rest of the body. This is the phase of the cardiac cycle that produces each peripheral pulse. But we cannot limit our view of cardiac output to a single snapshot of time and the volume of blood pumped from the heart in that one heartbeat. There is more to it than that. First, there is the volume of blood itself. There needs to be an adequate volume of blood in the ventricles in the first place. This, in part, is why it's important for the atria to squeeze as much blood as possible into the ventricles. For that, we need good strength of atrial contraction and that electrical pause at the AV node. Then there is the ***blood volume*** itself in general circulation and returning to the heart.

What affects the actual circulating blood volume? Well, ***hydration*** status for one thing. We said inChapter 2 that roughly 70% of the body is water. That water is present throughout all of the

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tissues of the body, and most certainly in the blood. Numerous factors maintain just the right volume of water in the bloodstream. The kidneys are targeted to limit the amount of water lost in the urine. Minimizing the amount of actual (urine) filtrate produced helps with water conservation. And because where sodium goes, water follows, the kidneys are also targeted to manage reabsorption of sodium, in part for water conservation. The digestive tract is targeted to reabsorb as much water as possible, to minimize the amount of water lost in the feces. And thirst centers in the brain are stimulated to make the animal drink more water. So, hydration is important in maintaining blood volume. But truth be told, even with perfect hydration status, there is simply not enough blood to fill all of the vessels of the body. It's true. If all of the vessels of the body were completely relaxed and fully dilated, there would not be enough blood to fill them.

So, the body maintains "***vascular tone***." Because this *vascular tone* is controlled by the sympathetic branch of the nervous system automatically, it is often referred to as ***"sympathetic tone"*** Most vessels of the body are rarely fully dilated, especially arteries. (They are the most muscular, remember.) The smooth muscle in vascular walls (arteries and veins) are always in various states of contraction. Some vessels are more constricted than others at any given moment in time. Blood is always routed to the most important tissues and organs in that moment in time. For instance, following a meal, ***vasodilation*** routes blood to the digestive tract to support digestion. At the same time, ***vasoconstriction*** diminishes blood flow to areas of the body nonessential for the digestive process (e.g., the brain and major muscles). This plays a role in making us sleepy following lunch, during that afternoon class. (Yeah, I've seen it and experienced it myself.) Ah, but if something frightens us, our bodies will sacrifice lunch for self-preservation.

In ***fight or flight*** (discussed in detail in Chapter 11), those tissues and organs of the body that are **not** essential for survival in that moment in time are sacrificed. Digesting lunch in a life-threatening situation is not top priority of the day. In sympathy for our survival, the *sympathetic* branch (get it?) of the nervous system targets key vascular areas. Arteries and arterioles to the skin and digestive tract experience profound *vasoconstriction*. Have you

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heard people say that someone "turned white as a sheet" or that "the color drained from her face"? It's all because of this necessary *peripheral vasoconstriction*, all geared toward maintaining an adequate circulating blood volume. In contrast, *vasodilation* occurs in the lungs and large muscles. In fact, even the airways dilate. How is this beneficial? We called this *fight or flight*, right? Well, we need use of great muscles to either fight the threat or run away from it. And we need better gas exchange for oxygenation and removal of CO_2 to support that activity. And to give us clarity of mind to make the decision to fight or flee, improved carotid arterial blood flow supports the brain. By the way, the sympathetic nervous system cannot sustain this response. So, blood supply to the ***adrenal glands*** (so named because they are near [*ad-* near, toward] the kidneys [*ren(o)-* kidney]) is also increased.

We'll talk about the ***adrenal glands*** in detail inChapter 10. For the purpose of our discussion here, we'll focus on the key adrenal hormone, ***epinephrine*** (or adrenaline). You see, when the sympathetic nervous system kicks into high gear, it also stimulates the adrenal glands. In response, the adrenal glands secrete *epinephrine*. There are specific ***adrenergic*** [*adren(o)-* adrenal + *erg(o)-* work, working + *-ic* pertaining to] ***receptors***, along vascular walls, that respond to *epinephrine*. Now, there is always a low level of epinephrine secreted by the adrenal glands. This helps to maintain that *vascular tone* we mentioned earlier. But in the case of fight or flight, a huge bolus of epinephrine is secreted and binds with more receptors along the vasculature. ***Beta adrenergic receptors*** are located in key areas and promote *vasodilation*. So there must be an abundance of *beta adrenergic receptors* along arteries and arterioles leading to great muscles, the lungs, and the brain, right? Right. That's how we increase circulation to those important areas. Beta-adrenergic receptors are also responsible for ***airway dilation***. Do you remember our discussion of the ***spleen*** inChapter 3? We said that at any given moment in time, the spleen may store roughly 30% of the circulating blood volume. Well, guess where we have more beta-adrenergic receptors—the spleen. And during fight or flight, stimulation of those receptors causes the spleen to contract, forcing stored blood into circulation, directly increasing blood volume. Remember, "*Beta is better*." In contrast, stimulation of

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alpha-adrenergic receptors by epinephrine causes *vasoconstriction*. Can you guess where we have an abundance of *alpha-adrenergic receptors*? Yep, along vessels in the skin and along the digestive tract, as well as other *nonessential areas*. And this is how, even in normal circumstances, vessels contribute to blood pressure homeostasis.

All of this increases the available blood volume for the ventricles to pump. But we said that volume is only one piece to the puzzle of *cardiac output*. ***Heart rate*** also plays a role. The faster the heart pumps (within reason, of course), the better the circulation and blood pressure. Did we not say that during ventricular systole (contraction) there is tremendous pressure exerted within the arteries? This is especially true for the aorta and pulmonary arteries. And because the left ventricle is more muscular, this is especially true for the aorta. We also said that for every beat of the heart, a peripheral pulse is created, right? That sensation is the transfer of *systolic pressure*, passed along from the left ventricle and aorta all the way to the peripheral artery we're palpating to detect that pulse. So by increasing heart rate, the transference of those systolic pressures throughout the body increases blood pressure. Simply put, increased heart rate over a window of time increases *cardiac output*. Increased cardiac output increases blood pressure.

Now let's look at cardiac output in light of fight or flight. Have you ever been frightened—say, by a sudden noise or by having to speak in front of a large group of people? Your heart probably raced and felt like it was going to beat out of your chest, didn't it? No matter the cause of our fear, founded or unfounded, that fight or flight response is engaged. And both the sympathetic nervous system and epinephrine dramatically affect cardiac output. In fact, *epinephrine* has what we call ***positive inotropic*** [*in(o)-* from Gr. *inos* fiber + *trop(o)-* influence + *-ic* pertaining to] ***effects*** on the heart. Well, if epinephrine has a *positive influence* on the heart, then there must be *beta-adrenergic receptors* on the heart, because "beta is better." And that's absolutely the case. Stimulation of these beta adrenergic receptors causes *vasodilation* of the *coronary arteries* to sustain the myocardium for a more intense "workout." And with regard to that workout, *heart rate* increases. *Strength of contractions* (myocardial contractility) also increase dramatically. Bottom line,

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cardiac output dramatically increases in response to the *positive inotropic effects* of *epinephrine*—from increased ***stroke volume***, increased ***heart rate,*** and increased ***contractility***. And all of that results in improved blood pressure. When the threat is gone or neutralized, epinephrine in the system dissipates and the parasympathetic branch of the nervous system takes the body's foot off the throttle and "hits the brakes," to slow down the heart and reduce all of its rigorous activity.

Of course, the body is not a "one-trick pony." We certainly can't rely on epinephrine alone. We said that we had built-in redundancies for blood pressure homeostasis. What are some of these redundancies? Well, because adequate blood pressure is absolutely essential for kidney function, believe it or not, the ***kidneys*** are going to contribute to blood pressure homeostasis. First, in response to ***antidiuretic*** [*anti-* against + *diure(o)-* urination + *-tic* pertaining to] ***hormone*** (***ADH***) from the pituitary gland, the kidneys reduce urine output. But ***renal*** [*ren(o)-* kidney + *-al* pertaining to] contributions to blood pressure go well beyond simply conserving water for the body. You see, another name for *ADH* is ***vasopressin*** [*vas(o)-* vessel + *press(o)-* pressure + *-in* the]. And *vasopressin* causes *vasoconstriction* of *renal arterioles* (thereby reducing water excretion), as well as other vessels in nonessential parts of the body. We've already said that vasoconstriction, like that, increases blood pressure.

Plus, the kidneys actually secrete hormones of their own that act on blood vessels. (The kidneys are rather self-serving.) You see, in response to adrenergic stimulation, kidneys secrete a hormone called ***renin*** [*ren(o)-* kidney + *-in* the; i.e., the kidney hormone]. In the presence of *renin*, ***angiotensinogen*** [*angi(o)-* vessel + *tensin(o)-* tension, pressure + *gen(o)-* producer] is transformed into the hormone ***angiotensin I***. That hormone has no direct effect. *Angiotensin I* is the *inactive* precursor for the active hormone ***angiotensin II***. To convert angiotensin I into angiotensin II, ***angiotensin-converting enzyme*** (***ACE***) is needed. Once *ACE* has provided us with angiotensin II, we target areas like the brain, kidneys, and peripheral blood vessels. ***Angiotensin II*** increases *thirst*, causes *peripheral vasoconstriction*, increases secretion of *vasopressin* (ADH), increases secretion of *norepinephrine* by the

adrenal glands (with similar adrenergic effects), and increases *sodium retention* by the kidneys. All of this serves to increase blood pressure—a win-win for the kidneys and the rest of the body.

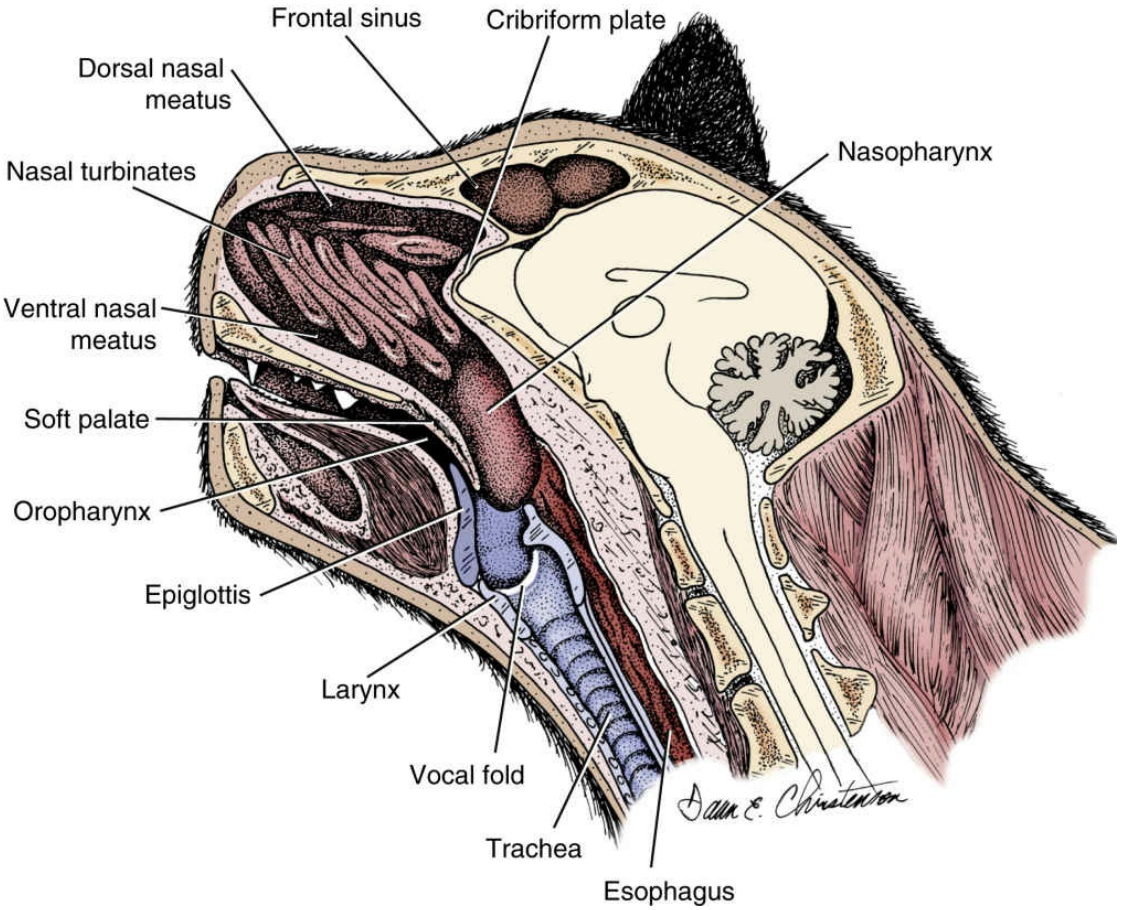

FIG. 5.18 Sagittal feline head.

I know this is a complicated story. Believe it or not, this is not complete. We'll talk about other factors in Chapters 6 and 10. Later in this chapter, we'll refer back to many of these homeostatic mechanisms when we discuss various cardiopulmonary diseases. I hope those discussions will help clarify blood pressure for you. Remember, the overarching need for adequate blood pressure is to sustain life by circulating blood for the delivery of oxygen and other nutrients and for the removal of wastes. And that sounds like a great segue to taking a closer look at the respiratory tract. In fact, this should give you just the *breather* you need right now (pun most definitely intended).

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Respiratory Tract

If the primary purpose of the cardiovascular system is to circulate blood, then the primary purpose of the ***respiratory tract*** must be gas exchange. As we said a moment ago, the cardiovascular system circulates blood for the delivery of oxygen and the removal of wastes, namely carbon dioxide. And that is why *cardiopulmonary* function goes together like bread and butter.

But I think after our challenging discussion on blood pressure, it might be wise to investigate some less-complex features of the respiratory tract first. We might as well begin with the nose.

Nose and Nasal Passages

It's my humble opinion, but I think noses are cute—especially certain animal noses. Think about bunny noses and the way rabbits wiggle them. That makes me smile. And I don't think anyone can argue about how cute that is. (Hold onto the bunny visual for a moment. It might help you to de-stress, after that blood pressure discussion.)

Noses come in all sorts of shapes and sizes. But they all have some things in common. Every nose has two holes called ***nares*** (nār'ēz) or ***nostrils***. (A single ***nostril*** is a ***naris***.) The *nares* funnel air into the ***nasal*** [*nas(o)-* nose + *-al* pertaining to] ***passages***. Those passages are separated right down the middle of the muzzle by the ***nasal septum***. And if you look at Fig. 5.18, you'll see that the nasal passages are not just hollow tubes. There are many thin folds of cartilage and bone, covered by mucous membranes, called ***nasal turbinates***.

Why on earth do we have these *turbinates* taking up space in the nasal passages? Well, the mucous membranes of the *turbinates* have three very important functions that protect the lower airways. First, the mucous membranes are highly *vascular*, with lots of *capillaries*. That rich blood supply exposes air to *warmth* from the core of the body, *warming* the inspired air. This is especially important during cold weather. Lower airways tend to constrict when exposed to cold air, to protect the alveoli (air sacs in the lungs) and to minimize

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loss of heat from the body. Plus, cold air tends to be quite dry. The colder the air, the less moisture it can support. And there are desert regions that can see extremes in temperature, from well below freezing to triple-digit high temperatures. Ah, but it's a dry heat, right? Well, hot or cold, we don't want the lower airways, especially the alveoli, to dry out. That brings us to the second purpose of the turbinates. Because the mucous membranes are *moist*, as air passes over and through all of those folds, the inspired air is *humidified*. This keeps the lower airways from drying out. Finally, things stick to moist surfaces, right? And there are cells to secrete ***mucus*** [L. "slime"] that coats the mucous membranes of the turbinates. Stop. Did you catch that? Mucus (ending in -us) is slimy stuff and mucous (ending in -ous) is an adjective used to refer to something that is covered by slime, like a mucous membrane. Getting back to our discussion, the final purpose of the nasal turbinates is to *filter* the inspired air using sticky mucus. Dust, pollen, and other particles stick to the mucus covering the turbinates. This protects the lower airways and lungs from particulate matter. So, why have turbinates in the nasal passages? To warm, humidify, and filter inspired air.

What happens to all of the gunk that gets stuck in the mucus? Well, the superficial cells of the mucous membranes in the nasal passages are ***ciliated*** [from L. *cilium* "eyelash"] ***pseudostratified*** [*pseud(o)-* false + *stratified* layered] ***columnar*** [*column* from L. *columna* "pillar" + *-ar* pertaining to] ***epithelium***. As you can see in Fig. 5.19, these hairy little cells falsely appear layered because their nuclei are not lined up neatly in a straight row. The constant, coordinated movement of the cilia of all those cells continuously sweeps and moves the mucus and everything trapped in it. Ultimately, everything is swept ventrally and caudally to the *pharynx* (throat), where it is intended to be swallowed. Of course, some irritants and large objects need to be removed by greater force, back out the way they came in. And that's where ***sneezing*** comes into play. Sneezing is an involuntary reflex, designed to forcefully remove irritants from the nasal passages.

I'll never forget the Basset Hound (no, not mine) that I saw early in my career. The dog had been sneezing repeatedly for a couple of days. The owners finally brought him in to be evaluated. Yep, he

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was sneezing—a lot! He sneezed so hard at times that his poor nose hit the floor. But aside from the sneezing, the dog was fine. There was only a slight, unilateral, watery nasal discharge. We were convinced there had to be some kind of foreign material in his nasal passage. That's not unusual, especially for a low-to the ground scent hound, like a Basset. He could have easily snorted up a grass awn or other such thing. The nose is a very sensitive thing, and his was already irritated to the max. So, we anesthetized him to do a ***rhinoscopy*** [*rhin(o)-* nose + *-scopy* a viewing of]. It didn't take long to find the foreign object. It was not a seed or a grass awn. In fact, it was not at all what we expected. What we found was a stick. But this was no tiny stick. It was nearly 4 inches long and as big around as a pencil! That was well beyond the capabilities of those little ciliated cells and mucus! I am still astounded that he didn't damage his turbinates to create ***epistaxis*** (ep″ĭ-stak'sis [Gr. *epistaxis* "nosebleed"]). Amazingly, he didn't bleed after we pulled the stick out either. He certainly felt better when he recovered from anesthesia that day—no more sneezing.

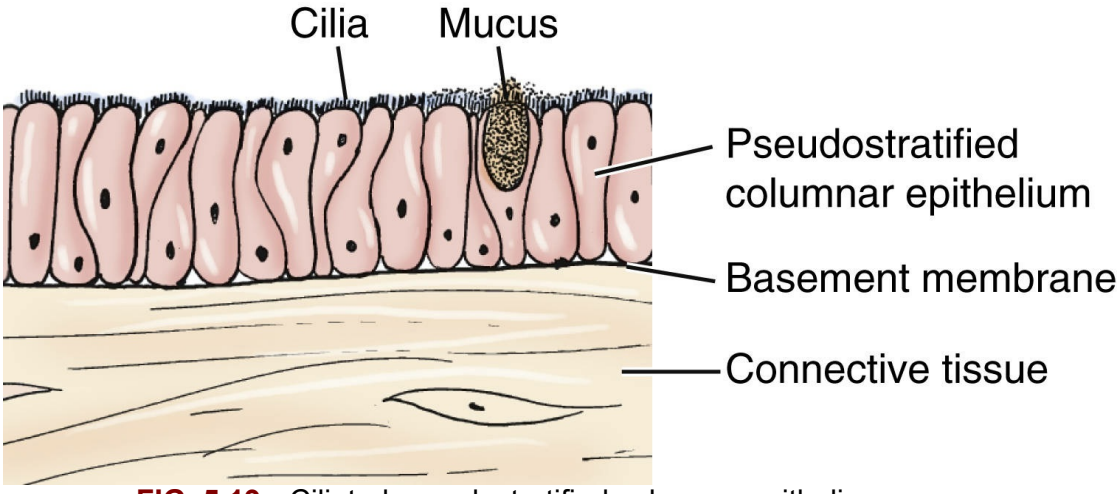

FIG. 5.19 Ciliated pseudostratified columnar epithelium.

In retrospect, if I had to guess, I'll bet that Basset shoved the stick into either the ***dorsal*** or the ***middle nasal meatus*** (me-a'tus [L. *meatus* "a way," "path"; i.e., a passageway]). Only the dorsal and the ***ventral nasal meatus*** are shown in Fig. 5.18. It's a fairly straight path into the dorsal and middle passages. And putting the stick in one of those larger passages would avoid damaging the turbinates.

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But both of those passages end abruptly at the ***cribriform*** [from L. *cribrum* "sieve" + *-form* shaped] ***plate***. So the stick must have stopped just short of the *cribriform plate*. You see, that plate of bone, separating the nasal passages from the *cranial vault*, is covered in highly vascular mucous membranes, just like the turbinates. So the stick must not have struck those mucous membranes either. Otherwise there would have been bleeding.

You must be wondering about a couple of things I mentioned just now. Like, if the turbinates are so important, why have three large passageways? Well, first, they're not that large. And second, we need someplace to sweep the contaminated mucus from the turbinates. The larger passages provide such places. And by the time all of that slimy stuff reaches the ventral nasal meatus, it's in a perfect location to be swept into the ***pharynx*** (throat) to be swallowed. Plus, the *ventral nasal meatus* also provides a great clinical use too. That's the passage we use when placing a ***nasogastric*** [*nas(o)-* nose + *gastr(o)-* stomach + *-ic* pertaining to] ***tube***. We simply slide the tube into the nose and through the ventral nasal meatus to the pharynx. When the animal swallows, we simply advance it until it's in the stomach. No, it's not as easy as it sounds. Most animals object to having a tube snaked through their noses. But there are times when we need a sure, accurate way to deliver nutrients and medications. And because animals would chew through an ***orogastric*** [*or(o)-* mouth + *gastr(o)-* stomach + *-ic* pertaining to] tube, the nose is the next best option. Once a *nasogastric tube* is secured in place with a couple of sutures, most animals tolerate them pretty well. It's the initial placement that they tend to object to.

The other thing you may be wondering about is the ***cribriform plate***. What on earth is a sieve-shaped bone, and why is it there? A sieve is a common kitchen utensil that is also commonly called a colander. That utensil is full of holes to drain pasta and produce. The *cribriform plate* is full of holes too. But it doesn't drain anything. Those holes are there as passages for nerves, specifically sensory nerves associated with the *sense of smell*. In the nasal passages are thousands of sensory nerve endings. When a dog sniffs, scent molecules are drawn into the nasal passages. And with all of the twists and turns through the turbinates, those molecules are

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brought in close contact with the sensory nerve endings enabling stimulation of them. Those nerves pass through the holes of the cribriform plate to the ***olfactory*** [from L. *olfacere* "to smell"] ***bulbs*** of the brain. The scent is then interpreted by the brain. By the way, the olfactory bulbs of most animals are much larger than those of people. And that is partly why animals have such an amazing sense of smell, especially scent hounds like that dear Basset Hound who had the stick up his nose.

Sinuses

What are sinuses and what purpose do they serve, aside from giving us headaches from time to time? Good questions. A ***sinus*** [L. *sinus* "a hollow"] is literally a hollow space. These empty spaces are lined with mucous membranes, keeping the space warm and moist. There are two major sinuses that we need to be familiar with. The ***frontal sinus*** is formed by the frontal bone of the skull. On us, that's found at our foreheads. It's shown in the cat in Fig. 5.18. But compare that tiny sinus to the bovine frontal sinus shown in Fig. 5.20. That's huge! And the size of that sinus makes clear its purpose —to make the skull lighter. Imagine if all of that space was solid bone. That would make it pretty exhausting for the animal to hold its head up, assuming it could lift it at all. So, being an "airhead" is actually a very good thing. We'll talk about the frontal sinus again inChapter 8, when we talk about dehorning. Notice how the sinus openly communicates with this adult's horn. That's a problem if we try to dehorn this animal at this stage. The open end would be whistling in the wind.

The other sinus to be familiar with is the ***maxillary sinus***. If you go back toChapter 4 and look at Fig. 4.27, you can see where the maxilla is. And the *maxillary sinus* is found under that cheek bone, just dorsal to that really big tooth and rostral to the zygomatic arch. It's not a very big sinus. Still, it helps lighten the skull. We'll talk about it again inChapter 7, when we talk about dental problems, associated with that really big tooth in its vicinity. A tooth root abscess there can create a nasty ***sinusitis*** [*sinus* + *-itis* inflammation of] for the animal.

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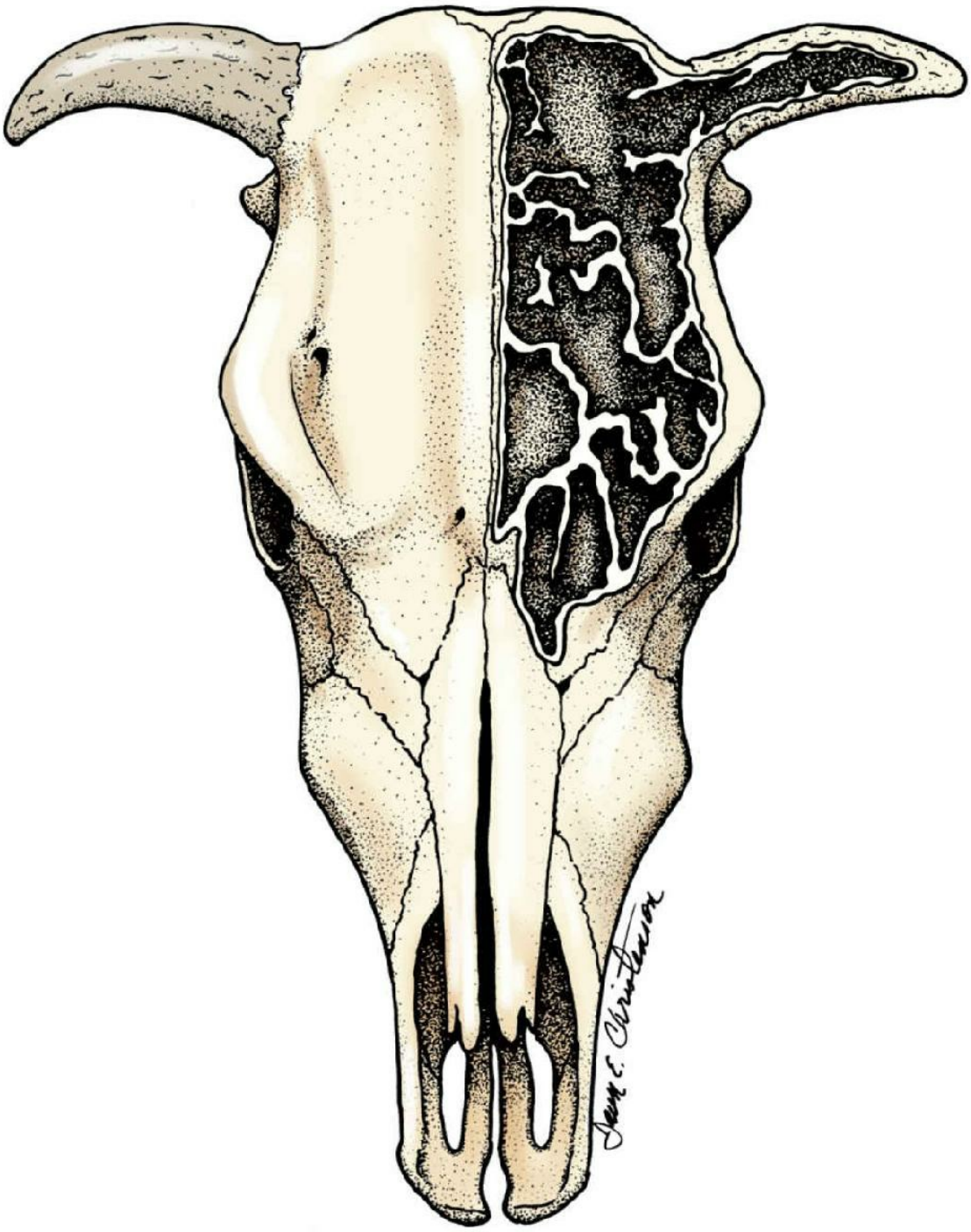

FIG. 5.20 Bovine frontal sinus.

Upper Airway

The ***upper airways*** include the nasal passages, and we already talked about those. We also mentioned the ***pharynx*** [Gr. *pharynx* "the throat"]. But we didn't mention subdivisions of the *pharynx*. You see, the rostral region of the throat is divided by the ***soft***

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palate, shown in Fig. 5.18. The area dorsal to the soft palate is the ***nasopharynx*** [*nas(o)-* nose + *pharynx* throat] and the area ventral to the soft palate is the ***oropharynx*** [*or(o)-* mouth + *pharynx* throat]. But why are the subdivisions of the throat important? It's all about function. When an animal breathes in (inspires), the soft palate is relaxed and drops ventrally. This directs inspired air from the nasal passages through the *nasopharynx* toward the *laryngeal opening*. But when the animal swallows, both the nasal passages and the lower airways need to be protected. And the job of protecting the nasal passages falls to the soft palate. During swallowing, muscles in the soft palate contract, lifting it dorsally to seal off the nasal passages. Is this fool-proof? No. You know this if you've ever laughed while drinking a beverage. A portion of what you were drinking probably came out of your nose. But protection of the nasal passages pales in comparison to protection of the rest of the airways and lungs. And that's where the larynx comes in.

Larynx

The ***larynx*** is the entryway to the trachea and lower airways. This is a very important "gatekeeper." If the larynx fails in its duties, there can be lethal consequences. The *larynx* is a chamber made of a number of ***chondral*** [*chondr(o)-* cartilage + *-al* pertaining to] ***plates***, muscles, and connective tissues. Its interior is covered by mucous membrane. Functionally, it's really the opening to the larynx that is most important. Fig. 5.21 shows our typical view of the pharynx and ***laryngeal*** [*laryng(o)-* larynx + *-al* pertaining to] ***opening*** (also called the ***glottis***). As you can see, the *laryngeal* opening has a number of parts. The actual margins of that opening are formed by the ***arytenoid*** [from Gr. *arytaina* ladle + *-oid* resembling] ***cartilages***. I always thought that was a really strange name for those cartilages. But I guess they really do look something like tiny ladles, if I use a little imagination. The rigidity of the *arytenoid cartilages* is important. They form the "door-jam" for the laryngeal opening. The shape of the arytenoid cartilages corresponds to the contours of the ***epiglottis*** [*epi-* on, upon + *glottis*]. The *epiglottis* serves as the "door" for the laryngeal opening (aka. glottis). Now does the name epiglottis make sense (*epi-* upon)? By having a rigid, sturdy door jamb (arytenoids) contoured to the shape of the door (epiglottis),

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when the door (also made of stiff cartilage) closes it creates a very good seal. But there is a little more to it than that. You see, during *swallowing*, the arytenoid cartilages are drawn closer together by adductor [*ad-* toward + *duct(o)-* to draw, lead + *-or* pertaining to; i.e. drawn medially] muscles, making the laryngeal opening smaller. At the same time, the epiglottis is drawn dorsally and caudally, to cover and form a tight seal against the arytenoids. When breathing in, the *arytenoids* are ***abducted*** [*ab-* away + *duct(o)-* to draw; i.e., drawn away laterally] and the epiglottis is drawn ventrally, away from the opening. This makes the laryngeal opening as *patent* (open) as possible for free movement of air.

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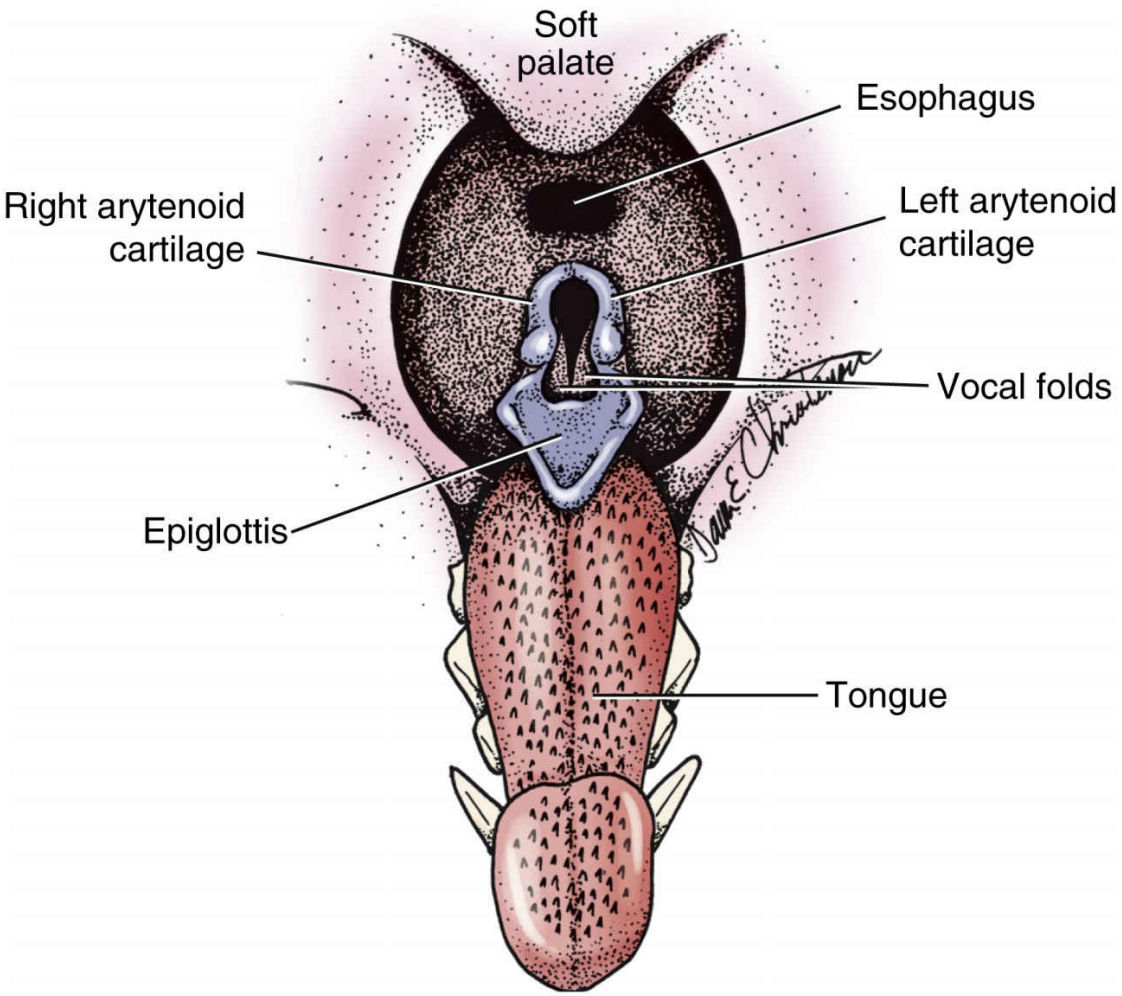

FIG. 5.21 View of the feline pharynx and laryngeal opening.

If you're noticing the ***vocal folds,*** or ***vocal cords,*** as they are often called, I'll bet you are wondering how they work. Well, during ***expiration*** [*ex*-out + *spira* breathing + *-tion* act of; i.e., exhaling, breathing out], muscles of the larynx may be used put tension on the vocal folds. As the expired air rushes past the taut vocal folds, they vibrate, creating sound. That's how we and animals *vocalize*. It's much like holding a blade of grass tightly between your thumbs and bases of your hands and blowing across the blade. It vibrates and makes noise. Through learned behavior, people and animals can consciously control the tension of the vocal folds as well as the volume and speed of expired air passing over them, to create different tonal qualities.

Brachycephalic syndrome

While we're in the neighborhood, let's talk briefly about some

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common anatomic problems. ***Brachycephalic*** [*brachy-* short + *cephal(o)-* head + *-ic* pertaining to] animals (e.g., English Bulldogs, Pugs, Boston Terriers, Persian cats) have, as the name implies, very short head structure. And it's really the muzzle that's been significantly shortened, making them look like they ran into a brick wall with their faces. While the skull itself through the muzzle has been shortened, not all of the soft tissues have been reduced. And the excess tissues tend to cause problems with many of these animals, resulting in what we refer to as *brachycephalic syndrome*. These animals often have difficulty moving air through the nasal passages and the pharynx. This tends to be more of a problem, even requiring surgical intervention, in *brachycephalic* dog breeds.

The first common anatomic abnormality for brachycephalic dogs and cats is their ***stenotic*** [*sten(o)-* narrow + *-tic* pertaining to] ***nares***. Some of these animals can barely inspire through their noses, because the nostrils are so small. (You know what this is like if you've ever had a bad cold or allergic ***rhinitis*** [*rhin(o)-* nose + *-itis* inflammation of]. You can't breathe through your nose because it's too stuffy from all of the swelling and excess mucus produced by the *rhinitis*.) For the *brachycephalic* animals, they have structural abnormalities making it difficult if not impossible for them to breathe through their *nares*. That means they have to breathe through their mouths. That's not good. Remember, the *nasal passages* are designed to *warm, humidify, and filter inspired air*. By open-mouth breathing, these animals bypass the nasal passages, bypassing the intended benefits. It can lead to lower airway disease. Normal mucus secretions dry up and become thick and sticky. That can actually obstruct smaller airways in the lungs. And that creates huge problems. So it is important to correct the abnormal nasal anatomy. ***Rhinoplasty*** [*rhin(o)-* nose + *-plasty* reconstruction of] is used to correct and remove the *stenosis*. Yep, it's a "nose-job." But it's not purely cosmetic "plastic" surgery. A *rhinoplasty* in a *brachycephalic* animal corrects an anatomic defect so they can actually close their mouths to breathe normally.

But this is usually just the "tip of the iceberg." Brachycephalic animals usually have ***pharyngeal*** [*pharyng(o)-* pharynx + *-al* pertaining to] problems too. Excess tissue in the *pharynx* reduces space for air movements. This is part of the reason these animals

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make so much ***stertorous*** [L. *stertor* snore + *-ous* pertaining to] noise when they breathe. ***Stertor*** is the loud snoring noise they make; except they snore even when they're wide awake. The sound is from excess air turbulence and pharyngeal tissues flapping and rattling around. And one of the biggest contributing factors to this is an ***elongated soft palate***. While some folks might think that the *stertor* is an endearing quality of their Bulldog, it can create serious upper airway obstruction or put the dog at risk for aspiration of foreign material into the lower airways. Sometimes the soft palate is so long, it actually blocks the laryngeal opening. If the laryngeal obstruction is not relieved, usually through coughing, the dog can die. And those moment-to-moment, breath-to-breath repeated obstructive episodes lead to ***edema*** [Gr. *oidema* swelling] of involved tissues. *Edema* further impairs air movements, especially inspired air. That can reduce the animal's ability to oxygenate adequately. Even if the elongated soft palate only partially obstructs the laryngeal opening, ***inspiration*** [*in-* in + *spira* breathing + *-tion* act of; i.e., inhaling, breathing in] is impaired. Plus, this prevents the epiglottis from creating a good seal against the arytenoids. That makes it really easy for the dog to aspirate foreign material. And that can result in life-threatening ***pneumonia*** [*pneumon(o)-* lungs + *-ia* condition of].

So, ***palatoplasty*** [*palat(o)-* palate + *-plasty* reconstruction of] is often required to restore free movement of air and normal laryngeal function. We literally remove a portion of the soft palate. But this may not completely correct the air movement problem. Remember the swelling we mentioned? Well, sometimes other pharyngeal tissues become ***edematous*** [*edema* swelling + *-tous* pertaining to] too, like the ***tonsils***. If you need to review the location of tonsils, please refer toChapter 3. With ***tonsillitis*** [*tonsill(o)-* tonsil + *-itis* inflammation of] the tonsils swell tremendously and bulge out of the ***tonsillar*** [*tonsill(o)-* tonsil + *-ar* pertaining to] ***crypts*** [L. *crypta* hidden]. They take up space in the pharynx and contribute to reduced and turbulent airflow, even with open-mouth breathing. So, we may also have to perform a ***tonsillectomy*** [*tonsill(o)-* tonsil + *-ectomy* to cut out, removal of] on these animals.

Inflammation and edema affect the larynx too. Have you ever had ***laryngitis*** [*laryng(o)-* larynx + *-itis* inflammation of]? If you

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have had it or know someone who has, you know that one of the classic signs of *laryngitis* in people is vocal hoarseness or even loss of voice. That makes it obvious that the vocal folds are inflamed. And the inflamed, *edematous* vocal folds simply can't vibrate normally to create normal vocalization. Big deal? No, loss of vocal ability is not a big deal for a Bulldog. Even a normal Bulldog will never sing at Carnegie Hall or with the Metropolitan Opera. But the inflammation in the region of the vocal folds does create something quite dangerous for the dog. It causes something that significantly reduces the lumen size of the larynx: ***everted*** (e-vert'ed [*e-* out + L. *vertere* to turn; i.e., turned inside out] ***laryngeal saccules*** [from L. *saccus* pouch, bag + *-ule* a small]. We mentioned earlier that the interior of the larynx is covered by a thin mucous membrane. That mucous membrane lines the surfaces of the vocal folds too. And if you look again at Fig. 5.18, you'll see that each vocal fold really lies against the lateral wall of the larynx. That creates a little pouch-like area between the vocal fold and the laryngeal wall. That "pouch" or *saccule* is lined with the thin mucous membrane. With inflammation, the mucous membrane becomes *edematous* and loses its "grip" on the underlying tissues. Eventually, it turns inside-out and everts like the throat of a frog, billowing into the laryngeal lumen. Talk about having a "frog in your throat!" Seriously, this can cause extreme ***dyspnea*** [*dys-* difficulty + *pnea* breathing]. So, part of our surgical intervention for brachycephalic animals also frequently involves a ***laryngeal sacculectomy*** [*saccul(o)-* saccule, pouch + *-ectomy* to cut out, removal of].

Surgery is traumatic. And trauma creates inflammation, as we discussed inChapter 3. What is one of the cardinal signs of inflammation? *Edema*. And if we've performed a *laryngeal sacculectomy*, a *tonsillectomy,* and *palatoplasty*, our patient may experience a tremendous amount of edema in the pharynx and larynx. Temporarily, our efforts to improve airflow may fail. ***Postoperative*** [*post-* after + *operat(o)-* surgery + *-ive* pertaining to] recovery of these patients can be challenging, to say the least. Our biggest concern is complete airway obstruction. If that happens, we need to place an ***endotracheal*** [*endo-* within + *trache(o)-* trachea + *-al* pertaining to] ***tube***. That means we need to anesthetize the patient again and maintain it under anesthesia for as long as the

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endotracheal tube is in place. If edema is so bad that we can't *intubate* via the *oropharynx*, we may need to perform an emergency ***tracheostomy*** [*trache(o)-* trachea + *- stomy* creation of a "mouth," Gr. *stoma* "mouth"]. The *tracheostomy* bypasses the pharynx and larynx by creating a hole (stoma) directly into the trachea. After the emergency surgical procedure, at least the patient can be awake with a ***tracheostomy tube*** in place. But we still need to be vigilant.

I've seen many *tracheostomy tubes* become plugged with blood clots and mucus in these patients. That's especially true for some of the smaller beasts, like Pugs, Boston Terriers, and French Bulldogs. I have literally checked on a *tracheostomy* patient and it was fine, breathing normally. Then I looked away for just a moment, and when I looked back at that patient, it was ***dyspneic*** [*dys-* difficult + *pnea* breathing + *-ic* pertaining to] and ***cyanotic*** [*cyan(o)-* blue + *-tic* pertaining to]. That's pretty scary. Blue is a *very* bad color for mucous membranes. I don't like having to scramble to change a *tracheostomy* tube under those circumstances. And there were times when I had just changed the tube minutes earlier! That's how quickly they can occlude. The tracheostomy is carefully maintained until pharyngeal and laryngeal swelling is gone and the animal can breathe normally. Then the tracheostomy tube is removed and the surgical site is allowed to heal. I have to say, it's pretty gratifying to see these patients after they've fully recovered. They breathe so much more quietly and easily. And that's a very good thing.

Laryngeal paralysis

In patients with ***laryngeal paralysis***, the muscles of the larynx do not work appropriately. Some of them no longer contract at all, usually affecting one or both arytenoid cartilages. Remember, when breathing in, laryngeal muscles should contract to *abduct* the arytenoids laterally. This opens the airway. When swallowing, laryngeal muscles should contract and draw the arytenoids closer together, to reduce the size of the laryngeal opening and help create a really good seal against the epiglottis. In *laryngeal paralysis* it is paralysis of the *abductors* that is of most concern for breathing. If the arytenoids are not abducted, they can partially or completely obstruct the laryngeal opening on *inspiration*. In fact, the more forceful the inspiration, the more severe the obstruction. A little air

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may squeak by the arytenoids, creating a loud, ***stridorous*** [*stridor* from L. *stridere* to creak + *-ous* pertaining to] sound. ***Stridor*** is the loud, harsh, raspy, sometimes high-pitched squeak that is associated with ***laryngeal obstruction***. When partially occluded, it tends to produce the really harsh, raspy noise that sounds a bit like Darth Vader.[1] The larger the animal, the louder the sound. In horses the harsh, raspy *stridor* sounds like a roar. That's why laryngeal paralysis is commonly called "***roaring***" in horses. More on this problem in horses shortly.

In dogs, *laryngeal paralysis* may be a *unilateral* or *bilateral* problem. But the laryngeal paralysis is often a symptom of a larger syndrome, resulting in profound weakness and loss of muscular tone in many parts of the body. And because this most often occurs in older dogs, we frequently refer to the syndrome as ***geriatric*** [*geriatr(o)-* old + *-ic* pertaining to] ***onset laryngeal paralysis polyneuropathy*** [*poly-* many + *neur(o)-* nerves + *-pathy* disease of] (***GOLPP***). Our Gordon Setter, Serena, was developing this. She was becoming very weak, especially in the rear. She would often fall down on uneven surfaces because of it. A support harness was helpful. But often she didn't realize that she was no longer as agile as she once was, making it difficult for us to offer her the support she needed. She often dragged us off into deep snow, until she (and almost we) got stuck. We had to limit Serena's physical activity because the more active she was, the more *dyspnea* she experienced. Deeper more powerful inspiratory movements tend to draw (suck) the flaccid arytenoids medially, obstructing the airway. I cared for numerous *GOLPP* dogs who were brought into the emergency room because of dyspnea. I'm convinced that some of them still had functional laryngeal *adductor* muscles, because some of those patients were in ***laryngospasm*** [*laryng(o)-* larynx + *spasm,* from L. *spasmus* a sudden, violent, involuntary muscle contraction], with the muscles holding those arytenoids tightly together, leaving little to no opening for air to pass through. Those dogs were extremely *dyspneic* and often *cyanotic*. The look on their faces is that of shear panic. Fortunately, Serena never experienced this.

Then there was the problem of Serena's weak ***esophageal*** [*esophag(o)-* esophagus + *-al* pertaining to] muscles. A weak or paralyzed esophagus cannot effectively move food into the

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stomach. Food tends to just sit in the esophagus causing it to dilate. Eventually, GOLPP dogs may develop ***megaesophagus*** [*mega-* enlarged + *esophagus*]. I don't know if you noticed where the *esophageal* opening is in relation to the larynx (Figs. 5.18 5.21). It's dorsal to the larynx. *Regurgitation* frequently occurs with *megaesophagus*. Remember, these dogs cannot protect their airways. The larynx isn't working properly. So, if a *GOLPP* dog regurgitates food, as they so often do, there is a HUGE risk for ***aspiration pneumonia***.

What do we do for dogs with GOLPP? Well, if laryngeal obstruction is putting the dog at grave risk, we can attempt surgical correction. What we do is suture one or both arytenoids laterally. This opens the larynx for free movement of air. Breathing improves dramatically for these dogs. Owners need to realize that the "tie-back" surgery can fail, especially if they do not limit the dog's physical activity. The other concern following laryngeal "tie-back" surgery is aspiration. Remember, during swallowing the arytenoids should be drawn medially to reduce the size of the laryngeal opening and create a good seal with the epiglottis. We've sutured one or both of the arytenoids open. So, a good seal may not be created between the arytenoids and the epiglottis. Aspiration is still a risk *postoperatively*, especially if the dog also has *megaesophagus*. Serena did not have this surgery. Her laryngeal issues were not that severe yet and were easily managed without surgery.

In horses, ***laryngeal hemiplegia*** [*hemi-* half + *pleg(o)-* paralysis + *-ia* condition of] is most common. Usually, only one arytenoid is affected (most often the left). And this frequently develops in performance horses (e.g., racing Thoroughbreds or Standardbreds). I think it stands to reason that the performance of these horses becomes poor. They simply cannot oxygenate as well enough, due to the partial laryngeal obstruction. Yes, we perform a similar "tie-back" surgery in horses too. But with or without surgery, that horse's racing career is over. If that postoperative horse is put back on the track, the tie-back will fail. The power of the inspiratory movements of a horse will place too much suctioning force on the sutured arytenoid. It doesn't take long for the suture to either break or rip through the arytenoid. So the horse needs to be retired. We still have aspiration concerns, especially when you consider the dry,

dusty nature of the hay that they eat. Fortunately, usually only one arytenoid is affected. Even after tie-back surgery, the risk of aspiration in these horses is still not as great as in GOLPP dogs. The "roaring" horses still have a good seal on the functional side of the larynx. And they don't develop megaesophagus. Still, it is wise for the horse owners to moisten dry hay, to reduce the amount of particulate matter that might be aspirated. And how does aspirated material make its way from the larynx to the lungs? Through the trachea.

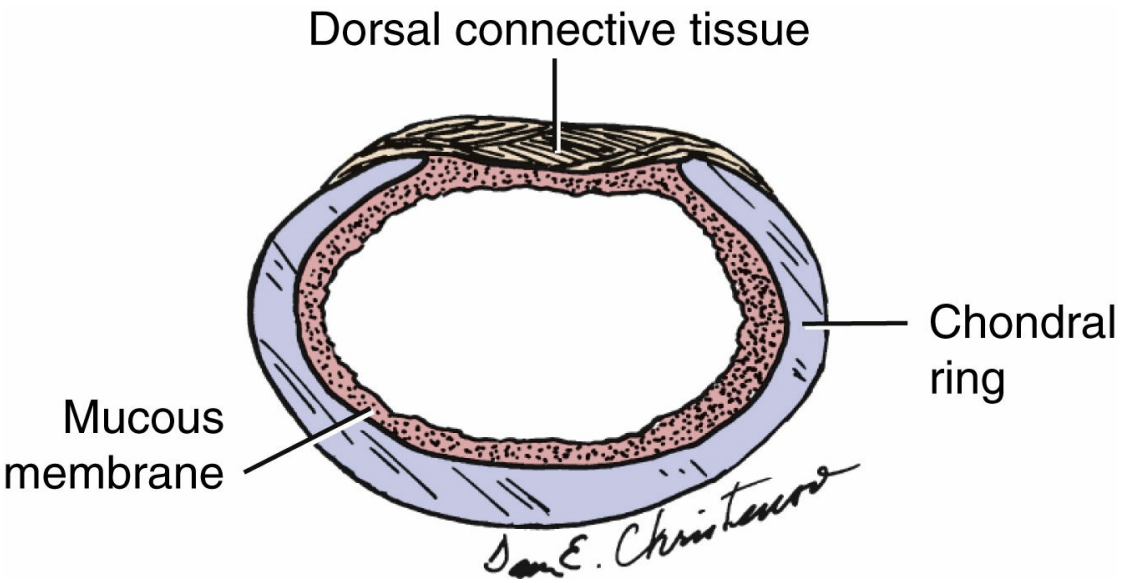

FIG. 5.22 Transverse tracheal schematic.

Trachea

The ***trachea*** (or "windpipe" as some people call it) is the large airway between the larynx and the ***tracheal*** [*trache(o)-* trachea + *-al* pertaining to] ***bifurcation*** [*bi-* two + L. *furca* fork + *-tion* state of]. That "fork in the road" (*tracheal bifurcation*) is where the trachea ends and the bronchi (plural of bronchus) branch off to the lungs. So the trachea is simply a long tube. Next! Not so fast; we really need to look more closely at its structure.

You see, the trachea is not a solid tube as you might expect. Like the larynx, it has cartilage to help it keep its shape. But it's not solid cartilage. The trachea has ***chondral rings***. And in animals those rings are C-shaped. If you look at Fig. 5.22, you can see that those

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C-shaped chondral rings have a gap along the dorsal border of the trachea. Remember, much of the esophagus is dorsal to the larynx and trachea (as you could see in Figs. 5.18 5.21). And because many animals, like dogs for instance, tend to gulp their food, having a little flexibility to the dorsal border of the trachea makes it a little less likely that food will get stuck in the esophagus as it passes over the trachea. No, that's no guarantee, but it helps. Between each chondral ring and connecting the dorsal margins of them is connective tissue. Like all airways, the interior of the trachea is lined with mucous membrane. The *ciliated pseudostratified columnar epithelium* in the tracheal mucous membrane creates a constant cranial sweeping action. This moves the mucus and anything stuck in it toward the pharynx where it can be coughed up and swallowed. (Yes, swallowed. Most animals do not spit.)

Okay, picture this: the inside of a giraffe's trachea while the animal is standing and trying to reach leaves on a tree to eat. That's a really long distance to the pharynx, even if we start in the middle, isn't it? You would think that gravity would win, making the mucus and anything stuck in it slide down into the lower airways. But those amazing ciliated cells keep sweeping the mucus up toward the head, like an escalator at a department store or airport. And that visualization is precisely why the upward sweeping action of the ciliated cells moving the mucus is often called the ***mucociliary*** [*muc(o)-* mucus + *cilia* hairs + *-ary* pertaining to] ***escalator***. Many parasites who either live in the airways or migrate through the lungs capitalize on the *mucociliary escalator*. How? Larvae (baby worms) and eggs are carried by the *mucociliary escalator* toward the pharynx. So, they don't need to work at all to get where they need to go. Whether they're coughed up and spit out or coughed up and swallowed, one way or another those tiny creatures get exactly where they need to go to finish their development and/or contaminate the environment. It fascinates me how they use normal anatomy and physiology toward their own ends.

By the way, ***coughing*** is a reflex action to forcefully remove ***phlegm*** (flem, [Gr. *phlegma* thick mucus]) and foreign particles from the trachea and lower airways. And the sensory receptors associated with coughing are most abundant in the larynx and

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trachea. This makes sense if we want to protect the lower airways and lungs from harm. Think about it. Especially if something foreign is aspirated, it would be best if we begin coughing as soon as it comes in contact with the larynx or cranial trachea. Even with infectious upper respiratory diseases, it's best if we can assist the *mucociliary escalator* by coughing the ***pathogens*** [*path(o)-* disease + *gen(o)-* producer] out of the trachea before they have a chance to set up housekeeping in the lower airways. Yes, of course that means we facilitate spreading the *pathogens* around, exposing others to the disease. (That's why it's best for people to cough into a tissue or their arm to minimize spewing the germs everywhere. No, don't cough into your hand; you'll probably touch something like a doorknob and contaminate it.) Fortunately or unfortunately, depending on how you look at it, many upper respiratory diseases cause ***tracheitis*** [*trache(o)-* trachea + *-itis* inflammation of]. And with *tracheitis* there's a lot of coughing. (Take that, you little *pathogens*!) And if the coughing becomes too severe that we can't get any rest, there are ***antitussive*** [*anti-* against + *tuss(o)-* cough + *-ive* pertaining to; i.e., cough suppressant] medications to reduce the coughing.

But *coughing* and *tracheitis* are not always indicative of infectious disease or aspiration of foreign material. Sometimes there is a structural problem with the trachea. And that is definitely the case with a collapsing trachea.

Collapsing trachea

It tends to be smaller toy and miniature dog breeds who experience ***collapsing trachea*** most often. I've seen numerous Yorkshire Terriers and Toy Poodles with this. And most of them were middle-aged or a little older. You can recognize a collapsing trachea dog a mile away. They sound like a honking goose. Coughing and breathing harder make the honking worse. The "honk" is produced by vibrations of the moist, flabby trachea. Wait. How can the trachea possibly be flabby? It's got strong chondral rings, right? Well, in a normal trachea, yes. But that's not the case in these dogs.

Collapsing trachea doesn't happen overnight, so to speak. It often develops over months or even years. For some reason, the chondral rings become weak. They develop something called ***chondromalacia*** [*chondr(o)-* cartilage + *malac(o)-* Gr. *malakos* soft + *-ia*

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condition of]. The cartilage literally becomes softened. And soft cartilage simply cannot hold its shape. If the tracheal rings were complete circles, they probably wouldn't collapse as easily. But they're not. The tracheal rings are C-shaped. So, as the cartilage softens over time, the trachea tends to flatten out. The dorsal connective tissue stretches and becomes flaccid too. That's the part that really vibrates to create the honk. At first, they only honk when they cough. But as disease progresses, and we factor in the forces of moving air and changes in air pressure produced by breathing, the *tracheal lumen* is easily reduced, impairing air movement. Unfortunately, this makes the animal struggle to breathe even harder and cough more. And *dyspnea* increases the forces placed on the tracheal wall, causing complete dorsoventral collapse. Now the dog squeaks and honks nearly all the time from breathing and coughing.

Most of these dogs experience ***extrathoracic*** [*extra-* outside + *thorac(o)-* chest + *-ic* pertaining to] tracheal collapse. So, it's only the trachea through a portion or all of the neck that is weak and collapses. And most of the time, the collapse develops first in the proximal trachea near the ***thoracic*** [*thorac(o)-* chest + *-ic* pertaining to] ***inlet*** (i.e., at the base of the neck). Forces produced by air movements during *inspiration* cause the full collapse. So, these animals experience ***inspiratory dyspnea***. Most of them can breathe relatively normally much of the time, when they are resting quietly. But if they exert themselves or become excited, the deeper breathing required for these behaviors causes collapse and occlusion of the trachea. And the more they struggle to inspire the needed air, the worse it gets. Coughing helps to blow the trachea open momentarily. The more they struggle and cough, the more they honk. The vibrations of honking are irritating. It turns into a catch-22. Sometimes, they can't oxygenate well because of it and require emergency care to break the cycle and provide needed oxygen. It's frightening for the dog and the owner, and can be life-threatening.

Most dogs, especially with minor *extrathoracic* collapse, can be managed medically. The goal is to minimize respiratory effort and coughing. Obviously, collars worn around the neck are an absolute no-no. These dogs should only wear a harness. Owners need to

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keep the dogs quiet, avoiding exertion and excitement. If the dog is overweight, as many of these lapdogs are, weight-loss will reduce respiratory effort. Sometimes *antitussive* medications are beneficial. For those dogs who can no longer be managed medically, surgery may be an option. The surgery involves placement of ***extratracheal*** [*extra-* outside + *trache(o)-* trachea + *-al* pertaining to] ***stents*** (splints). The plastic C-shaped stents support the trachea, helping to maintain a normal round, tubular shape and patent tracheal lumen. Even if we surgically correct the current area of collapse, the *chondromalacia* may progress along the trachea. So, continued medical management is important. Plus, owners need to realize that future surgery may be needed. If the *chondromalacia* eventually involves the whole trachea and progresses to include ***bronchial*** [*bronch(o)-* bronchus + *-al* pertaining to] collapse, there is very little we can do.

Lower Airways

All of the airways beyond the *tracheal bifurcation* are collectively referred to as ***lower airways***. Like the trachea they are tubular in shape, gradually getting smaller and smaller until finally reaching the alveoli (air sacs). In that regard, the lower airways are very much like the branches of a tree. That is why you may hear some folks refer to the branching and tapering nature of the lower airways as arborizing [from L. *arbor* "treelike"].

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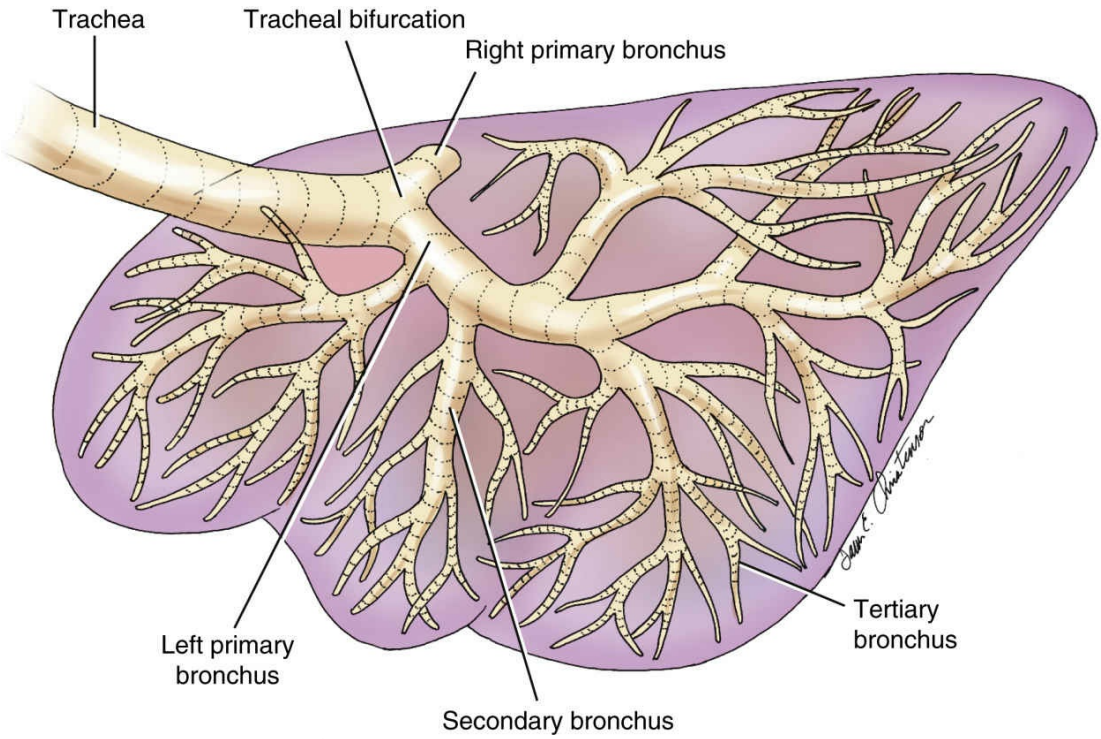

FIG. 5.23 Bronchial schematic.

Bronchi

If we think of the trachea as the tree trunk, then the bronchi are the large, medium, and small branches (Fig. 5.23). And perhaps that's why we refer to them collectively as the ***bronchial*** [*bronch(o)-* bronchus + *-al* pertaining to] ***tree***. There are two ***primary bronchi*** (i.e., mainstem bronchi) that divert inspired air into the right and left lungs. ***Secondary bronchi*** are the next in the series of airways that divert inspired air into the various lung lobes. Most animals, with the exception of the horse, have cranial, middle, and caudal lung lobes, as well as an accessory lobe. ***Tertiary*** [from L. *tertiarius* third in order] ***bronchi*** are smaller branches that divert inspired air throughout those lung lobes.

All of the bronchi have *chondral* support and smooth muscle in their walls. And their interior walls are lined with mucous membranes, complete with the *mucociliary escalator*. Primary and secondary bronchi are large enough to have chondral rings as their supportive structure. The smaller *tertiary bronchi* tend to have small patches and plates of cartilage. This can be problematic. Less defined chondral support may make it more difficult to maintain patency of these small bronchi. This is especially true in the

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presence of inflammation. With ***bronchitis*** [*bronch(o)-* bronchus + *-itis* inflammation of] involving tertiary bronchi, it is easy for ***bronchospasm*** [*bronch(o)-* bronchus + *spasm*; i.e., smooth muscle spasm] to severely impair air movements. The larger bronchi may experience *bronchospasm* too. But because primary and secondary bronchi are larger with better chondral support, their lumen size can't be reduced to the point that they're nearly obstructed. Add some edema and mucus, and obstruction of smaller bronchi becomes even more likely. But if you want to talk about potential for obstruction, we need to talk about the smallest of all the airways: the ***bronchioles*** [*bronch(o)-* bronchus + *-ole* a small].

Bronchioles

Bronchioles are so small, there is no space for cartilage. These tiny tubes are ***achondral*** [*a-* without + *chondr(o)-* cartilage + *-al* pertaining to], muscular airways. And being *achondral*, if smooth muscle of a *bronchiole* goes into spasm, the airway obstructs. In fact, bronchioles are so small, they can obstruct from edema alone. Worst-case scenario would combine *edema*, *mucus,* and *bronchospasm*. No air will pass through a *bronchiole* under those circumstances. Do you see why I said what I did about obstruction and bronchioles earlier? The effect of ***bronchiolar*** [*bronchiol(o)-* bronchiole + *-ar* pertaining to] obstruction on gas exchange can be devastating. These little guys are the gatekeepers for air movements to and from the alveoli. If air can't get past them, there is no gas exchange. We'll discuss gas exchange shortly. Since we just mentioned mucus, let's take a look at the mucous membrane of bronchioles and how mucus is affected by it.

The mucous membranes lining the interior of bronchioles are not like that of the bronchi and trachea. First, they don't produce any mucus. (That's probably a good thing, since they're so small. Mucus would easily plug them up.) Plus, the surface epithelium is different. A bronchiole only has ***ciliated columnar epithelium*** near the *tertiary bronchus*. Beyond that, they have simple *cuboidal epithelium* (Fig. 5.24) that eventually transitions to thin, flat *simple squamous epithelium* near the alveoli. This means that any mucus that slides down from the bronchi is going to be difficult to remove. There is practically no mucociliary escalator in bronchioles. If

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mucus is a concern, the same is true for fluid, like pus that accumulates in pneumonia. It's going to be difficult to get rid of it.

Finally, the terminal portion of a bronchiole that leads into the *alveolar duct* can actually participate (minimally) in gas exchange. So obviously, this terminal portion of the bronchiole can't have smooth muscle either. If it did, it couldn't play on the gas exchange playground. And since we're on the topic of gas exchange, let's look at the gas exchange aficionados, the alveoli.

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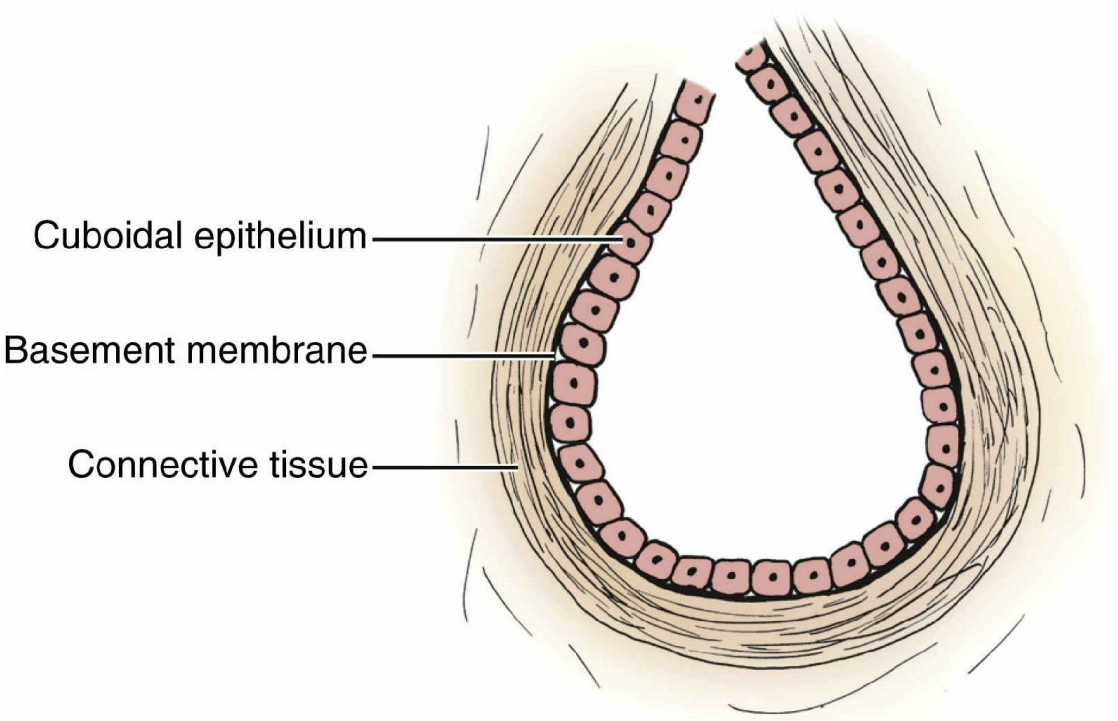

FIG. 5.24 Simple cuboidal epithelium.

Alveoli

This is it! This is *THE* place to be for pulmonary gas exchange. ***Alveoli*** make up the lung tissue we refer to as the ***pulmonary parenchyma*** (puh-reng'ki-muh, [Gr. *parenchyma* "anything poured in beside"; the functional tissue of an organ]). It is the functional tissue of the lungs. When fully inflated, the *pulmonary parenchyma* fills most of the space in the chest. And it takes billions of alveoli to take up that amount of space. We don't have as many bronchioles. So, in order to maximize available surface area for gas exchange stemming from each bronchiole, alveoli are clustered like a huge bunch of grapes at the terminal end of each bronchiole (Fig. 5.25). And in the close-up of an individual *alveolus*, you can see how each one is completely enveloped by an intricate *capillary network*. This is why the lungs are so efficient at gas exchange.

Within each alveolus is air. On inspiration, alveoli fill up with air like little balloons. On expiration, alveoli shrink into tiny balloons, kept ever-so-slightly inflated by a small ***residual volume*** of air. But even if somehow an alveolus could be completely emptied of its air, its moist walls won't stick together. Now, you might expect with moisture, ***surface tension*** would lock the walls of that alveolus

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together like Velcro. But there is a thin film of something called ***surfactant*** that prevents an alveolus from sticking shut for good. *Surfactant* keeps the walls of that alveolus from sticking together by reducing surface tension. You see, surface tension interlocks water molecules. That's just the nature of water. In fact, it interlocks the water molecules so well that if you were to place a pin on the surface of the water in a glass, the pin would be supported by the water. Really. It will stay at the surface of the water. But if we were to place a drop or two of dish soap in the water, the pin would immediately fall to the bottom of the glass. You're doubting me, aren't you? Go run your own experiment with this. I'll wait.

It worked, didn't it? But why did the pin fall to the bottom of the glass with the soap? The detergent reduced the surface tension. The soap removed the interlocking ability of the water molecules. And that is precisely what *surfactant* does in the alveoli. It reduces *surface tension*, making the surfaces slippery. That way, even without a *residual volume* of air, alveoli are less likely to stay collapsed. Because of slippery surfactant, with the next inspired breath, the alveoli should easily re-inflate. By the way, the word *surfactant* is actually an abbreviation of sorts. You see, it stands for "surface-active agent." Apparently, someone in the 1950s got tired of writing "surface-active agent." So, it was abbreviated *surf*(ace-)*act*(ive)*a*(ge)*nt*—*surfactant*. You have no idea how long I've struggled to find the etymology for that word, only to find out this is how it was created. One is never too old to learn.

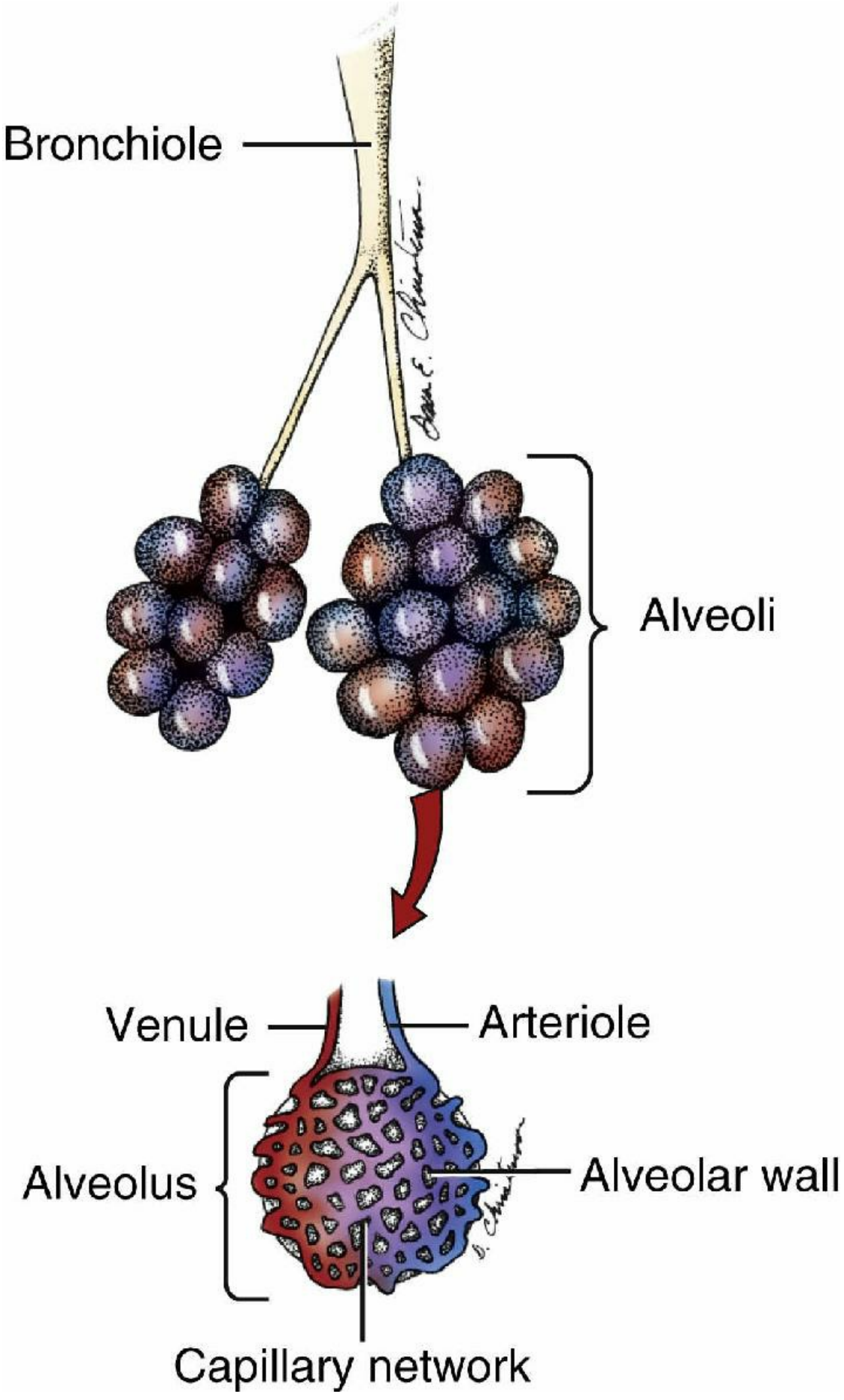
Bronchiole
Alveoli
Venule
Arteriole
Alveolus
Alveolar wall
Capillary network

FIG. 5.25 Alveoli.

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Now, before we can fully understand gas exchange, we need to have an appreciation for the ***respiratory membrane***. If you look at Fig. 5.26, you'll see an individual alveolus and a surrounding capillary. The red blood cells are shown squeezing single file through the capillary. The "squeeze" that they're subjected to maximizes contact with the ultrathin respiratory membrane. It's so thin it's transparent. What makes up the *respiratory membrane*? It's only the ***alveolar wall***, a tiny ***interstitial*** [*inter-* between + *stiti(o)-* tissue + *-al* pertaining to] ***space***, and the ***capillary wall***. By the way, there's not much to the alveolar or capillary walls—only a thin ***basement membrane*** and an ***epithelial cell*** (***endothelial cell*** in the capillary). And the interstitial space is an almost nonexistent gap. This ultrathin structure is what makes gas exchange so easy.

Gas exchange

Gas exchange is a matter of simple ***diffusion*** of ***oxygen*** and ***carbon dioxide*** molecules. Remember, diffusion requires an area of high concentration and an area of low concentration, separated by a semipermeable membrane. Molecules move from the area of high concentration through the semipermeable membrane to the area of low concentration. The thin *respiratory membrane* is our *semipermeable membrane*. So, in the lungs *inspired air* in the alveoli should contain a high concentration of O_2 and a low concentration of CO_2. Arterial blood coming to the lungs from the right ventricle contains a low concentration of O_2 and a high concentration of CO_2. As the blood squeezes through the alveolar capillaries, CO_2 rapidly diffuses into the alveolar lumen and O_2 diffuses into the red blood cells within the capillary. So, the venous blood returning to the left atrium is highly oxygenated. And *expired air* leaving the alveoli into atmosphere contains high amounts of CO_2.

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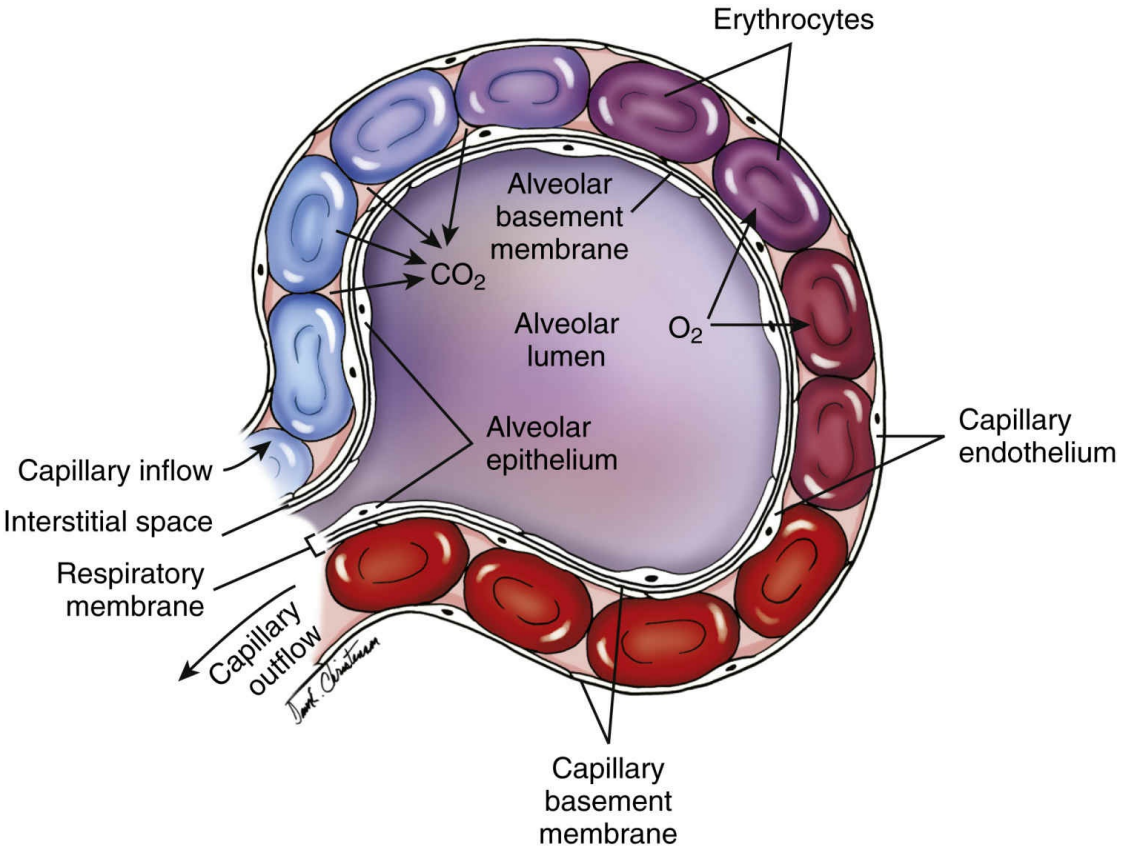

FIG. 5.26 Respiratory membrane schematic.

It's important to remember that gas exchange and blood flow are dynamic processes. They don't stop. They can't. If they do, we die. Yet in order to explain them, we try to take a "still photo" to capture a moment in time. But it's all transitional and ever-changing. Even for an individual red blood cell passing through that capillary, it's not like it stops for a "fill-up" and then merrily leaves. It's constantly moving with the blood flow and exchanging those molecules as it moves along. That's why Fig. 5.26 shows the red blood cells at gradual stages of gas exchange (depicted by the color sequence). Ideally, that diagram should show oxygen diffusing into the first cell entering at the capillary inflow. In fact, there should be arrows pointing into each and every RBC to show the movement of O_2 into them. Unfortunately, that many arrows would be distracting, confusing, and make it difficult to see detail. It's the best we can do with a two-dimensional static image. To give you another vantage point, you should look at the Evolve animation entitled *Gas Exchange.* That animation provides a nice review of the respiratory membrane too.

Of course, the lungs are not the only place gas exchange takes

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place. That wonderful oxygenated blood that returns to the heart is pumped from the left ventricle into the aorta and disbursed throughout the arterial network to the body tissues. Let's pause here before arriving at the tissues, to review how red blood cells carry oxygen. InChapter 3, we discussed the ***hemoglobin*** that fills mature RBCs. We said that the hemoglobin molecule is really big. It's made up of basically two compounds: the iron-containing *heme* and the protein called *globin*. We also said that there is a 4 to 1 ratio of heme to globin. The *iron* (Fe^{++}) of the heme can bind with one oxygen (O_2) molecule. That loose molecular bond forms ***oxyhemoglobin*** [*oxy(o)-* oxygen + *hemoglobin*]. (Remember, it's *oxyhemoglobin* that gives RBCs their bright red color.) And with a 4:1 ratio of heme to globin, each RBC can carry lots of *oxyhemoglobin*.

By the way, this is the stuff that a ***pulse oxymeter*** [*oxy(o)-* oxygen + *meter* measurer; also spelled *oximeter*] measures. It measures the *relative percent* of *oxyhemoglobin* in the blood. ***Oximetry*** [*oxi(o)-* oxygen + *-metry* measuring] assesses capillary blood flow in real-time. The lighted probe of the *oximeter* is placed in an accessible location. Nonpigmented mucous membranes usually work best, because capillaries are concentrated and very superficial there. So, a lip or vulva (on a female) works well. In an anesthetized animal the tongue is perfect. But even an ear, toe pad, or prepuce (on a male) will work. And because out in the tissue capillaries a small portion of oxygen has already diffused out of the blood, the oxyhemoglobin percentage will probably not be 100%. Most normal animals will have pulse *oximetry* readings of 95% to 98%. We start getting a little concerned as the reading approaches 90%. We would prefer a larger percentage of oxyhemoglobin arriving at the tissues. Anything below 90% is a big concern. *Oximetry* is no replacement for blood gas analysis. But it is a quick, easy, noninvasive means to determine roughly how well a patient is oxygenating its tissues. Okay, let's get back to our tissue gas exchange.

You know fromChapter 2 that two of the largest by-products of cellular activity are CO_2 and water. With regard to gas exchange in the tissues, it's the CO_2 that's of importance. There's lots of carbon dioxide in the tissues. And the greater the cellular activity, the more CO_2 is produced. Let's not forget that cellular metabolism uses up oxygen. All of this provides a really good concentration differential

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between the tissues and the blood to promote diffusion. So, as the arterial blood percolates through the *tissue capillaries*, oxygen easily breaks free of its loose bond in the *oxyhemoglobin* molecule and diffuses into the tissues. The CO_2 rapidly diffuses into the tissue capillary. A very small portion of that binds with the hemoglobin molecule to form ***carbaminohemoglobin*** [*carbamin(o)-* carbon dioxide + *hemoglobin*]. This too has a relatively loose molecular bond. We don't want to hang on to that stuff. It also imparts a slight bluish hue to the *carbaminohemoglobin* molecule. And because the red blood cells now contain both ***deoxyhemoglobin*** [*deoxy(o)-* reduced oxygen + *hemoglobin*] and *carbaminohemoglobin*, they appear a deep, dark almost purplish red. That's why *venous blood* is so much darker than *arterial blood*. But that's not an efficient means to transport CO_2 for removal.

The most efficient means of transport is to combine CO_2 with water in the *plasma*. And that, as you may recall fromChapter 2, forms ***carbonic acid*** (H_2CO_3). The beauty of this molecule is that it's versatile. If the body needs ***hydrogen ions*** (H^+), like for acid secretion in the stomach, hydrogen ions can break free and diffuse into the ***gastric*** [*gastr(o)-* stomach + *-ic* pertaining to] cells. The kidneys can excrete hydrogen ions in the urine if the body is too acidic from too much *carbonic acid*. And as you'll recall fromChapter 2, any time hydrogen ions are lost from the plasma, we're left with ***bicarbonate*** (HCO_3^-) a very alkaline compound. But if you want to talk about efficiency at reducing *carbonic acid*, you need to talk about the lungs. In the pulmonary capillaries, CO_2 rapidly breaks free of this molecule and quickly diffuses out into the alveoli. What does that leave us? Water. Boom! Carbonic acid reduced. How fast does CO_2 diffuse into the alveoli? Roughly 20 times faster than oxygen can diffuse in. And this is largely because most CO_2 is transported by plasma as *carbonic acid*. Controlling the amount of carbonic acid is a key to controlling ***acid-base balance*** in the body. And that leads us to a very important discussion about acidosis and alkalosis.

Acidosis versus alkalosis

We've talked about *homeostasis* many times in multiple chapters. In

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simple terms, *homeostasis* is the balanced, stable state of the body. Everything is in equilibrium—not too much of anything; not too little of anything; just right. And when it comes to *acid-base homeostasis*, we have an ongoing battle to keep things stable. Why? Because the cells of the body are constantly producing CO_2 and that means we're always creating lots of *carbonic acid*. Fortunately, we have both the lungs and the kidneys to alter that molecule to change *systemic pH*. We'd like pH to remain neutral. So, if there's too much carbonic acid, the kidneys remove some hydrogen ions, reducing systemic pH. And that's great! But let's think about this in terms of efficiency. Which do we do more—breathe or urinate? For your sake, I hope you said "breathe." We've already said that through the alveoli, CO_2 diffuses incredibly fast to be exhausted with each and every exhaled breath. That means the lungs play a pivotal role in acid-base homeostasis. And it doesn't matter how ***acidosis*** [*acid* + *-osis* condition or state of] or ***alkalosis*** [*alkal(o)-* alkaline, base + *-sis* condition or state of] develops. The respiratory system will respond accordingly to those changes. It can only do one thing with carbon dioxide: remove it. How effective the respiratory system is in its response really depends on the condition of the alveoli and circulation to and from them. So, this becomes a *cardiopulmonary* challenge. But let's stay focused on the *pulmonary* aspect for now.

Let's take a normal situation. Let's consider my Corgi pup doing "zoomies" through the house. She runs really hard and fast. Her muscles certainly need more oxygen. And her respirations will change in response. But that's not as great a driving force as the huge amounts of CO_2 building up in her muscles and body. Honestly, we need to be quite ***hypoxic*** [*hypo-* decreased + *ox(o)-* oxygen + *-ic* pertaining to] before the respiratory centers in the brain are activated to do something about it. First and foremost, it's CO_2 that drives the *respiratory centers*. If her respiratory centers do not respond appropriately to remove excess CO_2 (and thereby carbonic acid), she'll become ***acidotic*** [*acid(o)-* acid + *-tic* pertaining to]. So, that's what drives those centers in my pup to create her much-needed ***tachypnea*** [*tachy-* fast + *pnea* breathing]. And the longer she does zoomies, the more CO_2 and carbonic acid are produced and

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the more likely she will respond with ***hyperpnea*** [*hyper-* excessive, exaggerated + *pnea* breathing; i.e., faster and deeper breathing]. And because she needs to transport all of that CO_2 from her muscles to the alveoli and transport oxyhemoglobin to her muscles, she also becomes ***tachycardic*** [*tachy-* fast + *cardi(o)-* heart + *-ic* pertaining to].

When she happily finishes running her "zoomy" circuit, she lays down flat-out on the floor. Even though the exertion has ended, gas exchange has not. She is very busy at the tissue and cellular level. Her oxygen needs are no longer as great. But she still has an abundance of CO_2 that needs to be eliminated. To maintain a neutral state in her body, respiratory centers in the brain persist in making her ***hyperpneic*** for a little while. The deeper, rapid breathing pattern exposes more alveolar surface area in the lungs to facilitate better removal of CO_2. Eventually, she's simply ***tachypneic*** to blow off excess CO_2 rapidly. Once excesses of CO_2 (and carbonic acid) have been eliminated, she simply ***pants*** to cool off.

Panting is a very shallow, nonrespiratory breathing pattern that moves air rapidly over the mucous membranes of the mouth and upper airways. By rapidly moving air over the moist mucous membranes of the mouth and throat, through evaporation and convection heat is dissipated from the body. (This is the primary cooling mechanism for dogs and cats.) It's not long before my pup is breathing normally and sound asleep. Then her needs for gas exchange and acid-base homeostasis change dramatically. There is very little cellular activity going on. So very little CO_2 is being produced. She can't afford to exhaust large amounts of CO_2 through breathing. If she did, she'd probably become ***alkalotic*** [*alkal(o)-* alkaline, base + *-tic* pertaining to]. So, to maintain balance, the respiratory centers make her ***bradypneic*** [*brady-* slow + *pnea* breathing + *-ic* pertaining to] while she sleeps.

What about abnormal situations? Well, there are all sorts of ways that disease may cause *acidosis*. And even in *metabolic acidosis* that's caused by kidney disease, digestive disease, or endocrine diseases, the respiratory response to the acidosis will involve faster and perhaps deeper breathing (i.e., *tachypnea* and *hyperpnea*). It's the only way the system can respond. By the same token, the

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respiratory response to *metabolic alkalosis* will be to reduce the amount of CO_2 exhausted by slowing breathing (i.e., *bradypnea*). Really, changing rate and depth of breathing are the only ways that the respiratory system can effectively contribute to acid-base homeostasis. And this system provides the most efficient mechanism in response to ongoing acid-base changes. Which brings us to respiratory disease and how it negatively impacts gas exchange and acid-base homeostasis.

Obviously, anything that impairs or obstructs the movement of air along the respiratory tract will have a negative impact. Think about the upper airway diseases that we've already discussed: *brachycephalic syndrome*, *laryngeal paralysis,* and *tracheal collapse*. All of those in one way or another impair and potentially obstruct airflow to and from the lower airways. The more severe the impairment, the more dramatically alveolar gas exchange is affected. Let's think about one of those *tracheostomy* patients that I mentioned. I said that I've seen some of those patients acutely (suddenly) obstruct the tracheostomy tube. They rapidly become *cyanotic*. But why? And what does it tell us about oxygenation and acid-base status in the patient's body?

First, let's review the hemoglobin molecule and its color changes related to binding with oxygen and carbon dioxide. We said that *oxyhemoglobin* imparts a bright red color. *Carbaminohemoglobin* imparts a bluish color. Under normal circumstances, even *deoxygenated* venous blood still appears red. That's because only a very small portion of carbon dioxide typically binds with hemoglobin. Plus, there is still oxygen bound to hemoglobin. In order to produce a visible blue coloration, there needs to be very little oxyhemoglobin present and an abundance of carbaminohemoglobin. So, what assumptions can we make about our *cyanotic* patient? We can assume that the patient is *hypoxic*. We can also assume that the patient has excessive amounts of carbon dioxide in circulation and that means the patient is *acidotic*. Anytime we see ***cyanosis*** [*cyan(o)-* blue + *-sis* condition of] we know that the patient is critical. *Hypoxia* kills. Remember, all tissues of the body require oxygen. And that oxygen need is most important for vital organs, like the brain and the heart. In severe *hypoxia*, brain cells are lost. (Other cells of the body die too.) When we discussed

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cardiac arrhythmias, we said that hypoxia was one of the most common contributors to *VPCs*. The *myocardium* does not function well when hypoxic. The worse the hypoxia, the more VPCs; too many VPCs lead to *V-tach*; prolonged V-tach leads to ***ventricular fibrillation*** (uncoordinated quivering of the myocardium). The next step is ***asystole*** [*a-* without + *systole* contraction; on ECG a "flatline"] and death.

But it's not just the *hypoxia* that's lethal. ***Hypercapnia*** [*hyper-* excess + *capn(o)-* carbon dioxide + *-ia* condition of] kills too. And a *cyanotic* patient is most definitely ***hypercapnic*** [*hyper-* excess + *capn(o)-* carbon dioxide + *-ic* pertaining to]. Both *hypoxia* and *hypercapnia* can result in brain ***asphyxia*** (as-fik'se-uh [Gr. *asphyxia* "a stopping of the pulse"]. ***Asphyxiation*** (as-fik"se-a'shun [*asphyxia* + *-tion* act of] is basically ***suffocation***. *Asphyxiation* causes brain death. There is no coming back from brain death. Is this possible in a cyanotic patient? You bet it is. That's why *cyanosis* is always an emergency, with little response time before we reach a path-of-no-return. Plus, with *hypercapnia*, we know that there is an abundance of *carbonic acid*. Therefore *acidosis* also exists, as we stated a moment ago. But there are actually two sources contributing to the acidosis. Hypercapnia is one. And that plus the hypoxia will be very stimulating to the respiratory centers in the brain. The respiratory centers in our patient will produce extreme *dyspnea*. Of course, our *tracheostomy* patient with the acutely plugged tracheostomy tube won't be able to overcome the obstruction. None the less, the patient will engage in profound muscular activity, desperately struggling to take in oxygen and blow off CO_2. Not only does that muscular activity produce more CO_2, but it also produces ***lactic acid***. As you may recall fromChapter 4, *lactic acid* is produced by ***anaerobic*** [*an-* without + *aer(o)-* air, oxygen + *-bic* pertaining to] ***glycolysis*** [*glyc(o)-* sugar + *-lysis* breakdown, i.e., sugar metabolism]. So, between an abundance of carbonic acid and lactic acid, we have profound acidosis. Tissues do not function well in the presence of acidosis. Organs and muscles become less responsive. Some tissues become irritable, like the myocardium.

So, for any *cyanotic* patient, the combination of hypoxia, hypercapnia, and acidosis is a recipe for death. Our goal in this emergency situation is to supply oxygen and remove CO_2. In the

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case of our tracheostomy patient, we need to restore a ***patent airway***, first and foremost. Even in *cardiac arrest*, *airway* always comes first. (That's the "A" in the ABCs of CPR—"Airway.") We need to provide supplemental oxygen. And if the patient is unconscious and either ***hypoventilating*** [*hypo-* decreased, insufficient + *ventilate* from L. *ventilare* "to fan"; i.e., reduced respiratory movements/volumes of air] or ***apneic*** [*a-* without + *pnea* breathing + *-ic* pertaining to], we need to *ventilate* (breathe for) the patient. (That's the "B" in the ABCs of CPR—"Breathing".) If we don't maximize use of the alveolar surface area, we cannot rapidly infuse oxygen or remove CO_2. And we'd like to accomplish this before the patient develops *asystole* and/or brain death. By the way, upper airway obstruction can happen in happy, healthy animals too. The most common causes are slimy, sticky rawhide chews becoming stuck over the larynx of a dog or a plastic bag or container getting stuck over the dog's head. In the case of the rawhide chew, if the owners respond quickly with the ***Heimlich*** (hīm'lik) ***maneuver***, they may be able to save the dog's life. (The Heimlich maneuver is named after Dr. Henry Heimlich, who developed the technique of an abdominal thrust to dislodge objects from the larynx.) The bag or container covering the dog's head simply needs to be removed quickly.

That's just taking a brief look at possible upper airway contributors to impaired gas exchange and acid-base disturbances. Let's consider the most important players in gas exchange and acid-base homeostasis—the *alveoli*. How might alveolar changes disrupt normal respiratory physiology? Well, reduce available alveolar surface area and gas exchange is disrupted. How might surface area be reduced? ***Atelectasis*** [*atel(o)-* incomplete + *-ectasis* expansion; i.e., lung collapse] is a good example of this. I know, I know. We said earlier that *surfactant* prevented alveolar (and thereby lung) collapse by reducing surface tension. It makes alveolar walls slippery/less sticky. All true. But if we significantly dry out the airways, especially the alveoli, surfactant is insufficient to prevent alveolar walls from sticking tightly together. Here's a common scenario that might result in atelectasis: inhalant (i.e., breathed in) anesthesia during a long surgical procedure with the patient in lateral recumbency. The inspired air through the endotracheal tube is

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poorly humidified. In fact, that air is pretty dry. Evaporation within the alveoli is inevitable, especially during a really long procedure. And with the animal in lateral recumbency, the down lung fields will be poorly expanded. And gravity makes the rest of the *thoracic viscera* push down on the down, poorly inflated lung fields. *Residual air volumes* in those alveoli are eventually squished out. By the time, the animal is in *postoperative* recovery, many of the alveoli may be completely flat and stuck shut. This is especially true for peripheral margins of the lung lobes. The more ***atelectic*** [*atelect(o)-* incomplete expansion + *-ic* pertaining to] the lung lobes, the more impaired gas exchange. It can be overcome. But until it is, gas exchange will be reduced, sometimes significantly.

Another way to reduce functional surface area is to destroy alveoli. This is what happens in disease conditions like ***pulmonary emphysema*** [Gr. *emphysema* "an inflation"]. But the "inflation" that the Greeks were referring to is inflation to the point of bursting alveoli. With each alveolus that bursts, surface area is lost. Those lost alveoli cannot be regained. *Pulmonary emphysema* is a common disease in people and horses. And in the lungs of both horses and people, there can be huge cavernous open spaces throughout the lung fields. And if that's not bad enough, scar tissue adds insult to injury with regard to gas exchange. Bottom line, *pulmonary emphysema* patients have much less functional *respiratory membrane*, significantly decreasing gas exchange. I have seen a number of dogs who had ***emphysematous*** [*emphysema* an inflation + *-tous* pertaining to] ***bullae*** [L. *bulla* "a blister"; *bullae* is plural of bulla]. When a *bulla* near the pleural surface ruptured, it created a spontaneous ***pneumothorax*** [*pneum(o)-* air + *thorax* chest; i.e., air in the chest cavity surrounding the lungs]. As you will learn, when we discuss breathing in a little while, there should be nothing in the chest cavity itself. It should be for all practical purposes a vacuum. So, with free air in the chest cavity, the lungs will be *atelectic*. Hopefully, the *mediastinum* remains intact to isolate the *pneumothorax* to one side of the chest cavity. The patient's usable alveoli may be reduced by half, but that's better than nothing.

But if all of our alveoli are intact and inflated, what else might impair gas exchange? Making the normally thin respiratory membrane thicker will do it. Remember, the respiratory membrane

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is made up of alveolar epithelium and basement membrane, a tiny interstitial space, and the capillary basement membrane and endothelium. Edema is increased interstitial fluid, right? So, if there is ***pulmonary edema***, the interstitial space becomes larger, making the respiratory membrane thicker. This impairs gas exchange, especially oxygen. Carbon dioxide is affected too. But remember, CO_2 diffuses 20 times faster than O_2, so it's got "cushion," if you will. Ah, but as pulmonary edema worsens, it isn't limited to the interstitial space. Some of the fluid begins to seep into the alveoli too. That not only takes up valuable space, limiting the amount of air that can actually fill the alveoli, but it also increases the thickness of the respiratory membrane even further. The fluid within the alveoli is just one more layer for the gases to diffuse through. Gas diffusion is impaired even further.

Do you know what bacteria love? They love a warm, moist environment. Guess who lives in the airways. That's right—bacteria, ***commensal*** [*com-* together, with + L. *mensa* table + *-al* pertaining to] ***bacteria*** to be exact. *Commensal bacteria* are those that "sit at table with the host," living in a ***symbiotic*** [*sym-* with + *bi(o)-* life + *-tic* pertaining to; i.e., living in harmony with] relationship. But given a golden opportunity, like *pulmonary edema* in their gracious host, even these bacteria may colonize and overpopulate the area. We hope the body will respond with white blood cell infiltrates. As we discussed inChapter 3, ***macrophages*** [*macr(o)-* large + *phag(o)-* eater] are absolutely essential in destroying *pathogens*. Unfortunately, this creates thicker fluid (i.e., *pus*). The *pneumonia* that has now developed secondary to the pulmonary edema reduces gas exchange even further. It's really difficult for gases to diffuse through thicker fluid and a thicker respiratory membrane. The patient becomes more and more *dyspneic* and eventually ***orthopneic*** [*orth(o)-* straight + *pnea* breathing + *-ic* pertaining to]. In ***orthopnea*** [*orth(o)-* straight + *pnea* breathing], the struggle to breathe is so severe that the animal often sits up straight or stands with its head and neck held in extension and the elbows abducted. This is an attempt to move as much air as possible with the least amount of resistance. And to minimize resistance further, they usually open-mouth breathe. What will this do to the pus in the alveoli? It will dry it out, making the pus even thicker and

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stickier. There goes our gas exchange.

If the patient wasn't *cyanotic* before, it is now. And you know that guarantees the patient is *hypoxic*, *hypercapnic*, and *acidotic*. But how severe is it? Yes, we can use a *pulse oximeter* to determine a rough percentage of *oxyhemoglobin* in the blood. But it doesn't tell us an exact measurement of O_2 that's being circulated. By the same token, if the animal is intubated (as it very well may be, if we have to place the patient on a ventilator), we should and do use a ***capnograph*** [*capn(o)-* carbon dioxide + *graph* measurer] to measure CO_2 in the expired air. The sensor is placed at the end of the *endotracheal tube* for constant readings with each ventilated breathing cycle. But again, ***capnography*** [*capn(o)-* carbon dioxide + *-graphy* measuring] doesn't tell us precise amounts of carbon dioxide in the bloodstream. If we want precise blood gas measurements, we need to use *blood gas analysis*.

Arterial versus venous blood gases

Blood gas analysis provides precise measurements of *partial pressures* (p) of oxygen (pO_2), carbon dioxide (pCO_2), and hydrogen ion concentration (pH). (Partial pressures of O_2 and CO_2, because they are "pressure" readings, are reported in millimeters of mercury [mm Hg], just like blood pressure.) Blood gas analysis also provides precise measurement of *bicarbonate* (HCO_3^-), as well as blood glucose and many electrolytes, such as sodium (Na^+), potassium (K^+), chloride (Cl^-), and calcium (Ca^{++}). Now, we can obtain all of these measurements from either a venous blood gas or an arterial blood gas analysis. Measurements from both are quite precise. *Phlebotomy* is much easier to perform. And certainly, if all we really need to know is the state of various electrolytes, a venous blood gas will be quite sufficient.

Now consider this. If we want to know true *pulmonary function*, which analysis will be best? Which sample (venous or arterial) will have the least variability? To know the answer to these questions, you need to think about the number of physiologic changes, especially with regard to blood gases, that may occur between gas exchange in the lungs and our sample collection site. Venous blood is incredibly variable in that regard. Blood is returned to the left

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side of the heart from the lungs and then pumped through the aorta to most tissues of the body. In transit, tissue gas exchange will be consuming oxygen and releasing CO_2. Depending on where we draw the venous sample from, there could be tremendous alterations to blood gases. Arterial blood, on the other hand, is most reflective of the blood actually entering the left atrium from the lungs, and what's being delivered to all of the tissues of the body. Therefore arterial blood provides a more accurate assessment of pulmonary function. In pulmonary disease, we really need to know precisely how much oxygen is actually being delivered to the tissues. Only *arterial blood* can tell us that.

The cool thing about arterial blood collection is that we don't have to wonder if we've entered the artery with our needle. The heparinized syringe begins to fill automatically from the arterial pressure. When the needle is withdrawn, someone other than the blood collector needs to put immediate firm pressure over the site and maintain it for at least 5 minutes to prevent *hematoma* formation. The person collecting the sample immediately eliminates any air bubbles from the syringe and prevents any further air contact with the sample by plugging the needle with a rubber stopper. The sample should be placed in on ice and rushed to the lab for analysis. Often the lab will want to know the patient's body temperature at time of collection. *Arterial blood gas analysis* is temperature-compensated. Why? The higher the body temperature the more rapidly oxygen is consumed.

Normal ranges for arterial blood gas may vary slightly from lab to lab and species to species. But we can generalize by saying that for most normal dogs and cats breathing room air (21% oxygen), their arterial blood gas values will probably fall within the following range: pO_2 90 to 95 mm Hg, pCO_2 35 to 40 or even 45 mm Hg, HCO_3^- 21 to 27 mmol/L, and pH 7.35 to 7.45. We would expect pO_2 to be even better if the animal is receiving oxygen therapy (usually 30% to 40% for a therapeutic range). Believe it or not, I have seen arterial blood gas results from patients on a ventilator with ≥50% oxygen being delivered with pO_2 values of 70 to 75 mm Hg and pH of 7.1 or so. That tells me that the respiratory membrane was severely compromised. That is significant ***hypoxemia*** [*hypo-*

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low + *ox(o)-* oxygen + *-emia* a blood condition of] and *acidosis*. That cannot sustain life over the long term. And in most cases like that it didn't.

Ins and Outs of Breathing

Breathing (rate and depth) is controlled by the respiratory centers in the brain. We've already talked about those things that stimulate those centers, with CO_2 blood concentrations being the first and foremost powerful stimulant. Secondarily, low oxygen concentrations will also stimulate those centers. But what do those respiratory centers target? And how does that result in increased rate and depth of breathing?

Let's begin by looking at normal *inspiration* and *expiration*. A single breath (inspiration + expiration) is what we refer to as the ***respiratory cycle***. Both inspiration and expiration are completely involuntary acts. Normal ***expiration*** is a completely passive process. It relies on all of the elastic tissues (*pulmonary parenchyma*, *pleura*, relaxed muscles and abdominal viscera) simply springing back to their original state, prior to inspiration. It's really like a rubber band returning to its original size and shape after being stretched out. All of those tissues are stretched during inspiration and merely return to their original size and shape. But *inspiration* is facilitated by muscle activity.

The muscles involved in ***inspiration*** are the ***diaphragm*** (di'uh-fram [from Gr. *diaphragma* "a wall or partition"] and the ***external intercostal*** [*inter-* between + *cost(o)-* ribs + *-al* pertaining to] ***muscles***. The *diaphragm* is the principle muscle of inspiration. And it truly is a wall or partition, separating the *thoracic* cavity from the abdominal cavity. Only the aorta, caudal vena cava, and esophagus pass through the diaphragm at the ***hiatus*** [L. *hiatus* "gap or opening"]. That opening is completely sealed around those structures. And that's important. Otherwise, whenever we did abdominal surgery, the *thoracic cavity* would be open to the atmosphere and the animal would not be able to breathe on its own. What? It's true. You see, the thoracic cavity is for all practical purposes a vacuum. Pressure within that cavity at rest and during inspiration is always less than *atmospheric pressure*. Therefore the

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pleural cavity, aside from viscera, is empty—a void of negative pressure (a vacuum). And that vacuum-like state plus the thin film of fluid on the surface of the *visceral* and *parietal pleura* helps keep the lungs stuck to the rib cage and diaphragm. Yes, that thin film of fluid provides sticky *surface tension* between the lungs and the rib cage and diaphragm. So when the rib cage and diaphragm move, the lungs are along for the ride.

How does the ***inspiratory movement*** actually happen? Both the *diaphragm* and the *external intercostal muscles* are stimulated by the respiratory centers (via nerves of course) to contract. The diaphragm, when relaxed, is domed cranially into the thoracic cavity. When the diaphragm contracts, it pushes all of the abdominal viscera caudally. This is a big movement, and that's why the diaphragm is the primary muscle of respiration. At the same time, the external intercostal muscles contract, leveraging the ribs out laterally. Because the lungs are stuck to the diaphragm and rib cage, the lungs stretch with them, creating much more space within the lungs (i.e., alveoli). Well, that creates a significant drop in air pressure within the lungs. Air moves from high to low pressure (that's what creates wind in the environment). Because atmospheric pressure (normally 760 mm Hg or 14.70 psi at sea level) is higher than the current low pressure in the alveoli, air rushes in. At end-inspiration, pressures within the alveoli equalize with the surrounding air. Throughout inspiration, the fresh air (21% oxygen) mixes with residual air in the alveoli, facilitating gas exchange (O_2 into the blood, CO_2 out). Then, when all of the tissues and organs spring back during ***expiration***, pressures within the alveoli increase above atmospheric and air rushes out of the lungs, exhausting the CO_2. This is a ***passive expiratory movement*** that takes about twice as long as inspiration. There is a nice animation review of the respiratory cycle in the Evolve resources (*Respiratory Cycle*).

In times of increased need, like exercise (e.g., my pup running zoomies), respiratory centers increase the ***respiratory rate*** (*tachypnea*). If demand for even greater gas exchange is created, then the respiratory centers stimulate accessory muscles to facilitate increased rate and depth of respiration (*hyperpnea*). These accessory muscles were shown in and , in the Chapter 4. The ***serratus ventralis muscles*** can really expand the thoracic cavity by

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leveraging the ribs out much more than the external intercostal muscles can. So, the external intercostals expand the thoracic cavity as much as they can for inspiration, and then the *serratus ventralis muscles* pick up where the intercostals left off. A bigger thoracic cavity means increased space in the alveoli, which means lower pressure . . . and that means air rushes in quickly. By the way, ***stretch receptors*** in the visceral pleura stop the inspiratory movement, so that alveoli don't pop like overinflated balloons.

What about expiration? I mean, if we take more air in then we need to push more air out, right? And if the respiratory rate needs to increase, we can't exactly wait around for tissues to simply spring back into place. There has to be a shift, from a passive expiratory process to an active process. So, it only makes sense that we may need accessory muscles to force air out of the lungs to speed the respiratory cycle along. And increased levels of CO_2 cause the respiratory centers to stimulate respiratory muscles for forced expiration. This happens in both health and disease. Even my pup running zoomies will engage accessory muscles for expiration—namely the ***latissimus dorsi*** and ***abdominal muscles***. With these muscles engaged, expiration is no longer a passive activity. It is quite active and rapidly reduces the size of the thoracic cavity, forcing air out of the lungs. ***Internal intercostal muscles*** are also engaged, contributing to active expiration. But the major contributing muscles are the *latissimus dorsi* and *abdominal muscles*. Bye-bye CO_2!

Respiratory Volumes

During the *respiratory cycle* (i.e., a single breath—inspiration and expiration), a normal volume of air is moved. That normal volume is called the ***tidal volume***. Ocean tides ebb and flow, rise and fall, right? Well, with normal inspiration and expiration, the volume of air moving ebbs and flows too. That's why this is referred to as the *tidal volume*. The *tidal volume* of a normal animal is what's required to meet that animal's gas exchange needs at rest. We can actually estimate the tidal volume of an animal, based on body weight (i.e., *ideal* body weight). There are various formulas for that, depending on purpose. One of the most common purposes of estimating tidal volume is to select an appropriate-sized rebreathing bag for

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anesthesia. Then, when we assist or control ventilation of the patient while it is sleeping, we can ensure use of at least its *tidal volume* for gas exchange. Another purpose behind estimating tidal volume is to set minimal inspiratory volumes on a *mechanical ventilator*.

What about other volumes? Well, we said earlier that increased needs, from exertion or disease, could stimulate deeper breathing—and that means tapping into greater volumes of air over and above the tidal volume. Anything over and above the tidal volume is called a *reserve volume*, because it is held in reserve until we need it. For greater gas exchange, we definitely need ***reserve volumes***. And we separate those reserve volumes by the phase of the respiratory cycle in which they are used. So, to breathe in a larger volume of air, we tap into the ***inspiratory reserve volume***. To breathe out (forcefully, remember) a greater volume of air, we use the ***expiratory reserve volume***. And if we combine the tidal volume + inspiratory reserve volume + expiratory reserve volume, we have a total ***vital capacity***. It's vital, because it is needed to sustain life in times of greater need. If there is increased need and we can't meet that need, we may not survive, so *vital capacity* is vital for our survival. Don't forget, we typically have a tiny ***residual volume*** remaining in the alveoli at end-expiration, to keep gas exchange going and to make it easier for the next inspired breath. Remember, it's the *residual volume* and *surfactant* that keep the alveoli from sticking shut.

By the way, when estimating tidal volume for selection of an appropriate rebreathing bag, we typically select a bag slightly larger than just the tidal volume. This allows us to tap into the inspiratory reserve volume to improve gas exchange in an anesthetized patient. Of course, when we manually breathe for the patient, there is the risk that we could exceed that volume and burst alveoli. The stretch receptors won't help in this situation, because WE are doing the breathing, not the patient. To prevent damage to alveoli, we need to pay attention to pressures that we manually exert on the airways through the *endotracheal tube*. There is a ***manometer*** [Gr. *manos* thin + *meter* measurer] on every anesthesia machine. As we *ventilate* the patient with the rebreathing bag, we can watch the pressure increase on the *manometer*. We never want to

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exceed 15 to 20 cm H_2O pressure.

All right, let's try to put what we've learned thus far to use. Let's take a look at various cardiopulmonary diseases and how they adversely affect both cardiac and respiratory function.

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Integration in Health and Disease

In both the cardiovascular and respiratory sections, we talked about how we might alter function in times of need. Of course, many of our examples were normal situations of need, such as exertion. Disease states also create times of greater need. For example, inChapter 3 we talked about various types of ***anemia*** [*an-* without + *hem(o)-* blood + *-ia* condition of; i.e., insufficient hemoglobin or red blood cells]. Think about how the body will perceive insufficient numbers of red blood cells. With fewer RBCs, we have reduced oxygen-carrying capacity, right? So, what should we do in response to keep up with normal tissue demands for oxygen? If we speed up the delivery vehicles (RBCs), we might be able to keep up with supply and demand. So, an *anemic* animal will likely be *tachycardic*, even at rest. (I have observed patients who have been anemic for so long that they actually adapted to it. With adaptation, their heart rates were closer to normal.) The patient will also likely have *peripheral vasoconstriction*, to conserve available oxygen for vital core organs and tissues. So, between reduced RBCs and vasoconstriction, these animals look really pale. I've seen some, like the puppy with severe hookworm anemia discussed inChapter 3, whose mucous membranes were blanched (white). Vasoconstriction reduces demand for oxygen by limiting delivery to "sacrificed," nonessential tissues. To further reduce demand, the animal will likely be ***lethargic*** [Gr. *lethargia* drowsiness + *-ic* pertaining to]. Less activity means less demand for O_2. And if the animal exerts itself, the perceived tissue *hypoxia* will likely drive the respiratory centers in the brain to make the animal *tachypneic*. It is so gratifying to watch these things change during a blood transfusion. This is especially true for patients, like the puppy with hookworm anemia and the cat with fleas that were mentioned in Chapter 3. Over the course of the transfusion (usually 2 to 4 hours), the heart and respiratory rates slow down, mucous membranes become pink, and the patient's activity level increases—all because of improved oxygen-carrying capacity. Is carbon dioxide diffusion affected at all by the anemia? No. Remember, most the carbon dioxide is transported by the plasma. We have plenty of that in an anemic

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animal. So, that is a simple example of disease that affects both cardiovascular and respiratory systems. Now that you've tried your hand at problem-solving cardiopulmonary responses to disease, let's go deeper.

Cardiomyopathy

Cardiomyopathy [*cardi(o)-* heart + *my(o)-* muscle + *-pathy* disease of] is literally a disease of the heart muscle itself. Obviously, this directly affects function, especially *cardiac output*. Whether the heart muscle becomes flabby and weak, or really bulky, reducing ventricular chamber size, either way cardiac output is reduced. Of course, we can appreciate poor cardiac output with weak pulses and low blood pressure. But those assessment tools really don't tell us that it's the heart muscle itself causing those changes. ***Hypovolemia*** [*hypo-* low + *vol(o)-* volume + *-emia* a blood condition of] from excessive blood loss or severe dehydration could create *tachycardia*, weak thready pulses, and ***hypotension*** [*hypo-* low + *tens(o)-* pressure + *-ion* state of]. To know for certain that the *myocardium* itself is the problem, we need to look at it. And the best way to do that is to use ***echocardiography*** [*echo* echoes + *cardi(o)-* heart + *-graphy* recording].

Echocardiography

Echocardiography is similar to the abdominal ultrasound that we talked about inChapter 1. Both are noninvasive procedures that use sound waves bouncing off of organs and structures to visualize them. Of course, with *echocardiography* we're focusing exclusively on the heart. And the beauty of it is that we can see the heart functioning in real-time. We can watch the movements of chamber walls and valves. And by using ***Doppler*** (yes, just like Doppler radar for the weather) we can watch blood flow to see if there is any turbulence and *valvular regurgitation*. With still (stop-action) ***echocardiographic*** [*echo* echoes + *cardi(o)-* heart + *graph* record + *-ic* pertaining to] views, we can critically measure wall thickness and chamber size. All of the information we gather through *echocardiography* is essential for diagnosing cardiac diseases, like *cardiomyopathy*.

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Dilatory Cardiomyopathy

Dilatory [*dilat(o)-* dilation, expansion + *-ory* pertaining to] ***cardiomyopathy*** (***DCM***) is common in dogs. We tend to see it more frequently in certain dog breeds, like the Doberman Pinscher, Great Dane, and many others (mostly large-breed dogs). On occasion, dilatory cardiomyopathy may also be seen in cats, especially breeds like the Abyssinian, Burmese, and Siamese.[2] Generally in cats, ***hypertrophic*** [*hyper-* excess + *troph(o)-* development + *-ic* pertaining to] ***cardiomyopathy*** is far more common. We'll talk about that in a moment.

With dilatory cardiomyopathy, there is loss of *myocardial* tone in the ventricles. So, the ventricles have poor contractility and the chambers dilate. Obviously, this greatly reduces *cardiac output*, resulting in *hypotension*. With regard to cardiac output, the effects of these changes are most important in the left ventricle. Well, how does the body respond to hypotension? It engages every homeostatic mechanism it has to raise blood pressure. So, all of the sympathetic, hormonal, and renal mechanisms that we talked about earlier are engaged. Let's think about how all of those mechanisms contribute to raising blood pressure. They cause *tachycardia*, *vasoconstriction* (especially in the periphery), *increased thirst*, and *reduced urine output*. Now, in our earlier blood pressure discussion, we said that the hormone *epinephrine* had a *positive inotropic* effect on the heart. Unfortunately, in *DCM*, it's mighty hard to improve contractility of flabby, weak myocardium. So, all of these mechanisms do a poor job at best to improve blood pressure, especially as the disease worsens.

Echocardiographic imaging reveals dilated chambers and poor movement, especially of the ventricular free-walls. Radiographic imaging reveals *cardiomegaly*. Can you predict how the *QRS complex* on an electrocardiogram will be altered? That's right, the QRS will become wider (prolonged), because depolarization is covering more ground, due to the ventricular dilation. Plus, it's not unusual at all to see numerous *VPCs* or even *V-Tach*. Well, that reduces cardiac output even more. And even with normal valvular function, these animals often have a *third heart sound*, creating a ***gallop rhythm*** when heard. It's called a *gallop rhythm* because it sounds like a galloping horse (lub-dub-dub). The extra sound is actually created

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by blood splashing into the enlarged ventricles when the AV valves open. Amazing, huh? How do you think these animals will compensate in exertion? They can barely compensate at rest. So, under exertion their ability to maintain adequate circulation and gas exchange will, in a word—suck. In fact, it wouldn't be unusual at all for them to pass out. We call that ***syncope*** (sing'kuh-pe [Gr. *synkope* fainting]). By the way, if circulation sucks in these patients, so does their gas exchange. They go hand in hand, because we can't move the delivery vehicles efficiently. Eventually, these animals develop ***heart failure***. And we'll talk about the consequences of that in a little while. But first, let's take a quick look at ***hypertrophic cardiomyopathy (HCM)***.

Hypertrophic Cardiomyopathy

Hypertrophic cardiomyopathy (HCM) is quite common in cats, both pure breeds and mixed breeds. According to Dr. Jerold Bell, a veterinary ***geneticist*** [*genetics* + *-ist* specialist in], it is the most common cardiac condition of cats, with a 10% prevalence in the feline population.[3] Many of these cats develop *HCM* secondary to other underlying diseases. ***Hyperthyroidism*** [*hyper-* excess + *thyroid* + *-ism* state or condition of], to be discussed inChapter 10, commonly leads hypertrophic cardiomyopathy in cats. But there are those cats, like the Maine Coon, who have a *genetic* predisposition to HCM. These cats could be symptomatic as young as 1 to 2 years of age. That's young! I'm not talking about HCM in dogs because it's incredibly rare.

Whether primary or secondary for affected cats, the left ventricle tends to be most affected by the *hypertrophic* changes. Both the *interventricular septum* and the left ventricular free-wall become extremely hypertrophic (kind of like the upper arm of a WWF wrestler). But (and here's a really big but), the overall heart size doesn't increase that much. Sure, it'll be enlarged. But most of the time, the *cardiomegaly* would be classified as mild. So, if the *hypertrophic myocardium* isn't bulging out, then it must be taking up space within—and it does. The chamber size of the left ventricle can be extremely reduced. Let's see, what will that do to *stroke volume* and *cardiac output*? Yep, it sucks. Of course, that makes all of those blood pressure mechanisms kick into high gear. At least this muscle

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can attempt improving cardiac output and blood pressure. It's not weak and flabby like the dog's DCM heart. And because the all-important left ventricle is functioning so poorly, pumping out only small volumes of blood, blood tends to back up. Plus, due to left ventricular changes in shape, mitral regurgitation compounds the blood back up in the left atrium. And that usually causes left atrial dilation. It's a real domino effect that, like the dog with dilatory cardiomyopathy, often leads to *heart failure*. Certainly, heart failure is problematic and lethal. But for many of these cats, it's the blood pooling in the left atrium and pulmonary veins that creates the complication of ***saddle thrombus*** [*thromb(o)-* clot + *-us* a].

Saddle Thrombus

Saddle thrombus is probably more accurately described as a case of ***thromboembolism*** [*thromb(o)-* clot + *embol(o)-* thrown in + *-ism* act of]. As you may recall from our discussions of clotting inChapter 3, we spoke of vascular injury stimulating the clotting cascade to form a clot, to stop bleeding. But in the case of a cat with hypertrophic cardiomyopathy, there is no vascular injury. But there is ***stasis*** [Gr. *stasis* "a standing still"] of blood flow. The blood backflow and pooling in the atrium and pulmonary veins creates enough *stasis* for *thrombus* formation. Unfortunately, this is a precarious location for a *thrombus*. Think about where venous blood from the pulmonary veins and left atrium go next—the left ventricle. From there, blood is whisked away through the aorta and all of its tributaries. If a clot happens to be in that aortic blood, it will eventually be "thrown" into a smaller artery. That is what an ***embolus*** [*em-* in + *bol(o)-* to throw + *-us* a, an], or more accurately a ***thromboembolus*** [*thromb(o)-* clot + *embol(o)-* thrown in + *-us* a, the], is. It is a clot that is thrown into an arterial branch. And for most *HCM* cats that *thrombus* is typically thrown into the caudal aorta and final branches off the aorta—the ***iliac arteries***. If the term iliac made you think of the portion of the pelvis called the ilium, you are thinking appropriately. The iliac arteries branch off the aorta near the pelvis, providing arterial blood to the rear limbs.

So what happens with a *thromboembolus* in the *iliac arteries*? Well, it severely impedes blood flow to those legs and feet. (This may affect one or both extremities, depending on whether the embolus

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involves one or both iliac arteries.) That ***ischemia*** [*isch(o)-* from Gr. *ischein* to suppress + *-emia* a blood condition; i.e., suppression or deficiency of blood to a part] wreaks havoc with the tissues in those feet and legs. They progressively become *hypoxic*. And because blood flow is so poor, unable to deliver O_2 or remove CO_2 much if at all, those tissues also progressively become *hypercapnic*. Those factors may begin to cause cellular death. Sensory nerve endings in the area are stimulated and activate pain pathways to the brain. And if arterial pressures are strong enough to jam that clot in, such that it completely obstructs blood flow, then we have a really big problem called an ***infarction*** [L. *infarcire* "to stuff in"]. With an *infarction*, the ***acute*** (sudden) tissue hypoxia, hypercapnia, and stimulation of sensory nerves result in *acute* excruciating pain for the cat. Have you ever had one of your extremities "fall asleep" because you held it in an awkward position for too long? Do remember the burning, tingling, prickly sensations associated with that? Well, magnify that perhaps a hundred times and you probably have a good idea of what a cat with *saddle thrombus* feels. By the way, cats are not the only ones to experience this. I've seen dogs with dilatory cardiomyopathy develop this too.

When these animals are presented, the affected limb or limbs will be cool to the touch. Areas of their skin and paw pads without pigmentation will be pale, *blanched* (white), or *cyanotic*. Femoral pulses will be weak or absent. The animals typically cannot use the affected limb(s). And the patient may exhibit extreme outward behavior indicative of pain. Most cats express a loud, haunting yowl that sends chills down the spines of most veterinary professionals. We know what it means and that this may not end well. Is there anything we can do for these animals? Well, time is of the essence, especially with *infarction*. If we don't dissolve that clot rapidly and restore blood flow, tissue *necrosis* will develop. And if that happens, it can only mean either limb amputation and/or loss of life (usually humane euthanasia). There are a number of powerful ***anticoagulants*** [*anti-* against + *coagul(o)-* clotting + *-ant* one that] or "clot-busters" as some folks like to call them. If initiated quickly, we might be able to restore blood flow. And if recovery can be achieved, the patient is typically put on low-dose anticoagulant therapy for the rest of its life. Still, saddle thrombus may happen

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again. Sadly, many of these patients do not survive.

Heart Failure

Many things, including *cardiomyopathy* and *valvular insufficiencies,* can lead to ***heart failure***. In *heart failure*, the heart has lost its functional capacity to maintain the body for every day activity, let alone in disease situations. *Cardiac output* is poor, which means blood pressure is poor. Remember under normal circumstances, *hypotension* will activate multiple mechanisms to raise blood pressure, including increased *sympathetic tone*, increased secretion of *antidiuretic hormone* (*ADH*, *vasopressin*), and increased activation of the *renin-angiotensin system*. We said in a normal animal *peripheral vasoconstriction* (from sympathetic nervous stimulation, vasopressin, epinephrine, and angiotensin II) increases blood pressure. Also, ADH and angiotensin II *increase thirst* and cause the kidneys to *reabsorb water*, increasing blood volume and pressure. The kidneys also resorb more *sodium* (Na^+) to increase water retention and resorption. Plus, the *positive inotropic effects* of epinephrine increase heart rate and contractility to improve cardiac output. All of these homeostatic mechanisms are beneficial for a normal animal.

But in *heart failure*, we have poor contractility and cardiac output, with a weak heart unable to compensate. Unfortunately, those mechanisms just mentioned create an added level of difficulty for this weakened heart. All of the peripheral vasoconstriction creates more resistance that this weak heart must pump against. We call that "***afterload.***" Suffice it to say that in heart failure, there is simply no way for this weak heart to overcome increased *afterload*. The heart simply cannot muster greater *systolic pressure* to pump against the increased vascular resistance. *Systolic* pressures are poor and easily recognized clinically with weak peripheral pulses and prolonged ***capillary refill time*** (***CRT***). And because systolic pressures are so poor, blood backs up making venous return is poor. And poor venous return and pooling increases ***hydrostatic*** [*hydr(o)-* water + *-static* pertaining to standing] ***pressure*** in the capillaries. Remember inChapter 3, we said that increased *hydrostatic pressure* pushes water out of the capillaries into the

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interstitium [*inter-* between + *stiti(o)-* tissues + *-um* the]. We learned that *increased interstitial fluid* is also known as *edema*. And because of increased resorption of both sodium and water, in a heart failure patient, *edematous* fluids can be profound. (This is why *cardiac diets* are low in sodium, to minimize water retention and edema.) Development of edematous fluids due to heart failure is what we often refer to as ***congestive*** [from L. *congere* to heap together + *-ive* pertaining to] ***heart failure*** (***CHF***). And in CHF we have edematous fluids that are truly "heaped together." But where will edema develop? That depends on which side of the heart is failing. Yes, we can have right-sided or left-sided heart failure. Worst case scenario would be complete heart failure, with both sides involved. Let's take a look at some of the common edema consequences in heart failure.

Pulmonary Edema

So, let's see if we can figure out which side of the heart will result in ***pulmonary edema***. We need venous pooling that causes increased hydrostatic pressure in the *pulmonary* capillaries, right? So, which side of the heart causes this? That's correct—it's a *left-sided heart failure* that results in pulmonary edema. Two of the most common conditions that contribute to left-sided heart failure are hypertrophic cardiomyopathy in cats and *mitral regurgitation* in dogs.

What are the consequences of pulmonary edema? Let's see, there is increased thickness of the *respiratory membrane* reducing gas exchange, especially oxygen. So, *hypoxia* will be a problem. And when it becomes severe, water seeps into and begins filling the alveoli. The greater the amount of edematous fluid, the worse pulmonary gas exchange. But remember, the cause of this is ***cardiogenic*** [*cardi(o)-* heart + *gen(o)-* produced + *-ic* pertaining to] *pulmonary edema* because of heart failure. Even if mild to moderate pulmonary edema is not significantly affecting diffusion of CO_2, tissue *hypercapnia* will likely still be present to a degree due to poor circulation/poor venous return. So, a left-sided heart failure patient with pulmonary edema may present with *pallor* or *cyanosis*. And if the patient presents with *dyspnea* and *cyanosis*, we know that cardiopulmonary function is severely compromised.

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Pleural Effusion

Effusion [L. *effusio* "a pouring out"], as a consequence of congestive heart failure, is an accumulation of edematous fluid in a body cavity. ***Pleural effusion*** is accumulation of those fluids in the *thoracic* cavity (i.e., surrounding the lungs). This may happen in any animal with congestive heart failure. But it most commonly develops in cats, especially in those with *cardiomyopathy*. It also develops from other diseases, not related to heart disease at all, like certain cancers. And I've seen a number of cases of lung lobe ***torsion*** [from L. *torsio* to twist]. As the inflammation from the dying lung lobe develops, a great deal of fluid can effuse into the chest cavity with this. My point? *Pleural effusion* is not exclusive to congestive heart failure.

How does *pleural effusion* affect pulmonary function? Well, let's think about normal mechanisms of breathing for a moment. For normal inspiration, we need that thin film of fluid between the *visceral and parietal pleura*, just enough to stick the lungs to the *parietal pleura*. That *surface tension* will cause the lungs to expand with the chest cavity (i.e., diaphragm and rib cage). But with *pleural effusion*, there is too much fluid in the thoracic cavity. (Remember, there should be nothing in the pleural cavity except the organs that reside there.). Surface tension is lost and the lungs can't stick to the parietal pleura. *Atelectasis* results. The lungs for all practical purposes float in the effused fluid. Surface area for gas exchange has just been severely compromised. These animals will be *dyspneic* (*orthopneic* if effusion is severe), *hypoxic,* and *hypercapnic*.

Often, when the pleural effusion is severe, we need to remove some of the effused fluid. For that, we perform a procedure called ***thoracentesis*** [*thorac(o)-* chest + *-centesis* to puncture]. In this procedure we penetrate the chest cavity through an *intercostal* space with either a needle or a catheter attached to a collection line, a three-way stop cock, and a large syringe. This is an ultrasound-guided procedure. We can't afford to hit the lungs or the heart with our needle. If effusion is not severe, we may simply use a needle and syringe to collect a diagnostic sample. We need to know the character of the fluid. Pleural effusion will be treated quite differently than a ***hemothorax*** [*hem(o)-* blood + *thorax*] or ***pyothorax*** [*py(o)-* pus + *thorax*]. For pleural effusion, with our temporary

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collection system, we can withdraw as much fluid as possible. I've helped withdraw over two liters of fluid from some canine patients. It's quite remarkable to see them progress from extreme respiratory distress to relaxation as we remove the majority of the fluid.

Of course, in heart failure and many other conditions, like lung lobe torsion, we need to treat the underlying condition. Until then, the pleural effusion will develop again and again. There are many drugs that may be used to treat heart failure patients. ***Diuretic*** [*diure(o)-* urination + *-tic* pertaining to] drugs, like ***furosemide***, promote greater removal of fluid from the body through urination. Other drugs, like ***ACE-inhibitors***, break the *renin-angiotensin* cycle to reduce *vasoconstriction* and *afterload*. (*Angiotensin II* can't form without angiotensin-converting enzyme—ACE.) This reduces peripheral resistance that the heart needs to work against, improving its efficiency. There are also *positive inotropic* drugs, like ***digitoxin***, that improve contractility of the *myocardium* to improve *cardiac output*. Obviously, a *low sodium diet* for these patients is essential to minimize fluid retention in the body. The goal of drug therapy and diet is to improve heart function, which is a challenge with a failing heart. Eventually, as the heart failure progresses, our efforts will be insufficient.

This has nothing to do with heart failure, but this is a good time to talk about lung *torsion*. It can create significant pleural effusion. In the case of lung lobe torsion, a ***thoracotomy*** [*thorac(o)-* chest + *-tomy* to cut] is required to remove the ***torsed*** [twisted] lung lobe. This is challenging because as soon as we enter the thoracic cavity, exposing it to atmospheric pressure, the animal cannot breathe on its own. We have to breathe for the patient, throughout the procedure, usually using a *mechanical ventilator*. (This is a good example of a situation in which we need to carefully estimate tidal volume.) Generally, we can't simply untwist the lung lobe. These lobes are usually ***necrotic*** [*necr(o)-* death + *-tic* pertaining to]. Removal of the lobe is necessary, if that's the case (and it usually is). After the lung ***lobectomy*** [*lob(o)-* lobe + *-ectomy* to cut out; i.e., remove], we need to restore the vacuum-like environment for the chest cavity. Plus, from the surgical trauma, *postoperative* inflammation will probably result in further *pleural effusion* (temporarily). So, as the chest is being closed, a *chest tube* or *thoracic*

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catheter is usually placed. At the end of surgery, all air is withdrawn from the *pleural cavity* via the chest tube to restore "negative pressure" and needed surface tension, allowing the animal to breathe on its own. In recovery (perhaps for 1 to 3 days), the chest tube is maintained in place and used to evacuate any effused fluid that develops. Once fluid production is minimal, the chest tube is removed.

Ascites

Ascites (uh-si'-tēz [from Gr. *askites* "bag"]) is an accumulation of effused fluid in the abdominal cavity. *Ascites* is common in *right-sided heart failure*. That makes sense, when you consider the volume of venous blood returning to the right side of the heart from the caudal body. *Valvular insufficiencies* (*tricuspid valve* or *pulmonic valve*) can lead to right-sided heart failure. C*ardiomyopathy*, especially dilatory cardiomyopathy, can cause this too. *Heartworm disease* is another very common contributor to right-sided heart failure. We'll talk about heartworm disease shortly. Regardless of cause, *ascites* can also affect breathing. How? The more fluid builds up in the abdomen, the less the *diaphragm* can expand the chest cavity. The diaphragm can't move as far caudally when it contracts. There's no room because of the *ascites*. As with pleural effusion, excess fluid needs to be removed from the abdominal cavity. This time ***abdominocentesis*** [*abdomen(o)-* abdomen + *-centesis* to puncture] is used. I have seen liters upon liters of ***ascitic*** [*ascites* + *-ic* pertaining to] fluid removed via *abdominocentesis*.

Sometimes, ***bicavitary*** [*bi-* two + *cavit(o)-* cavity + *-ary* pertaining to; i.e., both *pleural effusion* and *ascites*] ***effusion*** develops (i.e., ascites AND pleural effusion). Those animals are severely compromised—often with both *hypotension* and poor gas exchange. Intense emergency care is required to stabilize such patients. The ***prognosis*** [Gr. *prognosis* foreknowledge; i.e., forecasted outcome] for patients with *bicavitary effusion* is usually very poor.

Heartworm Disease

Heartworm disease is one of the most common, preventable cardiac diseases of dogs and cats. The parasite, ***Dirofilaria immitis*** (di"ro-

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fil-ār'e-uh im'ĭ-tis), is intended to infect dogs. However, over the years it has adapted to infect cats as well. Still, only dogs can serve as reservoirs for the parasite, because the adult worms can only reproduce in the dog.

It is important to understand the life history of the parasite. In an infected dog, the adults of *Dirofilaria* live in the heart—primarily in the right ventricle and pulmonary artery, perhaps into the right atrium. All it takes is one adult female and one male to be reproductive and produce offspring—***microfilaria*** [*micro-* small + *filaria* from L. *filum* thread; i.e., 1st stage larva; microfilaria = singular, microfilariae = plural]. Even just two adult worms can interfere with heart function, especially valvular function. These worms may be skinny, but they're long. The average female may be close to 12 inches long. A male may be in the neighborhood of 7 inches long. They can easily stretch from the right atrium to the pulmonary artery, especially in a smaller heart. Obviously, the more worms occupying space in the heart, the more severe the consequences. I've seen dog hearts, on ***necropsy*** [*necr(o)-* death + *-opsy* viewing of; i.e., after death examination] of course, with the right chambers and pulmonary artery just packed with worms. That can create symptoms of right-sided heart failure, as well as respiratory symptoms. Blood flow to the lungs can be significantly impaired. These dogs have significant *exercise intolerance* and may become *cyanotic* from the slightest exertion. Cats don't tend to have as heavy a worm burden (i.e., large numbers of worms). But even one or two adults in a cat's small heart can create significant problems. Cats almost exclusively have ***occult*** [L. *occultus* "concealed"] ***infections***—meaning the adults are only one sex and cannot reproduce.

In a dog with a ***patent*** (i.e., "open", reproducing offspring) ***infection***, *microfilariae* are circulating in the bloodstream. And believe it or not, there are more microfilariae in circulation at night. This corresponds well to the feeding habits of the *mosquito*. The mosquito is required for transmission. When the mosquito feeds on the infected dog, microfilariae are consumed with the blood meal. Then over the next 3 weeks or so, the *microfilariae* go through development to third-stage larvae in the mosquito. It is the third-stage larvae that are infective. When this mosquito with infective

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larvae feeds on another dog, *Dirofilaria* is transmitted to that dog. The larvae enter the dog through the site of the mosquito bite and begin to migrate through the tissues. It will take several months before they reach the heart. Our goal is to kill them off long before that. *Heartworm preventive* medications are designed to kill off these migrating larvae. Most of the preventive drugs used are effective against the migrating larvae, during approximately the first month of their migration (i.e., ~30 days from the infective mosquito bite). That's why most heartworm preventative medications are given on a monthly basis. If we miss that window, the drug may no longer be effective and the larvae will eventually reach the heart to develop as juvenile worms in the right side of the heart. It takes a minimum of 6 months, from being bitten by an infective mosquito until there are reproducing adults in the heart. It is worth noting that climate change may alter mosquito activity, even in temperate regions, making it possible for transmission of *Dirofilaria* outside of traditional "mosquito season." As long as there is warm weather to support the mosquito population, heartworm transmission is possible. (Note: mosquitoes may be sluggish between 50 to 60° F, but they are still active.)

Can we do anything once a dog is infected with adult heartworms? Yes, we can treat them, if they are healthy enough to withstand treatment. These animals must be thoroughly screened with *thoracic radiographs*, *echocardiography*, and laboratory work (especially *serum chemistries* to evaluate liver and kidney function). If deemed healthy enough, treatment is often done in two stages. First, drugs are given to kill the adults residing in the heart. Second, drugs are given to eliminate circulating microfilariae. There are risks associated with each stage of treatment. Many of these drugs can be damaging to the liver. That is why the laboratory work is so important, to know if the liver is diseased before giving these drugs. And many drug metabolites (by-products of drug metabolism) are cleared by the kidneys. So, knowing *renal* function is important too. But the greatest risk of killing the adult worms is development of a ***pulmonary embolus*** (*emboli*—plural).

Pulmonary Embolus

Pulmonary emboli (***PE***), per TV commercials, are usually from deep

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vein thrombosis (DVT) in people. I've seen that happen once in a large dog who had been recumbent for an extended period of time, due to hit-by-car injuries. Between the recumbency and bandages on the rear legs that impaired venous return, the dog developed *thrombus* (DVT) formation. Soon after he became active for the first time *postoperatively*, he developed *bilateral* pulmonary emboli. Sadly, he did not survive. But in the case of heartworm disease, a *pulmonary embolus* usually results from portions of the dead worms obstructing tributaries of the pulmonary arteries. Like the *postoperative* dog with DVT, pulmonary emboli from dead heartworms can rapidly become life-threatening. This is why it is so imperative that following treatment to kill the adult heartworms, the dog must be kept absolutely quiet for at least 6 to 8 weeks. It takes that long for the dead worms to be removed, bit by bit, by *macrophages*. If the dog becomes excited or engages in exercise during this time period, the heart will be required to pump harder. And that could cause large portions of dead worms to break free from the right ventricle or pulmonary artery and produce ***pulmonary embolism*** [*embol(o)-* thrown in + *-ism* act or state of]. Owners need to be warned of this possibility. For high-strung, excitable dogs, sedation may be needed to get through the 6 to 8 weeks without incident. Over those weeks, ***macrophagic*** [*macr(o)-* large + *phag(o)-* eaters + *-ic* pertaining to] activity should remove the worms safely.

Following treatment for adult heartworms, in *patent* infections, we also need to eliminate microfilariae. Drugs used for heartworm prevention may be used for this. However, there is a catch. Dogs with active, patent heartworm infections have been sensitized to the parasite and its offspring. There can be billions of circulating *microfilariae* in such a dog. If we kill them off all at once, this could create a profound allergic reaction called ***anaphylaxis*** [*ana-* bac, up, again + *phylaxis* guarding; i.e. over-guarding, an excessive response]. This too is a life-threatening condition. Refer toChapter 3 to review *anaphylaxis*.

Bottom line: it is best to prevent heartworm disease. The disease itself and treatment for active infections can be deadly for both dogs and cats. And cats are nearly impossible to treat without serious consequences. For any patient who already has significant cardiac

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changes from the infection (e.g., chamber dilation), those changes may not go away after the worms are gone. That means the patient will have to live with chronic (long-term/life-long) heart disease. With all of the heartworm preventives on the market, preventing heartworm disease has never been easier. And most manufacturers stand behind their products. So, if we find a patient who has developed heartworm disease while on regular preventive therapy, the manufacturer will often pay for hospitalization and treatment. Fortunately, these situations are rare. Over the years, I've experienced those "difficult" pet owners who argue that heartworm prevention is just a money-making scheme. They think preventive medication is too expensive. So, they would rather gamble, playing the odds that their dog won't be bitten by an infective mosquito. For them, I try to share the cost of treatment (including all of the testing prior to, during, and after treatment) compared to the cost of prevention for a year. Prevention is always much, much less expensive. AND prevention is not life-threatening. Think about it. They could spend hundreds or thousands of dollars in treatment and still have a dead dog by the time all is said and done. Is that worth it? Somehow, I doubt it. I have a family member whose ex-husband thought it wasn't necessary to give heartworm preventive. Her dog is now infected with heartworms and she can't afford treatment. Until she can, she is at least treating to eliminate the microfilariae so that the dog is not a threat to other dogs in the area. All it takes is one mosquito to pass it on.

Infectious Respiratory Diseases

Infectious respiratory diseases are also quite preventable, through immunizations. Even if an immunized animal contracts one of these infectious organisms, the severity of disease tends to be quite mild, making it more of a nuisance than anything. And that is the point behind immunization—to prevent or at least significantly reduce the severity of disease. (Refer to Chapter 3 to review how immunizations work.) And prevention is important to protect the larger population, especially those who are most vulnerable (the young, the old, and those immunosuppressed from disease). The same is true for influenza in people. Immunizations protect public

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health. (Have you had your flu shot?) Most of these diseases tend to remain *upper respiratory* issues. However, even that can be quite problematic. In a cat, for instance, with an ***upper respiratory infection*** (***URI***), if it can't smell anything due to the *rhinitis*, it won't eat. And cats who don't eat, especially if they're fat, can develop very serious, even life-threatening problems. Most of the time dogs with ***infectious tracheobronchitis*** [*trache(o)-* trachea + *bronch(o)-* bronchus + *-itis* inflammation of] don't have much of a problem, aside from a nasty, dry persistent cough that lasts for weeks. We often refer to this as "***kennel cough.***" And that cough often keeps the dog and the owners from getting sufficient rest, because the dog is coughing so much day and night. Sometimes these dogs cough so much and so hard that they worsen the inflammation of the airways and burst capillaries in the tracheal mucosa. Then we might see mild ***hemoptysis*** [*hem(o)-* blood, bloody + *-ptysis* to spit; i.e., coughing blood]. Those dogs definitely need *antitussive* medication. But for animals with underlying *cardiopulmonary* disease and/or poor immunity, some of the diseases that cause upper respiratory infections, like "kennel cough," can progress to the lower airways and cause *pneumonia*. Some of them naturally rapidly progress to fulminating ***pneumonia***, requiring hospitalization and intensive care (like *canine influenza*).

Pneumonia

We mentioned ***pneumonia*** [*pneumon(o)-* lungs + *-ia* condition of; i.e., a deep lung condition with alveolar pus and inflammation] earlier, as a secondary complication to pulmonary edema and aspiration of material, such as vomit or regurgitation. More frequently, *pneumonia* develops from infectious diseases. And there are many ***etiologic*** [*eti(o)-* cause *log(o)-* knowledge + *-ic* pertaining to] *pathogens* of pneumonia—bacteria, viruses, and fungi. There are even some parasitic worms, like hookworms and roundworms, whose larvae migrate through the *pulmonary parenchyma* that may result in ***verminous*** [*vermin(o)-* worm + *-ous* pertaining to] ***pneumonia***. It is important to accurately diagnose the *etiology*, in order to treat the patient effectively. Until that diagnosis is made, intensive supportive care is needed.

Why is pneumonia so devastating? Well first, *pathogens* create

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profound inflammation in the alveoli, tertiary bronchi, and bronchioles. Due to alveolar inflammation, the *respiratory membrane* is thickened from *edema,* as well as ***purulent*** [L. *purulentus* pertaining to pus] ***exudates***, reducing gas exchange. ***Exudation*** [L. *exsudare* to sweat out + *-tion* act or state of] is a process in which fluid, protein, cells, and cellular debris escape the vasculature into the tissues. In the case of pneumonia, the *purulent exudates* accumulate in the alveoli and lower airways. And with regard to the tertiary bronchi and bronchioles, edema, exudates and *bronchospasm* all contribute to ***bronchostenosis*** [*bronch(o)-* bronchus + *stenosis* narrowing], making it very difficult to move air. So, *dyspneic* patients tend to open-mouth breathe. By bypassing the *nasal turbinates*, dry inspired air ***inspissates*** [L. *inspissatus* from *in-* intensive + *spissare* to thicken; i.e., extreme thickening of] the exudates. *Inspissated exudates* not only reduce gas exchange more (harder to diffuse through really thick stuff), but they may obstruct small airways, especially bronchioles. All of this has a profound negative impact on respiration and gas exchange. Patients with pneumonia are often very *hypoxic* and *hypercapnic*. And if that's not bad enough, in severe pneumonia cases the inflammation may progress to involve the pleura. ***Pleuritis*** [*pleura* + *-itis* inflammation of] is extremely painful. Imagine really inflamed visceral and parietal pleura rubbing against each other (squeak, squeak) with each and every inspiratory and expiratory movement. You can actually hear that rubbing with a stethoscope. It sounds like someone rubbing their damp hand over a balloon. We call that a ***friction rub***, because that's what it is. Most patients inhibit respiratory movements due to the pain. That's not good. *Hypoventilation* [*hypo-* low, under + *ventilation* act of breathing; i.e. shallow breathing] worsens the *hypoxia* and *hypercapnia* by further reducing functional alveolar surface area used. Remember, *cardiac arrhythmias* often develop in *hypoxia* and *acidosis*. And if ***pyothorax*** [*py(o)-* pus + *thorax* chest; i.e., pus accumulations in the chest cavity] develops, portions of the lung lobes become *atelectic*. Suffice it to say that these patients are critical.

Intensive care for *pneumonia* and ***pleuropneumonia*** [*pleura* + *pneumonia*] patients is multifaceted. Oxygen therapy is an absolute must. Remember, normal atmosphere contains only 21% oxygen.

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Therapeutic oxygen is usually between 30% and 50%. ***Intranasal*** [*intra-* within + *nas(o)-* nose + *-al* pertaining to] oxygen catheters sutured in place are usually tolerated much better than oxygen masks, especially if the patients are really *dyspneic*. And in severe cases, bilateral *intranasal* catheters are needed. If the animal will fit, an *oxygen cage* is another option. Second, *hydration* is also very important. *Intravenous* fluid therapy is important for circulation as well as liquification of airway secretions. Therapeutic oxygen must always be humidified. But that's not enough to liquify secretions. To further liquify airway secretions to facilitate easier removal, respiratory therapy also includes ***nebulization*** [from L. *nebula* "mist" + *-zation* act or process of; i.e., conversion of a liquid into a mist]. ***Sonic*** [*son(o)-* sound + *-ic* pertaining to] and ***pneumatic*** [*pneum(o)-* air + *-tic* pertaining to] ***nebulizers*** [L. *nebula* mist + *-izer* one that; i.e., a device for creating a mist] create mists with small enough droplet size to reach the bronchioles and alveoli. This is very important to facilitate removal of the exudates. Following nebulization, chest ***percussion*** [L. *percussio* to strike] helps to loosen the secretions from the walls of the airways and stimulate a cough. *Percussion* is a gentle, rhythmic rapping on the chest wall with cupped hands. The French call this ***coupage*** (coo-pahj' [Fr. *coup* to strike + *-age* act or state of; i.e., the act of striking, in this case the chest]). *Coupage* is best done with the cranial body slightly lower (~10 to 15-degree angle) than the rear, permitting gravity to help secretions slide from the alveoli and bronchioles to larger airways where we have the *mucociliary escalator* and more cough receptors. *Percussion* with the *drainage angle* also assists the mucociliary escalator with secretion removal (teamwork with those tiny cells). The combination of *nebulization* and *coupage* at a *drainage angle* is very effective. I have seen this mobilize such large volumes of secretions from the lower airways that suction was required to prevent upper airway obstruction. Be prepared.

Other therapeutics may include ***bronchodilators*** [*bronch(o)-* bronchus + *dilator* from L. *dilatator* expander] and antibiotics. Even if pneumonia develops from nonbacterial pathogens, like the *canine influenza* virus or the *equine rhinopneumonitis* [*rhin(o)-* nose + *pneumon(o)-* lung + *-itis* inflammation of] virus, secondary bacterial overgrowth of *commensal bacteria* frequently contributes to the

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pneumonia. So, antibiotics are usually administered. ***Antimycotic*** [*anti-* against + *myc(o)-* fungus + *-tic* pertaining to] agents are needed to treat *fungal pneumonia*. Fungal pneumonia is difficult to treat and often takes much longer for recovery. Plus, there may be concern for ***zoonotic*** [*zo(o)-* animal + *nos(o)-* disease + *-tic* pertaining to; i.e., transmission between animals and people] transmission with some fungal pathogens, like *Histoplasma sp.* ***Bronchoalveolar*** [*bronch(o)-* bronchus, bronchi + *alveol(o)-* alveolus, alveoli + *-ar* pertaining to] ***lavage*** [Fr. *lavage* "to wash out"] (***BAL***) is often performed to collect airway secretions for analysis. To do this, a small volume of sterile saline is flushed into the airways and aspirated back out. The sample can then be examined microscopically and cultured to identify the specific *pathogen*. For patients fortunate enough to recover from pneumonia, their respiratory tracts may never be completely normal. Often, they are more susceptible to developing pneumonia again.

Heat Stroke

Heat stroke can be life-threatening for anyone. But for those animals with limited temperature regulatory mechanisms, like dogs, cats, and pigs, very young and very old animals, *heat stroke* or ***hyperthermia*** [*hyper-* excess + *therm(o)-* temperature + *-ia* condition of] can kill rapidly. Dogs are probably the most vulnerable to heat stroke. Humans are the ones who put them at risk. The most common scenario involves a dog left in an automobile. Mind you, it doesn't even need to be an extremely hot summer day to kill a dog in a hot car. Even on a partly sunny day, if the ambient temperature is 68°F (20°C), a dog left in a car can die from heat stroke. The hotter and more humid the day, the more quickly heat stroke will develop and kill. Let's see if we can figure out why.

The only means dogs have to cool their bodies are to move away from a heat source and ***pant***. When locked in a car, the dog cannot move away from the heat. So, *panting* is its only option. We said earlier that panting was a very shallow nonrespiratory breathing pattern that moved air over the moist mucous membranes of the mouth and pharynx. Under normal circumstances with panting, through evaporation and convection, heat dissipates from the body

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through the superficial capillary beds of the mucous membranes there. But here's the catch—there needs to be a sufficient temperature and humidity difference for this to work. The ambient temperature must be significantly lower than the dog's core body temperature. And for evaporation to work, the surrounding air must be drier.

In an automobile in the sun, even with the windows partially open, the temperature rises dramatically in a matter of minutes. It's been proven that the interior temperature of a car parked in the sun can rise nearly 20 degrees in just 10 minutes. Depending on ambient temperature and time in the sun, the interior of that car can easily reach temperatures between 130°F and 170°F. The dog cannot escape. It begins to pant. But with the temperature inside the vehicle rapidly surpassing normal core body temperature (normal dog's body temperature is usually between 101°F and 102.5°F), the dog's panting has no cooling effect. So, the dog's body becomes ***hyperthermic*** [*hyper-* excess + *therm(o)-* temperature + *-ic* pertaining to]. We already said that oxygen is used more rapidly at higher body temperatures. Cellular metabolism accelerates in higher temperatures, rapidly using up O_2 and producing excess CO_2. So, this dog quickly becomes *acidotic*. What will that do? It will cause the respiratory centers in the brain to stimulate *tachypnea* and eventually *hyperpnea*, to blow off excess CO_2. That too will be incapable of reducing the body temperature. In fact, the muscular activity will only add to the rising body temperature AND the acidosis. *Vasodilation* in the periphery will try to dissipate heat to no avail. Did we not say that there is not enough blood volume to fill all of the vasculature? So, severe *hypotension* will likely develop. Many organs, especially the kidneys, cannot tolerate *hypotension*. They will begin to fail. If the brain becomes too hot, it will probably cause the animal to experience seizure activity. Convulsions will raise the body temperature exponentially, as well as make the dog extremely *hypoxic* and *hypercapnic*. Suffice it to say that it takes little time for a dog left in a car to reach lethal effects of heat stroke.

Even if we break into the car to render first aid, we may not be able to save the dog. The damage to the body, especially vital organs like the brain, heart, liver, and kidneys, may be irreversible. And how do we effectively reduce body temperature? You might

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think it's with ice. You'd be wrong. How will the superficial vasculature react to extreme cold? It will vasoconstrict. *Vasoconstriction* in the periphery will trap all of the extreme heat in the core. So, we need to use a method of cooling that won't promote major peripheral vasoconstriction. The best way to accomplish that is through cool running water. Running tap water over the body is the best method of cooling as a first aid measure. Then the dog must be transported as quickly as possible to a veterinary facility.

For us as veterinary professionals, emergency care revolves around *cardiopulmonary* support, as well as trying to lower body temperature to a safe level. That's roughly 103°F; no less. Lowering the body temperature below 103°F may cause the body to engage heating mechanisms as it does with fever. And not knowing the amount of brain damage, temperature control centers may not function appropriately, perhaps overreacting. IV fluid therapy to combat *hypotension*, as well as cool the animal, is important too. To promote better gas exchange and prevent hypoxia-induced *cardiac arrhythmias*, oxygen therapy is important. If the dog is experiencing respiratory failure, we may need to intubate and ventilate the patient.

Of course, even if we can stabilize this heat stroke dog, it could still die over the next few days. InChapter 3, we spoke about ***disseminated*** (widespread) ***intravascular coagulopathy*** [*coagul(o)-* clotting + *-pathy* disease of] (***DIC***) as a lethal consequence of heat stroke. With *DIC*, the dog could bleed to death in the next few days, because all of its platelets and clotting factors were used up in the extreme heat. *Edema* in the brain could easily result in death today or tomorrow. *Acute renal failure* could easily kill the dog in a few days. *Cardiac arrhythmias* could kill the dog at any moment. Our efforts to save the life of such a patient may be intense and on-point from the moment the dog is in our care. Yet all of our efforts may be in vain, simply because of secondary disease complications.

Dogs, cats, children, and geriatric individuals (human and animal) do not have the physiologic mechanisms to combat extreme heat exposure. So the best course of action is to never place them in situations that they are incapable of coping with. Panting for a dog is a poor antidote for a hot car. Many dogs die from heat stroke every year; children too. Our role as veterinary professionals is to

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educate our clients and the public, to prevent it from ever happening in the first place.

Case Study

Gordo is a male neutered 4-year-old Labrador retriever. He was brought into our emergency room due to dyspnea. On presentation, Gordo was orthopneic, tachypneic, cyanotic, and tachycardic. His cyanosis indicated to emergency personnel that Gordo was both hypoxic and hypercapnic, making him critically ill. Electrocardiography showed a normal sinus rhythm, with periodic VPCs. Pulse oximetry indicated only 80% saturation. Oxygen therapy was initiated immediately. An arterial blood gas was drawn for analysis. Bilateral intranasal catheters were placed for continuous oxygen therapy. An intravenous catheter was also placed for IV fluids. Once somewhat stable (i.e., still dyspneic and with mucous membrane pallor), thoracic radiographs were taken. Radiographically, Gordo had significant pulmonary consolidation. The veterinarian made a tentative diagnosis of pneumonia. Further diagnostics were ordered, including a complete blood count and serum chemistries.

Historically, Gordo was last normal 2 weeks ago, when the family was on vacation with him. They went on a camping trip. Early in the vacation, the family and Gordo capsized their canoe. Gordo was trapped for a few minutes under the canoe. When they pulled him out, Gordo seemed to be gasping for air and coughed a great deal. Later in the day, he seemed fine. His appetite was good, though he did seem to cough a little periodically. That was approximately 2 weeks ago. Since then, Gordo's activity level has progressively diminished. And since returning home several days ago, Gordo has been coughing more and more, though otherwise seemed fine. Today, he refused to eat and collapsed outside. He also seemed to struggle to breathe. So, the owners brought him in for care.

Based on history, the doctor assumes that Gordo has aspiration pneumonia. However, she still wants to collect bronchial secretions for analysis to determine pathogens contributing to the

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pneumonia. Gordo's initial arterial blood gas results were paO_2 78 mm Hg ↓, $paCO_2$ 47 mm Hg ↑, pH 7.21 ↓, and HCO_3^- 20.2 mmol/L ↓. Blood glucose and electrolytes were within normal limits. Serum chemistries were within normal limits. The complete blood count showed increased numbers of white blood cells, consistent with an infection. When Gordo was stable enough, bronchoalveolar lavage was performed to collect secretions for analysis. Results of that analysis confirmed a diagnosis of fungal pneumonia.

Gordo was treated with intravenous antimycotic medication. He was hospitalized for 2 weeks. During that time, intranasal oxygen therapy, IV fluid therapy, and antimycotic infusions continued, along with nebulization and coupage. Over the 2 weeks, Gordo made gradual improvements. By the end of 2 weeks in intensive care, he was oxygenating at 94% at room air (via pulse oximetry). He was still experiencing tachypnea, but no longer dyspneic. His coughing was productive, especially following coupage. So, no antitussive medication was prescribed. After several more days of hospitalization, he appeared well enough to be discharged. He was sent home on oral antimycotic medication. He eventually made a full recovery.

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Case Study Questions

1. Gordo was brought in for evaluation due to _____ (i.e., difficulty breathing).
2. In emergency, personnel observed extremely severe difficulty breathing, such that Gordo attempted to sit upright, with his head extended and elbows abducted. This type of severe difficulty breathing is medically termed _____.
3. Gordo was also _____ (i.e., pertaining to a fast heart rate).
4. Gordo's mucous membranes were _____ (i.e., bluish).
5. Both his mucous membrane color and pulse oximetry made it clear that Gordo was suffering from _____ (i.e., condition of low oxygen).
6. In addition to oxygen deficiency, Gordo's blue mucous membranes also indicated that he was _____ (i.e., pertaining to excess carbon dioxide).
7. Low oxygen levels were probably responsible for the VPCs observed via _____ (i.e., recording of electrical activity of the heart).
8. Oxygen therapy was delivered to Gordo via _____ (i.e., within the nose) catheters.
9. Fluid therapy was initiated via an _____ (IV; i.e., within a vein) catheter.
10. In order to determine the cause of Gordo's pulmonary disease, the doctor performed a _____ (2 words; i.e., washing the bronchi and alveoli) procedure

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to collect airway secretions for analysis.

11. Results of the secretion analysis confirmed a diagnosis of fungal _____ (a lung condition).
12. Gordo was then given _____ medication to eliminate the fungus.
13. Gordo was also treated with _____ (i.e., creation of a mist) liquify airway secretions.
14. We also performed _____ (a French word for gently "striking" his chest) to help mobilize secretions to be coughed up.
15. We continued to assess Gordo's oxyhemoglobin saturation with noninvasive pulse _____.

The Answer Key to these case study questions may be found in Appendix B.

[1] Darth Vader is the villain character from the *Star Wars* movies. *Star Wars* is a film series and franchise, created by George Lucas and distributed by 20th Century Fox. The first film in the series was released in 1977.

[2] Bell J, Cavanagh K, Tilley L, et al. *Veterinary medical guide to dog and cat breeds*, Teton NewMedia, Jackson, WY; 2012: 497, 517, 575.

[3] Bell J, Cavanagh K, Tilley L, et. al. *Veterinary medical guide to dog and cat breeds*, Teton NewMedia, Jackson, WY; 2012: 544.

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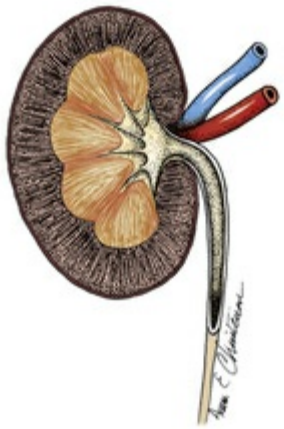

Applied Urinary Terminology

General Anatomy,
- Male Versus Female Urethral Anatomy,
- Urinary Bladder,
- Kidneys,
  - Nephron and Urine Production,
  - Urinalysis—Revealing Evidence,

Renal Contributions to RBC Production and Blood Pressure,
- Erythropoietin,
- Blood Pressure,

Common Urinary Diseases,
- Diabetes Insipidus,
- Urolithiasis,
- Urinary Tract Infections,
- Renal Failure,
  - Acute Renal Failure,
  - Chronic Renal Failure,

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Case Study,

GOALS AND OBJECTIVES

By the conclusion of this chapter, the student will be able to:

1. Recognize common root words, prefixes, and suffixes related to the urinary system.
2. Divide simple and compound words into their respective parts.
3. Recognize, correctly pronounce, and appropriately use common medical terms related to the urinary system.
4. Demonstrate an understanding of basic urinary anatomy.
5. Demonstrate an understanding of kidney function, with regard to urine production, water homeostasis, waste excretion, and electrolyte homeostasis.
6. Demonstrate an understanding of the kidneys' contributions to maintenance of blood pressure and red blood cell production.
7. Demonstrate familiarity and understanding of common urinary diseases and their impact on the urinary system and the body.
8. Demonstrate familiarity with diagnostic analysis of urine.

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General Anatomy

Ah plumbing, that's the ***urinary*** [*urin(o)-* urine + *-ary* pertaining to] system, and it is a very important bit of equipment for the body. Let's take a look at some of the basic pieces and parts. It may appear in Fig. 6.1 that ***kidneys*** are *in* the abdominal (peritoneal) cavity, but they're not. They are actually ***retroperitoneal*** [*retro-* backward, behind + *peritone(o)-* peritoneum + *-al* pertaining to]. In case you're wondering, the ***peritoneum*** is the connective tissue lining of the abdomen. The kidneys are, in essence, sandwiched between the peritoneum and the muscle layers along the dorsal abdomen. In spite of their retroperitoneal location, we still palpate the kidneys during routine abdominal palpation of dogs, cats and other small creatures. And if we're going to palpate them, we need to know where to find them. But kidney location is not bilaterally symmetrical. So, when viewing abdominal radiographs ("x-rays") or performing abdominal palpation, you need to remember the differences in their placement. To help you remember those locational differences, you may want to commit the following phrase to memory: "righty-tighty, lefty last, and loosey." Let me make sense of that for you. You see, the right kidney tends to be a bit more cranial and more tightly connected to the dorsum. In fact, it may be so far cranial that it is partially protected by the last rib. This can sometimes make palpating the right kidney difficult. The left kidney, on the other hand, is more caudal with loose connections to the dorsum. In a small to moderately sized dog or cat, in good body condition, the left kidney can be palpated relatively easily.

Each kidney has its own blood supply via a ***renal*** [*ren(o)-* kidney + *-al* pertaining to] artery. The *renal* vein for each kidney returns blood to general circulation. (If you need to review basic blood circulation, refer to Chapter 5.) These vessels, like the kidneys, are *retroperitoneal* too. And the kidneys need really good blood flow. Why? For now, think of the kidneys as little "water treatment plants." Blood with various impurities (especially nitrogen-containing impurities like ammonia and urea) enters the kidneys through the renal arteries. The kidneys process the blood, removing

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impurities and retaining or eliminating just the right amount of water, and then return the "treated" blood back into circulation via the renal veins. We'll talk about this in much greater detail later. The "wastewater" is funneled off to the ureters.

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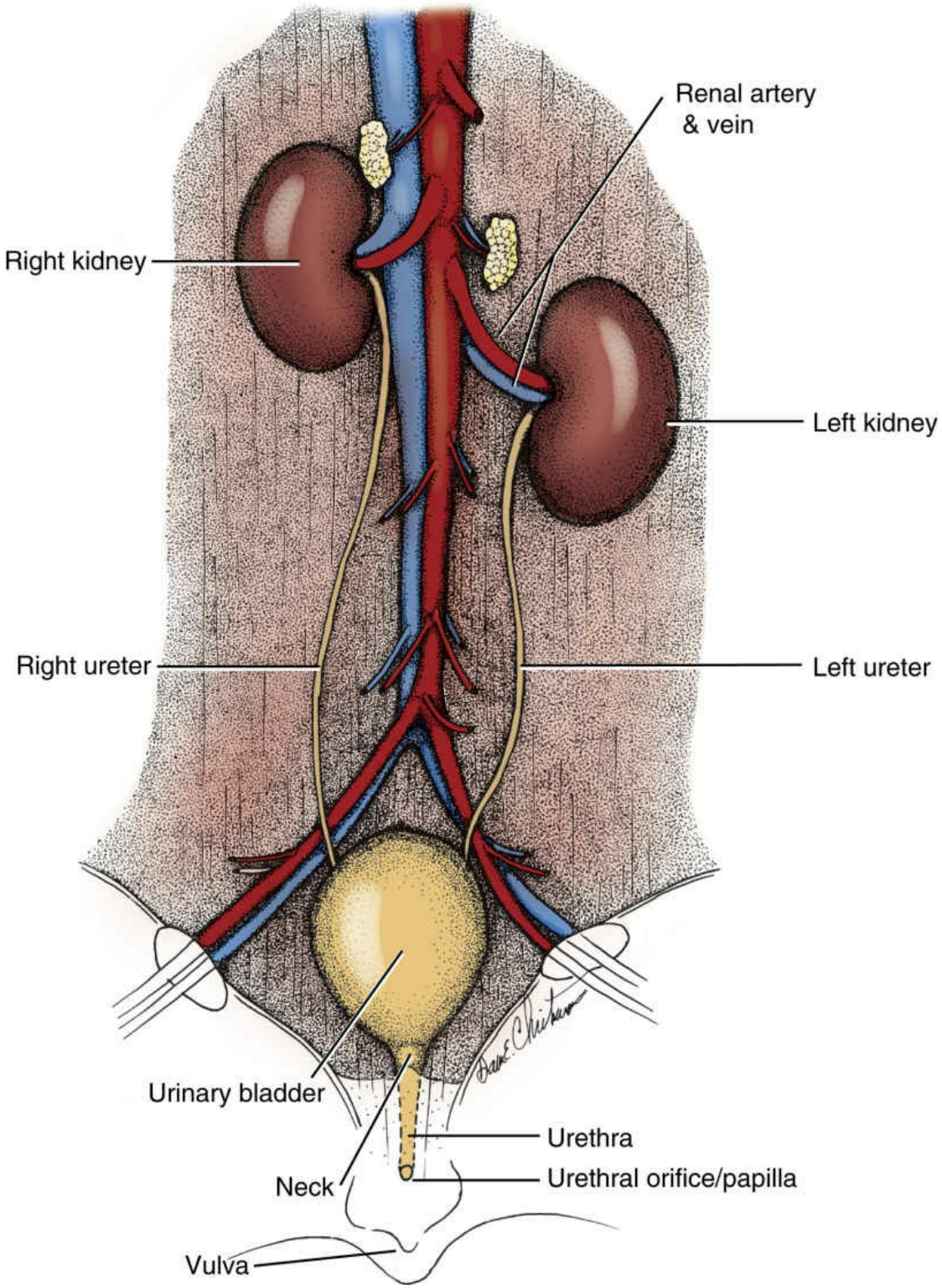

FIG. 6.1 Female urinary system (ventrodorsal view).

The ***ureters*** are small muscular tubes connecting each kidney to the urinary bladder. These tiny tubes are located *in* the abdominal cavity. And yes, you read that right. They are muscular. No relying on gravity here! The rhythmic, wavelike contractions, or ***peristalsis***

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[*peri-* around + Gr. *stalsis* contraction], of the ***ureteral*** [*ureter(o)-* ureter + *-al* pertaining to] muscles actually push the "wastewater" (i.e., urine) from the kidneys to the urinary bladder. On abdominal ultrasound, you can sometimes see the urine pulsate into the bladder in tiny little "puffs" or "jets." This is visual evidence of the rhythmic muscle contractions of the ureters.

Of course, the ***urinary bladder*** is merely a "wastewater" holding tank, if you will. But it's not just a flabby water balloon. It's muscular too. Muscular contraction helps in fully emptying the bladder during urination. And when that happens, the urine passes from the bladder to the outside environment through the ***urethra***. It's here where we find the greatest anatomic differences between male and female animals.

Male Versus Female Urethral Anatomy

If you compare the female anatomy in Fig. 6.1 to that of canine male anatomy in Fig. 6.2, you'll see that the biggest difference is the length of the urethra. By the way, the female anatomy found in Fig. 6.1 is pretty representative of females across animal species. In both the male and the female, the urethra passes over the floor of the pelvis. After that, in the male (as shown for the dog in Fig. 6.2) the urethra is quite long before it reaches the ***urethral*** [*urethr(o)-* urethra + *-al* pertaining to] ***orifice*** (opening) at the distal penis. Most male animals (canine, equine, bovine, porcine, caprine, ovine) have very long urethras, compared to females. An important and unique feature of the male dog is the fact that the urethra passes through the ***os*** [L. *os* bone] ***penis***—yes, an actual bone in the penis. That bone can become problematic, especially when it comes to passing urinary stones. Yeah, that can plug up the works. Now compare the male dog to the male cat, shown in Fig. 6.3. No, the feline urethra is not as long as that of the male dog (or any other male animal, for that matter), and the cat's penis does not contain a bone. However, the male cat's urethra is somewhat "S-shaped" and becomes rather narrow by the time it reaches the urethral orifice. As plumbing goes, this design can be problematic for the cat, as you'll learn a bit later. It can also be problematic for us, when we try to pass a urethral catheter. That said, the caudal orientation of the cat's penis

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is very useful for “spraying” urine to mark territory—just backup and go!

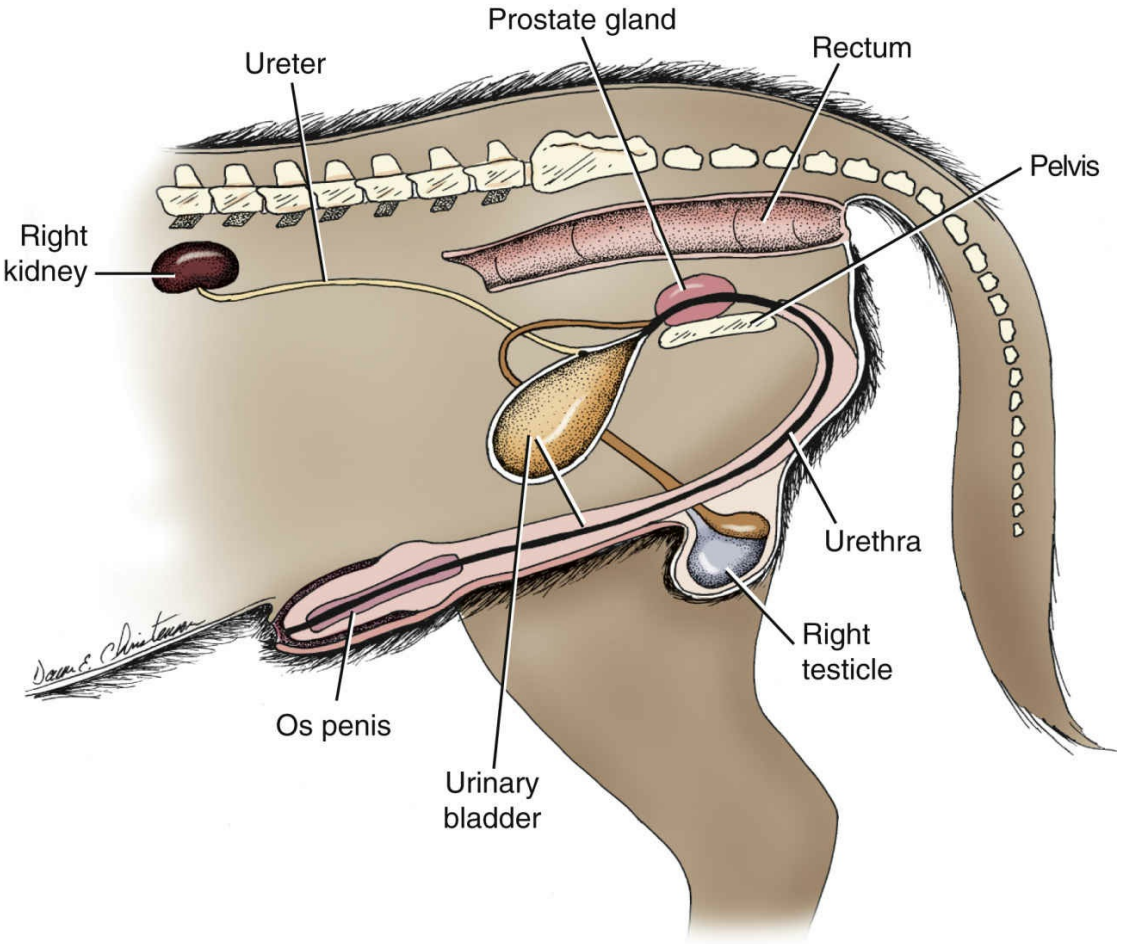

FIG. 6.2 Canine male urogenital tract (midsagittal view).

One other important difference to be noted between the urethra of female versus male animals is that the urethra is dual-purpose in males. In males the urethra provides passage for urine as well as semen (for reproduction). The reproductive aspect will be discussed in Chapter 9. In females the urethra is used exclusively for urination.

Urinary Bladder

We talked about the urinary bladder in brief, a moment ago. But there are a few details worth taking a closer look at. First of all, the layering of the bladder wall is important. Like all abdominal organs, its exterior surface is covered by ***visceral*** [*viscer(o)-* organ + *-al* pertaining to] *peritoneum*. Then, we find a layer of smooth muscle. (You may want to refer to Chapter 4 to review the nature of smooth muscle.) Finally, its interior is lined by ***stratified*** (layered) ***epithelium***.

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This type of epithelial tissue is *transitional* because it is constantly being lost and replenished. If you notice in Fig. 6.4, the cells at the deepest layer are plump little cubes. In this basal cell layer is where mitosis takes place. (Refer to Chapter 2 to review mitosis.) So, the youngest cells are deep in the stratification (layering). Older cells get progressively pushed toward the surface, as new cells are made. Those cells in the middle layers are referred to as ***transitional*** because they are transitioning from the depths toward the surface. They are also gradually transforming from plump cells, like those in the basal cell layer, to flatter and broader cells. Finally, the most superficial cells, nearest the bladder's interior, are flat like scales. In fact, that's why the most superficial cells are called ***squamous*** [*squam(o)-* scale + *-ous* pertaining to] cells, because they're flat like the scales of a fish or reptile. The most superficial of the squamous cells will eventually slough off and be passed in the urine, when the animal urinates.

If you look again at Figs. 6.1 and 6.2, you'll notice that the ***neck of the bladder*** is the most caudal point that lies near pubic bone of the pelvis. The apex (the cranial, rounded border) of the bladder will vary in its location. As the bladder fills with urine, the bladder expands cranially. Of course, the weight of the urine makes the bladder drop further ventrally in the abdomen the fuller it gets. The opposite is true when the animal urinates. This is something to remember whenever you collect urine via ***cystocentesis*** [*cyst(o)-* bladder + *-centesis* puncture of; i.e., puncture with a needle and syringe for sample collection]. The needle should be directed somewhat caudally so that when the bladder is evacuated, urine can continue to be safely collected. If the needle is mistakenly directed cranially, the bladder wall near the apex may actually be drawn down onto the bevel of the needle, as the bladder is evacuated. This could result in a secondary puncture or even a laceration of the bladder wall, not to mention possible damage to other structures.

Finally, please note that the ureters usually enter the dorsal bladder near the ***trigone*** (tri'gōn [L. *trigonum* triangle]). The *trigone* is a triangle formed by the ureteral orifices and the neck of the bladder leading to the urethra. This trigone region is frequently referenced during ultrasound imaging and surgery of the urinary

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bladder. By the way, there is a ***sphincter*** (sfingk'ter [Gr. *sphinktēr* that which binds tight]) in the neck of the bladder. Only when this muscular *sphincter* relaxes can urine pass into the urethra. Laxity in the sphincter may result in urinary *incontinence* or dribbling of urine. This is common in geriatric (old) dogs. On rare occasions, ***chronic*** [*chron(o)-* time + *-ic* pertaining to; i.e., over time, long-term] urinary incontinence may occur in puppies. This is most often due to an ***ectopic*** [*ec-* out, outside + *top(o)-* place, placement + *-ic* pertaining to] ***ureter***. Generally in these puppies, one ureter bypasses the bladder and actually enters the urethra. This requires surgical correction. Thankfully, *ectopic ureters* in puppies are the exception, not the rule. By the way, in normal puppies, good conscious control of the urinary sphincter does not develop until at least 6 months of age. Until then, you need to expect "accidents" in the house.

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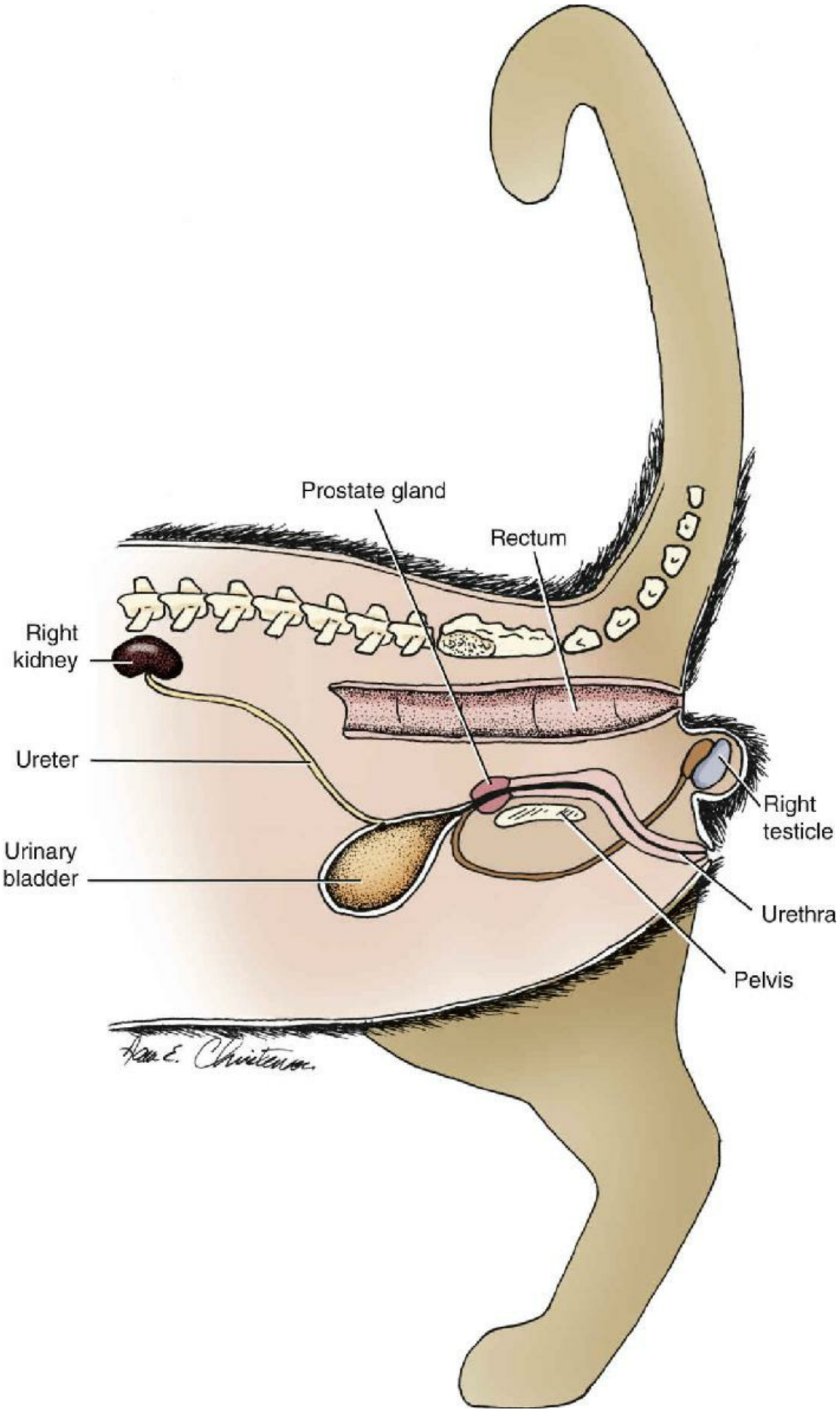
Prostate gland
Rectum
Right kidney
Ureter
Urinary bladder
Right testicle
Urethra
Pelvis

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FIG. 6.3 Feline male urogenital tract (midsagittal view).

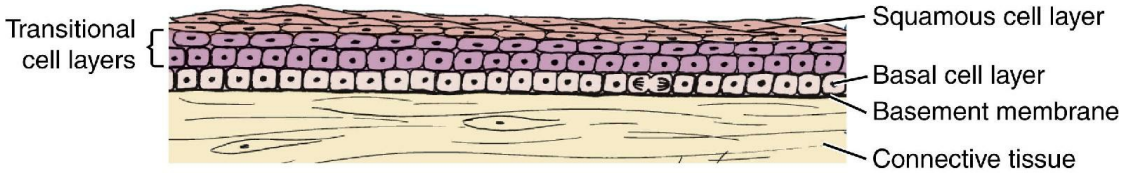

FIG. 6.4 Stratified squamous epithelium.

Kidneys

Okay, we have arrived at the main part of our "water treatment plant." Let's take a closer look at a kidney in Fig. 6.5. The renal artery carries blood into the kidney, and the renal vein carries blood from the kidney into general circulation. As you can see, the kidney has a fibrous connective tissue covering called the ***renal capsule***. Then we have the ***renal cortex*** [L. *cortex* "bark, rind, shell"]. This is where we find most of the major working parts of the kidney. Further inward, we find the ***renal medulla*** [L. *medulla* "inward part"]. The renal medulla contains some functional parts with regard to urine production. It also has numerous collecting ducts that direct urine to the ***renal pelvis***. I like to think of the renal pelvis as a funnel, guiding urine to the ureter. But we can't do that until we actually produce urine. To understand how that happens, we need to look at the functional unit of the kidney: the ***nephron*** [*nephr(o)-* kidney + *-on* a, the].

Nephron and Urine Production

In the world of the kidney, the nephron is where "the rubber meets the road." Each nephron (and there are potentially millions of them in each kidney, depending on species) is a self-contained "water treatment plant." Fig. 6.6 shows the basic plumbing of a nephron. Let's walk through the basic structure first. Then we'll talk about how those pieces and parts actually work.

First, notice the blood vessels leading to and from and twisted all around the "pipes." The ***arteriole*** [*arter(o)-* artery + *-ole* a small; i.e.,

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the smallest of arteries] carries blood in. The arteriole leads to a tangled ball of ***capillaries*** in the ***glomerulus*** [L. *glomerulus* "tiny ball"]. As you may recall, generally in circulation, an arteriole leads to capillaries (the smallest vessels of the body) and capillaries lead to venules [*ven(o)-* vein + *-ule* a small; i.e. the smallest of veins]. (Refer to Chapter 5 to review vessels and circulation.) Here there's actually an arteriole inlet and an arteriole outlet for the glomerulus. So to be accurate, the ***afferent*** [L. *afferent*, from *af-* toward + *ferre* to carry] *arteriole* takes blood to the glomerulus and the ***efferent*** [*ef-* out] *arteriole* carries blood out from the glomerulus. Arterioles, like bigger arteries, have thick muscular walls. Contraction of those muscles of either the afferent or efferent arteriole will help control blood flow through the glomerulus and the volume of glomerular [*glomerul(o)-* glomerulus + *-ar* pertaining to] filtrate produced. This process is shown in the Evolve animation: *Renal Filtration*. But I'm getting ahead of myself. Sticking with just the blood vessels for now, after the efferent arteriole we have ***peritubular*** [*peri-* around + *tubul(o)-* tube + *-ar* pertaining to] ***capillaries*** wrapped around the "water pipes" until blood finally reaches the ***venule***. Capillaries, remember, are the smallest and most permeable vessels in the body. Okay, that's the ***vascular*** [*vascul(o)-* vessel + *-ar* pertaining to] part. Then there is the actual plumbing of the nephron.

The plumbing begins with ***Bowman's capsule***, surrounding the glomerulus. I like to think of Bowman's capsule as a sink and the glomerulus as the faucet. Like any sink, its drain is connected to drainage pipes. Some of those pipes are convoluted (curved, twisted) at the proximal and distal ends. In the middle is the ***Loop of Henle***, much like the "J-trap" under your kitchen or bathroom sink. What? You've never looked at that? By all means, go look! I'll wait. See? Doesn't that look like this part of the nephron? Anyway, all of this plumbing ultimately drains into the main "sewage line," the collecting duct. Did I mention that I've done my fair share of plumbing? It's not one of my favorite things to do in the world. That said, doing my own plumbing work has given me great appreciation for well-designed plumbing that works. And that, my friends, is precisely what we have in the nephron. And it is way more complicated than simply turning on the faucet and draining away the water. Much of it involves ***hydrostatic*** [*hydr(o)-* water + -

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static pertaining to maintenance] and ***osmotic*** [*osm(o)-* impulsion, osmosis + *-tic* pertaining to] pressures, electrolytes, and diffusion. So if you're a little rusty on those things, you might want to go back and review them in Chapters 2 and 3. When you're ready, let's look at this plumbing in action.

It's important to note that one of the most important factors influencing renal function is blood pressure. Adequate blood pressure provides the *hydrostatic pressure* (i.e., water pressure, like in a fire hose) necessary to force "wastewater" (***glomerular*** [*glomerul(o)-* glomerulus + *-ar* pertaining to] ***filtrate***) from the glomerular capillaries into Bowman's capsule. (Faucet on!) In fact, if we hormonally target the *efferent arteriole* to cause ***vasoconstriction*** [*vas(o)-* vessel + *constriction* the act of constricting/narrowing], we "dam the river," causing blood to back up into the glomerulus. This increases the hydrostatic pressure within the glomerular capillaries and that translates into turning the faucet on "full." And that produces a bunch of filtrate. This filtrate contains water, electrolytes, and toxic wastes, like urea. Urea is the liver's answer for removing ammonia (NH_4). (Ammonia is a principal by-product of protein metabolism.) But then, in my opinion, the liver drops the ball and passes the buck to the kidney, because now it's up to the kidney to remove urea.

Alright, so blood under sufficient pressure enters the nephron through the *afferent arteriole*. The *glomerular capillaries*, like any capillary, are very permeable to water and electrolytes. *And* the glomerular capillaries also have larger gaps in their walls to permit larger molecules like urea, amino acids, and glucose to slip out with the filtrate. But really, really big things like cells and protein molecules cannot pass through. As blood flow carries the blood cells and plasma from the glomerulus to the *efferent arteriole* and onward to the peritubular capillaries, the blood still has a great deal of *osmotic pressure* from the plasma proteins. I like to think about osmotic pressure as "suck power." Those plasma proteins are like huge sponges—very attractive to water. This comes in very handy for reabsorbing water by osmosis from the *proximal convoluted tubule* and the *Loop of Henle*. But plasma proteins are not the only things that promote water reabsorption. Sodium is also very important. As sodium (Na^+) ions are reabsorbed by active transport through the

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proximal convoluted tubule and Loop of Henle, water and negative ions, like chloride (Cl^-), tend to follow. By the time we reach the *distal convoluted tubule*, any water needing to be reabsorbed should have been, because portions of the distal tubule are almost impervious to water.

Throughout the nephron's tubules, there is a progressive give and take of water and electrolytes, as noted in the Evolve animation *Urine Formation*. This is very important for overall ***homeostasis*** [*home(o)-* unchanged, sameness + *-stasis* state of standing; i.e., equilibrium]. Some of the key ions that may be excreted or reabsorbed, depending on the body's needs, include Na^+, chloride (Cl^-), potassium (K^+), hydrogen (H^+), calcium (Ca^{++}), phosphate (PO_4^-), and even some bicarbonate (HCO_3^-). Much of this ionic activity is under hormonal control. To learn more about some of these hormonal influences, please refer to Chapter 10. Ultimately, the filtrate we are left with, by the time it reaches the *collecting duct*, is nothing like it was at the start. The filtrate in the proximal tubule is generally ***isotonic*** [*iso-* equal + *ton(o)-* tonicity + *-ic* pertaining to; i.e., equal concentration of dissolved particles] with the plasma in the peritubular capillary. But especially under the influence of ***antidiuretic*** [*anti-* against + *diure(o)-* urination + *-tic* pertaining to] ***hormone*** (***ADH***) from the pituitary gland, water is drawn back into circulation. So, comparatively, the final filtrate (urine) is ***hypertonic*** [*hyper-* excess + *ton(o)-* tonicity + *-ic* pertaining to; i.e., greater concentration of dissolved particles], containing nothing but a bit of water and lots of waste. Of course, the effects of ADH also means the animal may be ***oliguric*** [*olig-* small + *ur(o)-* urine + *-ic* pertaining to] too. ***Oliguria*** [*olig-* small + *ur(o)-* urine + *-ia* condition of; i.e., small urine volume] and concentrated urine go hand in hand. Certainly in healthy animals, the degree of concentration and overall urine volume depend in part on water homeostasis for bodily needs.

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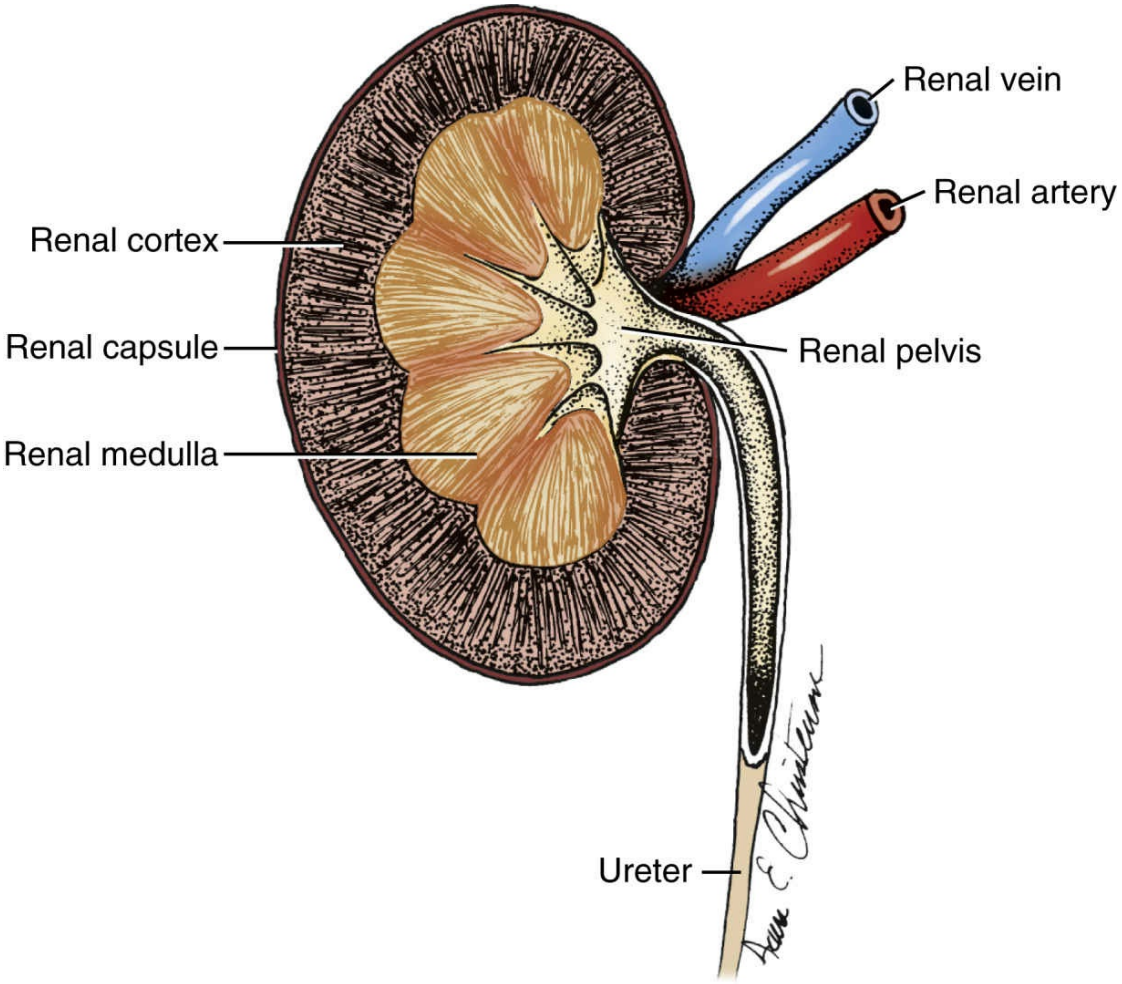

FIG. 6.5 Sagittal kidney.

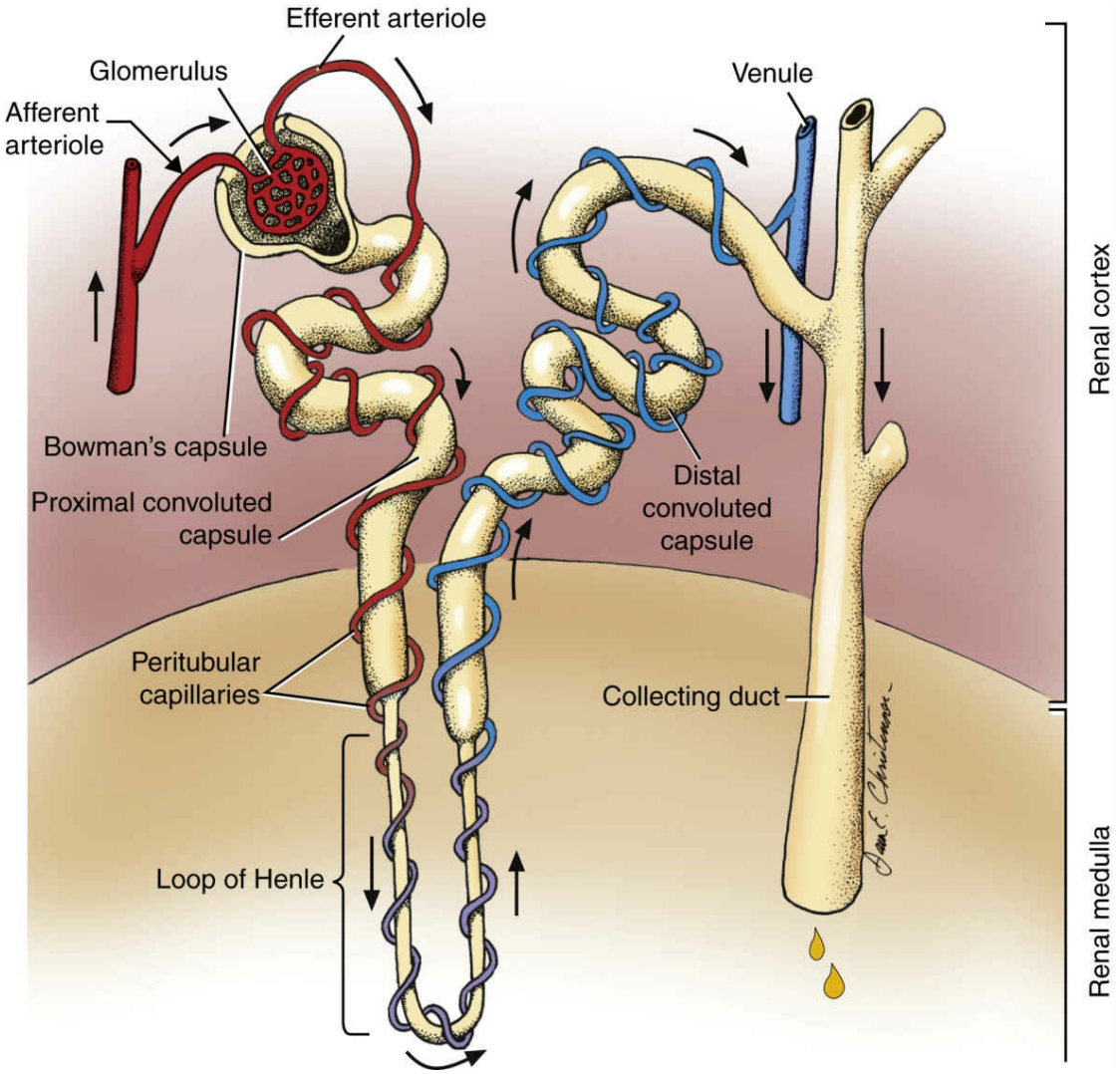

FIG. 6.6 Nephron.

Urinalysis—Revealing Evidence

So, what can examination of the urine tell us? More than you can imagine! Sure, you'd expect that it will give us a glimpse at the health of the bladder and kidneys. But it can also reveal evidence regarding liver function, acid–base homeostasis, and many other things. A complete urinalysis includes chemical analysis (screening via dipstick), specific gravity, and microscopic evaluation. Let's look more closely at what each component might reveal.

Urine Dipstick Screening

By the way, they are called "urine dipsticks" because that is how the manufacturer intends for them to be used. They are literally dipped into the urine. Each little chemical pad on the dipstick

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provides simple, quick chemical analysis of the urine. A standard dipstick evaluates for glucose, ketones, bilirubin, pH, protein, and blood. It is very important to time each indicator on the dipstick accurately. Many of the test pads will continue to change color over time. Inaccurate timing may create false positive or abnormally elevated results, and this may lead to inappropriate decisions with regard to patient care.

Glucose: Normal urine should not contain any glucose. The renal tubules are very efficient at reabsorbing glucose. In fact, they have a set threshold for glucose. The *renal threshold for glucose* varies from species to species. As an example, in dogs the renal threshold for glucose is about 180 mg/dL. Up to that set point, any glucose in the glomerular filtrate will be reabsorbed. But anything over 180 mg/dL will pass in the urine. ***Glycosuria*** [*glycos(o)-* glucose, sugar + *ur(o)-* urine + *-ia* condition of] is common in patients with diabetes mellitus. This disease syndrome is discussed in Chapter 10. But *glycosuria* isn't always due to disease. Patients receiving ***intravenous*** [*intra-* within + *ven(o)-* vein + *-ous* pertaining to] (IV) fluids containing dextrose will also have glycosuria. In that case, we're obviously putting more glucose into the system than is needed, surpassing the renal threshhold.

Ketones: Normal urine should not contain any ketones either. Ketones are a by-product of incomplete fat metabolism. In the normal patient, if fuel input equals energy output, there is no need to break down body fat. But in animals on a diet, ketones may be present. In starvation, ketones will definitely be present. More commonly, patients with unregulated diabetes mellitus frequently have ***ketonuria*** [*keton(o)-* ketones + *ur(o)-* urine + *-ia* condition of]. As we will discuss in Chapter 10, these patients can't utilize glucose for energy. The body breaks down fats to produce ketones as an alternate fuel. This is a very inefficient process, especially when it comes to utilizing ketones. And, long story short, ketones spill over into the urine.

Bilirubin: ***Bilirubinuria*** [*bilirubin* + *ur(o)-* urine + *-ia* condition of] may or may not be abnormal. What? How could that possibly be normal? Well, male dogs have a really low renal threshold for bilirubin. So we may see trace amounts of bilirubin in the urine of any male dog. Beyond that, bilirubinuria is abnormal. As you may

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recall from Chapter 3, bilirubin is a yellowish by-product of the breakdown of *hemoglobin*, the pigmented protein molecule that provides for oxygen transport by red blood cells (RBCs). We always have turnover of RBCs. So we almost always have trace amounts of bilirubin in the bloodstream at any given point in time. The liver is responsible for recycling bilirubin. Much of it is used to produce bile. But there are times when excessive RBC destruction, liver disease, or obstruction of bile ducts lead to ***hyperbilirubinemia*** [*hyper-* excess + *bilirubin* + *-emia* a blood condition of]. And if that develops, some of the excess bilirubin goes out with the wastewater (urine). Do you see how discovering *bilirubinuria* may lead to further diagnostics? We need to know if there is a problem with blood destruction, liver function, or bile duct flow. And because there may be very serious disease quietly lurking in the shadows, this shows why screening with routine urinalysis is so important.

Protein: ***Proteinuria*** [*protein* + *ur(o)-* urine + *-ia* condition of], especially larger quantities of protein, is another abnormal dipstick finding. However, if the urine is alkaline, the protein pad on the dipstick may change color, whether *proteinuria* is actually present or not. So take this dipstick screening with a grain of salt. If proteinuria is real, it points to a potentially serious problem with the kidneys. As you may remember, we said earlier that blood cells and protein molecules were too big to pass through the gaps in the glomerular capillary walls. If proteins are slipping through, we probably have some damage to the glomeruli. Further diagnositic testing may be warranted to confirm or rule out renal disease.

Blood: ***Hematuria*** [*hemat(o)-* bloody + *ur(o)-* urine + *-ia* condition of] is another abnormality. Could *hematuria* be ***iatrogenic*** [*iatr(o)-* physician + *gen(o)-* produced + *-ic* pertaining to; i.e., produced by us]? Sure! Urine collection via ***cystocentesis*** [*cyst(o)-* bladder + *-centesis* puncture] could cause a little bit of bleeding. Even just a drop or two of blood from our needle penetration will turn the dipstick. If we collect the urine by expressing the bladder or by placing a urinary catheter, we may cause just enough trauma that results in a little bleeding. Even soft, indwelling Foley catheters may result in minor bleeding, usually due to the patient's movement tugging against the balloon inside the bladder. But what if this is a voided sample (i.e., collected free-catch while the animal

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is urinating)? Even then, hematuria may not always point to disease. Take a female dog who's coming into heat, for example. She may have blood present from her normal reproductive cycle (discussed in Chapter 9). In any other circumstances, hematuria usually points to disease. Bleeding from the bladder wall frequently occurs from urinary tract infections (UTIs) or ***uroliths*** [*ur(o)-* urine + *lith(o)-* stone] in the bladder. Sometimes the dipstick shows a color change for blood when there are no RBCs present. Both *hemoglobin* and ***myoglobin*** [*my(o)-* muscle + *globin*; i.e., the pigmented protein molecule of muscle tissue] can turn the blood indicator on the dipstick. If we don't find RBCs under the microscope, we may need to pursue investigation of ***hemoglobinuria*** [*hemoglobin* + *-uria* a urine condition of] or ***myoglobinuria*** [*myoglobin* + *-uria* a urine condition of]. I've seen severe trauma victims (hit-by-car victims and those attacked by big dogs), who release large amounts of *myoglobin* into the system. Even the urine appeared reddish from the myoglobin. This just proves that you really shouldn't make assumptions. This type of screening test is designed to give us a very limited chemical look at the urine. The screening information may then guide us to further diagnostic testing.

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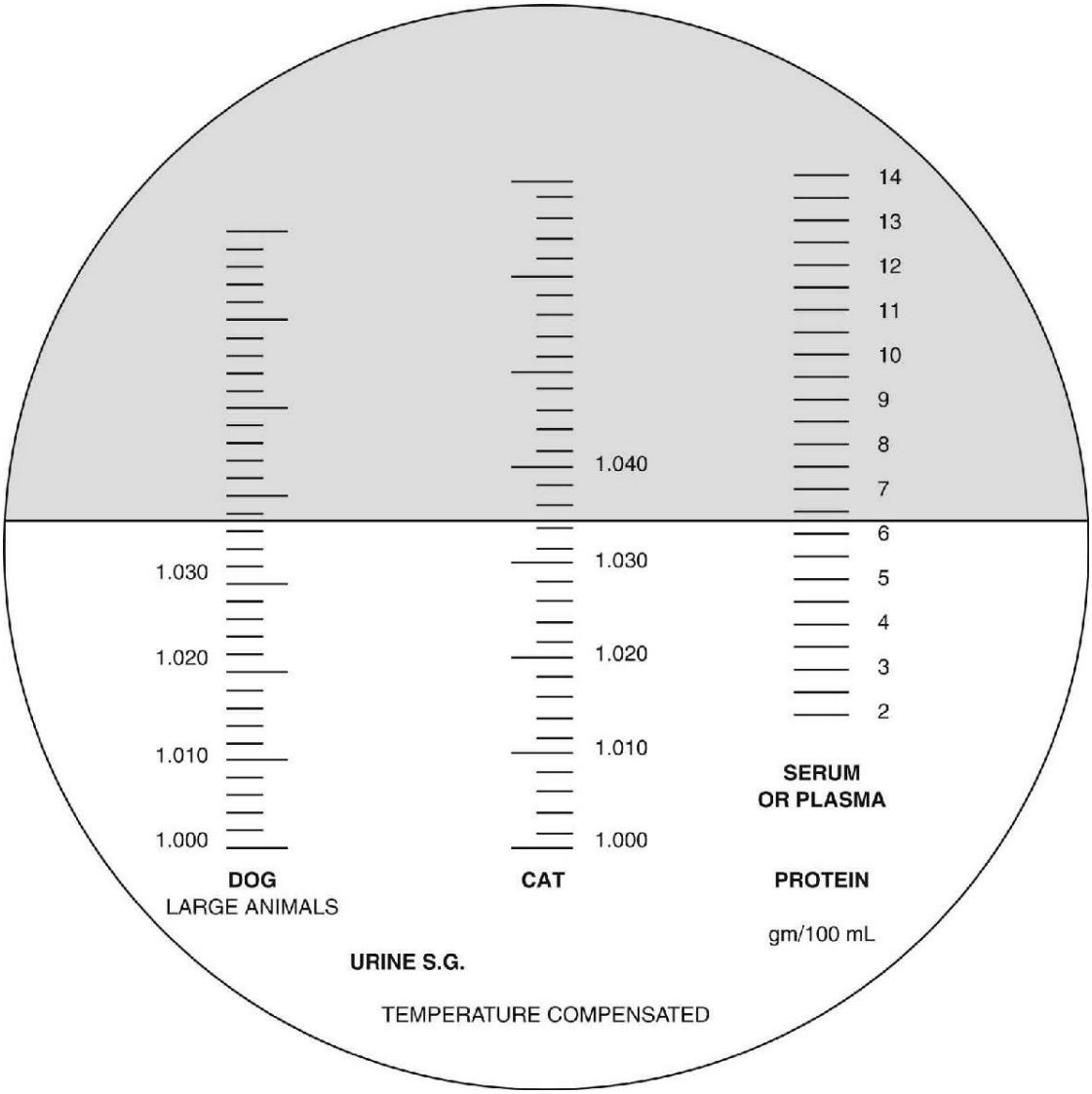

FIG. 6.7 Specific gravity (S.G.) using a refractometer.

pH: Urine pH is quite variable from species to species and patient to patient. Most herbivores tend to have neutral to alkaline urine. Dogs and cats may fall anywhere in the range from acidic to alkaline. It all has to do primarily with numbers of hydrogen ions excreted in the urine. The more H^+ in the urine, the lower the pH (i.e., more acidic). The less H^+ in the urine, the higher the pH (i.e., more alkaline). A relatively neutral pH is 7.0. Most dipsticks can determine acidic pH, from 5.0 to 6.5, and alkaline pH, which may range from 7.5 to 8.5. The importance of pH goes beyond just acid–base homeostasis. Homeostatic mechanisms alone can make urine pH fluctuate a bit throughout the day, especially following meals (usually shifting toward alkalinity after meals). One thing that can dramatically shift urine pH from acidic to alkaline is a UTI. I've

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seen dogs whose normal pH was 6.0 or 6.5 leap to 8.0 or 8.5, due to urinary tract infections. In fact, two of my personal pets experienced this. Fortunately, I frequently used their urine for teaching purposes, so I knew what was normal for them. And when I saw their urine becoming alkaline, I knew an infection was beginning to brew. I confirmed my suspicions with culture and sensitivity and microscopic evaluation.

Urine Specific Gravity

Urine specific gravity (S.G.) reveals the concentrating ability of the kidneys. We evaluate this using the same ***refractometer*** [*refract(o)-* bending + *meter* measure] that we used to determine plasma total solids in Chapter 3. All we need is a drop of urine, preferably after it has been centrifuged to remove any suspended particles. Suspended particles, especially crystals, may alter the refraction (bending of light) through the instrument, artificially altering the measurement. Fig. 6.7 shows a dog's specific gravity (1.037), as viewed through the refractometer. Notice that there are two scales for "urine S.G." (specific gravity)—the one in the middle for cats and the one on the far left for dogs and large animals. That's because cats often have more concentrated urine. But the bottom of both scales is 1.000. This is the specific gravity of distilled water and is our reference point against which everyone's urine is measured. Before checking urine specific gravity, the refractometer should always be calibrated with distilled water.

Healthy animals could have specific gravity readings as high as 1.065 (higher in cats).[a] The higher the reading, the more concentrated the urine. On average, the normal range of urine specific gravity is 1.020 to 1.035, for most animals.[b] In normal, healthy animals and people, it's all related to hydration status. As we dehydrate, water needs to be conserved. So, ADH is secreted, telling the renal tubules to reabsorb water. Greater reabsorption of water from the glomerular filtrate results in more concentrated urine with a higher specific gravity. With adequate hydration, for instance, an animal receiving a maintenance rate of IV fluids, patients will tend to produce an ***isosthenuric*** [*iso-* equal + *sthen(o)-* strength + *ur(o)-* urine + *-ic* pertaining to] specific gravity of roughly

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1.008 to 1.012.[c] In this range, the kidneys are "coasting along," with no need to concentrate or dilute urine, and the final urine osmolality is equal to that of the glomerular filtrate.

What if a patient is not receiving IV fluids, yet has a urine specific gravity of 1.008 to 1.012? Well that may make us wonder whether or not the kidneys are losing their concentrating abilities. There are times, in renal failure patients, that urine S.G. can become ***hypotonic*** [*hypo-* low + *ton(o)-* tonicity + *-ic* pertaining to] or, more accurately, ***hyposthenuric*** [*hypo-* below, insufficient + *sthen(o)-* strength + *ur(o)-* urine + *-ic* pertaining to]. Anything below 1.008 is considered *hyposthenuric*.[d] But *isosthenuria* and *hyposthenuria* may result from other diseases in animals with normal kidneys. Anything that results in ***polyuria*** [*poly-* much + *ur(o)-* urine + *-ia* condition of] can do this. For instance, diabetes insipidus (discussed later in this chapter and again in Chapter 10) is an endocrine disorder in which there is either insufficient *ADH* or the kidneys can't respond to it. Either way, the kidneys can't reabsorb water. So, water flows through the kidneys like a sieve, giving us lots of urine with a very low specific gravity.

Other conditions, like diabetes mellitus, can also result in *polyuria* and a low to normal specific gravity. Of course, the urine dipstick will give us a big clue about this disease by revealing the glycosuria. And it's the glucose creating polyuria in this case because glucose always tends to drag water with it. Wait. Why would a *polyuric*, diabetic animal have a normal specific gravity? If more water is being lost in a diabetic animal, why wouldn't it simply have *hyposthenuria*? It's all because of the glucose. Remember, glucose adds to the dissolved particles. More glucose means more solute (dissolved particles) and that means a falsely higher specific gravity. Let's not belabor this point. Simply remember that there are many diseases that can result in polyuria and ***polydipsia*** [*poly-* many, much + *dips(o)-* thirst + *-ia* condition of] and low specific gravity. Suffice it to say that any time an animal has PU/PD (polyuria/polydipsia), we should always investigate the cause. And anyone with persistent isosthenuria or hyposthenuria should definitely be evaluated further.

Microscopic Evaluation

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Finally, we cannot overlook the microscopic evaluation of urine. I don't know why, but many of my students dreaded the microscopic part of a urinalysis. There is far less to look for in urine than in a blood smear. In fact, there are only eight basic things we look for: epithelium, casts, crystals, white blood cells (WBCs), red blood cells (RBCs), bacteria, sperm, and fat droplets. On rare occasions, we may even find eggs from bladder or renal parasites. We always need to centrifuge the urine to have sufficient numbers of (what were suspended) things to look at. After the sample is spun, the liquid is poured off and the remaining concentrated sediment is gently mixed for microscopic evaluation. Let's look at the significance of each potential finding (i.e., those eight items mentioned a moment ago).

Epithelium: Remember, we said that the bladder wall is lined with stratified epithelium. The same is true for the urethra. The squamous epithelial cells at the surface are expected to exfoliate (fall off). We would expect greater exfoliation from the urethral epithelium, just like sediment is moved by a fast-flowing brook or river. So, we anticipate seeing more abundant squamous epithelium in *voided* or *catheterized* samples. A *cystocentesis* sample should have the least amount. We may also see a few transitional cells. Okay, so if these cells are going to show up in even normal urine, why bother looking for it and reporting it? Well, we already said that the method of collection will alter quantities of epithelium. What if we have collected a sample via cystocentesis, and microscopically we see large numbers of squamous and round (transitional) epithelium? Well, this might indicate some sort of trauma to the bladder wall. *Uroliths* rubbing and poking at the bladder wall could easily cause accelerated and deeper exfoliation. Some of the ***pathologists*** [*path(o)-* disease + *log(o)-* knowledge + *-ist* a specialist in] that I have collaborated with often said that if large "rafts" or "sheets" of round epithelium are observed in a cystocentesis sample, ***neoplasia*** [*ne(o)-* new + *plas(o)-* formation + *-ia* condition of; i.e., cancer] should be added to the rule-out list. Of course, increased numbers, including sheets, of transitional epithelium may result from catheterization. This is a good time to note that the collection method is extremely important for interpretation.

Casts: *Urinary casts* are formed in the renal tubules. The basic

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substance of a cast is a type of ***mucoprotein*** [*muc(o)-* mucus + *protein*] that is secreted by the distal convoluted tubules. As this mucoprotein solidifies, along with anything trapped in it, a "cast" or "mold" of the renal tubule is created. Because a cast is an actual "casting" or "impression" of the renal tubule, it is cylindrical in shape. Casts may be passed intermittently in the urine. While the absence of casts in the urine does not exclude renal disease, their presence does indicate changes in the renal tubules. Generally, when the cast forms, whatever is in the tubule becomes trapped in its matrix. The type of material trapped in the cast determines the type of cast. For example, if epithelial cells from the renal tubules get trapped, the cast is an *epithelial cast*. *Hyaline* [Gr. *hyalos* glassy] *casts* are just made of the mucoprotein. Hyaline casts are truly glassy, transparent structures. Like a glass held under water, their transparency makes them very difficult to see, even with reduced lighting. These may or may not be seen in normal urine. *Granular casts* are probably the most common type of cast to see. The granular appearance may be from protein precipitates, degenerated cells, or other tubular debris trapped in the mucoprotein matrix. *RBC casts* usually develop with renal bleeding or inflammation. *WBC casts* are also associated with renal inflammation. *Waxy casts* develop from degenerating cellular casts and usually indicate *chronic* renal disease.

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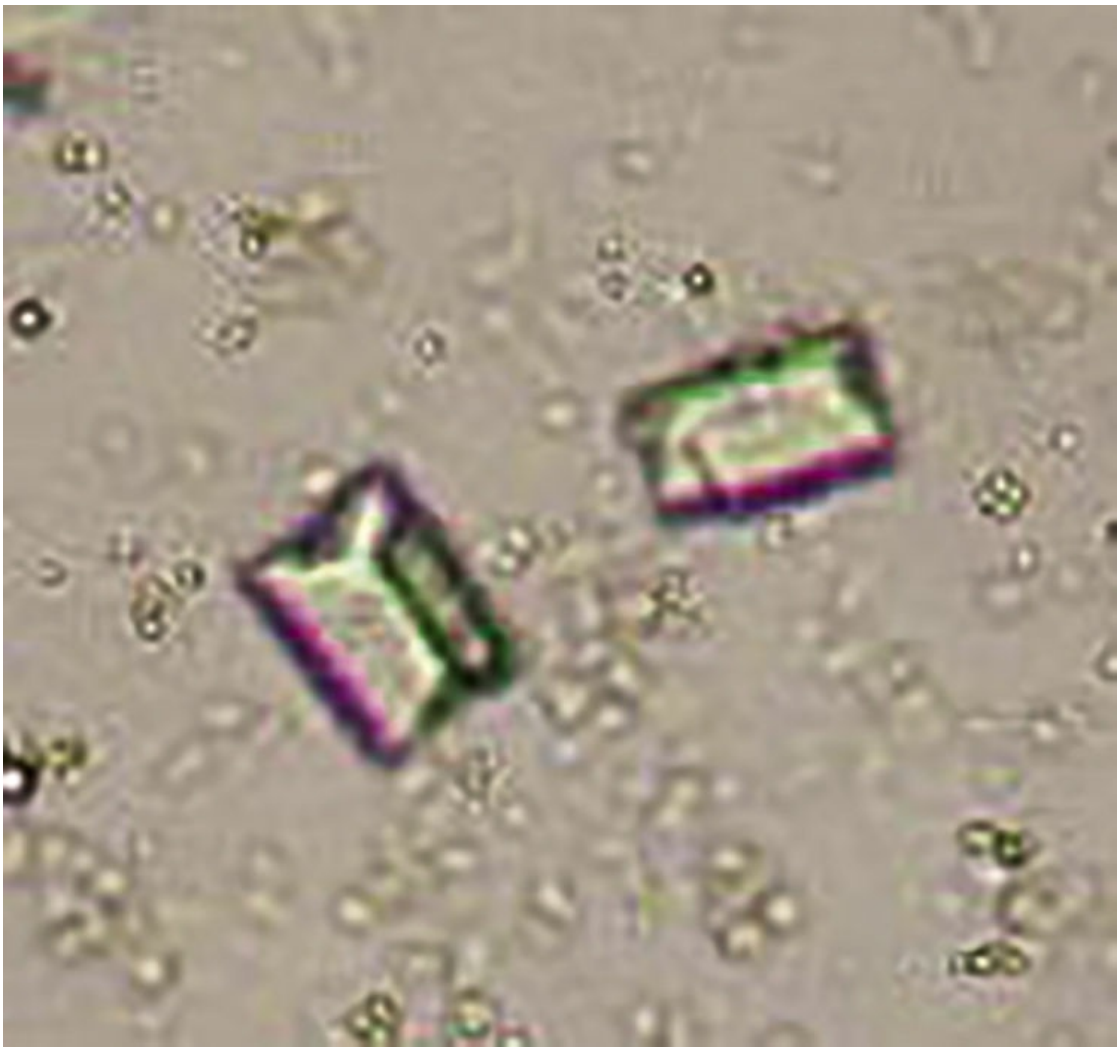

FIG. 6.8 Struvite crystals.

Crystals: Crystals may develop in the patient or in the sample once collected. We really need to know if a patient has actual crystalluria [*crystal* + *-uria* a urinary condition of], because that could put them at risk of stone formation. This is why urinalysis should be performed within 30 minutes of collection. The longer we wait and expose the sample to temperature changes, the more likely crystals will form and mislead us. There are many, many different types of crystals that may be seen in urine. Horses almost always have *calcium carbonate crystals*. Struvite (stroo'vīt) crystals (Fig. 6.8) are the most common crystal to be seen in alkaline urine of dogs and cats. Because of their structure, *struvite crystals* are commonly called "coffin lids." I have seen many cats and dogs with struvite ***uroliths*** [*ur(o)-* urine + *lith(o)-* stone] in their bladders. However, the presence of struvite crystals in a urine sediment does not always indicate ***urolithiasis*** [*ur(o)-* urine + *lith(o)-* stone + *-asis* condition of].

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The most common crystal to be seen in acidic to neutral urine is *calcium oxalate dihydrate* (Fig. 6.9 [*di-* two]). As you can see, these look like little square envelopes. But there are two types of calcium oxalate crystals that you need be familiar with. Calcium oxalate monohydrate [*mono-* one] crystals look more like the slats of a picket fence. Calcium oxalate monohydrate crystals are most commonly seen in ethylene glycol (antifreeze) poisoning. (If you ever see the monohydrate form of calcium oxalate, red flags and sirens should go off in your brain. Ethylene glycol is deadly.) There are many different types of crystals, each distinctive with its own diagnostic significance. Fortunately, most crystals are typically easy to see under low magnification on the microscope.

WBCs: White blood cells often indicate either inflammation or infection. We expect WBCs, especially neutrophils, to infiltrate tissues in the presence of inflammation or ***pathogens*** [*path(o)-* disease + *gen(o)-* producers]. That said, voided samples, particularly from females, may contain rare to few WBCs per high-power field. Their presence along the female reproductive tract is explained in Chapter 9. Refer to Chapter 3 to review detailed WBC information. Again, this reinforces how the collection method may influence our findings. Voided samples, even in healthy animals, naturally contain more stuff, including potentially WBCs.

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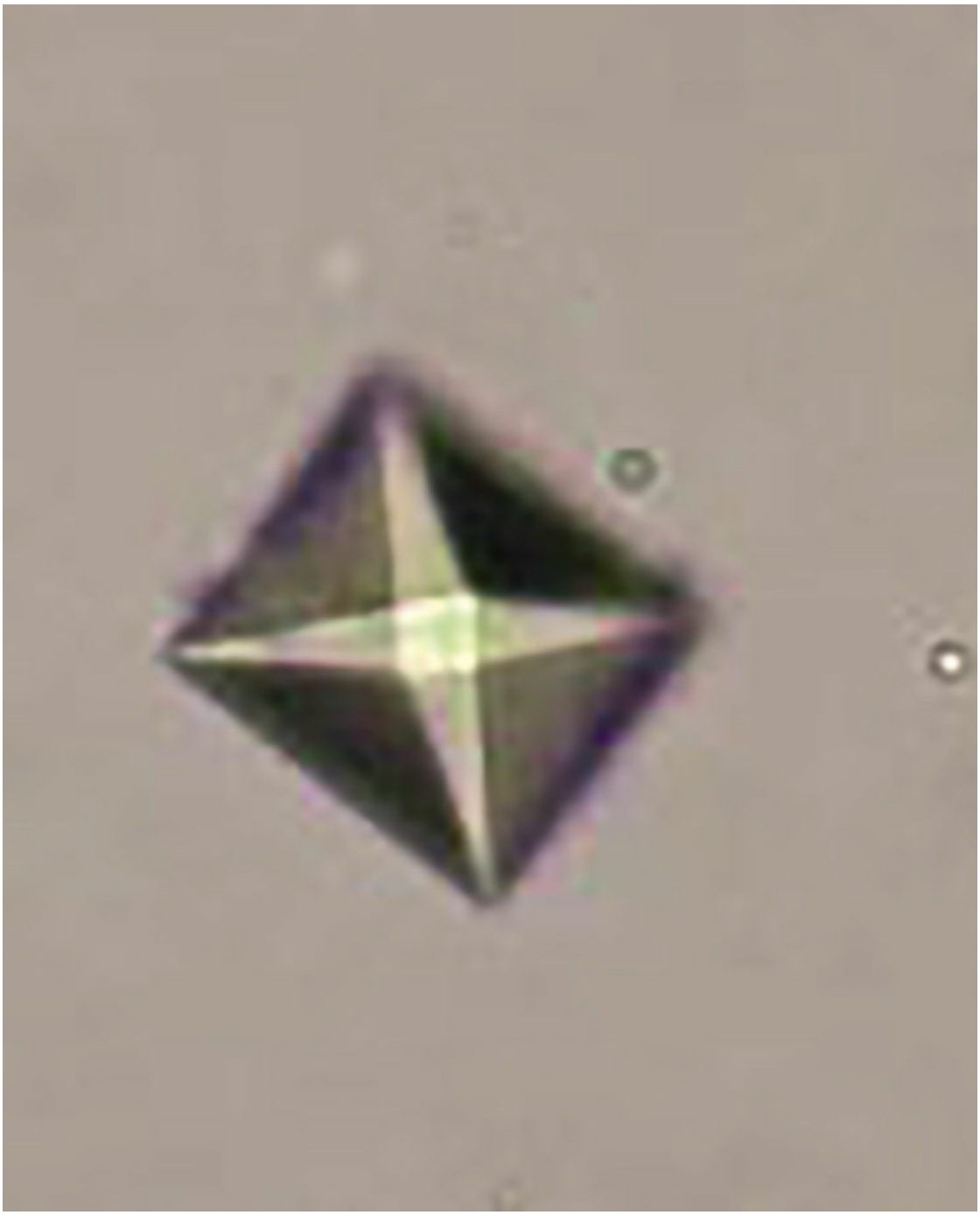

FIG. 6.9 Calcium oxalate dihydrate crystals.

RBCs: RBCs in the urine may be due to trauma or inflammation. As we stated earlier, traumatic *hematuria* can be *iatrogenic*, especially in catheterized or cystocentesis samples. Yet, finding blood on the dipstick does not always mean that we will see RBCs in the sediment even in the presence of bleeding. You see, the more dilute the urine, the more likely the RBCs will burst. Osmosis dictates this event. Remember, as discussed in Chapter 2, osmosis is the movement of water across a semipermeable membrane. That

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movement stops when equilibrium is achieved. When the urine is *hypotonic*, water will move into the RBCs. But they cannot possibly hold enough water to achieve equilibrium, so they burst. The opposite is true if the urine is *hypertonic*. In this instance, the RBCs tend to shrivel up. That shriveling process is called crenation [from L. *crenatus* "scalloped or notched" + *-tion* process of].

Bacteria: Bacteria may be merely a contaminant—certainly an expected finding in a voided sample. While we should not expect to see bacteria in a catheterized sample, contamination is still possible. Cystocentesis is the most ***aseptic*** [*a-* without + *sept(o)-* decay, bacteria + *-ic* pertaining to; i.e., sterile] method of collection. Bacteria (either rods, cocci, or both) observed in a cystocentesis sample is likely an evidence of an active infection. Of course, we cannot identify the specific type of bacteria present. We need cultures for that. All we can do is report the shape of the bacteria we see (i.e., rods or cocci; cocci look like tiny balls). Frequently, but not always, ***bacteriuria*** [*bacteri(o)-* bacteria + *ur(o)-* urine + *-ia* a condition of] will be accompanied by WBCs. Both bacteria AND WBCs are pretty strong evidence of an active urinary tract infection.

Sperm: Sperm would be a very normal finding from intact males and perhaps recently bred females. By the way, sperm will persist for a short time after neutering a male. But in a male who was neutered a long time ago, finding sperm in the urine may indicate that a retained testicle was missed.

Fat droplets: Fat droplets have no ***pathologic*** [*path(o)-* disease + *log(o)-* knowledge, study + *-ic* pertaining to] significance in urine. That said, it is important to be able to differentiate fat droplets from RBCs. RBCs should be in the same microscopic plane as all of the other cells, crystals, and such. Because oil and water do not mix, fat droplets will float right under the coverslip. Also, fat droplets refract light quite a bit, making them appear quite bright compared to cellular material.

Mucus: Mucus may be normal in a *voided* urine specimen, especially in equine urine. In dogs or cats, mucus may be normal, or it may indicate *urethral* inflammation.

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Renal Contributions to Rbc Production and Blood Pressure

Erythropoietin

Believe it or not, the kidneys do far more than simply make urine. And even that process of urine production has far-reaching effects in the body, beyond simple removal of wastes. As we've already discussed, electrolyte and water homeostasis are important renal duties. What you probably don't realize is that the kidneys also have important hormonal influences. As was mentioned in Chapter 3 and will be again in Chapter 10, the kidneys play an integral role in RBC production. The kidneys produce the hormone ***erythropoietin*** [*erythr(o)-* red + *poie(o)-* producer + *-tin* the] that influences the bone marrow to step up production of ***erythrocytes*** [*erythr(o)-* red + *cyt(o)-* cells; i.e., RBCs]. Because *erythropoietin* is so important for RBC production, it stands to reason that in significant renal disease, like ***chronic*** [*chron(o)-* time + *-ic* pertaining to; i.e., long term] renal failure, along with decreased renal function, we may also see nonregenerative ***anemia*** [*an-* without + *em(o)-, hem(o)-* blood + *-ia* condition of; i.e., decreased RBCs]. Refer to Chapter 3 to review regenerative versus nonregenerative anemias.

Blood Pressure

When it comes to hormonal influence with regard to the kidneys, there is probably nothing more important than the regulation of blood pressure. Adequate blood pressure is absolutely essential for renal function. So, it stands to reason that the kidneys would want to do everything in their power to maintain adequate blood pressure. And that they do, by producing hormones that contribute to blood pressure homeostasis. Self-serving? Perhaps. But the rest of the body and our very lives depend on it. That's also why blood pressure is being discussed in multiple chapters. It's really, REALLY important.

Earlier we talked about antidiuretic hormone (ADH). We learned that in the presence of ADH the kidneys conserve water for the

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body by absorbing water from the renal tubules. This is beneficial for blood pressure, by simply helping in maintaining the overall blood volume. After all, what's good for blood volume is good for blood pressure. But that's not all ADH does. Antidiuretic hormone has another name: ***vasopressin*** [*vas(o)-* vessel + *press(o)-* pressure + *-in* the]. As the name *vasopressin* implies, this hormone causes *vasoconstriction*. If vessels, especially peripheral vessels, are smaller, then less blood is needed to fill them. So, blood pressure goes up. These types of events happen even when something like dehydration develops. Both dehydration and low blood pressure will trigger the secretion of vasopressin (aka antidiurectic hormone, ADH) by the pituitary. Ah, but ***hypotension*** [*hypo-* low + *tens(o)-* pressure + *-ion* state of] can occur for many reasons beyond dehydration, like heart failure for example. And because adequate blood pressure is absolutely essential for all body tissues and major organs—like the brain, kidneys, liver, and heart—we need lots of mechanisms to help us raise and maintain blood pressure.

We have pressure receptors in key areas throughout the body. In the presence of *hypotension*, the posterior pituitary is stimulated to secrete vasopressin. At the same time, the anterior pituitary is stimulated to secrete ***adrenocorticotropic*** [*adren(o)-* adrenal + *cortic(o)-* cortex + *trop(o)-* influence + *-ic* pertaining to] ***hormone*** (***ACTH***). This hormone stimulates the adrenal glands to secrete ***aldosterone***. The hormone *aldosterone* influences the kidneys to reabsorb sodium and water. Where sodium goes, water follows. Resorption of both Na^+ and water helps improve the circulating blood volume. But wait, there's more.

Hypotension also triggers the ***sympathetic nervous system***. (This will be discussed in great detail in Chapter 11. For now, simply think of this branch of the nervous system having sympathy for us in this hypotensive situation.) Guess what—sympathetic nerve fibers are the only nerves connected to the adrenal glands. (By the way, the name ***adrenal*** tells us where they're located [*ad-* toward, near + *ren(o)-* kidney + *-al* pertaining to]. They are very near to the kidneys—very handy indeed, as we'll learn in a moment.) When the sympathetic nerve fibers stimulate the adrenal glands, the adrenals release some really powerful hormones. Have you ever heard of ***adrenaline*** [*adrenal* + *-ine* hormone]? Adrenaline is also

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called ***epinephrine*** (abbreviated "epi," when we're rushing to save a life in an emergency). Well, epinephrine targets specific ***vascular*** [*vascul(o)-* vessel + *-ar* pertaining to] receptors called ***alpha adrenergic*** [*adren(o)-* adrenal + *erg(o)-* working + *-ic* pertaining to] receptors in the periphery and other nonessential areas to cause *vasoconstriction* too. Again, this helps increase blood pressure. But epinephrine also targets ***beta adrenergic*** receptors on the heart, increasing heart rate and strength of contraction to help improve blood pressure. We're not done yet. Now for the kidney's self-serving part.

All of that adrenergic activity also stimulates the kidneys to secrete ***renin*** [*ren(o)-* kidney + *-in* a, the]. Decreased renal blood flow and other factors also stimulate the production of *renin*. *Renin* is needed as the first step in the production of the active form of ***angiotensin*** [*angi(o)-* vessel + *tens(o)-* pressure + *-in* a, the]: ***angiotensin II***. There are a few steps necessary before we get there. First, *renin* acts on ***angiotensinogen*** [*angiotensin(o)-* angiotensin + *gen(o)-* producer], a substance produced by other organs and readily available in circulating plasma. This creates ***angiotensin I***, the inactive form of angiotensin. Second, ***angiotensin-converting enzyme*** (***ACE***) transforms *angiotensin I* into active *angiotensin II*. Now we have something the body can work with. And the body's response to angiotensin II is significant.

Angiotensin II, as its name implies, causes vasoconstriction to improve blood pressure. Angiotensin II also increases thirst. Improved hydration through drinking water improves blood volume and pressure. Angiotensin II increases secretion of aldosterone. We learned a short time ago that aldosterone acts on the kidneys, causing reabsorption of sodium and water to improve blood volume and pressure. Angiotensin II also increases the secretion of vasopressin (ADH), causing more water conservation and peripheral vasoconstriction to improve blood pressure. Finally, angiotensin II also increases secretion of ***norepinephrine***. *Norepinephrine*, like epinephrine, has ***adrenergic*** effects that help improve blood pressure. When we finally achieve adequate blood pressure, all of these intricate hormonal activities are either reduced or disengaged. Wow, this ***renin-angiotensin cycle*** is really important for maintenance of blood pressure. And that means it's

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great for renal function.

Unfortunately, in heart failure all of these compensatory mechanisms compound problems for an already weak heart. As we discussed in Chapter 5, heart failure patients have poor ***cardiac*** [*cardi(o)-* heart + *-ac* pertaining to] ***output***. Poor cardiac output translates to poor blood pressure. And we just learned that in the presence of hypotension, there are lots of mechanisms that will be engaged, like the *renin-angiotensin system*. All of these mechanisms result in vasoconstriction and retention of sodium and water. A weak heart will have difficulty pumping against the increased resistance created by vasoconstriction. This can result in ***edema*** (swelling). If you remember, in Chapters 3 and 5 we talked about edema, or increased fluid in the tissues (interstitium). In patients with heart failure, those fluids often accumulate in the lungs, chest cavity, and/or abdomen. When fluids accumulate in the chest and lungs, it's very difficult to take in needed oxygen and get rid of carbon dioxide. So in heart failure, the renin-angiotensin cycle of events can be somewhat harmful. It's a delicate dance to maintain adequate blood pressure, without compounding fluid retention and edema formation.

To help the situation, most heart failure patients are fed a low-sodium diet. Less available sodium means less edema formation. Many heart failure patients are also medicated with *ACE-inhibitors*. By inhibiting the conversion of angiotensin I to angiotensin II, we minimize the catch-22 cycle of events that a weakened heart simply can't cope with. If fluid accumulation is still too great, we may place the patient on ***diuretic*** [*diure(o)-* urination + *-tic* pertaining to; i.e., a drug that promotes urine production] medications to promote removal of water from the body. Furosemide is a commonly used loop *diuretic*. It targets the Loop of Henle, decreasing the reabsorption of sodium and chloride. It also increases excretion of water and potassium. It's obvious that reducing Na^+ and Cl^- reabsorption and increasing excretion of water are beneficial in the removal of excess fluid from the body. But we do need to keep a close eye on potassium levels to avoid complications of ***hypokalemia*** [*hypo-* low + *kal(o)-* potassium + *-emia* a blood condition of]. Adequate potassium levels are very important for both heart and nerve function. There are other *diuretics* that can be

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used that are potassium “sparing,” so that we don’t need to be concerned with complications like *hypokalemia*.

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Common Urinary Diseases

There are numerous urinary diseases, and we certainly cannot discuss them all here. So we'll simply touch on a few of the major and most common problems.

Diabetes Insipidus

We mentioned ***diabetes*** [Gr. *diabētēs* a siphon] ***insipidus*** [L. *insĭpĭdus* tasteless] earlier. Animals with *diabetes insipidus* have *polyuria* and *polydipsia* (PU/PD). That's what the term diabetes refers to: water being siphoned—literally flowing through the body, seemingly unchanged as if it were flowing through a siphon hose. This is truly a water management issue. And water is tasteless, hence the second part of the name: *insipidus*. No, we don't ever taste urine. Gross! But if we did, the urine of a *diabetes insipidus* patient would be pretty tasteless. Let's pause for a moment. I'll bet you're thinking, "Wait! I thought diabetes was a problem with blood sugar. I know people who take insulin for it." Yes, most of the time when we use and hear the term diabetes, we refer to ***diabetes mellitus***, which is **not** a urinary disease. Diabetes mellitus **is** a disease affecting blood glucose. Unmanaged, diabetes mellitus results in ***hyperglycemia*** [*hyper-* excess + *glyc(o)-* sugar, glucose + *-emia* a blood condition of]. It's the second part of the name—*mellitus* [L. *mellĭtus* honeyed, sweet]—that distinguishes this disease from diabetes insipidus. If you recall, we said that the kidneys have a set threshold for glucose. Any glucose over and above that threshold will pass in the urine. If we were to taste the urine of a patient with diabetes mellitus, it would be sweet because of the *glycosuria*. Again, we don't taste it, but we can sometimes smell the sweetness and we can certainly measure the glucose with urine dipsticks. Both types of diabetes result in *polyuria* and *polydipsia*. But the actual mechanisms behind the PU/PD are dramatically different.

We'll discuss diabetes mellitus in detail in Chapter 10. Our focus here will be on the renal implications of the disease, to help you understand the difference between diabetes mellitus and diabetes insipidus. In brief, diabetes mellitus results from either insufficient

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insulin being produced by the pancreas or resistance to insulin by cellular receptors. You see, insulin is needed to facilitate active transport of glucose molecules into cells. If glucose cannot enter cells, *hyperglycemia* results. The kidneys can reabsorb some glucose, but everything over the renal threshold remains in the filtrate and is passed in the urine. Like sodium, glucose is very attractive to water. With large amounts of glucose in the urine, water will be dragged with it. So, diabetes mellitus patients have *polyuria*. But remember, the urine specific gravity could be normal because the glucose increases the osmolality of the urine. Often, *polyuric* patients are *isosthenuric* or *hyposthenuric*. And with small amounts of glucose in the urine, a diabetes mellitus patient could be isosthenuric. Just remember, the more glucose in the urine, the higher the specific gravity will be.

Diabetes insipidus is different. Obviously, it does not involve glucose at all. Instead, there is purely a problem with our "water management system." This system involves a portion of the brain, the pituitary gland, and the kidneys. If there is a problem with any of these players or their communication, diabetes insipidus may develop. It's rare that the brain (specifically the hypothalamus) is the problem. So let's take that off the table. If the kidneys are the problem, then something must be interfering with the kidneys' ability to respond to antidiuretic hormone. If we can figure out what's causing the interference (like ***nephritis*** [*nephr(o)-* kidney + *-itis* inflammation of]) and treat it, we may be able to resolve the diabetes insipidus. It does happen, though not as frequently as problems with the pituitary gland. Most of the time, diabetes insipidus results from too little production of ADH. This could result from trauma, tumors, or cyst formation in or near the pituitary gland. Many times we have no idea why the pituitary isn't producing enough ADH. It's often an ***idiopathic*** [*idi(o)-* one's own + *path(o)-* disease + *-ic* pertaining to; i.e., a disease of unknown origin] disorder. *Idiopathic* disorders are unique to the individual, and we have no idea how or why.

In idiopathic diabetes insipidus, if we do not have sufficient ADH, the kidneys' hands are tied, so to speak. With insufficient ADH, the tubules simply cannot reabsorb enough water. So, we have a patient with PU/PD. Some pet owners, unaware of the

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underlying disease, may misunderstand the pet's polyuria. To prevent accidents in the house, they may reduce the availability of water. This is a dangerous move. These patients need to drink lots of water to keep up with what's being lost in the urine. Remember, the renal tubules simply cannot conserve water for the body. They cannot concentrate the urine. Hence large amounts of isosthenuric or hyposthenuric urine will be produced. If that animal doesn't have enough water to drink, it will rapidly dehydrate. Dehydration leads to hypotension, and hypotension taxes the whole body, especially the brain, heart, and kidneys. And that can produce deadly consequences. Fortunately, we can treat diabetes insipidus with medication. By giving synthetic ADH, we make up for the deficiency. This restores relatively normal renal function, permitting the kidneys to conserve water as they should.

Urolithiasis

When we talked about urinalysis, we talked briefly about crystal formation. We mentioned struvite and calcium oxalate dihydrate crystals being two of the most common crystals to form in the urine of dogs and cats. There are many other types of crystals, including calcium carbonate (common in horses), urate, cystine, and others. The presence of *crystalluria* does not mean that an animal has or will form *uroliths*. Crystalluria simply indicates that the urine is saturated with a certain ***crystalloid*** [*crystall(o)-* crystal + *-oid* resembling; i.e., the dissolved substance of crystals]. Once the urine is saturated, the *crystalloid* precipitates out into what we observe under the microscope as crystals. And while those crystals don't mean stones will form or are forming, they certainly increase the risk of *urolithiasis* [*ur(o)-* urine + *lith(o)-* stone + *-iasis* condition of].

There are a number of factors that can play into the development of crystalluria and urolithiasis. First, hydration and urine concentration. Patients who are adequately hydrated produce less concentrated urine, right? If the urine is less concentrated, certain crystalloids are less likely to precipitate and form crystals. Obviously as the urine becomes more and more concentrated, we are more likely to reach and exceed the saturation point, resulting in crystal formation. Second, the animals should have the

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opportunity to evacuate their bladders as needed. Stagnation of urine (especially with crystalluria) in the bladder provides an environment for aggregates of crystals to form, resulting in urolith formation. Urine pH can also be a factor. Some crystal formation can either be inhibited or enhanced, depending on the pH. We gave a couple of examples of this when we discussed crystals in the urine sediment section. In those examples, we said that struvite crystals tend to form in alkaline urine and calcium oxalate dihydrate crystals tend to form in neutral to acidic urine. Again, the presence of these crystals does not mean stones are forming. With the right combination of factors, urolithiasis may very well develop.

Consider struvite urolithiasis, for example. This is one of the most common types of urolithiasis in dogs and cats. Obviously, these dogs and cats must have alkaline urine. This may be the animal's normal urine pH. And there are factors that can contribute to the alkalinity. Believe it or not, eating can play a role. As you will learn in Chapter 7, hydrogen ions (H^+) are needed to produce stomach acid for digestion. The stomach uses H^+ from the bloodstream to produce hydrochloric acid (HCl). Temporarily after eating a meal, this will result in a phenomenon known as the ***postprandial*** [*post-* after + *prandi(o)-* meal + *-al* pertaining to] ***alkaline tide*** (tide, because it ebbs and flows like an ocean tide). What does the *postprandial alkaline tide* have to do with urine? After eating a meal there are fewer H^+ in the blood stream. To maintain homeostasis, the kidneys have to reabsorb H^+. As a result, the urine is more alkaline. Generally, this creates only minor fluctuations in the urine pH. And the alkalinity doesn't persist between meals. But in someone who already has neutral to slightly alkaline urine, this shift in pH could make a difference. This is especially true if the animal is allowed to "graze" and eat free-choice throughout the day and night. This may create chronically alkaline urine, enhancing the possibility of struvite crystalluria and urolithiasis. Another factor that frequently sets up the perfect storm for struvite urolithiasis is a urinary tract infection (UTI). Even if the animal normally has quite acidic urine, a urinary tract infection can dramatically shift the pH to become very alkaline. I have known many animals who developed struvite urolithiasis in the wake of UTIs. We'll talk about UTIs a little later. For now, let's stay on track talking about urolithiasis.

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Why are uroliths such a big deal? Well, uroliths in the bladder traumatize the bladder wall, creating ***cystitis*** [*cyst(o)-* bladder + *-itis* inflammation of]. (UTIs create *cystitis* too.) *Cystitis* can be very painful. In response to the inflammation, cystitis patients tend to be ***pollakiuric*** [*pollaki-* often, frequent + *ur(o)-* urination + *-ic* pertaining to]. You see the inflammation can precipitate spasm of muscles in the bladder wall and the urethral sphincter, increasing the urge to urinate frequently. Also, the ***pollakiuria*** [*pollaki-* frequent + *ur(o)-* urination + *-ia* condition of] is only compounded if the stones take up too much space in the bladder. That's bad enough, but what's worse is urinary obstruction.

As an aside, I should note that uroliths may also form in the renal pelvis. (In humans, we call these "kidney stones.") It happens in animals too. These uroliths can and do cause ***ureteral*** [*ureter* + *-al* pertaining to] obstruction. This will have a very direct negative impact on the kidney. But it is very difficult to treat. Sometimes we may wait to see if the peristaltic action of the ureter can push the urolith into the bladder. If waiting is not an option, we may attempt to place a stent—a device that increases the inside diameter of the ureter for easier passage of the stone. In some cases, to prevent significant renal damage, we may surgically create a shunt, bypassing the obstruction. Think of it as a detour for urine flow from the kidney to the urinary bladder. Fortunately in animals, ureteral obstruction is not as common as urethral obstruction.

Urethral obstruction with uroliths is common, especially in male animals. Although I have seen my fair share of females develop urethral obstruction—one was a Labrador. Think about that. She was a big girl (in good body condition). Yet in spite of her size and the size of her urethra, she still managed to obstruct with uroliths. That just proves that with the right size uroliths compared to the size of the urethra, it is possible for anyone to obstruct. Obviously, males are more prone to it than females because the size of the male urethra is comparatively smaller than females of the same species and breed. Male dogs in particular are structurally at risk of urethral obstruction, just by virtue of the fact that the urethra passes through the os penis. That bone allows no wiggle-room. Tiny stones may pass. Though he may demonstrate ***dysuria*** [*dys-* difficult + *ur(o)-* urination + *-ia* condition of], as he attempts to pass smaller

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stones. Ultimately, if a large enough stone gets wedged in the os penis segment of the urethra, he will obstruct. Male cats obstruct more frequently than anyone, from stones and "sand." The sand is not from the beach. Its aggregates of crystals that build up and eventually plug up the urethra. If the owner of a male cat ever calls saying, "I think he's constipated, because lately he's been loudly crying out a lot in the litter box," tell the owner the cat needs to be seen ASAP. That's a classic sign of urethral obstruction in a male cat. No matter how or in whom urethral obstruction occurs, it is an emergency.

Why is it an emergency? First, urine continues to be produced. Inability to evacuate the bladder could result in a ruptured bladder. Second, the longer the obstruction exists, the more likely secondary problems will develop beyond the urethra and bladder. As the bladder distends and pressure builds up, urine may be prevented from flowing into the bladder. The backup of urine can distend the ureters and even result in ***hydronephrosis*** [*hydr(o)-* water + *nephr(o)-* kidney + *-sis* condition of]. (This is more common from ureteral obstruction.) I have seen ***hydronephrotic*** [*hydr(o)-* water + *nephr(o)-* kidney + *-tic* pertaining to] kidneys in which the renal pelvis is extremely dilated and the renal capsule becomes extremely distended with fluid. Imagine the pressure being exerted in such a kidney. It's extremely painful. And that pressure is going to damage lots of nephrons—never to be regained. Finally, if urine has nowhere to go, all of the things that should be excreted in the urine will remain in the body. We'll talk about some of this more when we discuss renal failure a little later. For now, let's focus on a very important electrolyte: potassium (K^+). The kidneys should excrete potassium. If they cannot do that, potassium will build up in the body. ***Hyperkalemia*** [*hyper-* excess + *kal(o)-* potassium + *-emia* a blood condition of] can be very deadly. *Hyperkalemia* significantly slows the heart and can slow it to the point of stopping. Hyperkalemia is most common in cats with urethral obstruction. Not only is the hyperkalemia potentially life-threatening by itself, it complicates our treatment of the animal by making sedation or anesthesia extremely risky. General anesthesia and most sedatives powerful enough for our procedure also slow the heart rate. Suffice it to say that electrolytes must be evaluated as quickly as possible

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before we risk complicating matters.

Beyond ***triage*** ([Fr. *triage* "sorting"] i.e., prioritizing medical problems) and stabilizing patients with urethral obstruction, our principal goal is to relieve the obstruction and decompress the bladder. (Do NOT attempt to decompress the bladder by manual expression. There is EXTREMELY HIGH RISK of rupturing the bladder.) Sometimes this is easier said than done. As indicated earlier, most of the time these patients must be either heavily sedated or sleeping under general anesthesia for this procedure. Generally, to relieve the obstruction we attempt to pass a urinary (urethral) catheter. Imagine how that will feel in an already painfully raw urethra. That's why we need sedation or anesthesia. With a catheter we may be able to push the stone(s) ***retrograde*** [*retro-* backward + *grade* to step, move] into the bladder. In the process, the urethra (already fragile and traumatized from the obstruction) may easily be torn by the stone or the catheter. Sometimes, even with a rigid catheter, the stone won't budge. In those cases, we may try urethral ***retropulsion*** [*retro-* backward + *puls(o)-* push + *-ion* act of; i.e., the use of fluid to flush the stone backward]. The movement of fluid is a powerful force to be reckoned with. *Retropulsion* is often very effective in flushing stones back from the point of obstruction all the way back into the bladder.

After the obstruction is relieved, we may leave a urinary catheter in place for a brief period to monitor urine output. Generally, with a catheter in place the urethra remains *patent* (open). But be forewarned, I have seen urethral catheters become plugged with stones, sediment, and even blood clots. And if we're going to administer IV fluids with the purpose of ***diuresis*** [*diure(o)-* urination + *-sis* process of; i.e., to cause increased urine production], we need to monitor fluid input and output very closely to avoid fluid overload. Plus, we need to monitor urine output, making sure that what goes in flows out. If that catheter becomes plugged, we could rupture the bladder. A temporary urethral catheter also permits urethral inflammation, especially swelling associated with it, to subside. This is particularly necessary if ***deobstruction*** [*de-*, L. *de* "away from"] was difficult. We want these animals urinating and evacuating their bladders. The ***urethritis*** [*urethr(o)-* urethra + *-itis* inflammation of] may make it feel to the animal like it's urinating

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razor blades. That may cause the animal to behaviorally inhibit urination. Obviously, that is not in the patient's best interest and may lead to further stone formation and subsequent obstruction. In spite of all of this, there is still debate over placement of indwelling urinary catheters in these patients. Those against indwelling catheters, even if placed for only 12 to 24 hours, are concerned with introducing an infection. However, if the catheter is placed and maintained in an *aseptic* manner, secondary infection should not be a concern. Plus, some of these animals already have UTI and are being treated for it, making the infection argument a moot point.

Okay, so let's say we have successfully relieved the urethral obstruction, adequately diuresed, and restored electrolyte homeostasis of our patient. Clinically, the patient may be stable. However, based on abdominal ultrasound and ***cystography*** [*cyst(o)-* bladder + *-graphy* recording of; i.e., radiographs of the bladder], we know that numerous stones are still in the urinary bladder. A ***cystourethrogram*** [*cyst(o)-* bladder + *urethr(o)-* urethra + *-gram* a recording of; i.e., radiographic imaging using contrast dye] may also reveal abnormal narrowing at certain points along the urethra. We know that the stones, especially at narrowed areas of the urethra, could result in future obstruction. So, what can we do? Well, we really need to know the composition of the stones. If we know that, a dietary change may be beneficial. For instance, if we know that we are dealing with struvite urolithiasis, there are commercial diets available that are designed to dissolve those stones. After the stones are gone, another diet that contains fewer of the elements needed to form struvite crystals as well as acidifiers to prevent their formation may be fed. Meal feeding rather than free-choice feeding may help too. But in cases with large numbers of uroliths, diet alone may be insufficient. We may need to perform a ***cystotomy*** [*cyst(o)-* bladder + *-tomy* to cut]. Certainly, if the patient is in imminent danger of obstructing again, a *cystotomy* is warranted. And if we don't know the composition of the stones, we'll need to perform a cystotomy. Once the stones are surgically removed from the bladder, they can be submitted for analysis. The results of that analysis will help guide the medical management of the patient, perhaps for the rest of its life. This is very true for dogs with urate or uric acid urolithiasis. Special diets and other measures

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are required to prevent future stone formation.

For those male animals who repeatedly experience urethral obstruction with uroliths, more aggressive measures may be needed. The distal urethra is the most narrow segment in both the dog and the cat. And we've already talked about problems associated with the os penis in dogs. If we can create a urethral opening in a wider, more proximal segment, we may be able to prevent future obstruction. That is precisely the purpose behind a ***urethrostomy*** [*urethr(o)-* urethra + *-stomy* creation of a "mouth" (stoma)]. In male cats, due to the caudal positioning of the penis, a ***perineal*** [*perine(o)-* perineum + *-al* pertaining to] *urethrostomy* is done. The penis is amputated, and a wide urethral opening is created through the *perineal* skin (i.e., the skin just dorsal to the prepuce). In dogs, the most common location of the *stoma* (mouth-like opening) is in the ***prescrotal*** [*pre-* before + *scrot(o)-* scrotum + *-al* pertaining to] area (i.e., the skin just cranial to the scrotum). Once recovered from the surgical trauma, most urethrostomy patients do very well. The dogs have the biggest learning curve in figuring out how to urinate without it running down their rear legs. Eventually, they adapt. Finally, note that some animals have the tendency to scar excessively. So, owners need to continue being observant for signs of dysuria or obstruction in urethrostomy patients.

Urinary Tract Infections

Urinary tract infections (UTIs) of the lower urinary tract (i.e., urethra and bladder) are relatively common, especially in dogs and cats. Of that population, dogs have the most frequent incidence of UTIs. Not everyone is predisposed to developing UTIs. In fact, for most, there are basic features of the urinary tract that make it less likely for infections to develop. First, let's consider the routine voiding and emptying of the bladder. This tends to wash bacteria out of the urethra. Strike one for bacteria. Second, the urine itself is not generally a good substrate to promote bacterial growth. This is especially true if the urine is acidic with a higher specific gravity. Strike two. Third, there are natural defenses with local and humoral immunity (refer to Chapter 3), as well as ***mucosal*** [*mucos(o)-* mucus + *-al* pertaining to; i.e., the lining of the bladder and urethra that is

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covered in a mucus-like substance] barriers that prevent colonization and promote removal of potential ***pathogens*** [*path(o)-* disease + *gen(o)-* producer]. Strike three—you're out! Still, UTIs do develop.

Females tend to have UTIs more frequently than males. Why the gender difference? Anatomically the ***urogenital*** [*ur(o)-* urinary + *genit(o)-* reproductive + *-al* pertaining to] anatomy of females is more favorable for contamination with bacteria or other microbes. We'll look at urogenital anatomy in greater detail in Chapter 9. For our purposes here, let's look at it briefly to see how the anatomy can play a role in development of a UTI. First, consider the fact that the anus is dorsal to the vulva in all female domestic animals. Depending on the distance between those features and the actual structure of the vulva itself, there may be increased risk of contamination of the vulva with feces. Second, excessive folds and crevices around the vulva may also provide a warm, moist environment that promotes bacterial growth. Finally, because female dogs squat close to the ground to urinate, they are at a greater risk than many other females for contamination of the ***vulvar*** [*vulv(o)-* vulva + *-ar* pertaining to] and ***perivulvar*** [*peri-* around + *vulv(o)-* vulva + *-ar* pertaining to] skin. Bacteria are the most common contaminants and cause of urinary tract infections. In fact, *E. coli* from feces is the most common *pathogen* associated with UTIs in dogs and cats. *Staphylococcus sp*. (staf"uh-lo-kok'us), a common skin inhabitant, takes second place as a lower urinary tract pathogen. With increased risk of contamination and promotion of ***bacterial*** [*bacteri(o)-* bacteria + *-al* pertaining to] growth, it sets many female animals up for the development of *retrograde* UTIs. Remember, the urethra opens into the vestibule/vaginal region. So, if she develops progressive inflammation and infection of the vulva and vagina, anatomically it is very easy for it to progress into the urethra. From there, it's a short hop to the urinary bladder.

Comparatively, urogenital anatomy of the male is highly integrated. The urethra passes all the way through the penis, making both dual purpose for urination and breeding. You might think that this integration alone provides perfect anatomy for UTIs. But it doesn't. There are several factors to be considered here. First, the penis and urethral orifice are covered and protected by the

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prepuce. Second, due to the location in most males (the cat is the exception), along the ventral abdomen, contamination of the prepuce and penis is less likely to occur. (Not true for Mini-Dachshunds.) Third, ***prostatic*** [*prostat(o)-* prostate + *-ic* pertaining to] secretions are ***bactericidal*** [*bacteri(o)-* bacteria + *cid(o)-* killing + *-al* pertaining to]. Finally, the urethra of most males is incredibly long, making retrograde *cystitis* due to infection far less likely to occur. That said, some males (especially dogs) do develop bacterial urinary tract infections.

Whether male or female, there are some common symptoms reported in the clinical history. Owners may complain about the *pollakiuria,* ***stranguria*** [Gr. *stranx* drop + *ur(o)-* urine + *-ia* condition of; i.e., painful, strained urination, straining to urinate], or *dysuria*. Accidents in the house by dogs who have been housebroken for years often prompt a veterinary visit. If the dog urinates in the house or on snow, the owners may notice *hematuria* [*hemat(o)-* bloody + *ur(o)-* urine + *-ia* condition of]. Sometimes owners may also report foul smelling urine. Aside from direct urinary symptoms, some animals become lethargic with decreased appetites. A thorough physical examination is always warranted. Because bacterial culture will be needed, our urine sample should be collected via cystocentesis. If a bacterial UTI is present, our urinalysis findings will probably include *hematuria*, ***pyuria*** [*py(o)-* pus + *-uria* a urine condition of; i.e., white blood cells], ***bacteriuria*** [*bacteri(o)-* bacteria + *-uria* a urine condition of], and increased numbers of transitional epithelium. Remember, in a cystocentesis sample, we typically don't see much transitional epithelium. Most often in a UTI the urine will be alkaline. The bacterial culture and sensitivity results will identify the pathogen and those antibiotics best to eradicate it. It's really important that the owners give all of the antibiotics for the full duration of treatment. If they don't, their pet's UTI will either not be cured or it will recur. Plus, not giving the antibiotics as prescribed could promote bacterial resistance.

Do UTIs ever progress beyond the urinary bladder? Yes. Fortunately, this is the exception rather than the rule. Why? There's a valve-like structure where each ureter enters the urinary bladder. This alone makes it more difficult for bacteria to move into the ureters. Do you remember that we said the ureters actually have

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muscular contractions pushing the urine from the kidneys to the bladder? That ***peristaltic*** [*peri-* around + Gr. *stalsis* contraction + *-tic* pertaining to] activity makes it mighty hard for bacteria to move retrograde along the ureters. It's like trying to swim upstream against a powerful current. That said, there are circumstances when bacteria, against all odds, do make their way to the kidneys creating bacterial ***pyelonephritis*** [*pyel(o)-* pelvis (renal) + *nephr(o)-* kidney + *-itis* inflammation of]. This is serious. With bacterial *pyelonephritis*, the inflammation and traumatic bacterial activity can significantly damage nephrons. Even once the infection is eliminated, it may leave the animal with significant and permanent damage. While this may occur, in my experience, most of the cases of bacterial ***glomerulonephritis*** [*glomerul(o)-* glomerulus + *nephr(o)-* kidney + *-itis* inflammation of] are from blood-borne pathogens like *Leptospira sp*. ***Leptospirosis*** (lep"to-spi-ro'sis [*leptospir(o)- Leptospira* bacteria + *-sis* condition of] is a ***zoonotic*** [*zo(o)-* animal + *nos(o)-* disease + *-tic* pertaining to; i.e., a disease transmitted between animals and people] disease. This bacterium easily penetrates the skin, especially where there are breaks in the skin. Once in the bloodstream, it tends to infect highly vascular organs like the kidneys and liver. *Leptospirosis* can be deadly for both animals and humans. Survivors may have significant kidney damage that leads to chronic renal failure. I have cared for numerous patients with leptospirosis. It's a devestating disease. Fortunately, a vaccine is available to help protect dogs against this deadly disease.

Renal Failure

Renal failure can be categorized as ***acute*** (sudden), chronic (long-term), and acute on chronic (i.e., a sudden worsening of existing disease). No matter how you categorize it, renal failure involves the loss of functional nephrons. Many would agree that when roughly three-quarters of the nephrons of both kidneys no longer work, the patient is in renal failure.[e] Loss of that many nephrons dramatically reduces the kidneys' abilities to do what they were intended to do —excrete wastes and help maintain water and electrolyte homeostasis. Sure, we could talk about how damage to each part of the nephron affects renal function. But frankly, if one part of the

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nephron is damaged, the whole thing is nonfunctional. There are lots of things that can damage nephrons and cause renal disease and failure. In general, we classify these causes as ***prerenal*** [*pre-* before + *ren(o)-* kidneys + *-al* pertaining to], *renal* (i.e., a direct insult to the kidneys), and ***postrenal*** [*post-* after + *ren(o)-* kidneys + *-al* pertaining to]. As we move through this discussion of renal failure, I'll try to point out some of the more common prerenal, renal, and postrenal causes.

Regardless of cause, there are some common threads in renal failure that we need to understand. First, if we've lost a significant number of nephrons, our ability to remove wastes and maintain electrolyte and water homeostasis is significantly impaired. Remember, the kidneys are responsible for removing nitrogenous wastes like ammonia, urea, and ***creatinine*** (kre-at'ĭ-nēn; a by-product of the decomposition of the amino acid creatine). In renal disease, these wastes build up in the bloodstream, resulting in ***azotemia*** [*azot(o)-* nitrogen, nitrogenous + *-emia* a blood condition of]. Believe it or not, ***azotemic*** [*azot(o)-* nitrogen, nitrogenous + *em(o)-*, *hem(o)-* blood + *-ic* pertaining to] animals can actually appear clinically normal on physical examination, especially with mild to moderate *azotemia*. In spite of their outward appearance, laboratory values of blood urea nitrogen (BUN) and creatinine for these patients will tell a different story. Often, it's not until an animal develops ***uremia*** [*ur(o)-* urine, urea + *-emia* a blood condition of; i.e., excessive levels of nitrogenous wastes in the blood] that they appear really ill. Even their owners can't miss it. ***Uremic*** [*ur(o)-* urine, urea + *em(o)-*, *hem(o)-* blood + *-ic* pertaining to] patients often have ***anorexia*** [*an-* without + *orex(o)-* appetite + *-ia* condition of] and vomiting. These digestive system signs develop from (1) direct stimulation of vomiting centers in the brain from the uremic toxins and (2) inflammation and even ulceration of the stomach. And remember, these patients have poor water homeostasis. So, they can dehydrate very quickly from the vomiting, which will only concentrate all of the noxious wastes in the bloodstream even more. By the way, those uremic toxins can also have a very negative impact on brain functions too.

Common electrolyte disturbances that develop in renal failure include *hyperkalemia*, ***hypernatremia*** [*hyper-* excess + *natr(o)-* sodium

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+ *-emia* a blood condition of], ***hyperphosphatemia*** [*hyper-* excess + *phosphat(o)-* phosphate + *-emia* a blood condition of], and ***acidosis*** [*acid(o)-* acid + *-sis* condition of]. In renal failure excretion of potassium, sodium, and phosphate is impaired, resulting in their excesses. The *acidosis* that develops is from the kidneys' failure to excrete sufficient hydrogen ions. All of these electrolyte abnormalities can have significant negative impacts on the rest of the body. Remember, hyperkalemia can dramatically slow or even stop the heart.

Renal disease can also alter hormones like renin. Renal failure often results in increased levels of renin. This can actually cause high blood pressure in some patients, compounding renal damage. And, as we discussed when we talked about blood pressure, if the patient has preexisting heart disease, stimulation of the renin-angiotensin cycle could be devastating. Another hormone that may be affected is *erythropoietin*. In chronic renal failure, there tends to be insufficient erythropoietin. As we mentioned in an earlier section, this results in a nonregenerative anemia. It's nonregenerative because the bone marrow is not being stimulated to create replacement red blood cells. As anemias go, nonregenerative anemia has a very poor ***prognosis*** [Gr. *prognosis* "foreknowledge," i.e., forecasted outcome]. As you can see, there is far more to renal failure than just a urinary problem. Renal failure is a disease that has a huge, body-wide impact. When it develops suddenly, the body simply can't cope.

Acute Renal Failure

In ***acute renal failure***, something has resulted in profound damage and an abrupt decline in renal function. One common cause of acute renal failure would be some sort of vascular event like profound, prolonged *hypotension*. Hypotension would be a *prerenal* cause of renal failure. We've already talked about the importance of blood pressure. Suffice it to say that the more rapidly we restore normal blood pressure, the better we minimize damage to the kidneys. Another common (renal) cause of acute renal failure would be exposure to a ***nephrotoxin*** [*nephr(o)-* kidney + *tox(o)-* poison + *-in* a, the]. Believe it or not, grapes can be very ***nephrotoxic*** [*nephr(o)-* kidney + *tox(o)-* poisonous + *-ic* pertaining to] for dogs.

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Really?! Yep. So **never** ever feed grapes (or raisins—they're dried grapes) to dogs.

But the single most common nephrotoxin to cause acute renal failure is ***ethylene glycol***—antifreeze. A puddle of this stuff is very attractive to animals. The sweet odor and taste mask the deadly nature of antifreeze. Ingesting even a little can be deadly for anyone (including humans!); as little as a teaspoon could kill an 8-pound cat. For an average 40-pound dog, only about 2.5 ounces (~5 tablespoons) may be deadly. That's not much! Similar small volumes may kill children. Antifreeze is an indiscriminate killer. That's why even if a pet owner or a parent is the least bit suspicious that a pet or child has consumed antifreeze, emergency treatment should be sought as soon as possible. Our goal is to prevent absorption of the toxin. But we need to intervene FAST!

The ***prognosis*** for any victim of ethylene glycol toxicity (poisoning) depends on how much was consumed and how quickly medical care is initiated. You see, antifreeze is rapidly absorbed from the digestive tract and rapidly metabolized. It's the metabolites and formation of calcium oxalate *monohydrate* crystals (remember—picket fence crystals) that ultimately damage and destroy the nephrons. The damage and destruction happen very quickly. Other organs are damaged too. So, not only do the victims rapidly develop acute renal failure, they also may suffer multiple organ failures. When presented to the emergency room, if the patient has already developed ***anuria*** [*an-* without + *ur(o)-* urine + *-ia* a condition of; i.e., no urine production], there is very little hope for recovery. Those with ***oliguria*** [*oligo-* small + *ur(o)-* urine + *-ia* a condition of; i.e., still producing small amounts of urine], may have a slightly better chance of survival. Because the chances of survival are slim to none (and "Slim" has often already walked out the door), the best way to deal with ethylene glycol toxicity is by preventing it. All chemicals like antifreeze should always be stored out of reach of children and animals. All antifreeze spills should be promptly cleaned up.

Obviously there are many other things that can result in acute renal failure. Leptospirosis is one such condition. As you may recall, I said that leptospirosis could be lethal (deadly) for animals and humans. During the acute stages of ***nephritis*** [*nephr(o)-* kidney

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+ *-itis* inflammation of] with this infection, ***dialysis*** (di-al'uh-sis [Gr. *dialysis* "dissolution"]) may be necessary to keep the patient alive. I have cared for numerous *dialysis* patients, many of whom were in acute renal failure from leptospirosis. These patients were critical and required round-the-clock dialysis and intensive care. We used *continuous renal replacement therapy* (*CRRT*) as the method of dialysis. How did it work? In brief, we surgically placed a very large-gauge catheter in the jugular vein (found in the neck). The catheter is specially made for CRRT, with an inlet port and an outlet port. As the blood from the patient circulates through the dialysis machine, diffusion and filtration remove wastes and restore ionic balance to the blood. The treated blood then flows back to the patient through the inlet port. Basically, the machine does the work that the kidneys temporarily (we hope) cannot. Monitoring of these patients, their laboratory values, and urine production is intense, to say the least. It was gratifying for me to see some of those patients recover and go home. Sadly, not all of them made it.

Chronic Renal Failure

Chronic renal failure is different in that it develops progressively over time. There are many factors, conditions, and events that may progressively "chip away" at the functional nephrons. Some common causes of chronic renal failure we've already mentioned, which include *pyelonephritis*, *leptospirosis*, and *urolithiasis*. Wait. How can urolithiasis cause renal failure? Well, this is a possible *postrenal* cause. Think about it. If we obstruct the ureters or the urethra, urine backs up, right? The backpressure from that can cause significant damage to nephrons. Repeated episodes of obstruction have a snowball effect, compounding the damage each time. Urinary obstruction is just one example. Heart disease is a common prerenal cause of chronic renal failure. Whatever the cause, chronic renal failure is often insidious (sneaky). Most owners don't realize that a problem is brewing until either their veterinary health care team detects changes in laboratory values or the animal appears sick from *uremia*. Many times it's a sudden insult creating an acute on chronic complication of the animal's renal failure that makes the animal clinically ill and that prompts a veterinary visit. That's not ideal. Ideally, we'll catch changes in renal laboratory values early,

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before we have significant azotemia or uremia.

Once we are aware of mild renal insufficiency, we can and should monitor more closely. BUN, creatinine, electrolytes, and urine specific gravity are important parts of that monitoring package. If we can identify contributing factors to the renal disease, we might be able to eliminate them before there is too much irreversible renal damage. Between close monitoring and identification of contributing factors, we might be able to minimize or slow progression of the pet's renal disease. This is why routine blood work is so important. In reality, chronic renal failure is often idiopathic. That said, close monitoring of the patient (including laboratory values) permits us to deal with systemic consequences, before they get out of hand. We might not be able to cure renal failure, but we can certainly minimize symptoms. One of the most important things we can do to reduce azotemia is to reduce nitrogenous by-products in the body. How? The solution is simple, really. If nitrogenous by-products like ammonia and urea are from the metabolism of proteins, then the best thing we can do is reduce protein in the diet. That's the hallmark of a renal diet—low protein. A low-protein diet won't reverse the renal damage, but it sure will improve the quality of life for that patient. And in chronic renal failure, that's important.

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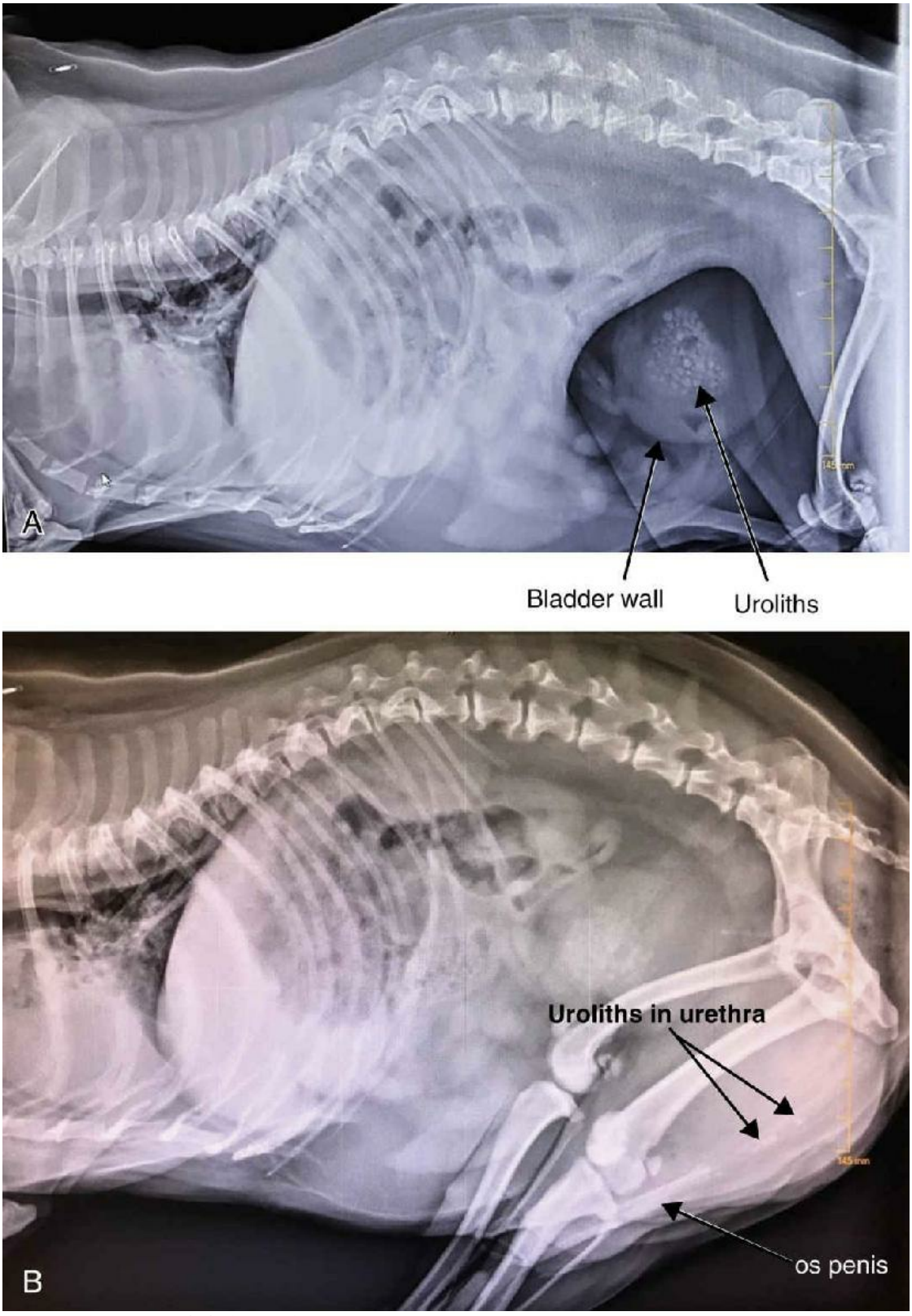

FIG. 6.10 (A) Case Study—Rt. Lateral abdominal radiograph with compression. (B) Case Study—Caudal Rt. Lateral abdominal radiograph.

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Case Study

Vader, a 13-year-old, neutered male pug was presented to our emergency room. His owner reported that he was unable to urinate. On physical examination Vader was bright and alert, though somewhat anxious. His abdomen was tense on palpation, particularly the caudal abdomen. On rectal examination, his urethra felt thickened with a focal firm object within the proximal urethra. The remainder of his physical exam was unremarkable. Radiographs revealed a full bladder with a number of uroliths present in the urinary bladder and the urethra. (See radiographs in Fig. 6.10A and B.) These findings confirmed a diagnosis of urethral obstruction due to urolithiasis.

An indwelling urinary catheter was passed, relieving the obstruction and evacuating his bladder. A urine sample was collected for urinalysis and culture. Urinalysis findings included S.G. –1.015, pH–7.7, protein–trace, glucose–negative, ketones–negative, hematuria, pyuria, bacteriuria, and crystalluria (struvite). Both preliminary and final culture results demonstrated *Staphylococcus* as the sole pathogen, with sensitivity to multiple antibiotics, including amikacin, amoxicillin/clavulanate, cefazolin, and others. This confirmed the diagnosis of bacterial cystitis.

To prevent subsequent obstructive episodes, a cystotomy was performed for urolith removal. (Subsequent urolithic analysis demonstrated exclusively struvite composition.) Immediately after recovering in the hospital from the cystotomy, Vader had persistent dysuria. A cystourethrogram was performed to determine the cause of his dysuria. There was no evidence of any uroliths. However, the cystourethrogram did reveal narrowing of the urethra near the proximal os penis. Another urinary cathether was placed and left indwelling for 24 hours. Following removal of the catheter, the next day Vader was able to urinate normally. He was discharged on antibiotics and pain relievers.

Vader continued to have pollakiuria for several days at home. He has since fully recovered. As this was a first-time event for Vader, no dietary changes were warranted at this time. It is believed that the urolithiasis was secondary to the UTI. We

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continued to monitor him, rechecking both urinalysis and cultures approximately 1 week after he had finished the antibiotics. After that, it was recommended that he be reevaluated every 6 months. The owner was told that if he observed pollakiuria, dysuria, decreased appetite, or lethargy, Vader should be seen immediately.

Case Study Questions

1. Vader suffered from a condition of urinary stones, which is medically termed _____.
2. On urinalysis, Vader had _____or bloody urine.
3. On urinalysis, Vader had _____or pus (WBCs) in his urine.
4. On both urinalysis and culture, Vader had _____or bacteria in his urine, specifically *Staphylococcus sp.*
5. Vader had _____or inflammation of the bladder for two reasons—an infection and urinary stones.
6. The medical term for a urinary stone is _____.
7. Vader's urinary stones were removed from his bladder via a(n) _____.
8. The medical term for difficult urination is _____.
9. The medical term for frequent urination is _____.
10. A _____ is a radiographic procedure in which a contrast dye is infused into the bladder and urethra to enhance visualization of abnormalities, if present.

The Answer Key to these case study questions may be found in Appendix B.

The Answer Key to these case study questions may be found in

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Appendix B.

[a] Latimer, Kenneth, Duncan & Prasse's Veterinary Laboratory Medicine: Clinical Pathology, 5th Ed., Wiley-Blackwell, 2011, 256

[b] Ibid, 256

[c] Ibid, 257

[d] Ibid, 257

[e] Nelson and Couto, Small Animal Internal Medicine, 2nd Ed., Mosby, 1998, 613

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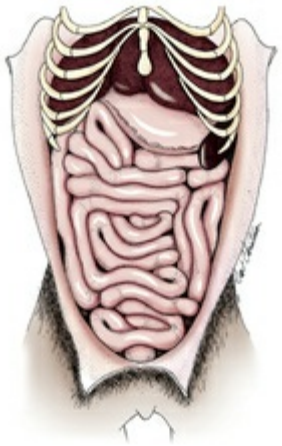
Applied Digestive Terminology



Basic Common Ground,
- Basic Nutrients,
  - Water,
  - Carbohydrates,
  - Lipids,
  - Proteins,
  - Vitamins,
  - Minerals,
- Carnivorous, Omnivorous, and Herbivorous Diets,
  - Carnivores,
  - Omnivores,
  - Herbivores,
- Structural Commonalities,
  - Abdomen,
  - Mesentery and Omentum,
  - Liver,

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Abomasum,
Small Intestine,
Cecum, Colon, and Rectum,
Scours,
Case Study,

GOALS AND OBJECTIVES

By the conclusion of this chapter, the student will be able to:

1. Recognize common root words, prefixes, and suffixes related to the digestive system.
2. Divide simple and compound words into their respective parts.
3. Recognize, correctly pronounce, and appropriately use common medical terms related to the digestive system.
4. Demonstrate a basic understanding of digestive anatomy, as it relates to domestic animals.
5. Demonstrate familiarity with comparative tooth structure, dentition, and dental formulas for domestic animals.
6. Demonstrate a basic understanding of digestive physiology, as it relates to domestic animals.
7. Demonstrate a basic understanding of nutrients and nutrition.
8. Demonstrate familiarity with common diseases of the digestive tracts of domestic animals.

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Basic Common Ground

It seems to be human nature to gravitate to differences. And certainly, where digestive anatomy and function are concerned, there are tremendous differences among domestic animals. Still, despite differences between humans and animals and among various species of animals, we actually share much in common. No matter who we are, we all share the same basic needs for shelter, water, and food. We all drink, eat, digest, and utilize nutrients and then eliminate wastes in the form of urine and feces.

Basic Nutrients

Our goal here is not to make nutritionists out of you. Our goal is to simply give you a foundation on which to build. As we talk about the digestive tracts and digestion of domestic animals, we'll refer back to some of this information as it becomes relevant. Nutrients are classified into six basic categories: water, carbohydrates, proteins, lipids, vitamins, and minerals. Everyone needs them in just the right balance for each life stage, circumstance, and species. Let's take a quick look at the basic importance of each one.

Water

Water is vital for life. Approximately 70% of the body, anyone's body, is water. Animals and humans may be able to survive for long periods without food, but we cannot survive for long without water. Even if water is available, when water losses from the body (through sweating, vomiting, or diarrhea, etc.) exceed water intake, the individual will still dehydrate. And dehydration can be deadly. Sources of water may include fresh drinking water as well as consumed plants and/or animal tissues.

Carbohydrates

Carbohydrates are the most easily digested fuel sources for energy. They are compounds made of simple and complex sugar molecules. Many types of food are rich in carbohydrates, including fruits,

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grains, and vegetables, such as root vegetables. You're familiar with potatoes, right? Potatoes are pure starch, i.e., pure carbohydrate. The bread that you may eat is made from flour, which is prepared from some sort of grains—again, pure carbohydrate. Rice is a grain and pure carbohydrate. That means it's easily digested and broken down into simple sugars and absorbed for use by cells of the body. But when I think of grains fed to livestock, I think of grains, such as corn and oats. We often refer to these as "concentrates". Why concentrates? Because each tiny little grain of corn or oats is a concentrated package of carbohydrates and fats. And that means they're loaded with fuel to burn! For carnivores, like the cat, they will gain a fair number of carbohydrates from the abdominal contents of the prey that they eat. They'll also gain some from the muscle tissue itself. As you may recall from Chapter 4, ***glycogen*** [*glyc(o)-* sugar + *gen(o)-* producer] is the storage form of glucose (sugar) in muscle tissue.

Lipids

Lipids (fats) are the most concentrated form of fuel for the body. Ounce for ounce, lipids contain the most calories, even compared to carbohydrates. True, they both provide great sources of energy. But lipids are the premium, high-octane fuel of the nutrient world. They also provide a sustained burn. You see, their molecular structure is more complex than carbohydrates. So, digesting and using them is a progressive process. To compare lipids to carbohydrates in terms of energy, carbohydrates burn quickly like a piece of paper. Lipids, on the other hand, would be more like throwing a large log on the fire. Dietary sources of lipids may be found in grains, some plant matter, and animal tissues. Beyond fuel, lipids are also important structurally for the body. Both lipids and proteins are important components of many cellular membranes.

Proteins

Proteins can also be used as fuel for energy. Cats are the only animals to use proteins for energy efficiently. Comparatively, the digestive process to break down a large protein molecule into usable amino acids is long and complicated. As you'll learn later, there are numerous enzymes and other digestive processes needed

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to digest proteins. Of course, *amino acids* derived from proteins are incredibly important as building blocks for cells, tissues, and organs. So, even if protein isn't used as much for fuel (that depends on the animal), it is essential for body growth and repair. Sources of quality protein may include meat products, dairy products, and some plants, especially legumes. Legumes are types of plants that tend to enrich and replenish soil with needed nitrogen. Alfalfa hay is a legume, commonly fed to horses. Soybeans are another common legume used in foods for both animals and humans. In fact, many beans are rich sources of protein. Nuts provide sources of both protein and lipids. That's good news for squirrels!

Vitamins

Vitamins are compounds found naturally in many different dietary sources. They are required for many of the normal metabolic processes of the body. Vitamins are classified as either water soluble or lipid soluble. Water-soluble vitamins include vitamin C and B vitamins. Any excess intake of water-soluble vitamins will be lost in urine and feces. Lipid-soluble vitamins include vitamins A, D, E, and K. Lipid-soluble vitamins are stored in the body, primarily in the liver. So, oversupplementation with lipid-soluble vitamins can result in ***toxicity*** [*tox(o)-* poison + *-city* state of].

Vitamin C

Vitamin C is found naturally in many fruits and other plants. It is important for numerous metabolic processes. It is also very important for the production and maintenance of connective tissues. Most animals do not require vitamin C in their diets, because they actually synthesize their own by converting a precursor to ***ascorbic acid*** (vitamin C). Humans and guinea pigs, however, do **not** produce their own vitamin C. Because vitamin C is unstable and is easily destroyed by oxidation and exposure to heat and light, fresh dietary sources are important. Yes, although that bag of guinea pig food is "fortified" with vitamin C, the vitamin C in it may no longer be viable, especially the longer the bag is open. So, in addition to the guinea pig pellets, fresh sources of vitamin C should be fed daily. Those vitamin C–rich sources may include citrus fruits, kiwi, strawberries, cabbage, tomatoes, potatoes (white

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and sweet), broccoli, brussels sprouts, cauliflower, parsley, green and red pepper, winter squash, spinach, turnip greens, and kale. Lettuce is a very poor source.

Scurvy is a deficiency of vitamin C. Because connective tissues rely on vitamin C, they will be most adversely affected by deficiency of it. So, vascular walls become weak, most often resulting in bruising in the skin and ***subcutis*** [*sub-* beneath + *cutis* skin]. Of course, cartilage and connective tissues of joints are adversely affected, resulting in joint pain. Connective tissues that are found throughout muscles are also adversely affected. Between muscle and joint soreness, it is very difficult for these animals to walk. These animals are very lethargic and may experience extreme weight loss. Without vitamin C supplementation, they will die.

B Vitamins

That's right, B vitamins—plural. You see, there's actually eight vitamins that make up B complex. They include B1 (thiamine), B2 (riboflavin), B3 (niacin), B5 (pantothenic acid), B6, B7 (biotin), B12, and folic acid. Most of the time, we think of them as a complex. In general, B vitamins are important for metabolism, immunity, and skin and coat health. Still, each one is important in its own right. For example, B12 is important for the production of red blood cells, and B1 is important for brain health. So, if we have a generalized deficiency of B vitamins, we'll probably be lethargic and may have reduced immunity. Specific deficiencies may create very specific and serious problems.

For example, B1 (thiamine) is found in various foods. It is also produced through fermentation. If a ruminant, like a sheep or a goat, were to suddenly eat a large amount of grain, the microbes in the rumen will be dramatically altered. Good guys that produce the thiamine die, and bad guys that produce ***thiaminase*** [*thiamin* + *-ase* an enzyme for] flourish. This results in a profound deficiency of thiamin that is important for brain health. (Did you notice that? Thiamin may be spelled with or without an "e" at the end.) The end result of this B1 deficiency is ***polioencephalomalacia*** (po"le-o-en-sef"uh-lo-muh-la'shuh [*poli(o)-* gray + *encephal(o)-* brain + *-malacia* softening of; i.e., softening of the gray matter of the brain]). These animals become weak, depressed, and blind. If untreated or treated

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too late, they will die. The moral of the story is to prevent sudden dietary changes. A friend of mine lost a llama to this recently, despite their rapid response and intensive care provided in the veterinary hospital. I point this out not to highlight the importance of this particular vitamin. I do it to demonstrate the importance of vitamins and balanced diets.

For those who do not produce their own B vitamins, what are good sources of B vitamins? Whole grains, vegetables and other plants, dairy products, eggs, meats, and animal tissues. For those who do produce B vitamins from fermentation (ruminants and hindgut fermenters), they gain some B vitamins from the plants they eat and the rest from their fermentation processes. That said, animals like rabbits (hindgut fermenter) recoup much of the B vitamins produced by their ***coprophagic*** [*copr(o)-* feces + *phag(o)-* eating + *-ic* pertaining to] habits. The first, soft fecal pellets passed are loaded with B vitamins and are important to "recycle" through ***coprophagia*** [*copr(o)-* feces + *phag(o)-* eating + *-ia* process of]. Some horses engage in this behavior too.

Vitamin A

Vitamin A is important for growth, immunity, and vision. Many foods are rich in vitamin A precursors, such as beta-carotene. That's right, compounds like beta-carotene give vegetables their bright yellow and orange colors. So, Bugs Bunny must have great eye sight from eating all those carrots! You see, Bugs and most other animals that eat plants like that actually convert carotenes into vitamin A. Cats lack this ability. So, cats need to consume vitamin A directly from their food such as fish and liver. Insufficient vitamin A can lead to blindness.

By the way, I knew of a cat owner who decided to feed canned tuna to her cat exclusively. First, exclusive diets like that are never healthy. Second, canned tuna (unless it's Skipjack tuna) can contain higher amounts of potentially toxic substances like mercury. Finally, this diet created an overdose of vitamin A. Remember, vitamin A is a fat-soluble vitamin. Any excess is stored in the body. And in the case of ***hypervitaminosis*** [*hyper-* excess + *vitamin* + *-osis* condition of] ***A***, an animal or a human can become very ill. This poor cat had a poor hair coat with ***alopecia*** (abnormal hair loss),

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flaky peeling skin, profound nausea, and dizziness (observed as wide stance and stumbling gait). It was the nausea and loss of appetite that prompted the owner to bring the cat in. Fortunately, our physical examination and history prompted us to ask what the cat was being fed. After educating the owner about an appropriate balanced diet, the cat recovered after several weeks of being fed a commercial cat food.

Vitamin D

Vitamin D is another fat-soluble vitamin. So, it is stored in the body, especially the liver. For most of us (animals and humans), vitamin D is produced by the skin. Well, truth be told, the precursor for the vitamin is stored in the skin and is converted to vitamin D on exposure to ultraviolet (UV) light. Vitamin D is very important for strong bones by promoting the absorption of calcium and phosphorus and mineralization of bones. While dietary intake of vitamin D is generally not necessary in animals, a balanced diet is essential for the body to produce vitamin D precursors.

Rickets is a bone disease that can result from vitamin D deficiency. It most often occurs in young, growing animals fed exclusive all-meat diets. Bones are poorly developed and poorly mineralized. Growth is stunted. Bones are weak, brittle, and painful. Angular limb deformities are common. Provided there are no complicating factors like fractures, the prognosis for an animal with rickets is good once it is fed a balanced diet. By the way, the most frequent animals I have seen through the years with rickets are reptiles, like iguanas. Many of them experienced ***pathologic*** [*path(o)-* disease + *log(o)-* knowledge, study + *-ic* pertaining to] fractures because of it. Yes, there are supplements containing vitamin D that can be added to their diets. However, it is very important to provide sufficient UV light exposure. In the wild, these animals frequently bask in the sun, providing an abundance of vitamin D synthesis.

Vitamin E

Vitamin E is another fat-soluble vitamin that is stored primarily in muscle and fat. This vitamin is often called an antioxidant (*anti-* against), because it binds quickly to oxygen-free radicals to prevent

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oxidation and damage to tissues and other substances. Vitamin A and vitamin C are highly susceptible to oxidation. So, vitamin E helps prevent their destruction. Sources of vitamin E are grains, fruits, nuts, vegetables, meats, and animal fat.

Vitamin K

We talked about vitamin K in Chapter 3, when we discussed *coagulation* (clotting). If needed, please review that information inChapter 3. There are numerous places along the whole clotting cascade where vitamin K is necessary. Unfortunately, unlike most of the other fat-soluble vitamins, storage of vitamin K in the liver is limited. So, regular dietary intake of vitamin K is important. Failure to consume enough vitamin K could result in significant, even lethal bleeding. Sources of vitamin K include leafy, green plants, soy, and eggs (yolk). Human foods rich in vitamin K are spinach, kale, broccoli, cauliflower, and brussels sprouts.

Minerals

Minerals are inorganic (i.e., non-carbon-containing) elements, including iron, calcium, and phosphorus. InChapter 3, we talked about the importance of iron in red blood cells, contributing to their oxygen-carrying capacity. Calcium is one of those minerals that seem to keep showing up in many important ways. Along with vitamin K, calcium is very important at key places in the clotting cascade. InChapter 4, we learned of its importance for muscle contraction and bone development. InChapter 11, we'll learn of calcium's importance in nerve transmission. Unfortunately, calcium is not absorbed from the diet as readily as phosphorus. The body needs just the right ratio of calcium to phosphorus, for many reasons. Suffice it to say that there are numerous minerals and trace minerals that are needed for ***homeostasis*** [*home(o)-* unchanged + *-stasis* a state of standing; i.e., equilibrium; balanced stable state of the body]. And while we share the importance of many of those minerals across animal species, there are some species variations. For instance, selenium is a trace mineral needed by domestic livestock for muscle integrity. But there are geographic regions where selenium is not present in the soil. Michigan, for example, is a selenium-deficient state. That means the plants grown there will

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not contain selenium. Livestock there need selenium supplementation, usually through trace mineral salts.

You see, there's more to nutrition than meets the eye. Diets need to be appropriate for the type of animal. Diets need to be balanced. Sometimes, that balance needs to be adjusted for individual needs and circumstances. Nutrition and its importance for the overall health of the body should never be taken lightly.

Carnivorous, Omnivorous, and Herbivorous Diets

Carnivores

A ***carnivorous*** [*carnivor(o)-* flesh eating + *-ous* pertaining to] diet is not just about feeding meat. We've already demonstrated through our discussion of nutrients that various tissues of the body contain various levels of certain nutrients. Meat (muscle tissue) is only one piece to the puzzle when feeding a carnivore. And that begs the question: who is a carnivore? Believe it or not, cats are the only true carnivores. They gain most of their fuel for energy from protein. So, a feline diet should have a high percentage of protein in its formulation—high, yes, but not 100%. Let's think about that for a moment. What do cats eat when they catch and kill their prey? Do they eat just the muscle? No. Whether it's a house cat catching a mouse or a lion catching a wildebeest, those cats eat many tissues. In fact, the house cat probably eats the whole mouse. That's not possible of course for the lion. But the lion will consume muscle, connective tissues, skin, organs like the liver, portions of the digestive tract, and blood, and will even gnaw on the bones. It's balanced and complete. Suffice it to say that if a cat owner tried to feed nothing but cleaned, boneless, skinless chicken breasts to the cat, that cat would develop significant nutritional deficiencies. Early in my career, researchers discovered the link between the ***essential amino acid*** taurine and ***cardiomyopathy*** [*cardi(o)-* heart + *my(o)-* muscle + *-pathy* a disease of] in cats. From that point forward, pet food manufacturers began adding taurine to their cat food products, and the incidence of cardiomyopathy declined.

Omnivores

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An ***omnivorous*** [*omnivor(o)-* all eating + *-ous* pertaining to] diet opens doors to all sorts of possibilities. Omnivores literally eat a variety of foods. Dogs and pigs (and humans) are omnivores. Among wild animals, numerous creatures are omnivorous, including bears, opossums, skunks, sloths, raccoons, squirrels, chipmunks, mice, and rats to name a few. Being an omnivore permits dietary flexibility, especially in the wild, depending on available resources. Take a grizzly bear for example. This bear will happily consume berries and other fruits when in season. Nuts, seeds, and insects could be on the menu some days. And when salmon are running, a fishing trip at the river will provide an excellent dinner. The thing with omnivorous diets is balanced variety. That balance provides just the right amounts of essential carbohydrates, proteins, fats, vitamins, and minerals.

Why belabor this? Because I've seen many number of dog owners who perceive their dogs to be carnivores and feed them exclusive raw beef or chicken (just the meat). Putting the potential for bacterial infections with *E. coli* or *Salmonella* aside for a moment, let's consider the nutritional impact of that. Dogs (omnivores) should gain nutrients from a variety of sources. They cannot use proteins for energy like cats can. They need carbohydrates and fats too. Can dogs be predators? Yes. My Basset Hound, to my dismay, became quite the predator. She developed an insatiable taste for rabbits. Most of the time Sadie was a lackluster, lazy beast. But put a rabbit in her sights, and she became the fastest, fiercest beast on short legs. Even when she was old and arthritic, she'd dart after a rabbit—successfully, I might add. And here's the thing about her eating habits; once the rabbit was caught and killed—she always ate the abdominal contents first. This is typical for dogs who prey on rabbits. It is so predictable that there are parasites like *Taenia pisiformis*, a tapeworm of dogs, which actually places its infective stage in the abdomen of rabbits. Let's consider the nutritional value of the rabbit's abdominal contents for a moment. There's plant matter and fermentation by-products, such as fatty acids and B vitamins. There's protein and fat in the tissues. Think of the wonderful concentrations of vitamins and minerals in the blood and liver of that rabbit. When Sadie (and other dogs) consumed the abdominal contents of rabbits, she ate a relatively balanced meal.

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On the occasion that she swallowed a bunny whole, she got a really complete meal. The only part of that bunny she didn't digest was the fur. (Yes, I checked to make sure that I didn't need to be concerned about an intestinal obstruction.)

I guess even dogs know that they're not pure carnivores. They're omnivores, whose digestive tracts are designed to digest and absorb nutrients from a variety of plant and animal sources. Pet food manufacturers know this. Reputable pet food manufacturers not only create balanced formulations from plant and animal sources, but also make sure that the formulations are appropriate for particular life stages. Puppies require much more energy from carbohydrates and fats as well as more protein for growth. I remember a ***neurology*** [*neur(o)-* nerves + *-logy* study of] patient. It was a mixed-breed dog who was 13 years old. The owner was concerned because of the seizure activity the dog was developing. We ran every test imaginable on that patient. Yet, the diagnosis and prognosis were completely dependent on one simple question: "What are you feeding the dog?" The owner was feeding a puppy diet to the dog. The high concentration of nutrients, especially of proteins, was too much for a ***geriatric*** [*ger(o)-* old age + *-atric* pertaining to] animal, whose liver could not cope with the excess ammonia and other by-products of protein digestion. The dog didn't need medication. It needed an appropriate diet. After placing the dog on a *geriatric* formulation, all of its seizure activity stopped. A balanced diet, formulated from a variety of sources, which is appropriate for not only the species but also the individual animal, is important. Fad diets often become popular among people. But they are generally not nutritionally sound. Grain-free diets have become popular in recent years. And we are discovering nutritional deficiencies and serious health problems, such as heart disease, developing from them. Sometimes, we need to remind owners that dogs are omnivores.

Herbivores

Now you might think that ***herbivorous*** [*herbivor(o)-* plant eating + *-ous* pertaining to] diets are easy, right? Just feed plants, and the animal will be fine. Not so fast. Not all plants are alike, and not all herbivores are alike. Hindgut fermenters, like horses, rabbits, and

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guinea pigs, need some high-quality forages (i.e., plant-based food, often gathered through grazing). Clover and alfalfa hay contain much higher levels of proteins and other nutrients compared to grasses, like Timothy hay. Given their digestive process, higher nutritional value to their forages is important. Their fermentation process takes place near the caudal end of the digestive tract. So, many of the fatty acids and B vitamins produced by fermentation may be lost in the feces. (Of course, those nutritional by-products make horse manure best for the garden.) *Coprophagia* can help recoup some of those by-products, but not all animals engage in that behavior. And for those living in temperate regions, concentrates are needed to sustain their energy needs through the cold winter months.

Ruminants (cattle, sheep, and goats) can glean more nutrients from forages and roughages of lesser quality. Why? Their fermentation processes of those roughages happen in the foregut, prior to those portions of the digestive tract where most nutrients are absorbed. Honestly, the only creature with a more efficient digestive process is the termite. Termites can actually digest all of the woody portions of plants, where most herbivores, including ruminants, cannot. But the efficiency of the ruminant digestive tract is otherwise unsurpassed. Are there times when even ruminants need a boost to their fuel resources for energy? You bet! Think of a cow, doe, or ewe during pregnancy or ***lactation*** [*lact(o)-* milk, milking + *-tion* state of]. They need way more fuel and building blocks for their developing young. So, moderate additions of concentrate and/or partially fermented feeds, such as silage or haylage, can provide just the nutritional boost of fuels they need. Still, these types of dietary needs and changes to meet those needs must be done gradually. In fact, abrupt dietary changes are not tolerated well by most animals.

So, it is a very long and complicated way of saying that all animals need basic nutrients. They each may have preferred foods that match their digestive processes, but the basic nutrients required by them are very much the same. Their diets must be balanced with regard to those basic nutrients. Excesses or deficiencies can lead to serious health problems. And consistency is important. Abrupt dietary changes can "upset the apple cart" with

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their digestive tracts. So, despite marked anatomic differences, animals have much in common. In fact, even portions of their anatomy have common ground.

Structural Commonalities

Abdomen

Every single animal has an abdomen with ***abdominal*** [*abdomen(o)-* abdomen + *-al* pertaining to] ***viscera*** [L. organs]. The abdomen or ***peritoneal*** [*periton(o)-, peritone(o)-* peritoneum + *-al* pertaining to] ***cavity*** of every animal is lined with the same connective tissue (***peritoneum*** [from Gr. *peritonaion* to stretch around]). And we name that peritoneal lining depending on its location. ***Parietal*** [*pariet(o)-* the wall + *-al* pertaining to] ***peritoneum*** lines the actual abdominal wall. ***Visceral*** [*viscer(o)-* organ + *-al* pertaining to] ***peritoneum*** covers the organs.

There are times when any animal for a variety of reasons could develop excess abdominal fluid. We need to know the nature of that fluid to help us diagnose and treat the underlying problem. We need to know the protein, water, and cellular makeup of the fluid. Is there blood, pus, or bacteria present? If it's whole blood, we know we're dealing with *hemorrhage* (severe bleeding). If there are bacteria with large numbers of white blood cells, we'll know that we're dealing with a ***septic*** [*sept(o)-* bacteria + *-ic* pertaining to] ***peritonitis*** [*periton(o)-* peritoneum + *-itis* inflammation of]. By the way, it is possible for an ***aseptic*** [*a-* without + *sept(o)-* bacteria + *-ic* pertaining to] ***peritonitis*** to develop from leakage of things like bile and urine that are very irritating to the peritoneum. Through analysis of the abdominal fluid, our findings point us to different conclusions. But to analyze the fluid, we need to collect it. This requires ***abdominocentesis*** [*abdomin(o)-* abdomen + *-centesis* puncture of]. It is a minor procedure but extremely valuable diagnostically.

If surgery is deemed necessary on the basis of history and diagnostic evidence, a ***laparotomy*** [*lapar(o)-* abdomen+ *-tomy* to cut] is performed. This means that we are entering the abdomen through a surgical incision. In dogs and cats, that incision is most often made on the ventral midline of the abdomen. And for most

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surgical procedures on the digestive tract, it usually involves a very long incision from the xiphoid to the pubis. (Refer to Chapter 4 to review skeletal anatomy.) The large abdominal incision provides the best access and visualization of the ***gastrointestinal*** [*gastr(o)-* stomach + *intestin(o)-* intestine + *-al* pertaining to] (***GI***) tract. Of course, a *laparotomy* is not used exclusively for digestive surgical procedures. Surgery for any of the abdominal viscera may necessitate a laparotomy. By the way, laparotomies in cattle frequently use a flank incision, with the animal standing. Yes, they're awake. We simply use a regional block to desensitize the flank.

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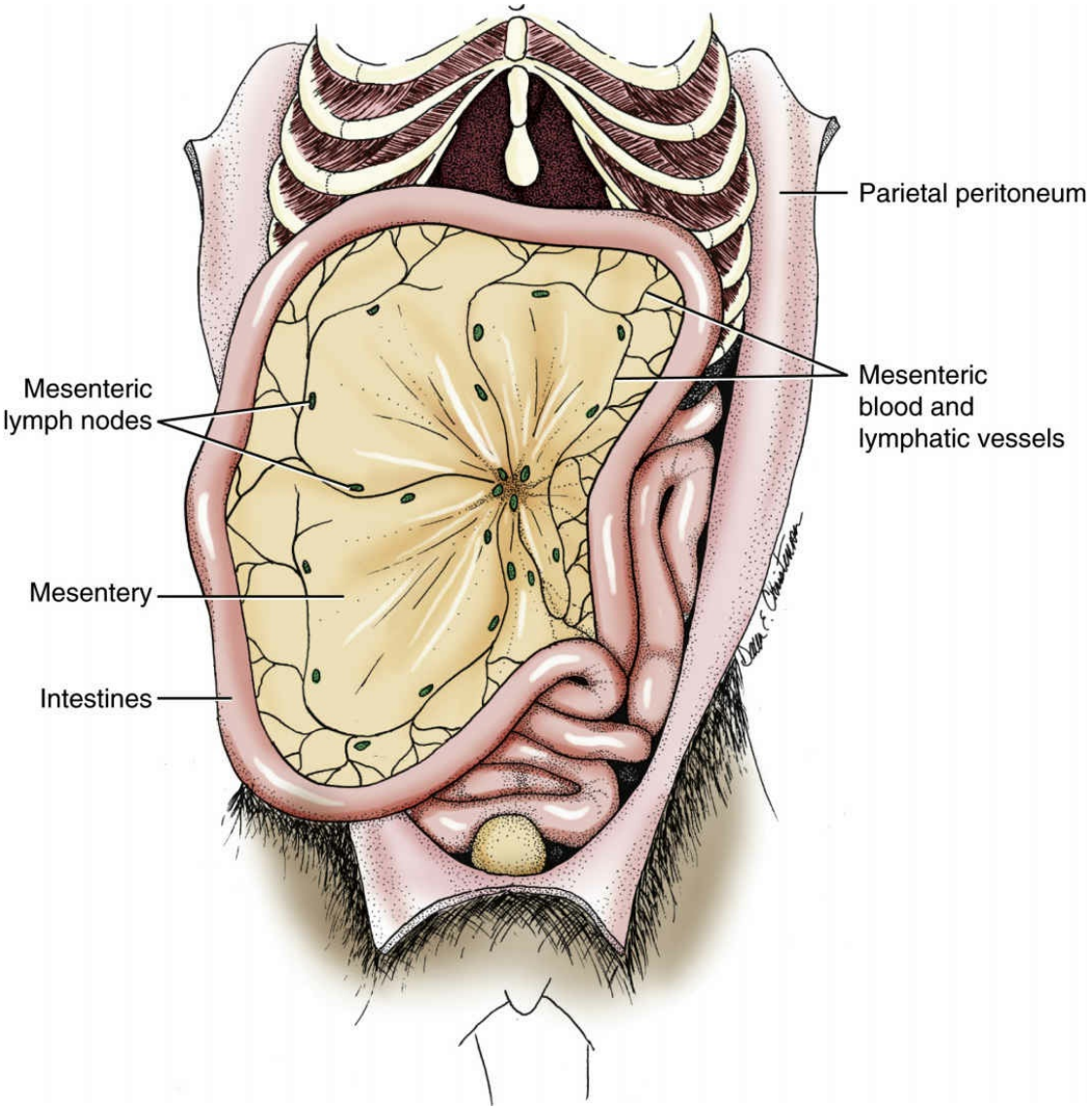

FIG. 7.1 Mesentery in the canine abdomen.

Sometimes, ***laparoscopy*** [*lapar(o)-* abdomen + *-scopy* viewing] is used. ***Laparoscopic*** [*lapar(o)-* abdomen + *scop(o)-* to view + *-ic* pertaining to] ***surgery*** involves the use of a ***laparoscope*** [*lapar(o)-* + *-scope* viewer]. This instrument requires only tiny surgical incisions compared to most laparotomies. The laparoscope has a fiberoptic light source and camera that allow for clear visualization of structures. Laparoscopes also have a number of accessory instruments, giving surgeons the ability to take tissue biopsies, cauterize vessels, apply vascular clamps, and much more. The most frequent laparoscopic procedure I've seen, with regard to any portion of the digestive tract, has been for liver biopsies. But there are many other applications for laparoscopy for other body systems. The biggest advantage of laparoscopic surgery over a

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laparotomy is the quick recovery time for the patient. It really speeds the recovery along when you're not opened up from stem to stern.

Mesentery and Omentum

Every animal has ***mesentery*** and ***omentum***. The ***mesenteric*** [*mesenter(o)-* mesentery + *-ic* pertaining to] tissue is the sheet of connective tissue that supports and suspends all of the vessels, lymph nodes, and digestive tract within the abdomen (Fig. 7.1). The *mesentery* is really just a fold of *peritoneum* that connects and secures structures like the intestines to the abdominal wall.

The ***omentum*** [L. "fat skin"] is a much more delicate tissue than the mesentery, although it is still a fold of peritoneal tissue. But comparatively, *omentum* is made up of loose connective tissue and adipose (fat). As you can see in Fig. 7.2, because of the loose woven nature of the connective tissue and fat, *omentum* has kind of a lacy appearance. It's not as fragile as it looks. And it has key attachment points. In ***monogastric*** [*mono-* one + *gastr(o)-* stomach + *-ic* pertaining to] animals, it attaches along the caudal border of the stomach. In ruminants, omentum is attached to the rumen and abomasum. In everyone, omentum also attaches to the spleen. For the rest, omentum simply loosely drapes over the rest of the abdominal contents. So, what is its purpose? Well, despite being made of loose connective tissue, it does help secure things in place. More importantly, the omentum has been shown to play a role in immunity. Not only does it secrete inflammatory mediators, but it also serves to wall off areas of infection in the abdomen. Small wounds are actually isolated, limiting the spread of infection in the abdomen. So, while the omentum may look like nothing more than a place to deposit lots of abdominal fat, it actually has some pretty important duties.

Liver

The liver is a multipurpose organ. We learned in previous chapters that it is responsible for producing many of the proteins used throughout the body. InChapter 3, we discussed plasma proteins produced by the liver, especially those important as clotting factors. In Chapter 12, we'll talk about the liver's importance for the

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metabolism, ***biotransformation*** [*bi(o)-* life, living + *transformation* changing], and excretion of drugs. In a general sense, we can think of the liver as the "furnace" for the body. Many metabolic processes occur in the liver, and those processes produce heat. So, the liver makes major contributions to the maintenance of core body temperature. InChapter 10, we'll talk about how the liver can actually produce glucose from scratch (***gluconeogenesis*** [*gluc(o)-* sugar + *ne(o)-* newly + *gen(o)-* produce + *-sis* process of]). This remarkable organ does many, many things. Its importance cannot be overlooked or underrated. In fact, if we were to name two things that are most important in the body, we would probably cite water and the liver. We cannot survive without either one, and each of them impacts virtually every facet of body activity.

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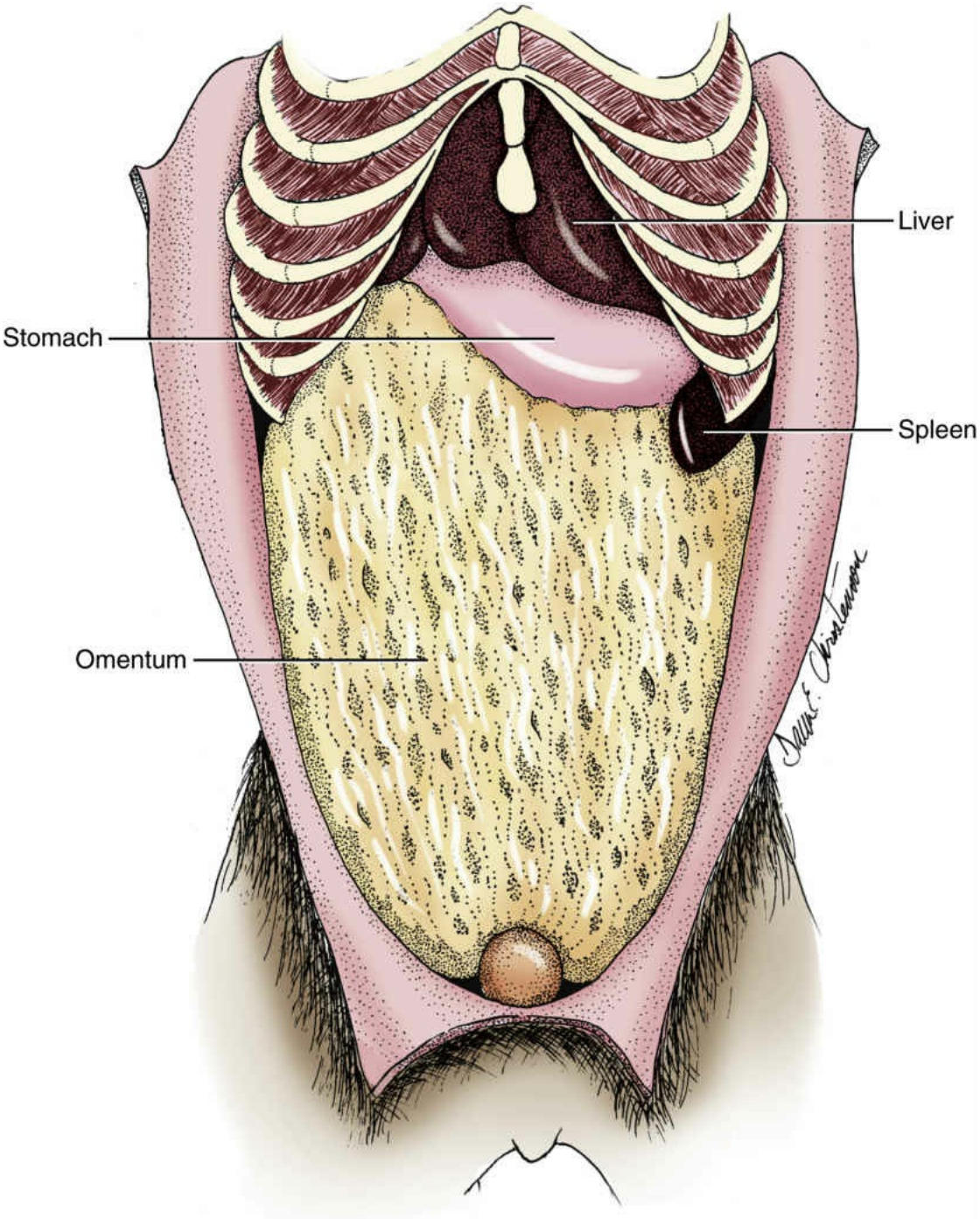

FIG. 7.2 Omentum in the canine abdomen.

Of course, our focus in this chapter is the liver's relationship to the digestive tract. It really is an accessory digestive organ. It is found in the cranial abdomen, as shown in the dog's abdomen in Fig. 7.2. For everyone, the liver is tucked closely against the diaphragm. And for the most part, the caudal margins of the liver should be protected by the caudal ribs. If it extends beyond the caudal ribs, ***hepatomegaly*** [*hepat(o)-* liver + *-megaly* enlargement of]

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probably exists. Of course, this is most easily discovered in small animals, such as dogs and cats, when palpating the abdomen. The liver has multiple, distinct lobes. This is valuable, if a given lobe is damaged. If it can't be repaired, it can be removed. The only times in my career that I've seen a liver ***lobectomy*** [*lob(o)-* lobe + *-ectomy* to cut out, remove] were in cases of ***hepatic*** [*hepat(o)-* liver + *-ic* pertaining to] tumors and severe trauma that resulted in significant bleeding. The only way to stop the life-threatening *hemorrhage* (bleeding) was to remove the bleeding lobe surgically. That said, the liver is the one internal organ of the body that has tremendous capacity to repair and regenerate itself when damaged.

Hepatoportal Circulation

The liver is a highly ***vascular*** [*vascul(o)-* vessel + *-ar* pertaining to] organ. And its ***vascularity*** [*vascul(o)-* vessel + *-arity* state of] plays a large role in *hepatic* function. You see, all of *mesenteric* blood from the digestive tract goes to the liver. This is advantageous for the liver—it has first dibs on all of the nutrients in the mesenteric blood. So, all of the mesenteric blood (from the digestive tract, spleen, and pancreas) enters the ***portal vein***, sometimes called the ***hepatoportal*** [*hepat(o)-* liver + *port(o)-* entrance + *-al* pertaining to] vein. Think of this as the "port of entry" into the liver. The blood is dispersed by branches of the portal vein throughout the liver lobes. Eventually the blood enters a capillary-like network of ***sinusoids*** [L. *sinus* a hollow + *-oid* resembling]. (Please refer to Chapter 5, if you need to review basic blood flow and vessels.) Of course the liver also receives fresh oxygenated blood through the ***hepatic artery***. All of the blood flows from the liver through the ***hepatic vein*** into the ***caudal vena cava*** and general circulation. But before it gets into general circulation, the liver has to do a lot of processing.

Hepatic sinusoids contain ***phagocytic*** [*phag(o)-* eater, eating + *cyt(o)-* cell + *-ic* pertaining to] cells. These ***macrophages*** [*macr(o)-* large + *phag(o)-* eater] are important to remove bacteria from the blood. Somehow bacteria always manage to move from the digestive tract into mesenteric blood. So, the liver's defensive role is very important. Just think of the profound infection that could develop anywhere in the body or throughout the body, if these *macrophages* didn't do their jobs. That would be devastating! If you

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want to review macrophages and all they do, please refer toChapter 3.

The *hepatic sinusoids* are lined by ***hepatocytes*** [*hepat(o)-* liver + *cyt(o)-* cell]. This is where the rubber really meets the road. First and foremost, *hepatocytes* have the first access to all of the wonderful nutrients in the blood. Through active transport and diffusion, they take the nutrients in. This gives them the opportunity to use, change, repackage, and store them as needed. For any toxins that may be in the portal blood, hepatocytes detoxify (i.e., remove toxins) the blood. It's the hepatocytes that metabolize, biotransform, and excrete many pharmaceutical (drug) compounds. And this is true, whether the drug was given by mouth or by injection. Either way, that blood goes through the liver, and the hepatocytes act on it. Ultimately blood leaving the liver should be clean and detoxified, with only usable nutrients in their simplest form (e.g., simple sugars and amino acids) for use elsewhere in the body.

Bile, Bile Salts, and Bile Acids

Hepatocytes are also responsible for the production of bile and bile salts. Bile and bile salts are necessary to emulsify fats for digestion. If you remember fromChapter 3, old and damaged red blood cells are removed from circulation by macrophages (primarily in the spleen, but also in the liver). A principal by-product of ***hemolysis*** [*hem(o)-* blood *-lysis* the process of breaking] is ***bilirubin***, a yellowish pigmented compound. It's the pigments of bilirubin that give bile its yellowish color. Hepatocytes are responsible for removing bilirubin from the blood. Most of it is combined with cholesterol to produce ***bile*** and ***bile salts***. ***Bile acids*** are a precursor compound of bile. In times of disease, bile acids may become elevated, if the liver has a reduced ability to use them in the production of bile.

The bile and bile salts are secreted into the ***bile ducts***. The ***biliary*** [*bili(o)-* bile + *-ary* pertaining to] ***tree*** (Fig. 7.3), created by all of the bile ducts, is very much like a tree. In a tree, the most peripheral branches are quite small and are supported by larger branches. The largest branches stem from the main trunk. The same is true for all of the bile ducts in the *biliary tree*. The most peripheral ducts are small. Progressively the bile ducts become larger and larger until

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they finally converge on the ***common bile duct*** (tree trunk). The common bile duct communicates with the first segment of the small intestine (the duodenum). The bile entering here, at the proximal end of the small intestine, permits better emulsification of fats. This is important to facilitate better digestion of dietary fats. Emulsification is really nothing more than taking a large oil slick and turning it into smaller, manageable globs of fat. In that respect, bile functions like a detergent. Like Dawn dish soap[a], it "cuts through grease", as they say in the ad. That creates more surface area for digestive enzymes to actually break down the lipids into the molecules that can be absorbed. The Evolve animation *Bile Ducts and Pancreatic Ducts* provides a nice overview of the biliary tree, gallbladder, and delivery of bile to the duodenum.

Gallbladder

What about the ***gallbladder***? Most animals (and humans) have a gallbladder. Horses are the exception. But for everyone else, some bile flows into and is stored by the gallbladder. Bile that is stored in the gallbladder becomes highly concentrated. So, when a meal high in fats is consumed, the gallbladder contracts, squeezing concentrated bile through the ***cystic*** [*cyst(o)-* bladder + *-ic* pertaining to] ***duct*** and on into the common bile duct and the duodenum. This is like turbo-boosted-bile! It will emulsify large amounts of fat very efficiently.

Cholecystitis and Cholecystectomy

There can be drawbacks to having a gallbladder. For those who are predisposed, the concentrated bile and bile salts can form thick sludge and even precipitate to form stones. When this happens, either the *cystic duct* or the *common bile duct* can become obstructed. As the gallbladder becomes distended with bile, inflammation develops. ***Cholecystitis*** [*chole(o)-* gall, bile + *cyst(o)-* bladder + *-itis* inflammation of] can be very painful. A distended, inflamed gallbladder in dogs and cats is often referred to as a ***mucocele*** [*muc(o)-* mucus + *-cele* a swelling or hollow], because the gallbladder is often filled with thick, ***necrotic*** [*necr(o)-* dead + *-tic* pertaining to], ***mucoid*** [*muc(o)-* mucus + *-oid* resembling] material. It looks like tar

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but smells like roadkill. Because these patients are at risk of having the gallbladder rupture, an emergency ***cholecystectomy*** [*chole(o)-* gall, bile + *cyst(o)-* bladder + *-ectomy* to cut out, remove] is usually performed.

Hepatopathy

Many factors can contribute to ***hepatopathy*** [*hepat(o)-* liver + *-pathy* disease of]. And whether primary or secondary disease, there are some typical consequences. First, when *hepatocytes* are damaged, they leak some of their enzymes. So, as we review blood work, related to hepatic function, we may see elevations in ***liver enzymes***. Hepatic enzymes include ***ALT*** (alanine aminotransferase), ***AST*** (aspartate aminotransferase), and ***ALP*** (alkaline phosphatase). (Note the *-ase* suffix in each of those names; it means *enzyme*.) As the liver's ability to detoxify the blood decreases, we often see elevations in ***blood ammonia***. As protein production decreases, ***hypoproteinemia*** [*hypo-* low + *protein* + *-emia* a blood condition of] develops. As the liver's ability to use bile acids in the production of bile, ***bile acids*** become elevated. Finally, ***hyperbilirubinemia*** [*hyper-* excess + *bilirubin* + *-emia* a blood condition of] often develops. And because of the bilirubin pigments, with excess accumulations, we can see this clinically as ***icterus*** or ***jaundice***. The skin and mucous membranes appear yellowish.

But *icterus* alone does not necessarily indicate primary hepatic disease. Refer back to our discussions inChapter 3 on *hemolytic* disease. Excess ***hemolysis*** [*hem(o)-* blood + *-lysis* breakage], whatever the cause, will overwhelm the system with bilirubin. The liver can only conjugate (transform) so much bilirubin in a given period of time. So, until the liver can get to it, bilirubin from hemolysis will back up in the bloodstream, causing a ***prehepatic*** [*pre-* before + *hepat(o)-* liver + *-ic* pertaining to] ***icterus***. It's *prehepatic* because the *hemolysis* happened before the blood even got to the liver. Certainly infectious diseases, such as viral ***hepatitis*** [*hepat(o)-* liver + *-itis* inflammation of], can be a cause of ***hepatic icterus***. There are many infectious diseases, toxins, and parasites that can directly damage the liver, resulting in *hepatic icterus*. In these cases, the liver is simply incapable of conjugating even normal amounts of bilirubin. Finally, if we obstruct the flow of bile, the liver will no

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longer conjugate bilirubin for the production of bile, because it has nowhere to put anymore bile. Obstruction of the common bile duct is the most common cause of ***posthepatic*** [*post-* after + *hepat(o)-* liver + *-ic* pertaining to] ***icterus***. I've seen stones and even parasites, such as roundworms and liver flukes, obstruct the common bile duct. And because the common bile duct enters the duodenum at the same point as the major ***pancreatic*** [*pancreat(o)-* pancreas + *-ic* pertaining to] duct (as you saw in the animation), I've seen patients with severe ***pancreatitis*** [*pancreat(o)-* pancreas + *-itis* inflammation of] obstruct flow of bile.

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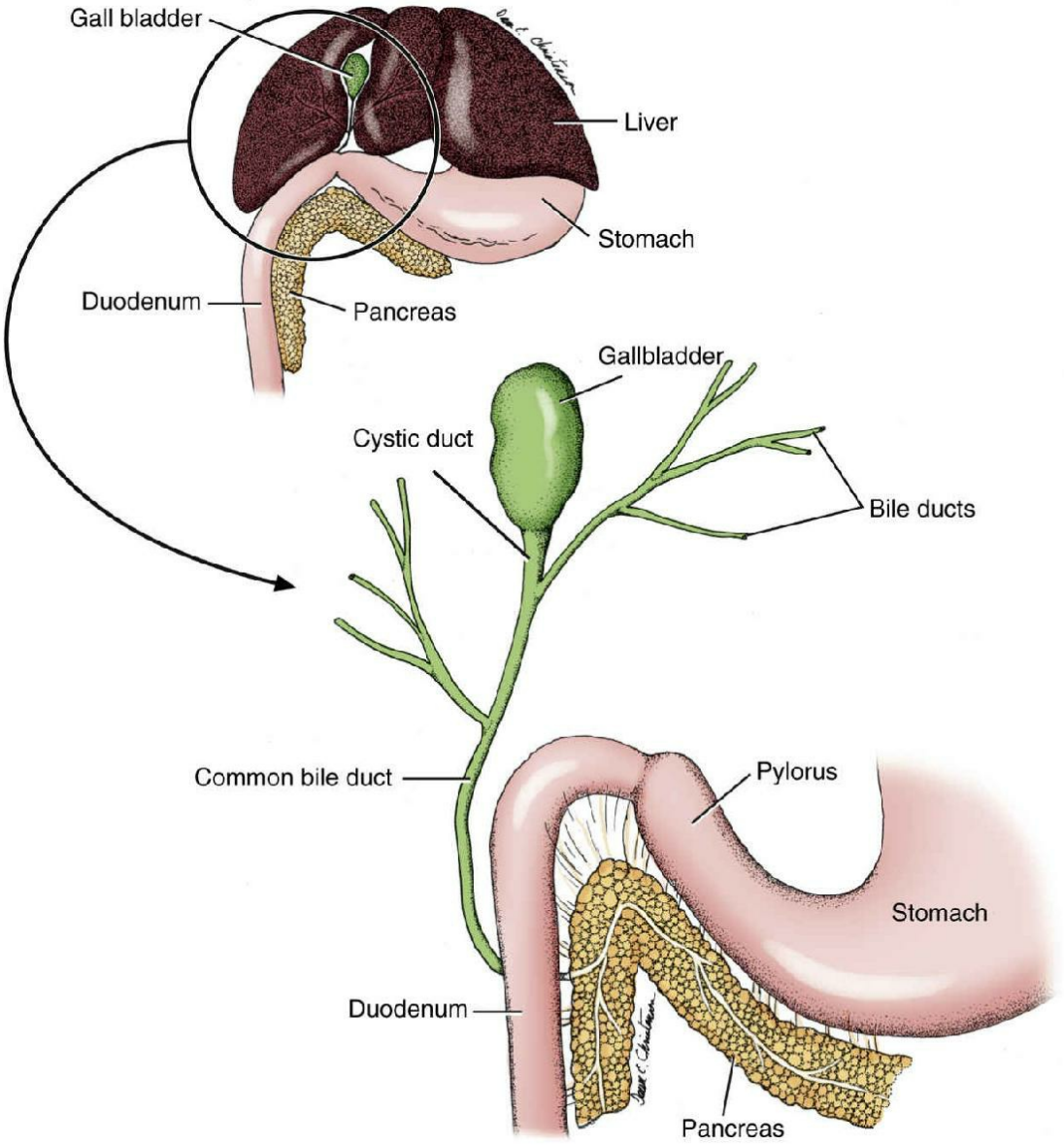

FIG. 7.3 Canine liver, biliary tree, and gallbladder.

Why spend so much time talking about icterus? We cannot jump to conclusions when we see it. We cannot assume that the liver itself is horribly diseased. There are factors both before and after the liver that may result in icterus. And our management of each of those patients will be dramatically different. Performing a *cholecystectomy* on a patient with ***immune-mediated hemolytic anemia*** [*an-* without + *em(o)-, hem(o)-* blood + *-ia* condition of; i.e., deficiency of red blood cells] will not only fail to resolve the icterus but will also probably jeopardize the life of the patient. By the same token, a dog with viral *hepatitis* poses a serious health risk to other dogs. That broadens our concern from an individual patient to all of our canine patients who might be exposed. So, long story short—

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we need to do our due diligence to figure out if the icterus we observe is *prehepatic*, *hepatic*, or *posthepatic*. Period.

Pancreas

The pancreas is a "switch-hitter" to coin a baseball phrase. It is an incredibly talented organ that plays dual roles very well. We'll talk about its hormonal role in detail inChapter 10. For our purposes in this chapter, we'll focus on its ***exocrine*** [*ex-* out + *crin(o)-* secrete] functions. So, we'll be discussing *pancreatic* enzymes involved in digestion.

Location is everything, right? Well the pancreas, as you could see in Fig. 7.3, is conveniently located alongside the stomach (abomasum in ruminants) and duodenum. Because much of the digestion and absorption of nutrients take place in the small intestine and the duodenum is the most proximal part of the small intestine, this is indeed a perfect location for the pancreas. The major pancreatic duct enters the duodenum at the very same spot as the common bile duct (i.e., at the ***major duodenal*** [*duoden(o)-* duodenum + *-al* pertaining to] ***papilla***, as shown in the animation). It also has a lesser duct, a little further along the duodenum (a variation from what you saw in the animation). So, what about these enzymes? Well, we'll need different enzymes for different nutrients. Remember, the principal nutrients that will require digestion (breaking them down into smaller, absorbable, usable molecules) are carbohydrates, lipids, and proteins. Let's look at the corresponding enzymes in that order.

Amylase

To digest carbohydrates, especially more complex starch molecules, we'll use ***amylase*** [*amyl(o)-* starch + *-ase* an enzyme for]. As food with various carbohydrates enters the duodenum, *amylase* is secreted into the duodenum. That enzyme progressively breaks down larger starch molecules and moderately sized glucose molecules into simple sugars that can be absorbed through active transport. Obviously, ***enteric*** [*enter(o)-* intestine + *-ic* pertaining to] cells get to use whatever glucose molecules they need first. The rest goes into mesenteric blood flow and then to the liver and beyond. Then cells throughout the body have access to and can absorb these

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valuable glucose molecules. Both the liver and the muscle tissue may store some of it away as ***glycogen*** [*glyc(o)-* sugar, glucose + *gen(o)-* producer]. As we'll discuss inChapter 10, the pancreatic hormone, *insulin*, is needed for the active transport of glucose into cells. *Glucagon* is another pancreatic hormone that can stimulate conversion of *glycogen* to usable glucose. It can also stimulate the liver to engage in *gluconeogenesis*. These two pancreatic hormones are keys for the regulation of blood glucose levels: insulin lowers blood glucose and glucagon raises blood glucose. Do you see the beautiful arrangement of exocrine and endocrine pancreatic activity? *Amylase* hits a bases-loaded home run by breaking down carbohydrates into small glucose molecules (not one but many baseballs). Then *insulin* and *glucagon* "field" the glucose molecules. It's a winning combination. (Groan.)

Lipase

Remember our pancreatic enzymes enter the duodenum at the same spot as bile from the liver. We said earlier that *bile* and *bile salts* emulsify the fats into smaller globs. Now we have greater surface area for ***lipase*** [*lip(o)-* fat + *-ase* an enzyme for] to work. This is a big job, because lipids have some really big molecules. That's why for those animals who have a gallbladder, the concentrated bile and bile salts are so beneficial. In their concentrated form, they are way more efficient at emulsification. But no worries. For those who don't have a gallbladder, fats are still emulsified. It's just that it may take more lipase and longer for it to digest those fats for absorption. We've got a really long segment of small intestine to give lipase the chance to do just that. Once the fats have been reduced down to fatty acids, they can be absorbed. And because the cell membranes of enteric cells are made of ***lipoproteins*** [*lip(o)-* fat + *protein*], we won't need active transport to absorb the fatty acids.

But absorption isn't a straight shot into the bloodstream. Once a fatty acid diffuses into an enteric epithelial cell, the cell's Golgi apparatus repackages it with a protein to create a ***chylomicron*** [*chyl(o)-* chyle, juice + Gr. *micron* a small thing]. Please refer to Chapter 2 if you need to review cellular organelles, such as the Golgi apparatus. We're still not ready for these repackaged *chylomicrons* to be in the bloodstream. Rather than entering a blood

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capillary, they are transported into a special lymphatic capillary called a ***lacteal*** [*lact(o)-* milky + *-al* pertaining to], shown in Fig. 7.13. That is an apt name, because *chyle* (the "juice" loaded with chylomicrons) is a very milky-looking fluid. The ***chylous*** [*chyl(o)-* chyle + *-ous* pertaining to] fluid is carried by lymphatic vessels to the ***thoracic*** [*thorac(o)-* chest + *-ic* pertaining to] ***duct***, where it enters the caudal vena cava and general circulation. It's the chylomicrons in the blood that create ***postprandial*** [*post-* after + *prandi(o)-* meal + *-al* pertaining to] ***lipemia*** [*lip(o)-* fat + *-emia* a blood condition of]. But this is temporary, until the chylomicrons are taken out of circulation. This is largely the liver's job.

By the way, I should note that there are occasions when a leak in the lymphatic vasculature (vessels) develops at or near the *thoracic duct*. This results in a ***chylothorax*** [*chyl(o)-* chyle + *thorax* chest]. I've seen this quite often in dogs and cats. Suffice it to say that fluid of any kind in the chest is a bad thing (as discussed in Chapter 5). In the case of a *chylothorax*, surgery may be required to correct the defect. Until the defect heals or is repaired, we often manipulate the diet to minimize the volume of *chylous* fluid produced. Obviously, a low-fat diet is key. Fewer dietary fats result in fewer chylomicrons and less *chyle*.

Proteolytic Enzymes

Proteins represent the largest and typically the most complicated molecules to digest. So, it typically takes not just one but numerous ***proteolytic*** [*prote(o)-* protein + *-lytic* pertaining to breaking] enzymes to do the job. In fact, this is such a big job; protein digestion actually begins in the stomach of *monogastric* animals. We'll address that when we get to the stomach. As to the pancreas, probably the two most important proteolytic enzymes that it secretes are ***trypsin*** (trip'sin) and ***chymotrypsin*** (ki"mo-trip'sin). But these enzymes aren't simply stored in pancreatic cells until needed. That would be a disaster. Why? Because cells are made largely of protein molecules. It's kind of hard to store something that can ultimately digest the storage container. So, there are actually ***proenzymes*** [*pro-* before + *enzyme*]—inactive precursors to the actual enzymes. The *proenzymes* for *trypsin* and *chymotrypsin* are ***trypsinogen*** [*trypsin* + *gen(o)-* producer] and ***chymotrypsinogen***,

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respectively. It's these precursors that are actually secreted into the duodenum. They're activated once they are in the lumen of the intestine. Then they can act on proteins in the food. Gradually and progressively, these proteolytic enzymes reduce proteins in the food down to ***amino acids*** to be absorbed.

Pancreatitis and Pancreatic Insufficiency

Like all organs, the pancreas can become diseased. *Pancreatitis* [*pancreat(o)-* pancreas + *-itis* inflammation of] is a very common and VERY painful condition. I've cared for numerous canine and feline patients with pancreatitis. I've also known people with it. People verbalized what I implicitly knew about the pain associated with pancreatitis. It is, in a word, EXTREME. On a scale of 0 to 10, with 10 being the worst pain ever, people have told me in all seriousness that pancreatitis pain is a 20 out of 10 on the pain scale. That means, in addition to managing their gastrointestinal (GI) symptoms and nutrition, we also need to manage the patients' pain.

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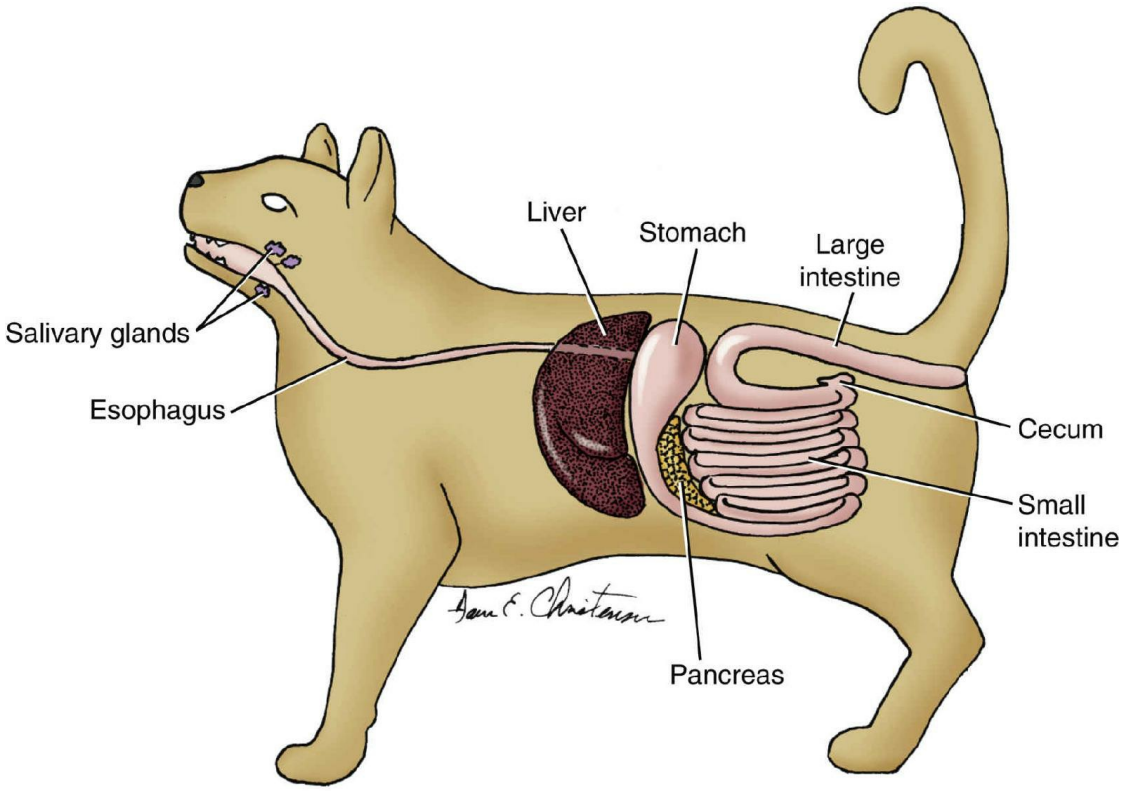

FIG. 7.4 Schematic of the feline monogastric gastrointestinal tract.

In my experience, middle-aged to older dogs and cats develop ***acute*** (sudden onset) ***idiopathic*** (unknown cause) ***pancreatitis***. Many, if not most, of these animals are overweight. And frequently, their histories include recent ingestion of a fatty meal; this is especially true for dogs. Early in the development of the disease, animals often become ***anorexic*** [*an-* without + *orex(o)-* appetite + *-ic* pertaining to] and depressed. With increasing pain, there is a reluctance to move. Even the slightest touch to the abdomen (like an owner petting the animal) may make the animal cry out or lash out. (Remember this, if you're going to attempt abdominal palpation or pick up that dog or cat. It's not at all unusual for these patients to bite in response to the pain you unintentionally inflict.) As the inflammation worsens, acute severe vomiting and diarrhea develop. Mind you, many of these are pretty nonspecific symptoms. Through numerous diagnostics from laboratory tests (CBC [complete blood count] and serum chemistry) to abdominal ultrasound, we may be able to make a rapid, accurate diagnosis of *acute pancreatitis*. Ultrasound can demonstrate pancreatic swelling and other changes. Serum amylase levels are elevated in over 80% of dogs with pancreatitis[b], making it a very valuable laboratory test.

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But some animals with pancreatitis have normal amylase and lipase levels. In some cases, inflammation affects the liver too, resulting in elevations of liver values as well. So, making a definitive diagnosis of pancreatitis may be difficult and takes time. Until then, we focus on supportive care and symptom management.

Those treated early in the course of disease have the best prognosis. Having pancreatitis once increases a patient's risk for it developing again. That's why those who have experienced recurrent bouts of pancreatitis should be maintained on a low-fat diet, as a preventive measure. The more severe the event(s) and the more times a patient experiences pancreatitis, the more damage is done to the pancreas. Ultimately this can lead to ***pancreatic insufficiency***. On the endocrine side, this means a patient may develop *diabetes mellitus*, requiring insulin injections for the rest of its life. If exocrine pancreatic insufficiency (i.e., reduced enzyme production) develops, the patient's ability to digest nutrients is impaired. Fortunately, we can supplement some pancreatic enzymes with each meal. Supplementation will be required for the rest of the patient's life.

Okay, fasten your seat belt. I think we're finally ready to journey "door-to-door" through the digestive tract-proper (i.e., the tubular path between the mouth and the anus).

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Simple Monogastric Animals

Simple *monogastric* domestic animals include dogs, cats, and pigs. It's a pretty simple design, as you can see in the crude schematic in Fig. 7.4. We'll begin our discussion of the digestive tract itself with monogastric animals for a reason. Because humans are simple monogastric creatures too, this will allow us to draw on some familiar aspects and make comparisons. Then, when we move on to discuss hindgut fermenters and ruminants, we'll be able to focus on how this basic system has been modified to suit their herbivorous needs. Ready? Let's go!

Mouth and Muzzle

This seems like a logical place to begin the journey of processing food. After all, the mouth is the instrument for taking food into the system. And muzzle design functionally impacts how an animal eats. Take the pig, for instance. Think about the large, flat nose so characteristic of pigs. It's like the blade on a bulldozer. Really! That pig's muzzle makes it possible for the pig to bulldoze in the dirt, when it searches for wonderful things like worms, grubs, and other such critters to eat. Dog and cat muzzles are obviously refined for a different manner of eating. The ***cleft*** (split) lip allows for ease of grasping foods, whether it be ripping tissues from the carcass of prey or nibbling on other foods. But once food is in the mouth, we find many similarities.

Oral Mucosa

The ***oral*** [*or(o)-* mouth + *-al* pertaining to] ***mucosa*** is kept moist with saliva. By the way, the ***oral mucosa*** (especially the ***glossal*** [*gloss(o)-* tongue + *-al* pertaining to] and ***gingival*** [*gingiv(o)-* gingiva, "gums" + *-al* pertaining to] *mucosa*) are probably THE most commonly used ***mucosal*** [*mucos(o)-* mucosa + *-al* pertaining to] tissue for assessment of ***mucous*** [*muc(o)-* mucus, slime + *-ous* pertaining to] ***membrane color*** and capillary refill time (CRT). This was discussed in Chapter 5, so we won't belabor it here. Please refer to that chapter, as well as Chapters 3 and 8 for further discussions of *mucous membrane*

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assessment.

Saliva and Salivary Glands

The ***salivary*** [*saliv(o)-* saliva + *-ary* pertaining to] ***secretions*** coating those mucous membranes are pertinent for our discussion here. First, they can give us insight into the hydration status of a patient. We expect the *mucous membranes* of the mouth to be moist with saliva, right? If they're not, we may very well have a dehydrated animal on our hands. Pavlov is famous for his research on canine (dog) ***salivation*** [*saliva* + *-tion* act of; i.e., salivating]. If you've never heard of him, Ivan Pavlov was a Russian ***physiologist*** [*physiolog(o)-* physiology study + *-ist* a specialist in]. Around the turn of the century (1900), his research bridged the realms of physiology and psychology and led to the development of what we know today as "classical conditioning." He stumbled across this, really. He was studying the salivation of dogs in response to feeding, measuring, and analyzing their *salivary secretions*. What he stumbled across was that the dogs began salivating whenever he walked into the room, even if he didn't bring any food. Now, it stands to reason that a dog will salivate when presented with food (we all do, right?). You know what I mean if you've ever had a food-motivated dog sitting near you drooling profusely while you eat. For eating, those salivary secretions are important to lubricate the food bolus for passage through the ***pharynx*** (throat) and esophagus. (Note: unlike human saliva, animal saliva does not contain any enzymes.) But Pavlov's scientific discovery was that dogs *learned* to associate him and other objects (e.g., food bowl) with food. Eventually, he used a bell as the stimulus. Once the dogs were conditioned to the sound of the bell, they would salivate profusely every time they heard it, with or without gratification of food.

Of course, clinically, we know that not all ***hypersialosis*** [*hyper-* excess + *sial(o)-* saliva + *-sis* a condition of] is Pavlovian. Nausea causes *hypersialosis* and nearly always precedes vomiting. *Oral*, ***pharyngeal*** [*pharyng(o)-* pharynx, throat + *-al* pertaining to], and ***esophageal*** [*esophag(o)-* esophagus + *-al* pertaining to] pain can cause *hypersialosis*. ***Stomatitis*** [*stomat(o)-* mouth + *-itis* inflammation of], ***glossitis*** [*gloss(o)-* tongue + *-itis* inflammation of], and ***esophagitis*** [*esophag(o)-* esophagus + *-itis* inflammation of] can be

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quite painful conditions that cause an animal to drool profusely. ***Dysphagia*** [*dys-* difficult + *phag(o)-* eating + *-ia* condition or process of] or esophageal obstruction can appear to cause hypersialosis. I said "appear," because in these situations, the animal may have normal volumes of saliva, but their impaired swallowing gives the saliva nowhere to go except dripping from the mouth.

This is definitely the case with the "frothing at the mouth" of animals infected with the rabies virus. They cannot swallow. I point this out for your sake. If an owner ever brings in a dog or a cat with *apparent* hypersialosis and claims that they think the animal has a "bone caught in its throat," red flags and flashing lights should go off for you. (In livestock, they usually claim that the animal has an apple or something caught in its throat.) Always double glove before examining that animal! Your life could depend on it. If the animal is rabid, the virus is shed in the saliva. Even if you're not bitten, the virus can enter your body through any minor cut, crack, or abrasion on your hands. With a deadly ***zoonotic*** [*zo(o)-* animal + *nos(o)-* disease + *-tic* pertaining to; i.e., a disease transmitted between animals and humans] virus like rabies, it is better to be safe than sorry. I know, everyone thinks about Cujo[c] and bite wounds when they think about rabies transmission. That novel and movie represent the "furious form" of the disease. (Of course, Stephen King embellished on the actual disease.) In veterinary medicine we often see the "dumb form" of the disease in our facilities, presented as a depressed, drooling animal. In a word—caution—is best practice.

There are a number of ***salivary glands***. The ***mandibular*** [*mandibul(o)-* mandible, "lower jaw" + *-ar* pertaining to] ***salivary glands*** are frequently mistaken by students for lymph nodes. The ***parotid*** [*para-* near + *ot(o)-* ear + *-id* the] salivary glands are on the lateral head, caudal to the ramus of the mandible extending dorsal to the base of the ear. Others are located in the ***sublingual*** [*sub-* below + *lingu(o)-* tongue + *-al* pertaining to] region of the mouth. The saliva from each gland enters the mouth through separate ducts. In canine and feline (cat) dentistry, the ducts from the parotid glands are important because the salivary secretions flowing in over the ***maxillary*** [*maxilla(o)-* maxilla, "upper jaw" + *-ary* pertaining to] fourth premolars promote tartar buildup there.

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The sublingual ducts are important for a different reason. They are associated with the ***frenulum*** [from L. *fraenum* "a small bridle"], that *sublingual* band of connective tissue that ties the tongue to the floor of the mouth. This tissue can easily be traumatized by oral foreign bodies like a string or thread wrapped around the base of a cat's tongue. This is why part of your oral examination, especially in cats, should always include sublingual inspection. The other frequent cause of sublingual trauma is during placement of an ***endotracheal*** [*endo-* within + *trache(o)-* trachea + *-al* pertaining to] ***tube*** for general anesthesia. We need to be careful to not drag and scrape the ventral surface of the tongue over the teeth, when we intubate. Whatever the traumatic cause, sublingual inflammation can impair or even obstruct the salivary ducts there. This is a frequent cause of something called a ***ranula*** (ran'u-luh [L. from *rana* frog + *-ula* a small; so-called because it looks like the distended throat of a frog when it croaks]) or ***sialocele*** [*sial(o)-* saliva, salivary + *-cele* a swelling; i.e., a cyst filled with saliva]. These often resolve when any foreign body is removed, and the inflammation subsides.

Palate

There are actually two distinct portions of the ***palate*** or "roof" of the mouth. The ***hard palate*** (shown in Figs. 7.5 and 7.6) is the rostral portion of the palate. It's hard because there's bone under the ribbed connective tissue and mucosa. Obviously, the hard palate is the dorsal border of the mouth that separates the oral cavity from the nasal passages. Caudal to the hard palate is a flexible, muscular ***soft palate***. The *soft palate* separates the ***nasopharynx*** [*nas(o)-* nose + *pharynx* throat; i.e., that region of the throat dorsal to the soft palate] from the ***oropharynx*** [*or(o)-* mouth + *pharynx* throat; i.e., that region of the throat ventral to the soft palate]. During swallowing, the soft palate becomes a barrier to protect the nasal passages from whatever is being consumed. Of course, if you've ever laughed while drinking something, you know that the protection provided by the soft palate is limited. The proof of that was provided by your beverage coming out of your nose.

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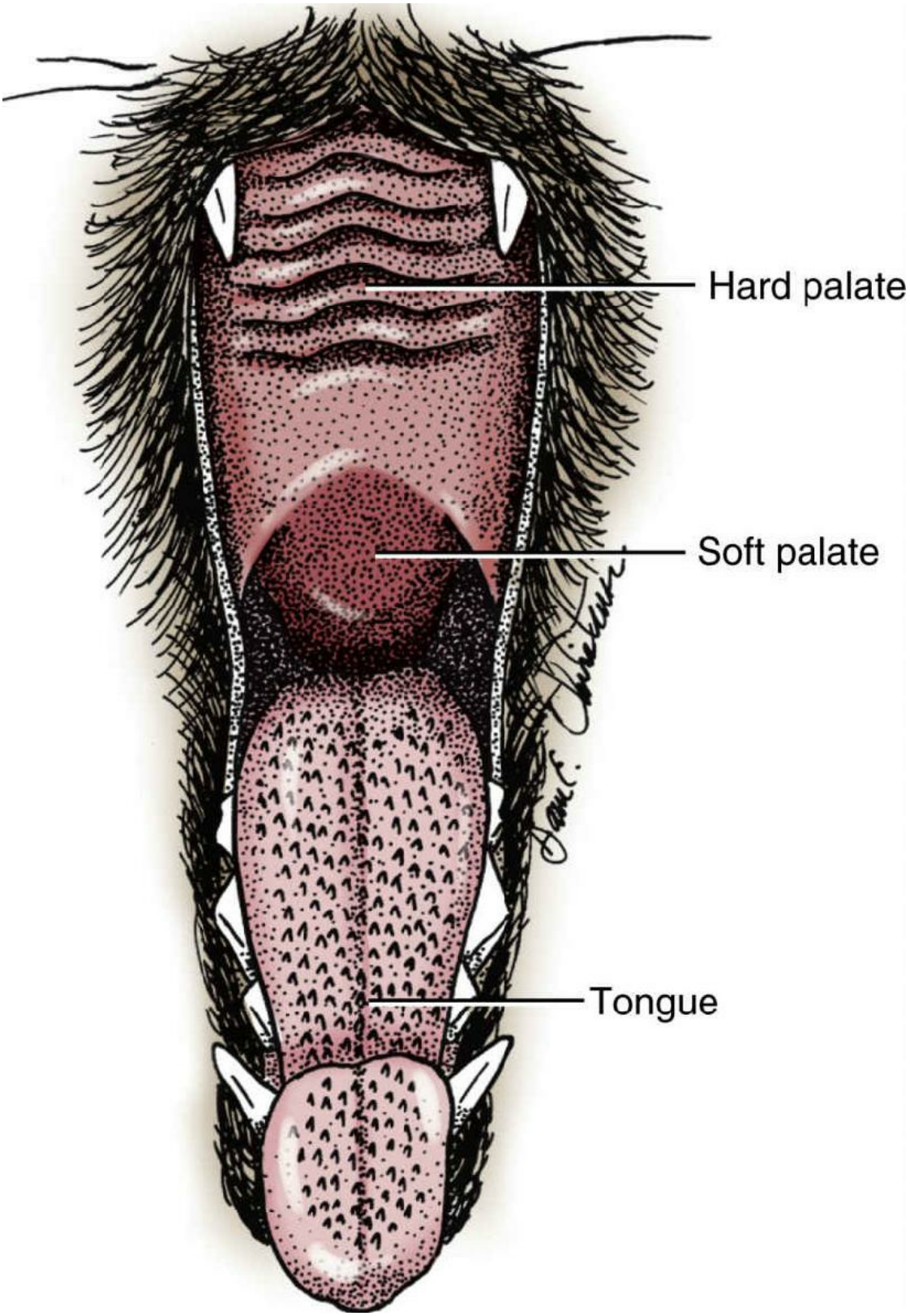

FIG. 7.5 Feline oral cavity (frontal view).

Are there ever problems associated with the palate? Sure. Obviously, either or both portions of the palate can be traumatized. Dogs that chew on sticks are notorious for that. But cats can have

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traumatic injuries too. I'll never forget the cat who we hospitalized for observation, due to vomiting. Her initial physical examination, including her mouth and pharynx, was unremarkable. After one of her vomiting episodes in the hospital, she was quite distressed and expressed some very strange behavior. She was pawing at her mouth and seemed to be gagging. When we reexamined her mouth and *oropharynx*, we discovered a sewing needle (thread still attached) embedded in her soft palate. (No, it wasn't there when we admitted her for observation.) We quickly anesthetized her and removed the needle. We were amazed that the cat was somehow able to swallow and vomit the needle, without any damage to anything but her soft palate. Of course, being an aspiring feline "sword-swallower" is the exception rather than the rule.

It's way more common to see ***palatal*** [*palat(o)-* palate + *-al* pertaining to] defects, such as ***palatoschisis*** [*palat(o)-* palate + *-schisis* a cleft]. *Palatoschisis* may involve either or both portions of the palate. Anyone can have a ***cleft palate***, as it is commonly called. And the greater the size of the defect, the greater the problem for the animal. Generally, we find *palatoschisis* in newborns or very young animals. Now, in piglets, the farmer usually discovers the cleft palate when the ***needle teeth*** are clipped. For litters of puppies and kittens, we don't always see them in the first week or two of life. The owner or breeder of the litter is typically alerted to a potential problem when the youngster has difficulty nursing and has milk bubbling out of its nose. Out of concern, the individual or the whole litter is usually brought to us. If you have the opportunity to examine newborns, always examine the mouth and *oropharynx* for palatoschisis. This is a ***congenital*** (birth) defect that requires ***palatoplasty*** [*palat(o)-* palate + *-plasty* reconstruction of] for repair. This can be a very challenging surgical procedure, especially in a tiny mouth. InChapter 5, we discussed *palatoplasty*, as it relates to animals with elongated soft palates. As you may recall, that surgery shortens and reshapes the soft palate so that it no longer interferes with the airway.

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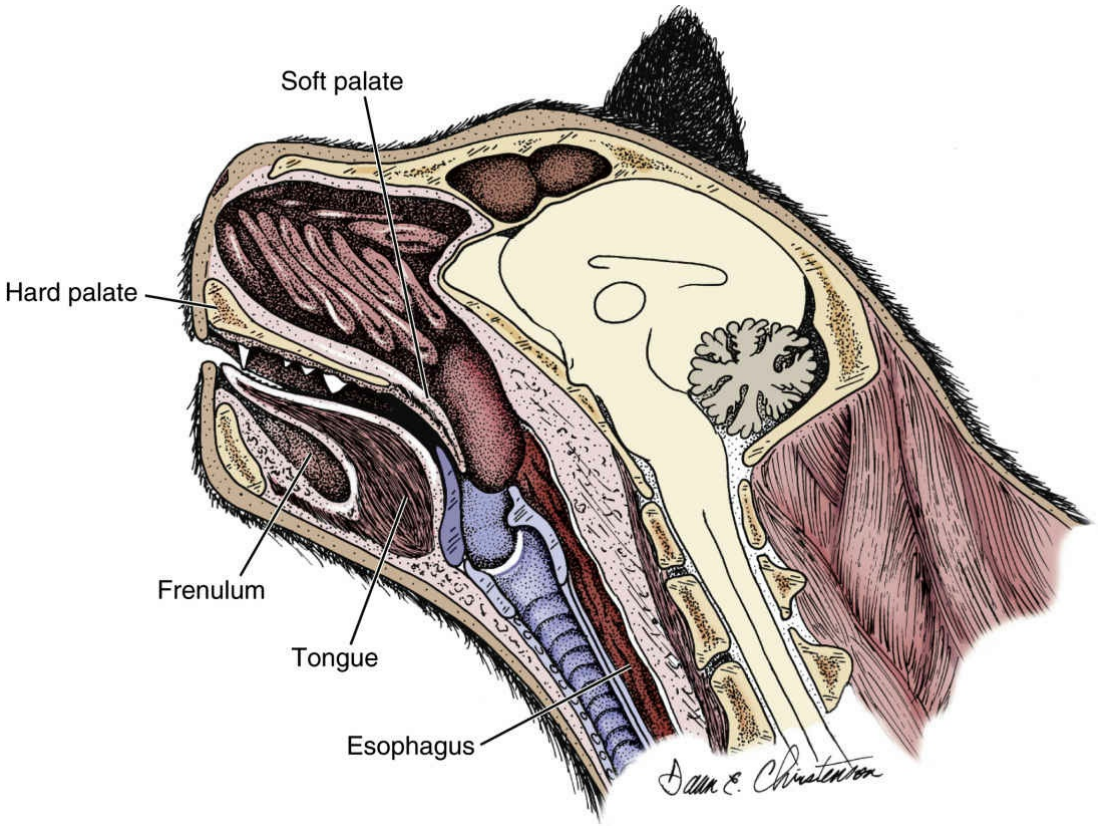

FIG. 7.6 Feline oral cavity (midsagittal view).

Tongue

The tongue is a pretty fascinating feature in the mouth. Think about it, it's covered in tiny ***papillae*** [L. *papilla* a small, nipple-like projection] that are integral for taste sensation. A cat's tongue, like the one shown in Fig. 7.5, has some very pronounced papillae that aid in their grooming behaviors. For dogs, their tongues can be quite expressive, as they gleefully lick their owners and new friends. And a dog's tongue, lolling out of its mouth and dripping wet with saliva, can aid in cooling the dog by evaporation as it pants. But as a muscular instrument, the tongue is absolutely amazing. Animals use this ***lingual*** [*lingu(o)-* tongue + *-al* pertaining to] prowess to consume food and drink. When consuming food, tongue movement is instrumental in moving the food bolus into the pharynx and esophagus. That muscular activity is so important that two of the twelve cranial nerves provide motor input for the tongue (the ***glossopharyngeal*** [*gloss(o)-* tongue + *pharyng(o)-* throat + *-al* pertaining to], ***nerve,*** and the ***hypoglossal*** [*hypo-* below + *gloss(o)-* tongue + *-al* pertaining to] ***nerve***). But tongue movement isn't just

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important for eating. When animals, like dogs and cats, drink, they actually form a little *lingual* ladle to scoop up the water. That's right, they actually curl the tip of the tongue ventrally to create that tiny ladle. When the tongue is drawn into the mouth, the water comes with it. And because the tongue is curled ventrally to do this, they almost always have little dribbles of water on their chins.

Dentition and Dentistry

Of course, we cannot talk about the mouth without talking about teeth. Teeth are important for grasping food (and perhaps prey) as well as chewing. Tooth structure and arrangement in the mouth will be different depending on the species of animal and the type of foods they are intended, by design, to eat.

Dental and Periodontal Anatomy

The schematic in Fig. 7.7 depicts a typical carnivore's or omnivore's tooth in the jaw. So that we're all on the same page, when I refer to the ***crown*** of the tooth, I'm referring to the portion that we can see in a normal animal above the "gumline" (***gingiva***). The ***roots*** are the portions of the tooth below the gumline. (Some teeth have only a single root.) We mentioned the gingiva earlier, when we discussed the oral mucosa. Gingiva is one of the most frequently used mucosal tissues used to assess mucous membrane color and CRT. The ***gingival sulcus*** [L. *sulcus* groove, furrow] is the groove formed between the free gingival margin and the crown of the tooth. The gingival attachment at the base of the gingival sulcus is important for protecting the tooth root and structures surrounding it. We'll focus on this later, when we discuss ***periodontal*** [*peri-* around + *odont(o)-* teeth + *-al* pertaining to] ***disease***.

The roots are attached to the jawbone by ***periodontal ligaments***. You may recall fromChapter 4 that most ligaments are made of dense connective tissue that holds bones together. In this case, the *periodontal ligaments* are holding the tooth in the "socket" of the jawbone. You also know, from our ligament discussion inChapter 4, that ligaments need a rough surface for strong, secure attachment. That's why the roots of a tooth are covered by a hard, rough, and tough material called ***cementum***. Yeah, it's kind of like cement—hard, rough, and tough. At the ***apex*** (point, tip) of each root is a

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collection of canals or passages called the ***apical*** [*apic(o)-* apex + *-al* pertaining to] ***delta***. If you're familiar with a river delta, you know that it has a number of tributaries or smaller streams that lead from the river to a body of water. The *apical delta* of a tooth provides access of vessels and nerves into (and out of) the pulp/pulp cavity of the tooth.

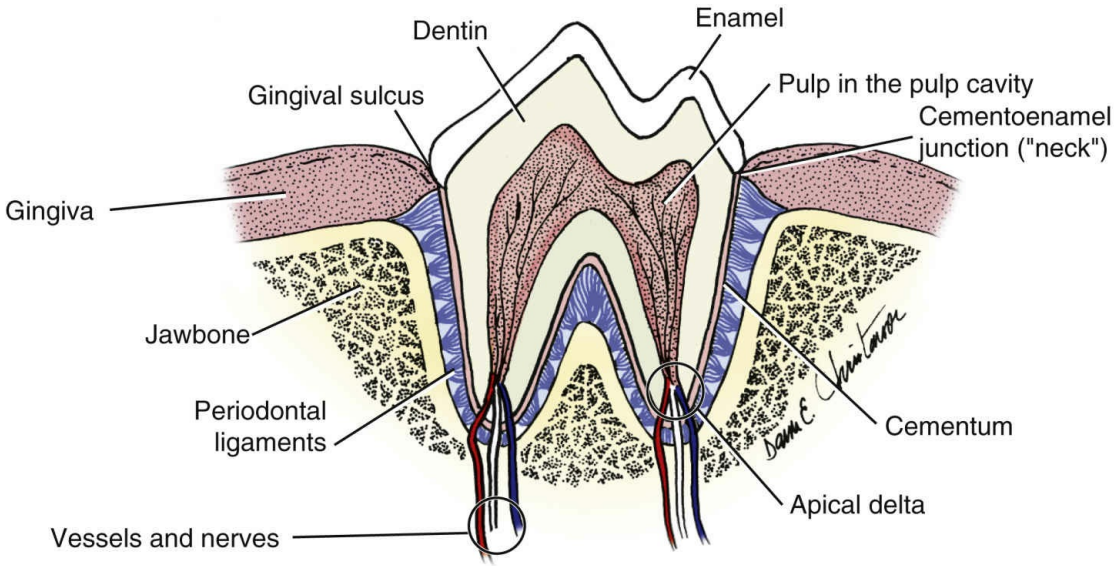

FIG. 7.7 Basic tooth and periodontal anatomy for carnivores and omnivores.

Yes, the ***pulp*** of a tooth is living, highly vascular tissue. If you have ever broken a tooth, exposing the pulp in the ***pulp cavity***, you also know that it has nerves in it too. Tooth fractures are very painful. In fact, you don't even have to expose the tissue in the pulp cavity. Just breaking through to the ***dentin*** that makes up the bulk of the tooth is painful. You see, dentin is hard but porous bony material, and there are tiny tubules through it that communicate with the pulp cavity. So, when dentin is exposed through a fracture, nerve endings in the pulp can still be stimulated. Can you say "ouch"? Been there; done that. It's not fun. That's why the ***enamel*** covering the crown is so important. Enamel is the hardest material found in the body. It is a rock-hard protective layer that doesn't allow for exposure and stimulation of nerves within the tooth. Unfortunately, if we wear through or break off some of the enamel, we can't repair or replace it. What you see is all you get. There are

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odontoblasts [*odont(o)-* tooth + *blast(o)-* germ, shoot; i.e., a cell that builds dentin of the tooth] in the dentin. So, if an animal slowly wears through the enamel and into the dentin, the *odontoblasts* will deposit ***reparative dentin***. If you've ever seen a dog with worn teeth and a dark brown color to the worn surface, then you've seen *reparative dentin*. Now you know that those teeth were worn down slowly.

Are there differences between carnivorous and omnivorous teeth? Yes. They all have the basic structure that we just described. But the crowns of a carnivore's (the cat being the only true carnivore) teeth are designed exclusively for ripping, tearing, and shearing flesh from prey. They don't have ***occlusal surfaces*** on their teeth (i.e., a flattened, bumpy surface designed for contact with another tooth), not even on their molars. *Occlusal surfaces* are designed for chewing—trapping the food between two teeth and grinding it into smaller particles. Well, cats' teeth simply aren't designed for chewing. Omnivores, on the other hand, do have teeth with occlusal surfaces. In fact, the greater the variety of foods an omnivore is likely to eat and chew, the more occlusal surfaces its teeth will have. Pigs (and bears) are great examples of this. They eat many bugs, fruits, and other vegetation that require ***mastication*** [L. *masticare* to chew + *-tion* act of; i.e., chewing] when consumed. The whole point of *mastication* is to pulverize the food into smaller particles for ease of digestion down the pike. (Humans are omnivores. So, chew your food!) All of the molars and many of the premolars in an omnivore's mouth, depending on species, have occlusal surfaces. They still have some teeth for shearing (otherwise, the bear would have a tough time eating that salmon it caught in the river). Comparatively, dogs don't tend to consume foods that require a lot of chewing. In fact, they simply tend to gulp their food. So, occlusal surfaces in the dog are limited to their molars. The rest of a dog's teeth are designed for flesh.

In fact, because of their size and familiarity for most of us, let's use the canine schematic in Fig. 7.8 to acquaint you with the different types of teeth found in everyone's mouth. We've already mentioned molars. ***Molars*** are the most caudal teeth in the mouth. (Carnivores have the fewest molars of any animal.) Molars generally have occlusal surfaces. Rostral to the molars are

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premolars [*pre-* before + *molar*]. Depending on the animal, premolars of some omnivores may have occlusal surfaces (e.g., pigs, bears). Rostral to the premolars are the ***canine*** teeth. Yes, other animals besides the dogs have canines. These teeth tend to be quite long and sharp. Their roots are equally long if not a little longer—a challenge if we need to extract one. Canines in the cat actually have longitudinal grooves in the enamel. Those grooves can harbor much bacteria. (Remember that, if you're ever bitten by a cat, you should seek medical attention.) The most rostral teeth in the mouth are the ***incisors***. Incisor, like the word *incision*, is derived from the Latin *incidere*, meaning "to cut open". And being the most rostral teeth in the mouth, the incisors will probably be the first to cut open someone' or something's flesh. Think of the long incisors of squirrel (another omnivore). Their incisors are valuable for cutting through the outer shell of seeds and nuts to get at the flesh. Those incisors also come in handy when they're eating insects or even other small mammals or snakes. (I'll bet you didn't know squirrels ate anything but nuts.)

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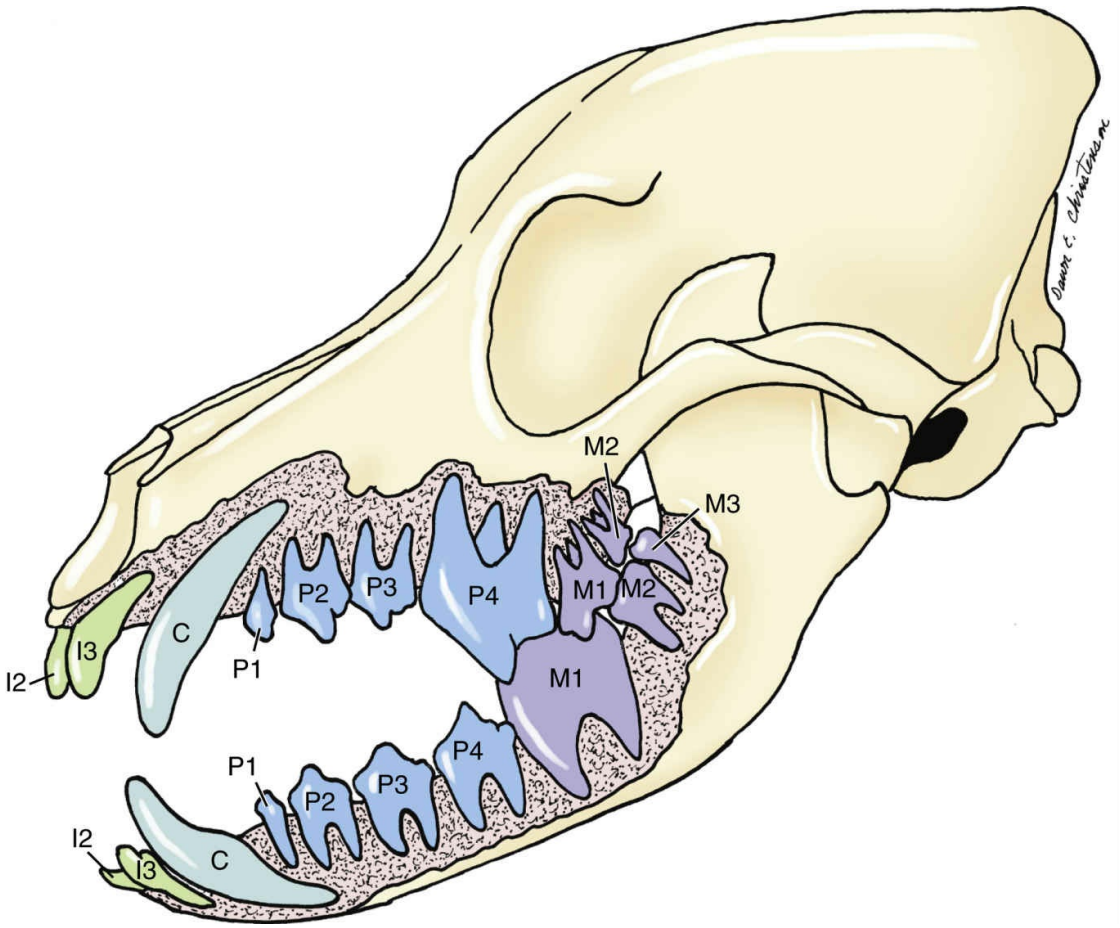

FIG. 7.8 Canine dental schematic. *C*, Canine; *I*, incisor; *M*, molar; *P*, premolar.

Canine, Feline, and Porcine Dental Arcades

What is meant by the term ***dental arcade***? Well, this kind of arcade has nothing to do with video games or pinball machines. In anatomy, an ***arcade*** is something that has a *series of arches*. I think the ventral view of the canine maxilla in Fig. 7.9A really demonstrates "a series of arches" quite well. The teeth on the right side of the mouth form an arch, as do the canines and incisors and the teeth on the left side of the mouth. So, when we think about dental arcades, we have a series of arches on the maxilla ("upper jaw") and another series on the mandible ("lower jaw"). Sometimes, folks will refer the maxillary teeth as the "***upper arcade***" and the mandibular teeth as the "***lower arcade***."

Continuing to look at Fig. 7.9A, you'll notice that we've labeled the teeth sequentially on each side of the mouth using two different methods. Certainly, we can simply reference the right fourth

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premolar of the maxilla to distinguish it from the left maxillary fourth premolar and the two mandibular fourth premolars. But in dentistry, that method of identification can be cumbersome and confusing. If someone writes in the medical record: "4th premolar," which one are they referring to? There are four of them! So, a numeric system was developed for canine and feline dentistry. Each tooth has a unique number, minimizing confusion. And when it comes to recording information in the medical record, the numeric system is way more streamlined and efficient. I know, you're probably looking at all of those numbers with a deer-in-the-headlights look on your face, wondering how you'll ever keep the numbers straight. Don't panic. Simply split the mouth and arcades into four quadrants. Teeth in the upper right quadrant are all **100**s. The teeth in the upper left quadrant are all **200**s. Teeth in the lower left quadrant are all **300**s, and those in the lower right quadrant are all **400**s. Within each numeric series, we number rostral to caudal. And there is a convenient little "rule of 4 and 9," to help you keep the teeth straight, especially if some teeth are missing. The canines (easy to spot in any mouth) are always numbered ending with a "4." And the first molars are always numbered ending with a "9." If that's true (and it is), then 104, 204, 304, and 404 are all canine teeth, and 109, 209, 309, and 409 are all first molars. If I need to record a fracture on the left lower mandibular canine, I would much rather simply record a 304 fracture. Why give myself writer's cramp? What about ***deciduous*** (baby) ***teeth***? That's easy. Just add 400 to the number of an adult tooth, and you've got the number for the corresponding deciduous tooth. For example, 504 is the deciduous right maxillary canine tooth. (See? 504 is easier to write.)

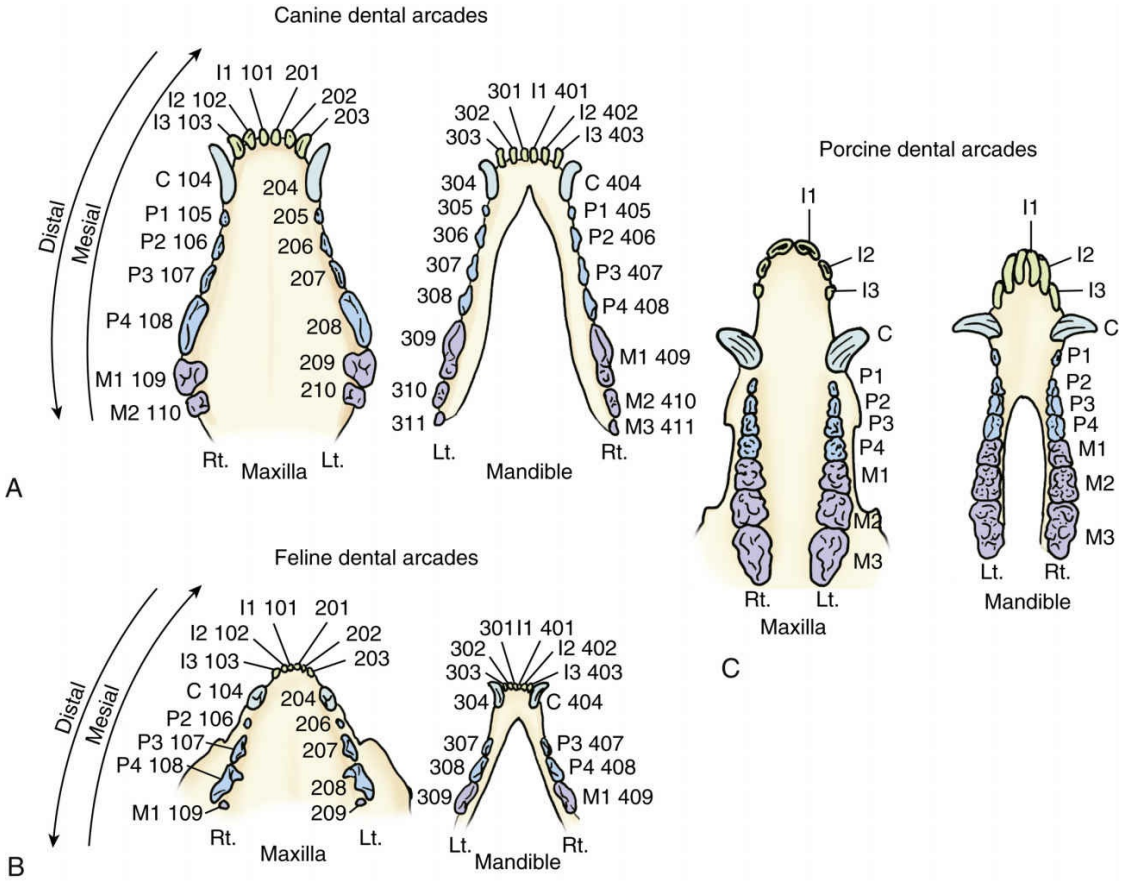

FIG. 7.9A (A) Canine dental arcades. (B) Feline dental arcades. (C) Porcine dental arcades.

If you compare the dental arcades of the dog to the cat (Fig. 7.9B), you'll notice that the cat has fewer teeth. Remember, a cat has no need for molars for chewing. Even if a cat wanted to chew, 109 and 209 won't do much for the animal. (I wish I had a nickel for every student who told me a cat was missing those molars in the upper arcade. But in their defense, those teeth are tiny and rather hidden, sitting medial to the fourth premolars.) Cats don't have as many premolars as dogs either.

Comparatively, the porcine (pig) dental arcades, shown in Fig. 7.9C, are loaded with teeth and occlusal surfaces. The "full mouth" of the pig is needed for the wide variety of foods (from worms and bugs to plants, mostly plants) that they tend to eat. In fact, later when we look at herbivores, you'll see that pigs have more teeth than most herbivores, to accommodate such a varied diet. Also notice that we've not used the numeric dental system for the pig. That system was developed for canine and feline dentistry. We simply don't do dentistry in pigs like that. But I will warn you about their canines (tusks) and incisors. Those teeth may be

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crooked, but they can do an awful lot of damage to you, if you're not careful. I've seen people with deep gashes in their legs from those tusks. And those crooked incisors can penetrate a leather boot.

TABLE 7.1

Comparative Adult Dental Formulas for Carnivores and Omnivores

Cats	
Dogs	
Ferrets	
Pigs	

Dental Formulas

Refer to Table 7.1 for comparative dental formulas of carnivores and omnivores. Did you look? Are you confused by what you saw? Go ahead, admit it. Those darn formulas look confusing as can be! They look like some algebra equation gone bad. But they're really not as bad as they seem.

Think about the dental arcades. If you were to split each arcade down the middle and then place one half of the maxilla over one half of the mandible, you'd have the basis for the dental formula on the inside of the brackets. Maxillary teeth are represented by the upper number of each "fraction," and mandibular teeth are represented by the lower number of each "fraction." And if you notice, each "fraction" within those brackets is preceded by a letter. As shown in Figs. 7.8 and 7.9, "I" refers to incisors, "C" refers to canines, "P" refers to premolars, and "M" refers to molars. Because those "fractions" for each type of tooth represent only one half of the mouth, the whole formula is bracketed and "multiplied" by two.

So, as we look at the dental formula for the cat, we find that in one-half of the mouth, there are three maxillary incisors over three mandibular incisors. Each half of the mouth has one maxillary canine and one mandibular canine. Each half of the mouth has three maxillary premolars over two mandibular premolars. Finally, each half of the cat's mouth has one maxillary molar over one

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mandibular molar. And that is precisely how each dental formula is interpreted. I hope this makes those dental formulas just a little less intimidating for you. Having those dental formulas tucked away in your brain (or a handy pocket notebook) can be very helpful when evaluating an animal's mouth.

Dental Surfaces

Now, we may not use that numeric system for every species of animal. But we can use the same references for tooth surfaces (Fig. 7.10) for everyone. Each tooth has four surfaces, kind of like a 4 x 4 fence post. Most of those tooth surfaces are named on the basis of their proximity to other structures in the mouth. For instance, all of the mandibular teeth have one surface that lies next to the tongue. That's the ***lingual surface***. All of the maxillary teeth have a surface that lies nearest to the hard palate. That's the ***palatal surface***. All of the premolars and molars have a surface that touches the mucosa of the cheek. That's the ***buccal*** [*bucc(o)-* cheek + *-al* pertaining to] ***surface***. The surface of the canines and incisors that comes in contact with the lips is the ***labial*** [*labi(o)-* lip + *-al* pertaining to] ***surface***. Those are easy and logical, right? In all the years I've taught dentistry, it's been the surfaces *between* the teeth that have been most confusing for my students.

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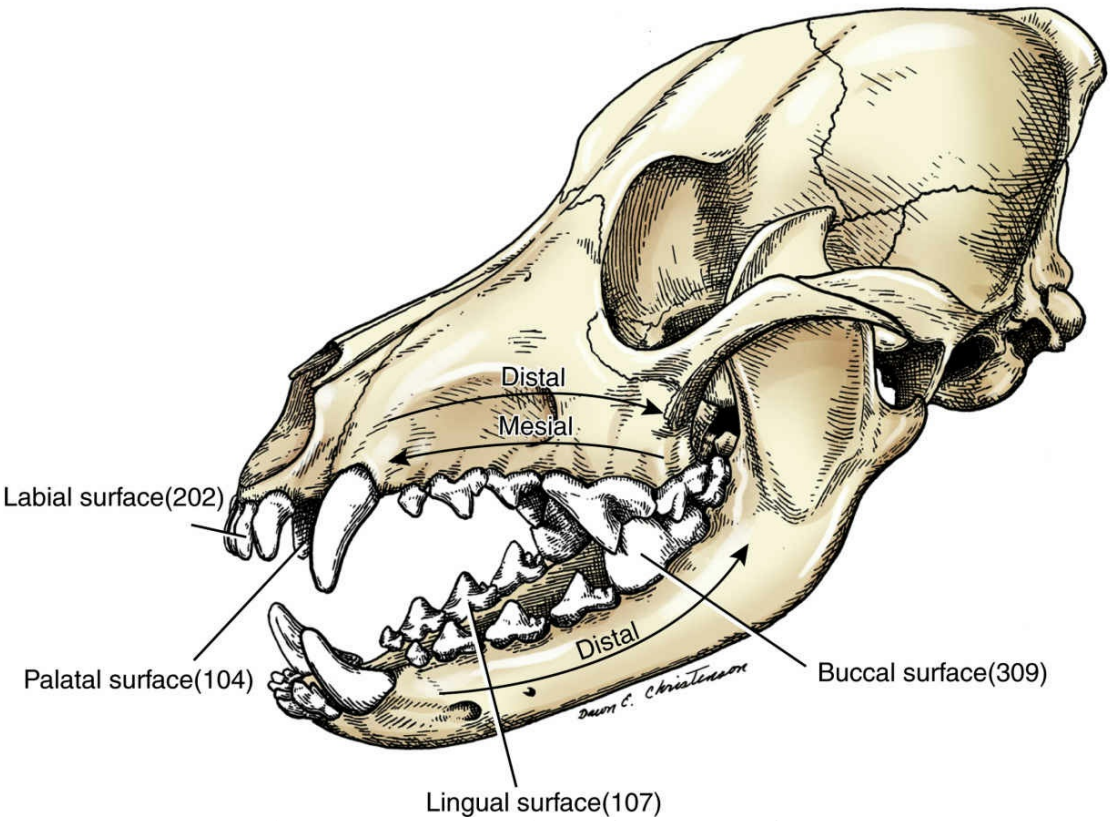

FIG. 7.10 Dental surfaces (canine skull).

The terms ***mesial*** and ***distal*** are simply more difficult to comprehend because the roots of those words have been used in other contexts to mean other things (*mesi(o)-* middle and *dist(o)-* distant). But middle and distant just don't seem to apply well in the mouth. So, how the heck do we apply these terms mesial and distal to the surfaces between teeth? As you look at the lateral teeth of the arcades, each tooth has what we might like to call a "rostral" surface. Don't do it. To be correct, that's the ***mesial surface***. The medial surface of each incisor is also the *mesial surface*. (At least *mesi(o)-* and *medi(o)-* both mean "middle." So, mesial is a good "fit" for the incisors.) Now, if thinking medial and rostral helps you remember where the mesial surfaces are, then go ahead and think it. Just don't write it in a medical record. What about the distal surface? Well, the lateral teeth of the arcades all have what appears to be a "caudal" surface, right? To be correct, that is the ***distal surface***. For the incisors, distal just doesn't seem to work. So, only for the incisors, the surface opposite from the mesial surface is … wait for it … the ***lateral surface***. Don't hate me, I'm just the messenger.

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Periodontal Disease

Any animal or human can develop ***periodontal*** [*peri-* around + *odont(o)-* teeth + *-al* pertaining to] ***disease***. As the name implies, it is a disease of the tissues around the teeth. It is a progressive disorder of the mouth. *Periodontal disease* is the most common oral disease of dogs and cats, especially dogs. And it is one of the leading contributors to heart and kidney diseases in dogs and cats. That is a sad statement since periodontal disease is preventable.

What causes it? In a word—bacteria. Have you noticed all of the plaque-fighting human oral hygiene products marketed to people? That's because ***plaque*** harbors bacteria in the mouth. *Plaque* is an invisible salivary film loaded with bacteria and food particles that coats all of the teeth and mucous membranes of the mouth. By removing plaque and the harmful bacteria that it harbors, we know that periodontal disease can be prevented. The same is true in animals.

You see, if oral bacteria are left unchecked, they inflame and destroy periodontal tissues. ***Gingivitis*** [*gingiv(o)-* gingiva, the "gums" + *-itis* inflammation of] is the first symptom. Imagine the warm, moist environment provided for bacteria, especially in the *gingival sulcus*. There and at the gingival margin, bacteria can flourish. The activity of overpopulated bacteria produces an overabundance of inflammatory mediators. (If you need to review inflammation, please refer toChapter 3.) The gingiva provides an important protective barrier for the body. If it becomes inflamed, the barrier can be breached. And if bacteria breach the gingival barrier, because of the ***vascularity*** [*vascul(o)-* vessels + *-arity* state of] of the gingiva, bacteria have free and easy access to the whole body. Highly vascular organs, such as the heart and kidneys, become prime targets of the invading bacteria. But here's the thing—plaque, that slimy bacterial film, is easily removed. Simple brushing removes much of it. Routine plaque removal reduces the bacterial population, thereby minimizing and preventing their harmful effects, such as *gingivitis*.

Let's say the bacteria are allowed to colonize and gingivitis develops. Visibly the gingival margins become red and ***edematous*** [*edema* swelling + *-tous* pertaining to]. It's no secret that inflammation weakens tissues, making them more friable (fragile).

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So, as the gingivitis worsens, the gingiva easily bleeds, creating a superhighway into the body. But think about the structure of that gingival sulcus. We said earlier that the gingival attachments at the base of the gingival sulcus are important to protect the rest of the periodontal tissues. Inflammation weakens those attachments. And if we break the gingival attachments, inflammation will easily progress and begin to weaken and destroy other periodontal tissues. And there's a contributing factor to breaking down the gingival attachments—***tartar*** (also called ***calculus***). You see, over time, plaque becomes thick and hard. It takes only 12 hours for plaque to harden into *tartar*. And once that happens, there's a snowball effect—with more and more plaque and tartar buildup. As time goes on, the tartar actually mineralizes into rock-hard concretions. It's a great cover for the bacteria to multiply under, further inflame tissues, and etch away at the enamel of the teeth. The gingival sulcus becomes packed with tartar. Between inflammation and the physical stresses of tartar buildup stretching the tissues, the gingival attachments give way. We said just a moment ago that routine plaque removal reduces the bacterial population and its harmful effects. And we now know that plaque removal within 12 hours prevents it from hardening into tartar. So, it's not just the physical removal that's important. It's also the timing of it that's important.

If we fail to remove plaque in a timely manner, the progressive cascade of events can and will be devastating. Systemically, we've already said that through gingivitis alone, harmful bacteria can enter the body and greatly contribute to heart and kidney diseases. How many dogs have you seen with "rotten" mouths who also have heart murmurs? I've lost count over my long career. And we know that there is a direct correlation between the oral disease and the ***valvular*** [*valvul(o)-* valve + *-ar* pertaining to] insufficiencies creating the murmurs. All ***venous*** [*ven(o)-* vein + *-ous* pertaining to] blood goes to the heart. Talk about a rich environment for bacteria! And where do they tend to "set up housekeeping"? —right at the edges of the valves. The *valvular* irregularities created by this make the valves leak, creating the murmurs. Over time, this reduces ***cardiac*** [*cardi(o)-* heart + *-ac* pertaining to] function and often leads to ***heart failure***. The kidneys are also highly vascular organs.

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Bacterial damage there leads to ***renal*** [*ren(o)-* kidney + *-al* pertaining to] ***failure***. Either (or more often both) of these organ failures reduces quality of life and leads to a shorter life span. How sad, when these consequences could be avoided with simple, timely removal of plaque.

But what about serious oral disease? After all, we are talking about *periodontal* disease, right? Right. Okay, let's look at the domino progression of periodontal disease in the mouth. We said earlier that *gingivitis* and *tartar* buildup result in loss of the all-important *gingival attachments*. If we could intervene as the gingival attachments are being damaged and broken, they might heal and restore the integrity of the *gingival sulcus*. If we don't and the gingival attachments are lost, bacteria have direct access to the ***periodontal ligaments*** and ***bone*** of the jaw. As the ligaments are damaged and destroyed, deep pockets develop around the teeth. During a routine dental cleaning, this is the very reason why we measure the gingival sulcus at several points around every tooth. A normal, intact gingival sulcus averages 1 to 2 mm or less in depth. I've seen periodontal pockets that were 9 mm deep! In a case like that, we've lost all of the periodontal ligaments down to the vicinity of the root apex. Of course, the bacteria don't limit their damage to the periodontal ligaments. They attack the *bone* of the jaw too. It's loss of both the ligaments and bone that creates loose teeth. Loss of those tissues also leads to visible ***gingival recession***. Intervention at this point cannot reverse the effects. All we can hope to do is slow the progression of the disease. Hopefully, we can minimize the chances for development of an ***apical abscess*** (a collection of pus at the root apex). This really accelerates the destruction of bone. I've seen animals who developed *apical abscesses* of maxillary teeth, where the infection actually broke through into the nasal passages and sinuses. Maxillary sinus infections are usually associated with the roots of the fourth premolar (108 or 208). These two premolars have 3 long roots that lie very close to the floor of the maxillary sinus. It's easy for an *apical abscess* there to break into that sinus. I've seen many dogs with draining, smelly, nonhealing cheek wounds just below the eye, where the infection has actually "eaten" through the dorsal bone of the sinus and the skin covering it. If you ever see a wound like that, flip the lips and look at the fourth premolar. I'd

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lay odds that it looks diseased. Apical abscesses of mandibular teeth can actually destroy so much bone that the jaw breaks. I've seen it. And I've seen dogs who have required removal of nearly half of the mandible because of it. Infected bone is very, very painful and difficult to treat—bacteria have lots of hiding places in bone.

Prevention

Do I have your attention yet? I hope I've convinced you that prevention of periodontal disease is very important for the health and longevity of our patients. It involves so much more than teeth. For our part, providing routine professional dental cleanings is important. In people, professional cleaning is recommended every 6 months. This would be ideal in dogs and cats too. But there's a catch—we need to perform the cleaning with the animal under general anesthesia. Because of the risks associated with this, we're probably limited to annual cleanings. We'll thoroughly assess and record the integrity and abnormalities of the teeth and periodontal tissues. We'll physically remove all of the tartar from the crowns and gingival sulci. Then we polish the teeth to reduce surface imperfections that provide a foothold for plaque and bacteria. And while this is very important, it is insufficient to prevent periodontal disease. The key in prevention lies with the owners.

We said earlier that routine removal of plaque, within 12 hours, reduces the bacterial population and minimizes tartar formation. There are commercial foods and treats available. When chewed, the abrasives in the food aid in the removal of some plaque. But remember, canine and certainly feline mouths are not designed for chewing kibble. Most chew-treats and toys for dogs usually only abrade tooth surfaces of the caudal portions of the arcades (i.e., the molars and some of the premolars). So, chewing alone is insufficient. The owners need to be directly involved by brushing the pet's teeth. On the basis of what we know, ideally, they should brush the teeth 2 to 3 times daily. Even if they only use a really soft toothbrush alone, this would have a dramatic impact. A toothpaste (dentifrice) designed for pets will add to the benefits. Owners should NOT use human toothpaste. Fluoride and other chemical compounds in human toothpaste can be toxic if swallowed. Plus,

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pet toothpastes don't foam (foam is very annoying). And pet toothpastes taste good to pets, making brushing a pleasurable activity. An added bonus to pet toothpaste is that they contain enzymes. Remember, unlike humans, canine and feline saliva does not contain any enzymes. Enzymatic toothpastes aid in the breakdown and removal of food particles in plaque that provide "food" for the bacteria. Needless to say, starving the little buggers is beneficial. It's pretty easy to acclimate a pet to routine toothbrushing. My new Corgi pup has become so habitual about toothbrushing that she actually patiently waits in the bathroom to have her teeth brushed while we brush our teeth. Prevention is key. And the lion's share of prevention rests with the owners.

For those dog owners who love to argue that wild canids, such as foxes, coyotes, and wolves, don't get periodontal disease and the systemic consequences that stem from it—really? How do they know that? Now, admittedly, by biting, ripping, and chewing on the tissues of their prey using the whole dental arcade, they probably do remove some plaque and maybe even some softer tartar. Periodontal disease may still develop. And if the average life span of many wild canids ranges from 2 to 6 years or so, how do they know that periodontal disease is not a factor contributing to the early demise of those animals? And let's consider domestication. We routinely feed commercial diets (canned, soft-moist, and dry) to dogs. Particles from these foods in plaque provide a great energy source on which bacteria can flourish. (This is really true for soft-moist diets, because they contain more carbohydrates in the form of sugars.) Owners simply need to take responsibility for their pets' oral hygiene. Period.

Feline Resorptive Lesions

Cats have a very unique dental disease that, for them, is probably more common than periodontal disease. Cats frequently develop ***resorptive lesions***. Inflammation from bacterial activity in their mouths stimulates ***odontoclastic*** [*odont(o)-* tooth + *clast(o)-* breaking + *-ic* pertaining to] activity. *Odontoclasts*, like their "cousins" the ***osteoclasts*** [*oste(o)-* bone + *clast* breaker] discussed inChapter 4, actually dissolve the mineralized structure of teeth (enamel, dentin, and cementum). Like periodontal disease, the development of ***feline***

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odontoclastic lesions begins with plaque and the bacteria it contains. The unique oral bacterial population and a cat's unique inflammatory responses promote odontoclastic activity. So, once again, gingivitis develops early on. Odontoclasts in the vicinity of the ***cementoenamel*** [*cement(o)-* cementum + *enamel*] ***junction***, under the cover of the gingival margin, are stimulated. So, odontoclastic lesions begin at the *cementoenamel junction*—often called the "neck" of a tooth (Fig. 7.7). That is why these lesions are often called "***neck lesions***."

There is an unusual interplay between the odontoclastic activity and the gingiva. As you would imagine, exposure of dentin is painful. We said so earlier. And these lesions can be very painful for cats. They may stop eating because of it. But for some, the pain is minimized by the gingiva. How? Well, ***gingival hyperplasia*** [*hyper-* excess + *plas(o)-* formation + *-ia* condition of] frequently develops, creating a "Band-Aid" of sorts over the odontoclastic lesion. So, if you ever see *gingival hyperplasia* in a cat (anesthetized of course!), carefully use your dental probe to peek under the ***hyperplastic*** [*hyper-* excess + *plas(o)-, plast(o)-* formation + *-tic, -ic* pertaining to] gingiva. I'll bet my bottom dollar that you'll find an odontoclastic lesion there. I have peeked under *hyperplastic gingiva* many times. Sometimes, the odontoclastic lesions I've seen were so large and deep that the pulp was exposed and bulging from the defect. No wonder those cats had oral pain! Just imagine the throbbing they must have felt.

How do we treat odontoclastic lesions? Well, there are two schools of thought. Because the whole tooth will eventually be resorbed, we can wait it out. Of course, this is provided the cat is not symptomatic (i.e., experiencing significant pain). The other option is to remove the diseased tooth. This is a very traumatic procedure, because the roots of these teeth are well attached. So, we physically have to use dental elevators to break down the periodontal attachments and basically "dig" the roots out. The remaining exposed periodontal bone and tissues will be painful, until the site heals. So, the question is: which is the lesser of two evils for an individual cat? The best solution is preventing resorptive lesions in the first place. How? Plaque and the harmful bacteria it harbors need to be removed before it causes disease. The

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bulk of that burden falls to the owner. Brushing the cat's teeth is the best preventive measure an owner can employ.

Gastrointestinal Transit

Now that we've talked about the mouth, let's follow a bolus of food through the rest of the GI tract. We'll pause at key places along the way to discuss the importance of each location. In the mouth, we said that the tongue helped form and move the food bolus into the oropharynx and pharynx.

Pharynx and Esophagus

In the pharynx (throat), there is some really important muscular activity that takes place. First, we know that the soft palate moves dorsally to protect the nasal passages. Simultaneously, ***laryngeal*** [*laryng(o)-* larynx + *-al* pertaining to] muscles draw the arytenoids together and draw the epiglottis over them to protect the trachea and lower airways. (Refer toChapter 5 to review airway anatomy and function.) ***Pharyngeal*** [*pharyng(o)-* pharynx + *-al* pertaining to] muscles also contract to push the food bolus into the esophagus.

The ***esophagus*** is a muscular tube that connects the oral cavity and the throat to the stomach. But this is no passive tube relying on gravity to move contents. First, gravity doesn't work, when most animals (especially domestic animals) walk on all fours. The esophagus is virtually horizontal, especially as it traverses the chest. So, we need a mechanism that pushes food and liquids to the stomach. That mechanism is called ***peristalsis*** [*peri-* around, near + *-stalsis* contraction]. *Peristalsis* is a rhythmic, coordinated wave of muscular contraction that pushes things along (Fig. 7.11A). In the case of esophageal muscles, that ***peristaltic*** [*peristal(o)-* peristalsis + *-tic* pertaining to] wave pushes food and liquids into the stomach.

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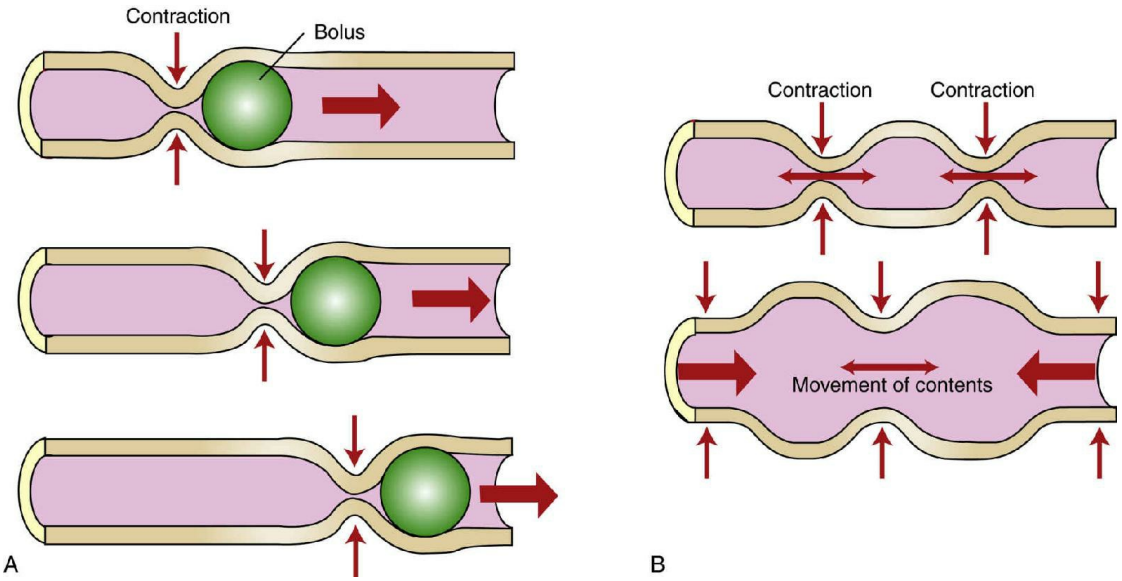

FIG. 7.11 (A) Peristalsis vs. (B) segmentation.
From Colville & Bassert, *Clinical Anatomy and Physiology* 2e, Mosby/Elsevier, 2008, 267.

Stomach

The ***stomach*** (Fig. 7.12) is a muscular temporary holding-tank/mixing chamber in the left cranial abdomen. As the food bolus is pushed through the esophagus, the muscles of the distal esophagus (sometimes called the ***cardiac sphincter*** [sfingk'ter, Gr. *sphinkter* that which binds tight]) relax to allow easy passage of the food into the stomach. The rest of the time, those muscles keep the entrance to the stomach tightly closed. This is important to prevent ***gastroesophageal*** [*gastr(o)-* stomach + *esophag(o)-* esophagus + *-al* pertaining to] ***reflux***. Acidic ***gastric*** [*gastr(o)-* stomach + *-ic* pertaining to] juices can do an awful lot of damage to the esophagus.

The stomach is divided into regions. The gastric ***fundus*** is the cranial curved, billowing portion of the stomach near the cardiac sphincter. The gastric ***body*** is the largest, middle portion of the stomach. And the ***pylorus*** (pi-lor'us [Gr. *pyloros* gate guard]) is the tapered, funnel-shaped portion that leads to the small intestine. And the *pylorus*, by virtue of the ***pyloric*** [*pylor(o)-* pylorus + *-ic* pertaining to] ***sphincter***, really is a "gate guard." You see, the *pyloric sphincter* only allows small volumes of stomach contents at a time to squirt into the duodenum.

The stomach wall itself has a number of layers. The ***serosal***

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[*seros(o)-* serosa + *-al* pertaining to] ***surface*** on the exterior is covered by *visceral peritoneum*. This layer is called ***serosa***, because cells in the tissue actually produce small amounts of slippery, ***serous***-like [*ser(o)-* serum + *-ous* pertaining to] fluid that allows *serosal surfaces* of abdominal organs to slide over one another. Next, there is a powerful muscle layer. The ***gastric muscles*** are important for mixing gastric contents with the gastric juices. And, as mentioned earlier, coordinated muscular contractions through the pylorus are responsible for squirting gastric contents into the duodenum. Then, we have the ***submucosal*** [*sub-* below + *mucos(o)-* mucosa + *-al* pertaining to] ***tissue layer***. The ***submucosa*** is a rich vascular layer made of supportive connective tissues. Finally, the inner most is the ***mucosal layer***. Here is where we find a variety of secretory cells, for mucus, acid, and proenzyme secretions. All of those cells are important. But if I had to choose one type that's most important, I'd pick the ***mucus***-secreting cells. Because without a really thick layer of mucus covering the mucosal tissue, the tissue would be seriously damaged by the gastric acid and activated enzymes. That's how gastric ulcers develop, you know. Anything that disrupts mucus production and the thick layer of mucus reduces protection of the underlying tissues.

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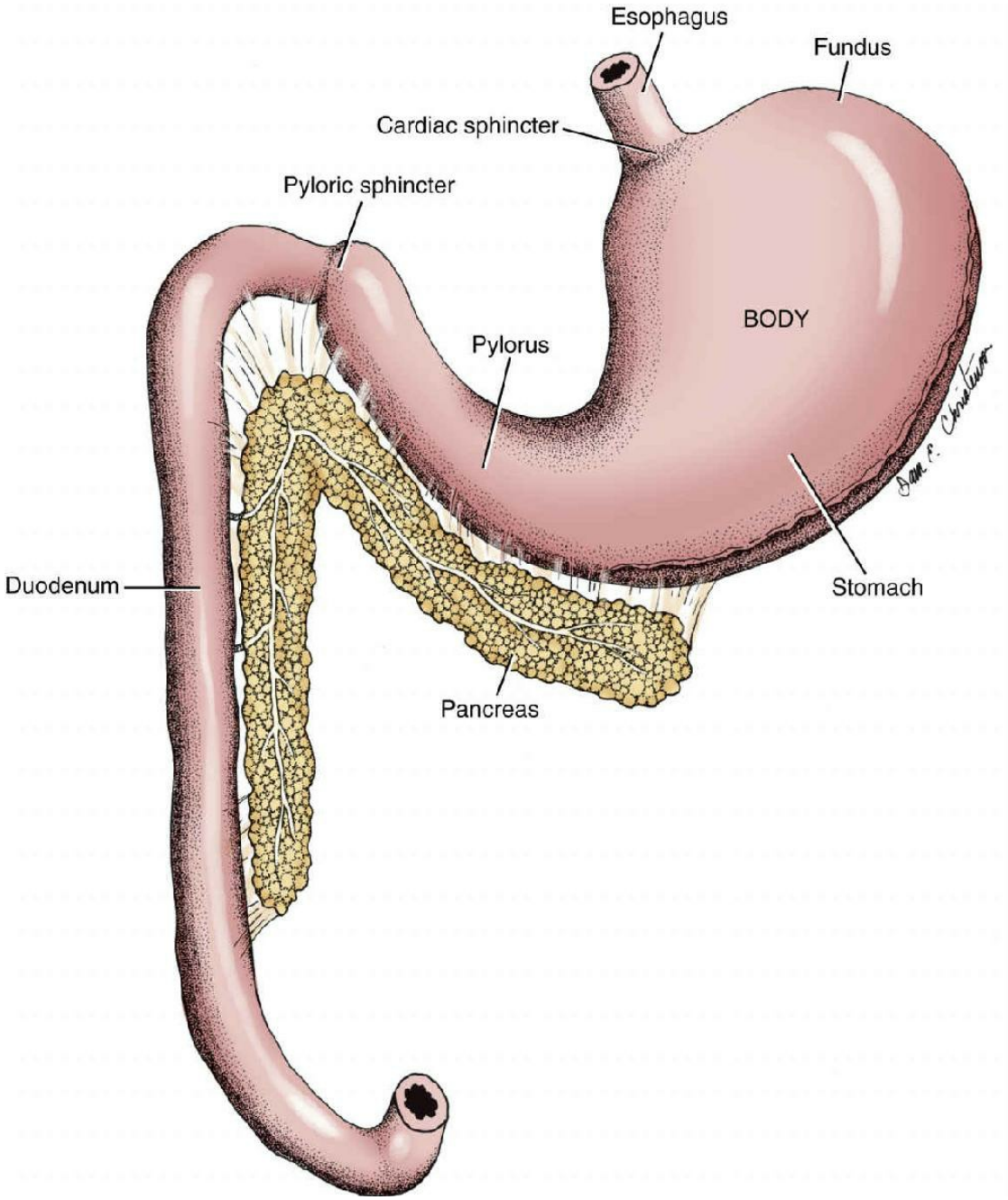

FIG. 7.12 Canine stomach, duodenum, and pancreas.

Now, when the stomach is empty, these tissue layers form all sorts of folds, called ***rugae*** (roo'ga [from L. *ruga* a ridge, wrinkle or fold]). These folds go away as the stomach fills. I like to compare rugae to all of the wrinkles of a raisin. A raisin is a shriveled-up grape, right? But if you put a raisin in water, it will soak up water like a sponge and become plump, stretching out all of the wrinkles. Of course, the stomach isn't a raisin or a sponge. But when the stomach gets full, all of the *rugae* get stretched out. Long story short, it gives the stomach a large temporary storage-tank capacity.

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Gastric Secretions

There are three primary gastric secretions: *mucus*, ***pepsinogen*** [*pepsin* + *gen(o)-* producer], and hydrochloric acid (HCl). Plus, to a lesser extent, bicarbonate is secreted. As I said earlier, there are a variety of gastric mucosal cells that are responsible for secreting these things or components of them.

Mucus and Bicarbonate

The thick ***mucus*** provides a protective barrier for the *gastric mucosa*. But when the gastric acid with a pH of around 2 (that's pretty darned acidic) could peel paint, what's going to keep it from destroying the mucus? ***Bicarbonate*** is at the other end of the pH spectrum, right? It's alkaline. So, to minimize complete destruction of the mucus, bicarbonate is secreted to bathe the surface of the mucus. This helps neutralize some of the acid coming in contact with the surface. Obviously, protection is a constant battle in such a harsh environment. So, both mucus and bicarbonate are secreted constantly. Anything that interferes with their secretion puts the mucosa and underlying tissues at risk. And if those tissues are exposed to gastric acid and enzymes, ***gastritis*** [*gastr(o)-* stomach + *-itis* inflammation of] will develop. And if the exposure is significant and prolonged, ***gastric ulceration*** will probably develop. The stomach wall is relatively thick but not impenetrable. Imagine the consequences if a ***gastric ulcer*** becomes so deep that the stomach wall actually perforates. Not good. Not good at all. Do you see why I said earlier that the mucus layer is so important?

Pepsin and Pepsinogen

We talked about proteolytic enzymes earlier, when we discussed the pancreas. Well, ***pepsin*** is another *proteolytic enzyme*. If you remember, we said that pancreatic proteolytic enzymes couldn't be stored by the secretory cells because they'd be digested from the inside out. The same is true for *pepsin*. So, like the pancreatic enzymes, gastric secretory cells produce a *proenzyme*: ***pepsinogen***. When *pepsinogen* interacts with HCl, it is converted to the active proteolytic enzyme: ***pepsin***. And while pepsin is proteolytic, it only *initiates* protein digestion. It's a lot of work to break down large protein molecules into amino acids. Pepsin won't get us there. But it

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will make a dent in the process. We'll have to rely on pancreatic enzymes to complete the job. But here's the cool thing—the smaller protein molecules that are produced by pepsin activity actually stimulate more release of the hormone ***gastrin***. And gastrin, in turn, stimulates more secretion of pepsinogen and HCl. This is a great example of a feedback loop. (We'll talk more about feedback loops inChapter 10.)

Hydrochloric Acid

Obviously, if ***HCl*** can peel paint and "eat" through all sorts of substrates, gastric cells can't store HCl ready for secretion. Then, how do we get HCl? It's simple chemistry. (Go toChapter 2 if you want to review molecules and molecular bonds.) Gastric cells, specific for the task, secrete the ***hydrogen*** (H^+) and ***chloride*** (Cl^-) ions separately. Opposites attract. So, when H^+ and Cl^- meet in the lumen of the stomach, they rapidly form a ***covalent*** [*co-* together + *val(o)-* strong + *-ent* one that is] ***bond***. It's a match made in heaven. And that's how HCl is "born."

But the cells secreting these ions aren't doing it all of the time. Three receptors on each of those cells need to be engaged. All three receptors are on the vascular side of the cells (i.e., opposite from the secretory side near mucosal surface). One receptor is activated by ***gastrin*** [*gastr(o)-* stomach + *-in* the]. Gastrin is actually a hormone, secreted into the bloodstream by the stomach wall. When it circulates back around to the gastric mucosa, it binds to receptors on the secretory cells. Then, there is a ***cholinergic*** [*cholin(o)-* acetylcholine + *erg(o)-* work + *-ic* pertaining to] receptor. We mentioned ***acetylcholine*** (uh-se'tul-ko"lēn) inChapter 4. (Please refer toChapter 4 to review that discussion. You may also read ahead toChapter 11 for a more detailed discussion.) We learned there that *acetylcholine* is a major ***neurotransmitter*** [*neur(o)-* nerve + *transmitter*] in the body. And it is the sole neurotransmitter used to stimulate activity along the digestive tract. In the case of our secretory cells for HCl, *acetylcholine* is necessary to activate their secretion of H^+ and Cl^-. But that can't happen until the final receptor is activated by ***histamine***. I know, when we discussed immunology inChapter 3, we talked about *histamine* as it relates to inflammation and allergic responses. Well, gastric production of

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HCl is another very important role for histamine. And this is the one place of activation that's easy to target, if we want to reduce the amount of gastric acid. You've probably heard of numerous drugs, such as famotidine (Pepcid), cimetidine (Tagamet), ranitidine (Zantac), and others. These types of ***antacids*** [*ant-*, *anti-* against + *acid*] are also referred to as ***H_2 blockers***. ***H_2 receptors*** are the specific receptors on these gastric secretory cells that are activated by histamine. So, by blocking the H_2 receptors, histamine can't activate them. No histamine activation, game over—acid production of HCl is dramatically reduced.

If all three receptors are activated, where do the ions that the cells secrete come from? The cells draw them from the bloodstream. As far as hydrogen ions are concerned, meals actually deplete circulating hydrogen ions, resulting in a temporary shift in systemic pH. It becomes a tad alkaline after eating. This phenomenon is called the ***postprandial*** [*post-* after + *prandi(o)-* meal + *-al* pertaining to] ***alkaline tide***. This is really a minor shift from neutral. And the body has numerous ***homeostatic*** [*home(o)-* unchanged + *-static* pertaining to standing; i.e., equilibrium, balance, stability] mechanisms in place to shift the body's pH back to a normal, neutral state. I point it out, because there are times when this alkaline state may become prolonged and contribute to other problems. In Chapter 6, we discussed bladder stones. We said that struvite crystals and stones form in an alkaline environment. So, if a cat or a dog who is predisposed to struvite crystal formation is allowed to graze on food all day, that eating behavior will perpetuate the *postprandial alkaline tide*. Now, the alkalinity is no longer a tide that comes and goes. It's a constant, resulting in alkaline urine. And in predisposed animals, it may contribute to struvite crystal and stone formation.

A more severe ***alkalosis*** [*alkal(o)-* alkaline + *-sis* condition of] creates greater problems for the body. Let's face it; the body doesn't like being "off-balance." But how could gastric activity contribute to *alkalosis*? Let's think about *gastritis* for a moment. If you've ever had it, you know that it can make you pretty nauseated. And when the nausea gets really bad, it usually makes us vomit. With ***emesis*** [from Gr. *emein* to vomit + *-sis* process of], we lose tons of HCl with the gastric contents. With each ***emetic*** [Gr. *emein* to vomit + *-tic*

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pertaining to] episode, more HCl is lost, and that means the body is losing significant quantities of hydrogen ions. So, if gastritis is severe enough to cause lots of *emesis*, *alkalosis* is bound to develop. Other electrolyte imbalances, such as ***hypokalemia*** [*hypo-* low + *kal(o)-* potassium + *-emia* a blood condition of], can accompany this. *Emesis* is almost always preceded by *hypersialosis*. And salivary secretions contain numerous electrolytes, potassium being an important one. So, excessive *hypersialosis* and *emesis* may precipitate *hypokalemia* in addition to the *alkalosis*. This is why, due to concerns over dehydration, alkalosis, and other electrolyte disturbances produced by vomiting, we often administer ***antiemetic*** [*anti-* against + *eme(o)-* vomiting + *-tic* pertaining to] medications—by injection of course! Otherwise, the animal will simply vomit up an oral drug.

Emesis vs. Regurgitation

Since we're talking about vomiting, what is the difference between emesis and regurgitation? ***Emesis*** is the abnormal, ***retrograde*** [*retro-* backward + *grade* to step, move] forceful evacuation of stomach contents. If the stomach is empty, vomiting episodes can actually bring up bile from the proximal duodenum. The actual act of vomiting is physically intense. (Believe it or not, I actually had a student who claimed to have never experienced vomiting. I found that amazing. Perhaps her mind simply blocked the awful experience.) Emesis involves contraction of both gastric and abdominal muscles. Most dogs and cats usually go through a series of strong abdominal contractions that you can easily see. And then there's the characteristic sound they make—sort of an "uhmfrook, uhmfrook, umfrook" noise—with each contraction. It's an unmistakable sound that is better than any alarm clock on the planet for most pet owners who hear it in the middle of the night. Of course, we dart out of bed in hopes that we can get the animal off of the bed and/or the carpet, before the beast actually vomits their payload. But the payoff of their retching is usually there faster than we are. No matter if it's us or our pets who are vomiting, it is a *very* unpleasant experience to say the least.

By the way, if vomiting is projectile (i.e., shooting out of the mouth over quite a distance), we often suspect a problem with the

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pylorus. Obstructive foreign bodies are one of the more common causes. Many gastric foreign bodies can be removed with ***gastroscopy*** [*gastr(o)-* stomach + *-scopy* viewing]. And they probably should be, even if they're not obstructive yet. Depending on the object, if it passes into the small intestine, there is a high probability that it will create an intestinal blockage. But if we're unable to remove a gastric foreign body with *gastroscopy*, a ***gastrotomy*** [*gastr(o)-* stomach + *-tomy* to cut] may be necessary. Abdominal ultrasound is very valuable in isolating the location of such foreign bodies.

Regurgitation [*re-* back + *gurgit(o)-* flooding + *-tion* the act of] is different. *Regurg*, as we so often abbreviate the word, has very little physical activity associated with it. There is no preparatory abdominal contraction or noise to warn us of what's coming. Often, animals simply appear to gag and deposit a pile of food or other material in one easy motion. Much of the time, regurg comes from the esophagus. That's usually pretty easy to spot, especially if it's been sitting in the esophagus for a while. It looks like a ***mucoid*** [*muc(o)-* mucus, slime + *-oid* resembling] tube of food. The *mucoid* coating is a combination of mucus and saliva. *Regurgitation* might contain some stomach contents, if the animal has *gastroesophageal reflux*. But generally, the contents of whatever the animal regurgitates are undigested material from the esophagus.

Why distinguish between regurgitation and vomiting? Well, they point to completely different problems. Causes of vomiting are numerous and may be either local (i.e., the stomach) or systemic in origin. Regurgitation usually points to an esophageal problem. ***Esophagitis*** [*esophag(o)-* esophagus + *-itis* inflammation of] and *esophageal obstruction* are two common causes of regurg that are treatable and probably curable. But ***megaesophagus*** [*mega-* large, enlargement of + the *esophagus*] is a serious problem that usually requires life-long management. Why is this such a serious problem? Well, first of all, food and liquids are not advancing, as they should into the rest of the GI tract for digestion and absorption of nutrients. Food and liquids sitting in the dilated esophagus are awfully close to the larynx. With that close proximity and the act of regurgitation itself, these animals can easily aspirate material into their airways. That can be deadly. Our geriatric Gordon Setter was developing

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megaesophagus late in her life. Plus, she was developing ***laryngeal*** [*laryng(o)-* larynx + *-al* pertaining to] ***paralysis***. That magnified her risk of aspiration. Often, the two conditions go hand in hand. So, in answer to the question "is it important to distinguish between vomiting and regurgitation?"—you bet it is. But be prepared, many pet owners don't know the difference. So, your history-taking needs to be extremely detailed. I often demonstrated the actions (and sounds) associated with them for owners. You need to do whatever it takes to help make an accurate diagnosis.

Gastric Dilatation Volvulus

GDV stands for ***gastric dilatation*** [from L. *dilatare* to spread wide] ***Volvulus*** [from L. *volvere* to twist around]. The term *dilatation* indicates the state of a structure that is dilated and stretched beyond its normal dimensions. For that reason, *GDV* is commonly called "***bloat***." But *GDV* is not simple like food bloat. At least with food bloat, we can typically pass an ***orogastric*** [*or(o)-* mouth + *gastr(o)-* stomach + *-ic* pertaining to] tube to relieve the gastric distention, evacuate all of the gastric contents, and ***lavage*** [Fr. *lavage* to wash out] the stomach. That's not possible in *GDV*. Both GDV and simple bloat can be life-threatening. But unless we perform an emergency *laparotomy* for the dog with GDV, it will die.

Who is most at risk of developing GDV? Technically, any dog could develop GDV. But deep-chested breeds, such as Great Danes, German Shepherds, Bloodhounds, Doberman Pinschers, Irish Wolfhounds, Newfoundlands, and other such breeds are most at risk. Even my Basset Hound, Sadie, developed GDV. (I loved that dog to pieces, but wow she was a problem child!) Deep-chested breeds are most at risk, because the deep chest structurally creates more room in the cranial abdomen for the stomach to flip over and twist (volvulus) on its own axis.

How does GDV develop? There are many factors that may contribute to it, including anatomy (as we already mentioned), gastric emptying time, prior "bloat" events, regular ingestion of large meals, and so forth. Slow gastric emptying keeps food in the stomach longer than normal. Anything that stretches gastric ligaments (like prior bloat events and eating large meals) creates greater freedom of movement of the stomach within the abdomen.

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With or without predisposing factors like these, the most common scenario in GDV cases is this: the dog consumes a large meal or large volume of water and then engages in physical activities, such as running or playing. The weight of the stomach, with the physical activity, makes it swing like a pendulum. End result: the stomach swings too far (beyond 180 degrees) and flips/rotates on its own axis (volvulus). With gastric ***torsion*** [L. *torsio* to twist], both the gastric entrance (gastroesophageal) and exit (gastroduodenal) are twisted tightly shut. Nothing can enter, and nothing can exit. With the "front door" shut tight, the dog can't belch to relieve the gas that develops, and we cannot pass an orogastric tube to evacuate the stomach. With the "back door" shut tight, gastric contents have nowhere to go. Secretions pour in, food (especially kibble) expands, and gases build up—all of that stretching the stomach like an overinflated basketball.

What symptoms might alert an owner that the dog has GDV? Abdominal distention is easily spotted in a dog with a normally "skinny waist" like a Doberman or Greyhound. In a heavier dog with a thicker coat like a Newfoundland, it may be more difficult to see right away. There are other subtle signs, such as depression, reluctance to move, the head held at an odd low angle, and hypersialosis. But the one symptom that should set off red flags, sirens, and flashing lights is ***unproductive retching***. These dogs will make many attempts at vomiting, going through all of the extreme muscular activity that we discussed earlier. But there is no way anything is going to leave that stomach. It can't. The stomach is twisted at both ends like a candy wrapper. If an owner ever tells you over the phone that their dog has unproductive retching, tell them to take the dog to the **nearest** veterinary facility as quickly as humanly possible. Make it crystal clear—**THIS IS AN EMERGENCY**! By the way, this is no time to be territorial about our veterinary clients. That dog needs emergency care NOW, preferably in 30 minutes or less. I've seen dogs die from GDV in 30 minutes. This is no joke. So, if our facility is 45 minutes to an hour away from the owner, but a competitor's is 15 minutes away, the owner needs to take the dog to our competitor. The patient and its needs always come first. And this dog needs emergency care ASAP to save its life.

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Why is GDV so life-threatening? If you think that this is just a digestive problem, you're wrong. GDV will impact virtually every body system. And when it comes to those systems essential for sustaining life, such as the ***cardiopulmonary*** [*cardi(o)-* heart + *pulmon(o)-* lungs + *-ary* pertaining to] system, things can deteriorate rapidly to the point of death. Let's see if we can figure out why. (If you need to, please refer toChapter 5 to review topics such as blood pressure, breathing, and gas exchange.) There are a number of factors impacting circulation and gas exchange.

First, the stomach is bloated beyond belief, right? I mean it is so stretched that if you tap your finger on it through the abdominal wall, it will "***ping***" like a fully inflated basketball. (We call that ***tympany***. Yes, like a drum.) With that kind of extreme distention (*dilatation*), excessive pressure is placed on the caudal vena cava. At first, the pressure exerted on it by the stomach will decrease blood returning to the heart from the abdomen, but eventually, it will block ***venous*** [*ven(o)-* vein + *-ous* pertaining to] return from the abdomen completely. This markedly reduces circulating blood volume.

Second, the spleen, by virtue of its proximity and ***omental*** [*oment(o)-* omentum + *-al* pertaining to] attachments to the stomach, often becomes involved in the *torsion*. We said inChapter 3 that the spleen stores roughly 30% of the body's blood volume. If it too is twisted with the stomach, cutting off circulation to it, we have created an acute (sudden) 30% reduction in circulating blood volume. Between this and all of the blood trapped in the abdomen because of the gastric dilatation, we do not have enough blood volume to maintain blood pressure. Circulating blood volume can easily be reduced by 50% or more. So, life-threatening ***hypotension*** [*hypo-* low + *tens(o)-* pressure + *-ion* state of] develops very rapidly. Does the body engage ***cardiovascular*** [*cardi(o)-* heart + *vascul(o)-* vessels + *-ar* pertaining to] mechanisms to raise blood pressure? Yes, of course. The heart speeds up and pumps harder, and peripheral vessels constrict. All *cardiovascular*, hormonal, and neurologic mechanisms are put to use to elevate the blood pressure. But it's not enough. We simply don't have enough blood volume available.

Third, excessive pressure exerted on the diaphragm inhibits its

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movement. The dog cannot breathe in or out with normal volumes of air. The dog's gas exchange is severely compromised because of it. The dog can't take in enough oxygen, and it can't exhaust enough carbon dioxide (CO_2). It struggles for each breath. The dog's struggle to breathe along with all of its unproductive retching produces excessive amounts of CO_2. ***Acidosis*** [*acid* + *-osis* condition of], profound *acidosis*, is inevitable. Finally, adding insult to injury, the *acidosis* that develops and the ***hypoxia*** [*hypo-* low + *ox(o)-* oxygen + *-ia* condition of] often lead to serious abnormal heart rhythms. Now, the heart is unable to compensate at all.

So, our initial emergency response to stabilize the patient for surgery is by improving the circulating blood volume with IV fluids and partial decompression (pressure relief) of the stomach. Gastric decompression is also beneficial for respiratory function by allowing room for the diaphragm to move. Deeper breathing facilitates better gas exchange. Because we cannot decompress the stomach with an orogastric tube, we need to ***trocarize*** [*trocar* + *-ize* act of]. A ***trocar*** (tro'kar) is an instrument used to puncture the wall of a body cavity. In this case, we'll be puncturing the abdominal wall and stomach simultaneously. A large gauge (e.g., 18 Ga) needle works quite well. It's held in place momentarily, while much of the pressure and gas are evacuated. If conscious, the patient usually looks relieved, and its breathing becomes less labored. Could *trocarizing* result in peritoneal contamination? Yes. But that is the least of this dog's worries right now. Abdominal radiographs may be taken at this point, but only if the dog is stable and the imaging can be done quickly. The classic "double bubble" produced by the abnormal position of the gas-filled pylorus over the gastric body and fundus is helpful in confirming the *volvulus***.** But—and this is a big but—imaging should not jeopardize the patient. In GDV, our goal is to stabilize and get the dog prepped and into surgery as quickly as humanly possible. Every. Second. Counts.

During the *laparotomy*, our first order of business is to ***detorse*** [*de-* reverse + *torsio* to twist; i.e., untwist] the stomach. Then the stomach is examined carefully for restoration of circulation to it. If any portion of the stomach appears *necrotic*, a partial ***gastrectomy*** [*gastr(o)-* stomach + *-ectomy* to cut out, remove] will be needed. The

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same is true for the spleen. So, a ***splenectomy*** [*splen(o)-* spleen + *-ectomy* to cut out, remove] may also be necessary. The very last thing most surgeons do, before closing the abdomen, is a ***gastropexy*** [*gastr(o)-* stomach + *-pexy* fixation of]. In a *gastropexy*, the stomach wall is actually sutured to the abdominal wall. This is done to minimize the dog's risk of future GDVs. Aside from this emergency surgery situation, *gastropexies* are often done preemptively in young, healthy dogs (especially deep-chested dogs). No *gastropexy* can ever guarantee that a dog will not develop a GDV, but it definitely reduces the chances of it.

If our GDV patient survives the surgery, the dog could still die, especially over the next 48 hours. Abnormal heart rhythms may persist during this time. Remember the severe *hypotension* that developed in the patient? Do you also remember from Chapter 6 the most important factor influencing kidney function? That's right —blood pressure. So, with the profound low blood pressure experienced by this patient (that probably persisted through much of the surgery and perhaps after), the dog could develop ***acute*** (sudden) ***renal*** [*ren(o)-* kidney + *-al* pertaining to] ***failure***. Oh, and let's not forget all of the blood, especially mesenteric blood, that was trapped and sitting stagnant in the abdominal vessels. This is the perfect storm for activating the clotting cascade. During surgery, when the stomach was *detorsed* and decompressed, and the stagnant blood returned to general circulation, the clotting cascade may have been activated body-wide. If that happens, the dog could easily use up all of its clotting factors and platelets. InChapter 3, we talked about a clotting disorder called ***disseminated*** (widespread) ***intravascular*** [*intra-* within + *vascul(o)-* vessels + *-ar* pertaining to] ***coagulopathy*** [*coagul(o)-* clotting + *-pathy* a disease of] or ***DIC***. If *DIC* develops (and it easily could after a GDV), our patient could bleed to death. The dog has no clotting factors. And it's going to take the dog some time for its liver to produce enough to stop and prevent bleeding. Until then, life-threatening bleeding is a very real concern.

Gastric dilatation volvulus is no picnic. Many dogs don't survive it or its secondary complications. And survivors are more likely to experience GDV again. The next time the dog may not be so lucky. So, beyond a gastropexy, there are some things the owners should

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do to reduce the dog's risk of future GDV. First, feeding smaller, more frequent meals may help. (The stomach will be less pendulous.) Once-a-day feeding is too great a volume. Second, they should discourage drinking large volumes of water, especially after a meal of kibble. Food, especially dry kibble, absorbs water and expands in volume. Third, owners should prohibit play and exercise for at least a couple of hours postprandial. Finally, owners need to be educated in the warning signs of GDV and seek prompt emergency care if they observe any of those signs.

Small Intestine

The ***small intestine*** is the longest portion of the digestive tract. How long? Well, some anatomists calculate that a dog's small intestine is roughly 3.5 times the animal's body length. That would mean my Cardigan Welsh Corgi, measuring 35 inches from nose to rump, has over 10 feet of small intestine in her belly. That's long! Boy, I hope she never gets small bowel diarrhea! Over that length, the small intestine is subdivided into three segments. We'll discuss the importance of each segment in a moment. In general, when thinking about the small intestine, let's think of it as the workhorse of the digestive tract. It's in the small intestine that most of the digestion and absorption of nutrients take place. And especially when it comes to absorption of nutrients, we need an awful lot of surface area. Now, there's a great deal of surface area in the small intestine, by virtue of the tremendous length of the tubular structure. But if we were to look at a ***histologic*** [*hist(o)-* tissue + *log(o)-* knowledge, study of + *-ic* pertaining to] sample of the ***enteric*** [*enter(o)-* intestine + *-ic* pertaining to] mucosa under the microscope, we'd see how the small intestine has boosted its surface area. That amplification of surface area along the small intestine is all because of the ***villi*** (plural of ***villus*** [L. *villus* tuft of hair]).

In Fig. 7.13, you'll find a schematic of two *enteric villi*. As you can see, villi are finger-like projections. These little "tufts of hair," as ancient anatomists described them, are the true workhorses of the small intestine. And while they boost the surface area exponentially, it doesn't end there. If we were to look at these villi under higher magnification, we'd see that the surface of each epithelial cell is covered in ***microvilli*** [*micro-* small + *villi* "tufts of

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hair"]. Talk about surface area! And all of that surface area is necessary to expedite absorption of nutrients. And look at the center of each villus. We have capillaries and *lacteals* readily available to carry off absorbed nutrients. The epithelial cells are the gatekeepers. Nutrients need to be absorbed through diffusion or active transport, before they can make their way into circulation. Finally, notice the crypts at the base of each villus. Here is where we have a large number of specialized epithelial cells, called ***goblet cells***, which produce mucus. The mucus on the mucosal surface of the small intestine is not near as thick as that of the stomach. Yet, it is still protective. After all, think about all of the enzymes that the mucosa is exposed to. So, a protective film of mucus is necessary.

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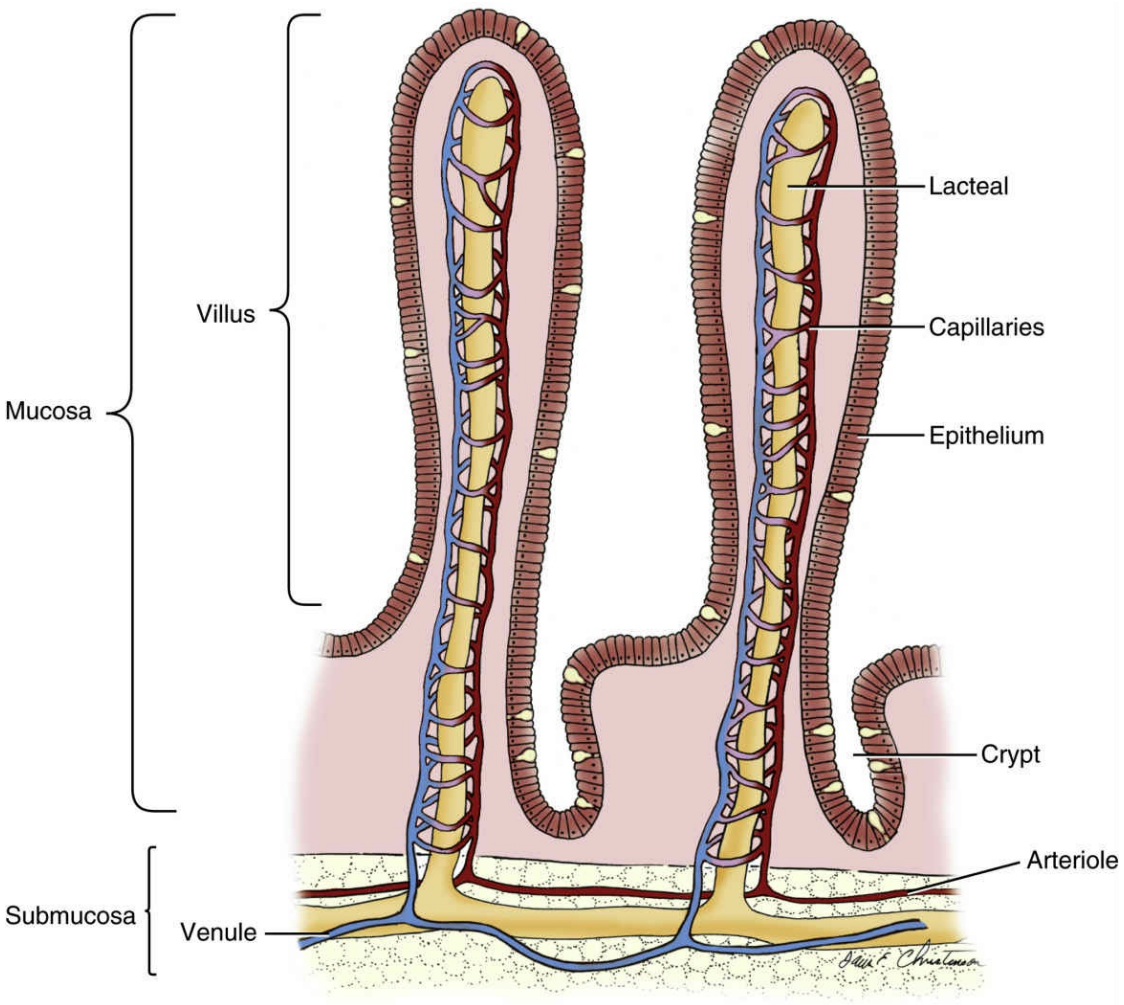

FIG. 7.13 Enteric villi.

Like the stomach, the small intestine has layers. On the interior, we have the *mucosa*, as you saw in Fig. 7.13. Under that is the *submucosa*. And under the submucosa is not one, but two muscle layers. Why on earth are there two? Well, it's not redundancy, in case one fails. The muscle fibers are actually arranged differently in each layer. Working in tandem, both muscle layers facilitate ***segmental*** [*segment(o)-* segment, segmentation + *-al* pertaining to] mixing action (Fig. 7.11B) and powerful *peristaltic* action (Fig. 7.11A) to mix and move enteric contents along. These movements are shown in the *Peristalsis* animation in the Evolve resources. Finally, on the exterior surface of the small intestine is the serosa.

Enteritis

Before we discuss each section of the small intestine, let's dwell a little longer on its powerful peristaltic action. Anything that causes

enteritis [*enter(o)-* intestine + *-itis* inflammation of] typically accelerates peristalsis. There are any number of viruses and ***pathogenic*** [*path(o)-* disease + *gen(o)-* producing + *-ic* pertaining to] bacteria that can cause *enteritis* and ***gastroenteritis*** [*gastr(o)-* stomach + *enter(o)-* intestine + *-itis* inflammation of]. Some common viral ***pathogens*** [*path(o)-* disease + *gen(o)-* producer] include *transmissible gastroenteritis* (*TGE*) in pigs, *canine parvovirus* and *coronavirus* in dogs, and *feline panleukopenia* in cats. And when any *pathogen* like these viruses create enteritis, they accelerate peristalsis and promote profound diarrhea. Many lumen-dwelling intestinal parasites create enteritis and screaming diarrhea too. *Coccidia* (kok'sid-e-uh; a class of microscopic, protozoal parasites) are significant in this regard. These tiny pathogens actually invade and destroy the mucosal epithelium by the truckloads. Inflammation? —It's severe.

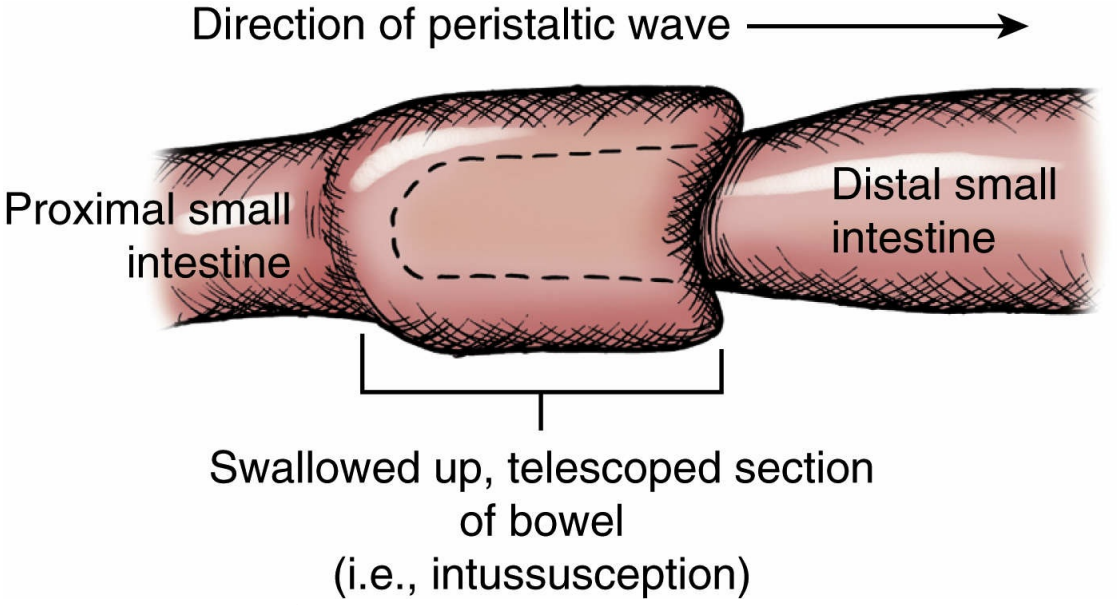

FIG. 7.14 Intussusception schematic.

Intussusception and Intestinal Foreign Bodies

The more intense the enteritis, the more powerful the peristaltic activity in response to the inflammation. It can be so powerful that a really strong, tsunami-like, fast-moving peristaltic wave can actually overrun an adjoining section of small intestine. That's right,

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the fast-moving section "swallows" the adjoining section (Fig. 7.14). We call this "telescoping" or "swallowing up" of one section of bowel into another an ***intussusception*** [from L. *intus* within + L. *suscipere* to receive + *-tion* act of]. I've seen numerous dogs and cats suffering with enteritis from viral and parasitic *pathogens*, who developed *intussusceptions*. Another more common cause of intussusceptions is an enteric foreign body. With obstructive foreign bodies (often toys and other unusual objects), as the peristaltic wave grows stronger and stronger in unsuccessful attempts to move the object along, the proximal section of bowel envelopes (swallows up) the adjoining section with the object. Linear foreign bodies, such as string, yarn, ribbon, socks, and pantyhose, are notorious for creating intussusceptions. Have you ever climbed a rope or perhaps watched someone climb a rope? The rope is gripped at successive points along the rope, with the hands, legs, and feet. With each successive grip, the climber moves up the rope. In the case of an *intussusception* caused by a linear foreign body, each successive "grip" made by the intestinal wall with each peristaltic wave strengthens the propulsion of the proximal section of bowel over the adjoining segment.

Regardless of cause, one of the greatest concerns with an intussusception is ***necrosis*** [*necr(o)-* death + *-sis* condition of]. The physical nature of the folded-over, telescoped section of intestine disrupts blood supply to the involved segment. The longer the intussusception exists, the more likely the tissues will become *necrotic*. Dead tissue falls apart. And in a necrotic loop of bowel means one thing—leakage of intestinal contents (loaded with bacteria) into the abdominal cavity. Leakage is compounded with linear foreign bodies. With linear foreign bodies, the intestinal wall becomes ***plicated*** [L. *plicatus* folded] in a series of pleated, crinkly, and wrinkly folds. Think about continued movement of those folds over a tightly stretched piece of string or other linear foreign body. Each movement drags the folds over the string, eventually wearing through the wall. That means, there's a whole bunch of tiny perforations contributing to contamination of the peritoneal cavity. I have seen way too many severe cases of *peritonitis* from intestinal foreign bodies and intussusceptions. Fortunately, intussusceptions and most foreign bodies (at least a suspect area of obstruction) can

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be found with diagnostic imaging. The density of foreign object determines how well it may be seen on radiographs. Abdominal ultrasound sometimes provides better visualization. This is especially true in the case of intussusceptions. An intussusception, on transverse view of an abdominal ultrasound, has a distinct, unmistakable circle-within-a-circle, "target-like" appearance.

Obviously, because *necrosis* and *peritonitis* are very real concerns with enteric foreign bodies and intussusceptions, they require emergency surgery. Postponing the *laparotomy* to "wait and see" is a very dangerous choice, for many reasons. I've seen the end result of owners and referring veterinarians who tried the "wait-and-see" approach. Many of those patients did not survive. Our overarching goal behind this laparotomy is to remove the obstruction (whether from foreign body or intussusception) and repair or restore the integrity of the small intestine. A simple ***enterotomy*** [*enter(o)-* intestine + *-tomy* to cut] is sufficient to remove a foreign object. The incision is made, object removed, and the intestinal wall carefully sutured closed to make sure it does not leak. If any portion of the intestine appears damaged, fragile, or necrotic (as it often is with intussusceptions), that segment of bowel needs to be ***resected*** (removed). An ***enterectomy*** [*enter(o)-* intestine + *-ectomy* to cut out, remove] is way more complicated than an *enterotomy*. The potential for abdominal contamination is much greater during an *enterectomy*, simply because we are physically removing a portion of intestine, fully exposing the remaining open ends of bowel.

Then comes the challenging ***anastomosis*** [Gr. *anastomosis* opening, outlet; from *ana-* back, up, again + *stoma* mouth, opening + *-sis* condition, process], when we reconnect the free, open ends of intestine. This used to be more challenging, when surgeons had to painstakingly suture the bowel back together. Now, we have specialized surgical devices to quickly reconnect and close the intestinal loops. These devices have multiple, alternating rows of staple-like clips, which provide strong and leak-free closure, without disrupting blood supply. Some of the devices cut and staple the tissues simultaneously. Now, it is preferred that an *anastomosis* site has a double-lumen width. Even with inevitable scarring at the site with a double lumen, the scar tissue will not create a stricture, because the width of the lumen at the *anastomosis*

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is twice that of normal bowel. Here is how we create a double lumen anastomosis. Picture this: hold your index fingers side by side, with the finger tips level with one another. This is how we position the free ends of the intestine. Then, we insert the "jaws" (one jaw in each free end of intestine) into the intestinal openings. When we close the device, it makes a longitudinal cut through the intestinal walls and securely staples them together on each side of the longitudinal incision. We've just created a double-lumen width. Then we use another device across the open ends of the intestine to staple it closed. It's quick, easy, and effective. I was just giddy in the operating room the first time I saw these devices in action! Thank goodness for continued advancements in medicine and surgery!

Okay, that's enough of the generalities of the small intestine. Let's finally take a look at the importance of each segment.

Duodenum

You're already somewhat familiar with the ***duodenum*** [from L. *duodeni* twelve at a time + *-um* the] from our prior discussions. You've seen it and its physical relationship to the stomach and pancreas (see Fig. 7.12). So, you already know that it is the most proximal segment of the small intestine. Are you wondering what the heck its name has to do with the number twelve? Early anatomists found that in most humans the average length of that segment of small intestine was 12 fingerbreadths wide. Hence, they named it the *duodenum* [L. *duodeni* twelve at a time]. Obviously, there is tremendous variance in the length of the duodenum across the spectrum of animals. Yet, even in a tiny mouse, we still call it the duodenum.

So, let's take it from the top. There's food in the stomach mixed with hydrochloric acid and pepsin. We'll call this slurry of stuff *ingesta*. The pylorus titrates the ingesta into the duodenum. Why titrate it? Well, we don't want to squirt it in all at once. That would be way too much volume. And if the duodenum gets stretched out, it usually stimulates vomiting. Plus, a large amount of ingesta would be very irritating (because of the HCl and pepsin). And our ratio of ingesta to bile and pancreatic enzymes would be way off, if we squirted it in all at once. That wouldn't be good at all for digestion. So, we titrate the ingesta into the duodenum.

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Titration allows water and *pancreatic* secretions to rapidly buffer and neutralize the HCl and pepsin. If we don't, we'll be in a world of hurt—literally. Small volumes of ingesta mixed with just the right amount of *bile* initiate emulsification of fats in the duodenum. And small volumes of ingesta mixed with just the right amounts of *amylase* and *lipase* initiate digestion of carbohydrates and fats in the duodenum. Pancreatic enzymes, such as *trypsin* and *chymotrypsin* secreted into the duodenum, carry on where pepsin left off with further digestion of proteins. Duodenal activity is an important beginning in the actual digestive and absorptive process. What happens here prepares the foundation for digestion. We can't possibly digest and absorb all of our nutrients in such a short segment of bowel. So, on the ingesta goes to the next segment of small intestine—the ***jejunum*** (juh-joo'num [L. *jejunum* empty]; Empty? Sorry, I've got nothin', because the jejunum usually has lots of liquid ingesta in it.).

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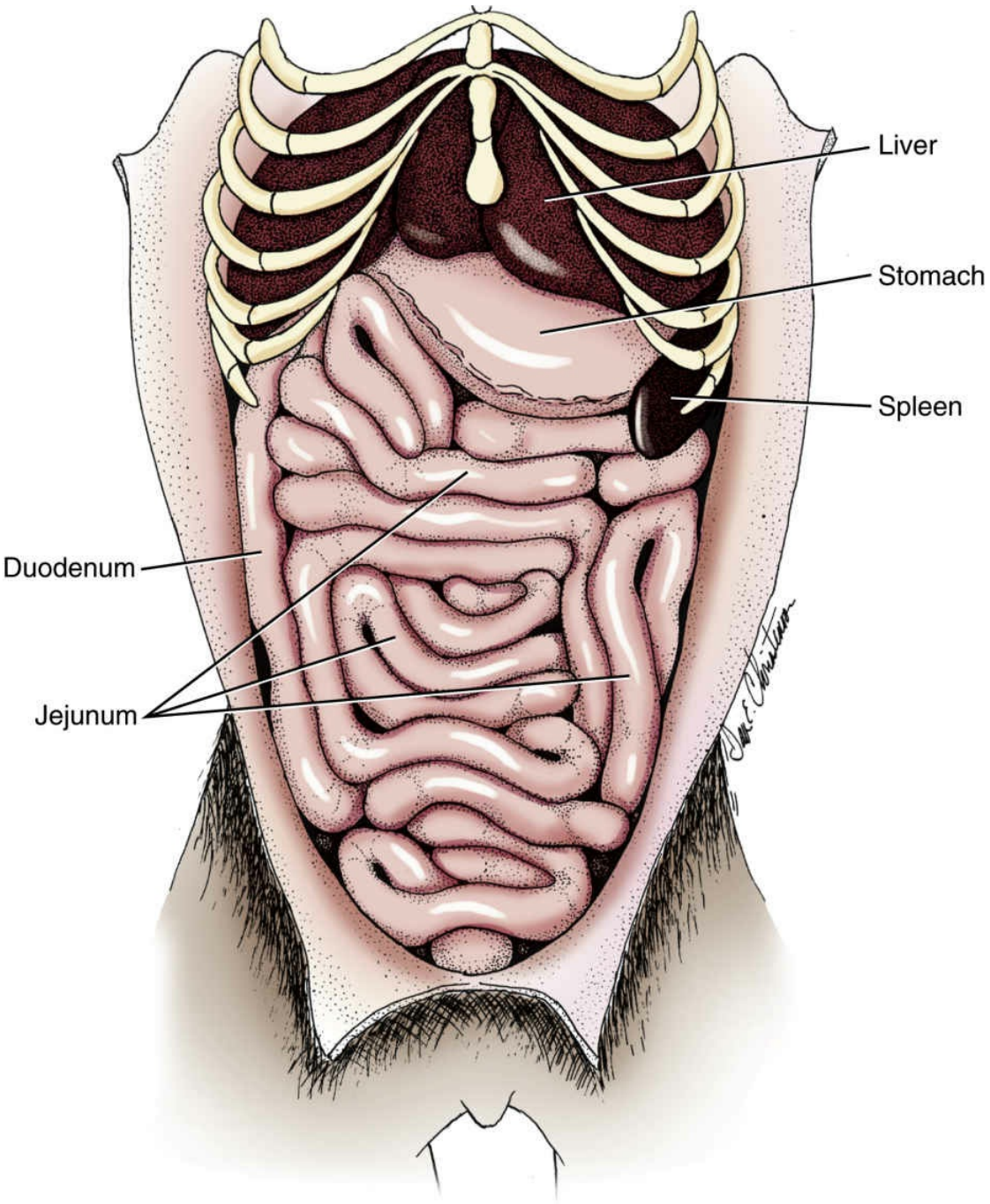

FIG. 7.15 Abdominal viscera of a dog (omentum removed).

Jejunum

The ***jejunum*** is the longest section of small intestine (perhaps 87% of the small intestine, give or take). As you can see in Fig. 7.15, the jejunum occupies most the space in the abdomen. Why have so much jejunum? Well, if this is the segment where most of the digestion and absorption of nutrients take place, we want enough length to buy time for thorough digestion of the ingesta to free up

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available nutrients. And we want sufficient surface area to facilitate absorption of as many nutrients as possible. And remember, the jejunum is not a static structure. It's like a conveyer belt, constantly moving ingesta along. So, all of that digestion and absorption are done on the fly. If we don't have a long enough jejunum, all of those valuable nutrients will be there and gone before we have a chance to do anything with them. ***Jejunal*** [*jejun(o)-* jejunum + *-al* pertaining to] length buys us transit and contact time.

Ileum

The very last little segment of small intestine is the ***ileum*** (Fig. 7.16). Please note the spelling of *ileum* here. It is spelled with an "e," just like the word *enteric*, which means intestines. Why point this out? If you remember fromChapter 4, the pelvis also has a portion called the ilium, spelled with an "i." Make sure that you spell those two words correctly, in the right context. Obviously, they are two very different things.

Can we tell the *ileum* apart from the *jejunum*? Not grossly. ***Histology*** [*hist(o)-* tissue + *-logy* study of] or abdominal ultrasound might help us distinguish them, because the mucosa is slightly different. But we know that the ileum of the average dog is roughly the last 5 to 6 inches of small intestine. Yes, it will be much shorter in a Chihuahua or a cat. Knowing that the ileum is the very last few inches of small intestine is more important than trying to mark its borders definitively. It is also important to know its primary function. The biggest task the ileum has is for the reabsorption of *bile* and *bile salts*. Yep, it recycles bile. The ileum of animals and humans has been recycling bile since long before recycling ever became popular.

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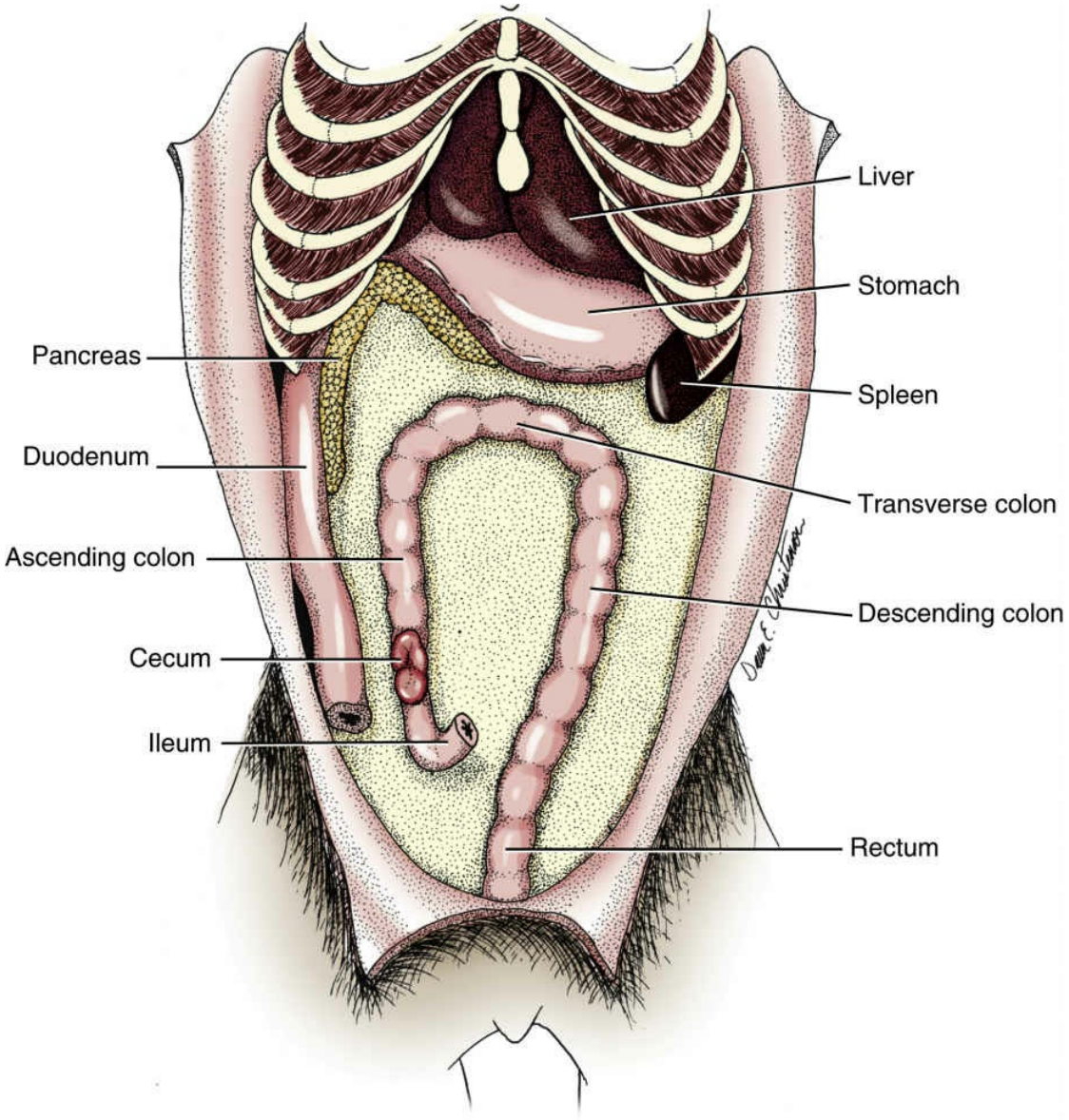

FIG. 7.16 Canine ileum, cecum, and colon.

Cecum

The ***cecum*** [L. *caecum* blind, blind gut] marks the end of the small intestine and the beginning of the large intestine or colon (see Fig. 7.16). It's pretty small in most *monogastric* animals. To be honest, in most simple monogastric animals, like the cat, dog, and ferret, the cecum is really a ***vestigial*** [L. *vestigia* a trace; i.e., remnant] structure. It is a tiny little blind pouch. Very little ingesta actually enters the cecum in these animals. Pigs, on the other hand, have a slightly larger cecum. And ***ileal*** [*ile(o)-* ileum + *-al* pertaining to] contents actually pass through the cecum of the pig, before entering the colon. One fun fact that most animals have in common,

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regarding the cecum—it provides a warm, happy home for ***whipworms***.

Colon, Rectum, and Anus

The ***colon*** (see Fig. 7.16) or large intestine is subdivided into three sections too. The ***colonic*** [*colon* + *-ic* pertaining to] sections in dogs and cats are named on the basis of orientation in the body and movement of material through it. The ***ascending colon*** is on the right side of the abdomen. Material moves in a cranial direction through that section. The ***transverse colon*** crosses the abdomen from right to left. And the ***descending colon*** is closer to the left side of the abdomen, moving contents caudally toward the rectum. The ***rectum*** is really the terminal portion of the colon, where fecal material is temporarily stored. The colon of pigs is similar. But the ascending colon of pigs is longer and somewhat spiraled compared to dogs and cats.

Structurally, the colon is quite different from the small intestine. It's not a smooth tube like the small intestine. The colon has ***haustrations*** [from L. *haustor* "drawer" + *-tion* state of]. *Haustrations* are pouch-like areas. The "pleats" (for lack of a better word) indenting the colon at the margins of each pouch are created by connective tissues and muscle fibers. Speaking of muscles, the musculature of the colon is quite different too—it's not as strong, especially for moving material along. And the arrangement of muscle fibers along the colon is much better at ***segmentation*** (see Fig. 7.11B) than it is at peristalsis. This is by design. You see, in the colon, we want to absorb water from the fecal material, before it leaves the body. The combination of the *haustrations* and the segmental muscular contractions creates a slow turning and squeezing of the fecal material. I liken it to using a shovel in the garden to turn the soil. We dig into the soil with the shovel, lift, and turn the soil over. The surface of the soil exposed to the air dries out. A similar thing happens in the *colon*. The muscular contractions squeezing at the bands of the haustrations dig into the fecal matter and turn it over. The surface of the fecal matter in contact with the *colonic* wall becomes dried out as water is absorbed.

During ***defecation*** [*defecat(o)-* defecating + *-tion* the act of; i.e., a bowel movement, passing stool], the haustrations actually relax.

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Then a coordinated, gentle peristaltic wave, called a ***mass movement***, forces the feces toward the ***anus***. The anus actually has two sphincters. The ***internal anal*** [*an(o)-* anus + *-al* pertaining to] ***sphincter*** is under automated neurologic control. The ***external anal sphincter*** is under conscious control. When potty training a puppy or a kitten, great patience is required. Those youngsters literally need to learn how, when, and where to control the external anal sphincter. Not only do they need to learn when and where to defecate, but also they need to develop some strength of that external anal sphincter. Until they learn and develop some "pucker-power," they will have accidents. Now, it is helpful to know that there is something called the ***gastrocolic*** [*gastr(o)-* stomach + *col(o)-* colon + *-ic* pertaining to] ***reflex***. This reflex connects the dots between food in the stomach and the mass movement of the colon. So, when potty training a puppy, it is wise to take the pup outside within 15 to 20 minutes or so after a meal. The mass movement usually happens within that *postprandial* period. If you put that knowledge to use, it will save you from having to clean up as many messes. Fortunately, adult dogs and cats can behaviorally control the external anal sphincter and inhibit *defecation*.

Constipation, Obstipation, and Ileus

An important note to make: the longer feces sits in the *rectum* and *descending colon*, the more it dries out. When feces is too dry, it becomes difficult to ***defecate***. ***Dyschezia*** [*dys-* difficult + Gr. *chezein* to defecate + *-ia* process of] is the medical term for difficult defecation. And that is what happens in ***constipation*** [from L. *constipatio* a crowding together; *con-* together]. If constipation is not relieved, the feces dries even more. Now, it's nearly impossible to defecate and that's ***obstipation*** (intractable constipation; i.e., obstruction with feces). Clear as mud? Okay, let's use a traffic analogy. *Constipation* is like a slowdown on the highway around a construction zone. Movement is slow. Sometimes, it stops for brief periods. But eventually, traffic makes it through. *Obstipation* is a traffic jam from a multicar pileup. And on a busy freeway, traffic can be backed up, at a dead halt for miles. On the highway, traffic can be rerouted to detour the accident. There are no detours with the *colon*. It's the back door (anus) or nothing.

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Unfortunately, there can be serious problems that develop from a colonic traffic slowdown or traffic jam. If an animal is ***obstipated*** too long, ***ileus*** may develop. With *ileus*, all peristaltic activity along the whole intestinal tract stops. And it may be very difficult to get it started again. Whether it results from obstipation or another cause, if we cannot restart motility, ileus is not survivable. How do we know if ileus has developed? We listen. We use a stethoscope to listen patiently to all four abdominal quadrants, on both sides of the animal. If we do not hear any ***borborygmus*** (bor"buh-rig'mus [L. *borborygmus* rumbling; i.e., the growling, rumbling, gurgling noises made by movements of the digestive tract]), we know we have no movement along the GI tract (i.e., ileus).

Megacolon

Megacolon [*mega-* large, enlargement + *colon*] is most common in cats. This is a condition that may develop secondarily to repeated episodes of *constipation* and *obstipation*. The colon can become extremely dilated in some of these cats. The fecal material building up first in the rectum, then progressively retrograde along the colon is what stretches it and causes the dilation. Why does it build up so badly? Well, the small intestine doesn't stop its peristaltic activity, not yet anyway. So, contents from the small intestine continue to be pushed into the colon. In the colon, the fecal material dries out and piles up like bricks. And if these bricks can't move out the back door, the only way to accommodate more bricks coming down our digestive conveyor belt is to expand the room (i.e., the colon itself).

Unfortunately for these cats, once *megacolon* develops, they get caught up in a catch-22 cycle of constipation and obstipation and further dilation of the colon. The dilated section of colon typically loses muscle tone and peristaltic activity completely. Manual evacuation of the rectum and descending colon along with other measures, such as stool softeners and laxatives, becomes the cat's new norm. It's not pleasant for anyone. For some cats, especially if a portion of the colon is still functional with motility, a partial ***colectomy*** [*col(o)-* colon + *-ectomy* to cut out, remove] may be done.

Small vs. Large Bowel Diarrhea

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Of course, bowel control for defecation goes right out the window when it comes to ***diarrhea*** [*dia-* through + *-rrhea* the flow]. Both *enteritis* and ***colitis*** [*col(o)-* colon + *-itis* inflammation of] can cause *diarrhea*. And both can create some powerful peristaltic waves (yes, even in the weak, wimpy colon). But there are differences in the character of the diarrhea, depending on where it originates. And knowing the difference between small bowel (small intestine) and large bowel (colon) diarrhea is helpful in making a diagnosis. Yes, we need to make a diagnosis, because diarrhea is merely a symptom of disease. Determining the disease causing it is important for treating and managing the patient. In some cases, it is also important for protecting public health, because some GI pathogens are ***zoonotic*** [*zo(o)-* animal + *nos(o)-* disease + *-tic* pertaining to; i.e., a disease transmitted from animals to humans].

Let's consider ***small bowel diarrhea*** first. The character of small bowel diarrhea, from *enteritis* or *gastroenteritis*, creates feces that is more ***liquid*** than normal. Yes, I know, all diarrhea is more liquid than normal. But in the case of small bowel diarrhea, the liquid nature of it results from two factors. First, there are increased secretions and water added to the ingesta, due to enteritis. That is the nature of inflammation, right? Second, in response to inflammation, peristaltic movements are stronger and more rapid, pushing *enteric* contents into and ultimately through the colon with great force. The ***volume*** can be quite large. That makes sense, when you consider that the small intestine is the longest portion of the digestive tract. So, it holds a large volume at any one time. And because the volume of small bowel diarrhea is so great, it can rapidly *dehydrate* a patient. And if the animal is vomiting as well, the *dehydration* can become profound—rapidly. Certainly, the ***frequency*** of defecation with small bowl diarrhea will likely be increased from normal. But generally, there is not extreme urgency associated with it. All of this contributes to tremendous loss of water and poorer digestion and absorption of nutrients (things are moving through too rapidly). Sometimes, the ***maldigestion*** [*mal-* bad, poor] and ***malabsorption*** can physically alter the appearance of the diarrhea.

This is definitely the case with ***steatorrhea*** [*steat(o)-* fat, fatty + *-rrhea* the flow]. ***Steatorrheic*** [*steat(o)-* fat, fatty + *rrhe(o)-* flow + *-ic*

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pertaining to] feces actually looks greasy. Fat digestion and absorption are poor. Why? Well, perhaps, there could be a problem with the *bile*. If we don't emulsify fats well, we can't digest them well. *Pancreatic insufficiency*, with regard to secretion of *lipase*, could cause it. Insufficient lipase means we can't digest fats adequately. There are even parasites, such as *Giardia sp*., which interfere with the absorption of fats. In the case of the latter, these fats usually become rancid, making the fecal material smell awful (not that it smells great to begin with, but this is really bad). By the way, *Giardia sp*. is a *zoonotic* organism. Does recognition of *steatorrhea*, as an example, provide us with a definitive diagnosis? No. But it points us in some key directions.

The nature of ***large bowel diarrhea*** is different. First, let's think about water. After all, we said that all diarrhea is more ***liquid*** than normal, right? But why is there more water in this case? Think about the principal role of the *colon*: absorption of water. In the presence of *colitis*, inflammation reduces the functional ability of the colon. If the colon has a reduced ability to absorb water, water remains in the feces. Fortunately, the ***volume*** of large bowel diarrhea is less. Why? Well, the colon is not as long as the small intestine. So, the volume contained by it is less. That means fecal volume with large bowel diarrhea is less. But the colon is right near the "back door," right? Even if the inflammation is in the region of the cecum and ascending colon, there's not a great distance to get from point A to point B. So, with large bowel diarrhea, there is marked increased ***frequency*** of defecation. There is also ***urgency*** associated with large bowel diarrhea. The inflammation is very near to the "back door," and it increases the strength of peristaltic movements through the colon. Another thing frequently associated with *colitis* is ***tenesmus*** [Gr. *teinesmos* straining]. An animal exhibiting *tenesmus* continues to strain for a prolonged period of time, with or without producing a small amount of stool. *Tenesmus* is usually a big clue, in patients with diarrhea, that the inflammation is in the colon or rectum. What about secretions? Well, there's really only one thing secreted along the colon, and that's mucus. In the presence of inflammation, mucus secretion can dramatically increase. So, large bowel diarrhea is often quite ***mucoid*** [*muc(o)-* mucus, slime + *-oid* resembling].

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Okay, so what might cause large bowel diarrhea? Well, in the colon, there is a wonderful ecosystem of bacteria. Anything that alters the bacterial population (e.g., antibiotic therapy) may result in colitis and large bowel diarrhea. With antibiotics, we may kill off "good" bacteria and permit others ("bad guys") to flourish. You see, the "good guys" keep the "bad guys" in check. So, until we reestablish a balanced ecosystem, the antibiotic-induced diarrhea will persist. ***Probiotics*** [*pro-* for + *bi(o)-* life + *-tic* pertaining to] can be very valuable in reestablishing that balance. *Probiotics* basically 're-seed' the colon, adding good bacteria into the mix. Bacteria are often a big factor in large bowel diarrhea. But there are parasites that target the *cecum* and *colon*. ***Whipworms***, mentioned earlier, are a good example of this. They live in the cecum and colon. And they cause inflammation, by penetrating and weaving a portion of their bodies through the mucosa. Very irritating indeed. They are most active during reproduction, but that activity waxes and wanes. So, the highly mucoid large bowel diarrhea that they create in their host also waxes and wanes.

Is it possible for an animal to have both small and large bowel diarrhea? Yes. Generally, diarrhea tends to be isolated in its origin to one segment of bowel. If both are involved, we need to be very concerned over the hydration of the patient. In fact, because diarrhea can create extreme water loss and *dehydration*, we often give ***antidiarrheal*** [*anti-* against + *diarrhe(o)-* diarrhea + *-al* pertaining to] medications. These agents vary in how they work, depending on the product. But probably the most effective means of relieving diarrhea is to alter the motility of the colon. If the colon is forced to slow down muscles associated with peristalsis, without affecting muscles associated with segmentation, we can improve water absorption and slow down transit time. That's how *antidiarrheal* agents, such as loperamide hydrochloride (Imodium), work.

Hematochezia and Melena

Bleeding along the digestive tract can be a serious problem. There can be significant loss of blood that's life-threatening. It is important to be able to recognize the difference in the appearance of

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the blood in the feces. The appearance distinguishes where (roughly) the bleeding is occurring. With ***hematochezia*** [*hemat(o)-* bloody + *chez(o)-* feces + *-ia* condition of], there is frank, red blood in the stool. If it's red, we know that it is undigested. Therefore we can conclude that the bleeding is probably coming from the colon, rectum, or anus.

Melenic [*melen(o)-* black + *-ic* pertaining to] feces is black because the blood is partially digested. Sometimes, it is so black that it looks like tar. If the blood is being digested, then it is in direct contact with digestive enzymes. Digestive enzymes are in the proximal digestive tract, right? So, when we see ***melena*** [Gr. *melaina* black], we can conclude that the bleeding is somewhere along the proximal GI tract, most often the stomach and/or small intestine. If the stomach is the source, there is likely *gastritis*. And with gastritis, there may be vomiting.

If there is bleeding in the stomach, we may also see ***hematemesis*** [*hemat(o)-* bloody + *emesis* vomit]. Remember, the blood is exposed to gastric acid and pepsin, so generally it is not frank, red material. There usually has to be significant ***hemorrhage*** [*hem(o)-* blood + *-rrhage* flow; i.e., bleeding] for *hematemesis* to actually look bloody. Usually, the blood is partially digested and clotted, giving it a "coffee-ground" appearance. Bleeding gastric ulcers are often associated with *hematemesis* and can contribute to melena as well.

A common cause of melena in dogs is a parasitic infestation with ***hookworms***. Hookworms are blood-sucking worms that live in the small intestine. They consume and cause significant bleeding and blood loss. In fact, the blood loss, especially in puppies, can produce a life-threatening ***anemia*** [*an-* without + *em(o)-, hem(o)-* blood + *-ia* condition of; i.e., RBC deficiency]. Another common cause of *melena* is bleeding ulceration in the small intestine. This is often caused by administration of nonsteroidal antiinflammatory drugs (NSAIDs), such as *aspirin* and *carprofen*. NSAIDs like these interfere with mucosal protection, resulting in bleeding ulcers. Bleeding is never good. Determining the location of the bleeding is the first step in trying to stop it. So, it is really important to be able to recognize both *melena* and *hematochezia*.

There. You did it! You have successfully waded through the simple monogastric digestive system. Hang on to what you have

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learned, because we are going to put it to use in the next sections. Yes, much of the information is still relevant. This gives us the opportunity to build on it, by focusing on those aspects of the digestive system that are different—modified for ***herbivorous*** [*herbivor(o)-* plant eating + *-ous* pertaining to] diets.

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Monogastric Hindgut Fermenters

We mentioned monogastric hindgut fermenters earlier. To refresh your memory, monogastric hindgut fermenters are herbivores, like the horse, rabbit, and guinea pig. For our discussion here, our main focus will be on horses and other Equidae (e.g., donkeys and mules). However, much of this information applies to other animals like rabbits and guinea pigs too. Where there are specific nuances or concerns for those smaller hindgut fermenters, I'll try to point them out.

Mouth

The mouths of these *herbivores* are modified for foraging, grazing, and chewing plants. Equine (horse) mouths are unique because they have ***prehensile*** [from L. *prehensio* to grasp + *-ile* pertaining to] lips. Their lips are so adept that they can select the best, most tender blades of grass or the tastiest nuggets of grain. If you've ever been around a horse, you know that they actually explore their environment with their lips—wiggling and touching them over your hand and clothing when they meet and greet you. And if you offer a treat like a sugar cube, apple, or carrot, they use those lips to pluck the treat gently from your hand. Considering the fact that because of their head structure, especially the eye placement, horses can't see directly in front of them. So, having *prehensile* lips is very important for them. By the way, horses drink water by suction. They don't have agile tongues like dogs and cats. The same is true for guinea pigs and rabbits, when it comes to drinking. Comparatively, these little friends have a cleft upper lip. The cleft lip may not be prehensile, but it sure adds to their cuteness, especially when they're eating.

Dentition and Tooth Structure

Once a horse has grabbed some food with its *prehensile* lips, it uses its teeth for breaking off the grasses and hay and pulverizing them through *mastication*. As you can see in Fig. 7.17, their teeth including the incisors have large *occlusal* surfaces. (Refer to Table

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7.2 for comparative adult dental formulas for herbivores.) But for ***chronic*** [*chron(o)-* time + *-ic* pertaining to; i.e., long-term], repeated grinding of forages, we need more than just a broad *occlusal surface*. We need teeth built for lots of wear and tear. If their teeth were designed like carnivorous and omnivorous teeth, these herbivores would quickly wear through the enamel and dentin, leaving them with no crowns left for mastication. Wouldn't it make sense to build their teeth with more of the really hard materials like enamel and cementum? And let's do it so that there's not just a superficial coating of these materials, but align rows of them throughout the length of the tooth. That is precisely how their teeth are constructed. As you can see in Fig. 7.18, we have vertical rows of rock-hard, durable *enamel*, surrounded by hard *cementum*, with the gaps filled in with *dentin*. As a horse (or any other herbivore) grinds the occlusal surfaces of their premolars and molars, it's going to take a long time and a lot of grinding to wear down that crown.

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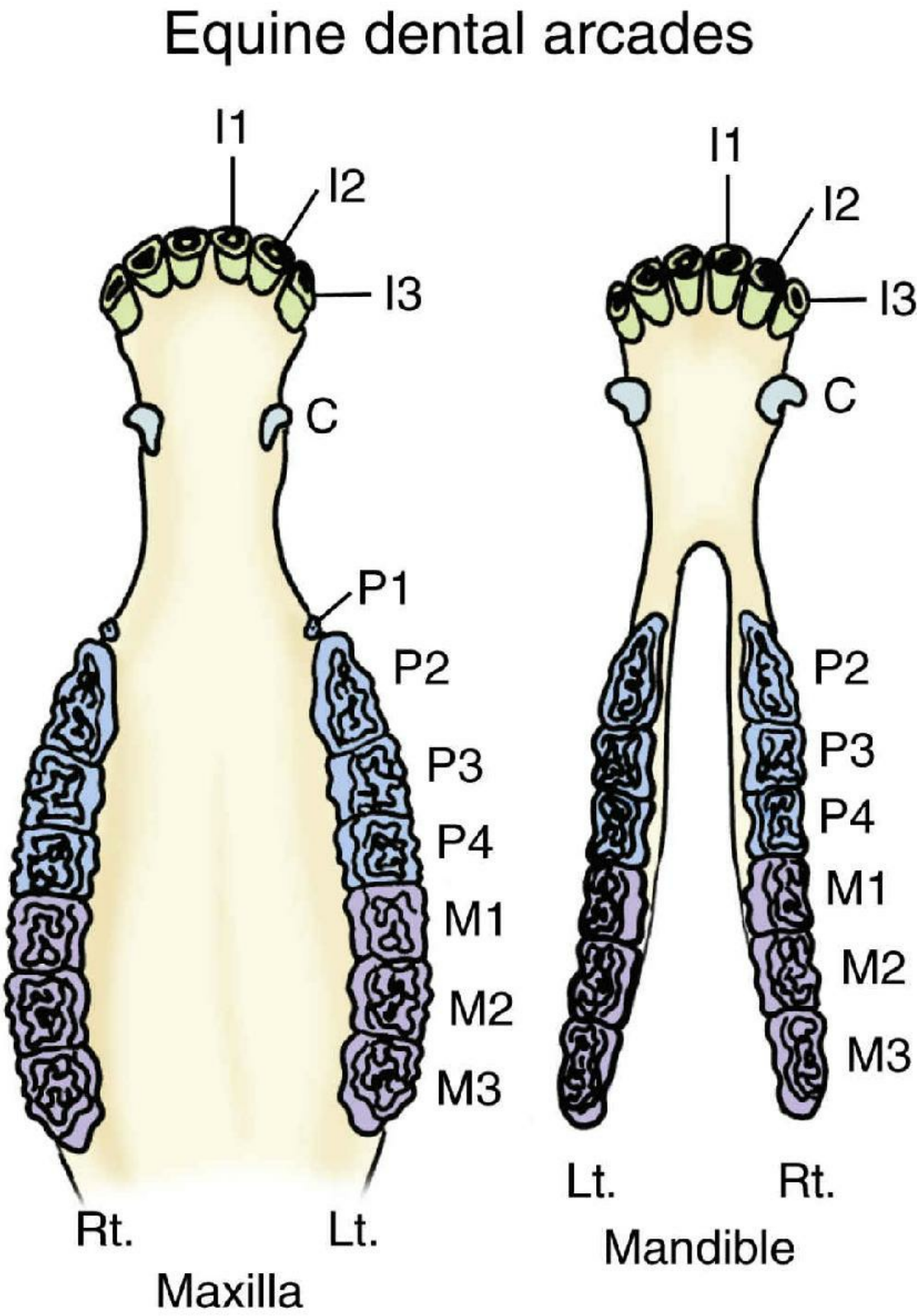

FIG. 7.17 Equine dental arcades.

TABLE 7.2

Comparative Adult Dental Formulas for Herbivores

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Horse	
Rabbits	
Ruminants	

Here's a really cool thing—their teeth continue to grow, even as adults! Can you believe that?! In fact, rabbits' teeth grow their entire lives! If you notice in Fig. 7.18, the tooth does not have an apical delta like carnivorous and omnivorous teeth. They have open root ***apices*** (a'puh-sēz; plural of apex). This permits the growth plates to remain open and active. So, their teeth are constantly, slowly erupting—pushing through the gingival margin to maintain a constant depth of the crown. How cool is that?! For most domestic herbivores (horses and ruminants), their teeth continue to grow well into adulthood. But eventually, that growth will stop. So, if the animal lives long enough, it could wear its crowns down. What about the *pulp* and *pulp cavity*? That remains constant too. Reparative dentin simply fills the distal region of the pulp cavity as it grows, so that the pulp is never exposed.

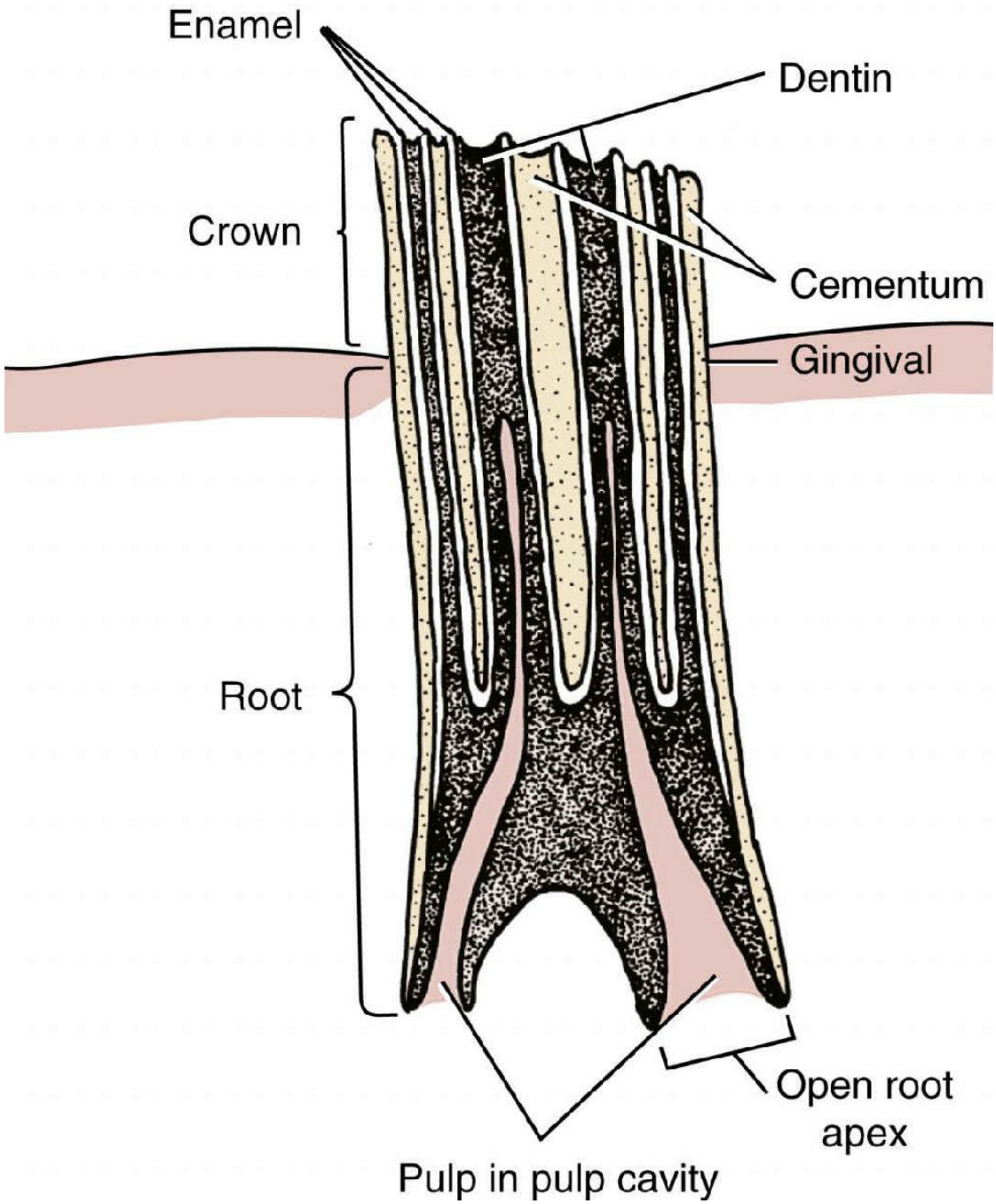

FIG. 7.18 Herbivorous tooth structure (for molars and premolars).

One last structural comment about the *incisors*. The incisors of horses are smaller than the premolars. There's no room for rows and rows of enamel, cementum, and dentin. So, the outer surfaces of the tooth are covered in enamel and cementum. There is also a cup of these materials (filled with dentin) at the center of the occlusal surface of each incisor. Some people use the wear pattern through that cup to estimate the age of a horse. ***Dental aging*** of horses provides a rough estimate. Sometimes, dental aging is quite

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accurate. But if a horse is chronically eating feed from the ground, especially really sandy ground, the wear of the incisors may be accelerated. So, the interpretation of incisor wear will falsely lead us to conclude that the horse is older than it actually is. Finally, notice the premolars in the schematic and the dental formula for the horse. Notice that the first premolar is absent on the mandible. The maxillary first premolar may or may not be present in every horse's mouth. That tooth is sometimes called the "***wolf tooth***." Because it can interfere with a "bit" (a metal bar) in the horse's mouth, the *wolf teeth* may be extracted.

Ah, there are two sides to every coin, right? It's great that their teeth continue to grow, but take a close look at Fig. 7.17 again. Notice how the maxillary dental arcade is wider than the mandibular dental arcade. The occlusal surfaces do touch when chewing. In fact, during mastication, the mandible moves in a rotating manner to maximize grinding between the occlusal surfaces, despite the mismatch of the dental arcades. However, the *buccal* aspect of the maxillary molars and premolars goes untouched. And the *lingual* aspect of the mandibular molars and premolars goes untouched. With continual growth of these teeth, the untouched areas will become high points. Those ***dental points*** can become quite tall and sharp. The mandibular dental points can actually ***lacerate*** (cut) the tongue. Maxillary dental points can *lacerate* the buccal mucosa. In horses (and sometimes domesticated rabbits), we remove these points with a procedure called "***floating***." When we "***float***" teeth, we use a rasp (like a really big file) to wear those points down. Manual floating takes a lot of muscle. There are powered (electric) rasps that require less elbow grease from us. But still, we need pretty good upper body strength to keep the rasp on the dental points. Oh, did I mention that most horses object to this procedure? Yep, muscle and patience (and probably a little sedation) are required.

Transit

Once the horse has grabbed and thoroughly chewed its forages and feeds, the actual transit through the GI tract is very much the same as simple monogastric animals. Much saliva is added to the food.

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(Much of what the horse eats is quite dry.) These salivary secretions moisten the food, so that the tongue is able to form a nice food bolus.

Proximal Gastrointestinal Tract

That food bolus is pushed into the pharynx by the tongue, and off into the esophagus it goes. Esophageal peristalsis pushes it into the stomach. Sound familiar? Now, the equine stomach is a bit different from the dog and the cat. The structure of the fundus and gastroesophageal areas form a much tighter cardiac sphincter. So, horses cannot vomit or ***eructate*** [from L. *eructation* "cast up wind"; i.e., belch]. (Neither can rabbits or guinea pigs.) So, if a horse were to eat something that produced a lot of gas in the stomach, the horse won't be able to relieve the pressure. In that case, we may need to intervene. But there is no way we can pass an orogastric tube. The horse would bite right through it. So, we usually pass a ***nasogastric*** [*nas(o)-* nose + *gastr(o)-* stomach + *-ic* pertaining to] tube. *Nasogastric* tubes are frequently used in horses to relieve gastric pressure from gases and administer medications. (We use them in dogs and cats too, especially to administer constant rate infusions [CRI] of water, critical care liquid diets, and medications.) Beyond that, the stomach of these herbivores is quite similar to simple monogastric animals.

As ingesta moves from the stomach into the duodenum, it is mixed with bile and pancreatic enzymes. Remember, we said earlier that horses do not have a ***gallbladder***. So, their bile is not as concentrated. But that's okay. They're not eating foods high in fat. (Pizza is not on the menu!) So, emulsification with bile and digestion with lipase are easier. Throughout the small intestine (duodenum, jejunum, and ileum), horses digest and absorb nutrients just like simple monogastric animals. Mucosal structure and villi are pretty much the same as dogs and cats. And it's really the concentrates (i.e., grains, like corn and oats) that are best digested here. The horse might be able to glean a little in the way of nutrients from the other forages (plant matter). But to really extract all of the needed nutrients from ***roughages*** (plant matter like grasses and hay), we need a big team of microbes to help us out. And that's where the cecum comes in.

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Cecum

Comparatively, the ***cecum*** of monogastric hindgut fermenters is HUGE! Just look at it in Fig. 7.19. Now, because of its size and function, we need better delineation between the ileum and cecum. When ingesta is ready to move into the colon, we don't want any retrograde movement into the ileum. So, monogastric hindgut fermenters have a strong, well-defined ***ileocecal*** [*ile(o)-* ileum + *cec(o)-* cecum + ***-al*** pertaining to] ***valve***. It opens to receive ingesta from the ileum. But when ***cecal*** [*cec(o)-* cecum +***-al*** pertaining to] muscles contract to move ingesta into the colon, the *ileocecal valve* is tightly closed.

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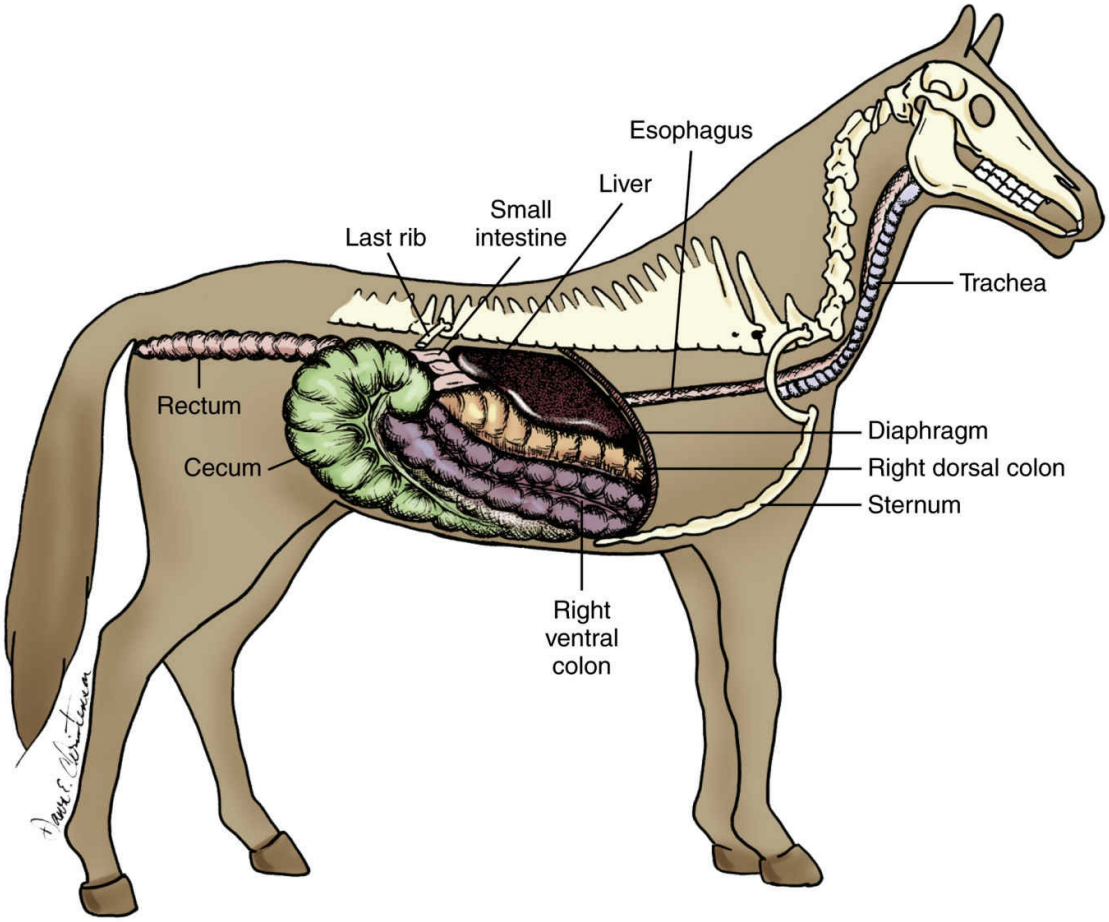

FIG. 7.19 Equine abdominal viscera.

The *cecum* (and *colon*) is the reason we refer to these animals as monogastric *hindgut fermenters*. The cecum and colon are pretty close to the "backdoor" of the digestive tract. And these "hind" portions of the gut are where these animals have huge populations of happy, wonderful microbes (bacteria and protozoa) to "ferment" the roughages. It's the best way to break down all but the woody parts of plants to free-up all of the nutrients.

In terms of nutrient value, especially with regard to protein content, there is a difference in roughages. Ounce for ounce, ***alfalfa hay*** has more protein than ***Timothy hay***. Now, that doesn't always mean alfalfa should be fed to every horse. An old gray mare, who gets little in the way of physical activity, probably doesn't need alfalfa. It may be too rich for her geriatric system. Timothy hay may do just fine. But for a mare who's pregnant, a protein-rich hay might be best. So, the alfalfa may be preferred for her. In fact, for her, we may want to pay attention to the cutting of the alfalfa. You see, during the average season (in a temperate region, like the

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upper Midwest US), we may be able to have the alfalfa grow well enough to harvest three cuttings. First cutting alfalfa may contain roughly 16% crude protein compared to around 20% or more in 3rd cutting alfalfa. Most grass hays average around 8% crude protein.

The fermentative process of roughages also produces very important and valuable by-products, such as ***volatile fatty acids*** and ***B vitamins***. Those *volatile fatty acids* provide a wonderful source of calories for energy. In fact, it's estimated that 75% of a horse's caloric needs are met by volatile fatty acids. How amazing is that?! The not-so-valuable by-products are CO_2 and methane—which can be problematic if they get trapped in a blind sac like the cecum. Can you say: gas-colic?

Colon and Rectum

The fermented *cecal* contents eventually move into the colon. Whatever roughages the microbes in the cecum didn't fully ferment and digest, *colonic* microbes will finish off. And in hindgut fermenters, the colon is much longer with more twists and turns to accommodate that. But, let's face it; the cecum and colon are not designed for major absorption of nutrients other than water. Will we absorb some nutrients and fermentative by-products, such as volatile fatty acids and B vitamins? Yes. But we can't absorb all of them. Many nutrients, other than water, will be lost in the feces. (Again, this makes horse manure better for the garden.) Of course, as we mentioned in the nutrient section earlier, rabbits regain more nutrients and fermentative by-products from the feces, by virtue of their *coprophagic* habits. Ah, but across the board having deeper, more well-defined *haustrations* throughout the colon and rectum, along with fantastic muscular *segmentation* activity, hindgut fermenters can make fantastic contact with the fecal material to maximize the absorption of water. This is why fecal material from horses, rabbits, and guinea pigs tends to be pelleted ("road apples" from horses). Sorry, that's probably more than you ever wanted to know about feces.

One thing to keep in mind, with monogastric hindgut fermenters—because they have a more complicated hindgut (cecum and colon), with more twists and turns, it is often easier to "plug up the plumbing." This is especially true if they don't have access to

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enough water. If they don't drink enough to maintain hydration, the colon will dry out the fecal material more. Dry feces is difficult to move along. Remember, peristaltic action for mass movements is weak in anyone's colon. Dry, sticky feces might not move along. It may simply get stuck, especially at one of the narrow, tight turns of the colon. And if we plug up the plumbing, the animal won't feel well and will probably develop abdominal pain or ***colic***.

Colic

Colic (kol'ik [from L. *colica passio* "colon suffering"; i.e., abdominal pain]) is a very generalized term for abdominal pain. Many factors can cause abdominal pain, even reproductive and urinary problems. But more times than not, in a horse with *colic*, it stems from the GI tract. But that can be anywhere along the GI tract, not just the colon. Remember, horses can't eructate. So, gas accumulations in the stomach could cause colic. But gas can accumulate anywhere along the GI tract. In fact, that's more likely in the cecum, as we mentioned earlier. Obstruction is another possible cause of colic. I've seen foals with such a huge number of *roundworms* in their small intestines that they obstructed. Enteric obstruction is painful. Colonic obstruction can happen too—especially from constipation or ingestion of too much dirt and sand. (Yep, sand tends to accumulate at places like the ***pelvic flexure*** [one of the sharp turns of the colon], creating an obstructive "sand-bar.") But one of the most common causes of colic in horses is an abrupt dietary change. This would be a typical scenario: a horse has been fed dry hay through the winter months and turned out onto lush green pasture in the spring. That is a tried and true recipe for colic.

To add insult to injury, whatever the cause of the abdominal pain, many horses will respond to that pain by rolling on the ground. If they do that, with all of the weight in the cecum and colon, rolling makes it really easy for a horse to create ***torsion*** (tor'zhun [*tors(o)-* twisting + *-ion* act of]) of those structures. Yep, that can be just as deadly as GDV in the dog. Believe it or not, even a huge draft horse is a pretty delicate creature. Most horses don't have a great pain threshold, especially abdominal pain. They tend to overreact to pain, compounding all of the systemic effects and

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consequences. So, prompt veterinary intervention is needed. And if the horse managed to ***torse*** (torz; twist) the cecum or colon, an emergency *laparotomy* is absolutely necessary to save the horse's life. Like the dog with GDV, every moment counts. And like GDV dogs, many of these horses will not survive. It is very important to listen for *borborygmus* in colic cases. *Ileus* can develop in a variety of colic cases. But if there is a torsion and there are no gut sounds, portions of the GI tract may already be *necrotic*. This is why *abdominocentesis* is such a valuable diagnostic tool in horses with colic. The contents and character of the fluid can help us assess the condition of the GI tract and *peritoneal* cavity rapidly.

The best way to deal with colic is to prevent it. Avoidance of abrupt dietary changes is the single most important thing that any horse owner can do. They should secure grain bins so that horses can't help themselves to large quantities of concentrates. (It's like a kid getting into the cookie jar.) They should gradually introduce horses to lush, green pasture in the spring. Seriously, horses need to be time limited when first turned out on pasture (e.g., perhaps an hour on the first day, with gradual increases in time each subsequent day over a week or two). If the paddock has really sandy soil, feeding from bins and troughs off the ground will minimize ingestion of sand. And throughout the year, it's really important to provide adequate drinking water, especially when feeding dry hay. These measures won't prevent colic in everyone, but they sure will reduce the chances of the most common forms of it.

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Ruminants

Ruminants have one of the most unique and very efficient digestive systems of any domestic animal. They are called ruminants, because they have a large chamber called the ***rumen***. For our purposes, we'll focus primarily on cattle, sheep, and goats for this discussion. We can generalize in many respects to include others, such as llamas and alpacas. But technically, llamas and alpacas are modified ruminants. We'll distinguish how they differ from the other ruminants as we move along through our discussions of the digestive tract.

Mouth

The mouths of ruminants are definitely built for an *herbivorous* diet, but they do differ among the species. For example, most ruminants have a *cleft* (split) upper lip. Cattle are the exception to this. The bovine muzzle has a flat, stiff upper lip. This means that grazing and foraging for cattle are quite different than other ruminants. With a stiff upper lip, cattle have difficulty grasping plants and grasses close to the ground. Other ruminants can nip off plants and grasses very close to the ground, all because of the cleft upper lip. The cleft lip makes the lips more maneuverable, to access and grasp forages better. (Still, nothing compares to the prehensile lips of horses.) By the way, like horses, ruminants drink by suction.

Dentition

Again, refer to Table 7.2 for the adult ruminant dentition formula. Of course, if you look at the formula, before looking at Fig. 7.20, you might think that someone made a mistake with the formula. No mistake made. As you can see, ruminants don't have any maxillary incisors. What?! How on earth do they bite off grasses and other plants? They have a tough, connective tissue, ***dental pad*** on the rostral hard palate. Believe it or not, they can grab and bite/tear off grasses and plants pretty well with the mandibular incisors and maxillary dental pad. Over time, they'll actually wear down their mandibular incisors by rubbing them over the dental

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pad. And because those incisors (and canines that look like incisors) have only a single layer of enamel covering the crowns, once they wear through the enamel, it doesn't take long to wear them down. The rest of the premolars and molars have broad occlusal surfaces for excellent grinding and pulverizing of foods during *mastication*. And those teeth are built just like the one shown in Fig. 7.18. It is worth noting that ruminants do not have any first premolars. It is also worth noting that we typically do not have to float ruminant teeth. With freer mandibular movement, ruminants can make a broader sweep while chewing. And they spend the better part of their days chewing. Because of this, they tend to wear the full occlusal surface. Minimal to no points—no floating required.

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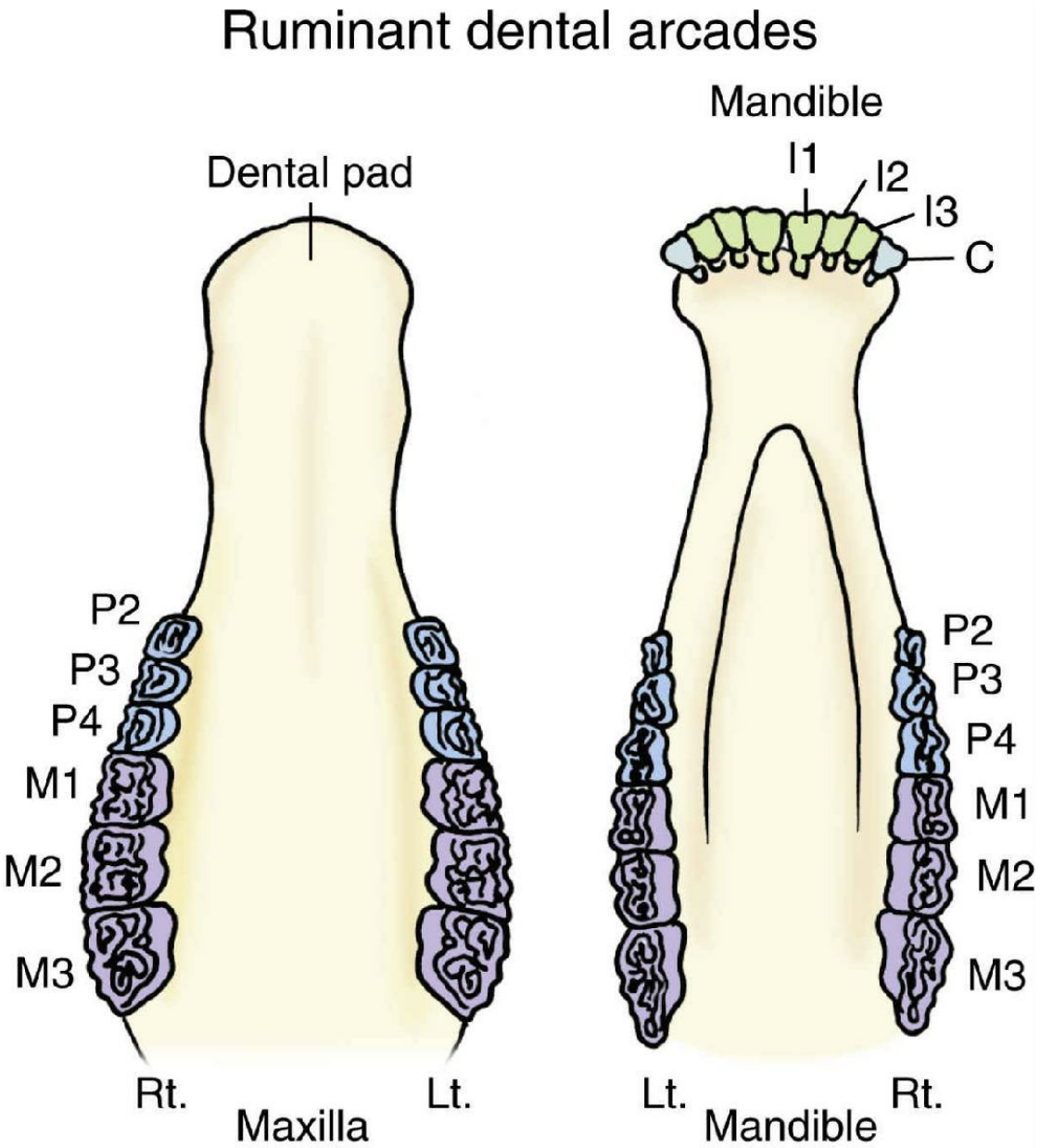

FIG. 7.20 Ruminant dental arcades.

While we're talking about *mastication*, this is one of the factors that help with the efficiency of ruminant digestion. They don't chew their food once and done. No. They ***eructate*** (i.e., belch up) a portion of what they've already swallowed and chew it again. This is what we refer to as "chewing their cud." ***Eructation*** (belching) is essential for this. So, the pattern of ruminant chewing is chew, swallow, eructate, chew, repeat, again and again and again. They can spend the better part of a day chewing their cud. And each sequential act of chewing pulverizes the food into smaller and smaller particles. That exposes way more surface area for microbes to ferment and digest.

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By the way, ruminants secrete lots of saliva too. Yes, like all of the other animals we've discussed so far, those salivary secretions help ruminants to form slippery food boluses. Plus, ruminant saliva contains more electrolyte buffers to support the microbes in the rumen. A slightly alkaline environment is perfect for them. And that's what ruminant salivary secretions help maintain. How much saliva do ruminants produce? Well, depending on the animal, it could be anywhere from 26 to nearly 40 gallons of saliva per day. That's a lot of saliva!

Transit

So, on the basis of cud chewing, we already know that transit is not quite as straightforward in ruminants as it is in monogastric animals. Chewing their cud adds a few extra, but valuable steps.

Pharynx and Esophagus

In the mouth, forages, roughages, and other foods are chewed, mixed with saliva, and formed into a bolus. The tongue helps to push the food bolus into the pharynx and into the esophagus. Esophageal peristalsis pushes the food bolus along and into the rumen. By the way, the opening between the esophagus and rumen is wide open. This facilitates ease of *eructating* rumen contents, so the animal can chew its cud. And remember, the eructation, cud chewing, and swallowing cycle happens many, many times.

Rumen

Ruminants have a multichambered ***forestomach*** (*fore-* before). The first and largest of those chambers is the ***rumen***, hence the name: *ruminant*. As you can see in Fig. 7.21B, the rumen takes up nearly half the space in the abdominal cavity. The rumen is virtually the only thing occupying the left side of the abdomen. The size of it is representative of its importance in the digestive process of ruminants. You see, here is where the bulk of the *microbial fermentation* of roughages takes place. If you were a microbe who ferments roughages, this is THE place to be! Microbes in the rumen can "party 'til the cows come home"! (Yes, pun intended.) Do you remember when we discussed microbial fermentation in the horse,

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we said that CO_2 and methane were negative by-products of fermentation? Well, in ruminants, those gases are actually a good thing. Gases rise, right? So, with these gases bubbling through and building up in the rumen, they help with eructation. Without those gases, it would be much more difficult for a ruminant to belch up cud to chew again.

The rumen is a muscular organ that slowly and methodically turns and churns its contents. The slow, methodical, muscular churning is called a ***rumination*** [*rumin(o)-* rumen, ruminate + *-tion* act of]. The mixing, created by ruminations, ensures that all of the contents are chewed adequately and thoroughly exposed to the microbes. The mixing also helps keep the microbial population happy and healthy. But *ruminations* are relatively infrequent. An adult cow, for example, may *ruminate* only once or twice, maybe even three times a minute. Smaller ruminants are similar. So, if you are examining a ruminant, be patient as you auscult (listen with a stethoscope) the left side of the abdomen. You really do need to listen for a full minute or two to know if that rumen is doing what it needs to do. What does it sound like? Well, have you ever listened to the low, barely audible rumble of a distant thunderstorm? You may even say to yourself or someone with you: "Did I hear thunder?" Then you listen intently again. Ruminations are like that —low, barely audible rumbles. Of course, if you're in a noisy barn, it will be impossible to hear them. So, an alternative evaluation for *ruminations* is to push your fist firmly into the left ***paralumbar*** [*para-* near + *lumbar*] ***fossa*** [L. *fossa* trench, depression, hollow]. (The paralumbar fossa is the depression of the lateral abdomen, just ventral to the lumbar spine.) On a cow, go ahead and lock your elbow and lean into it with some body weight. When the muscular contraction of a rumination happens, it will push your hand out. Yep, the contraction is strong enough to push your hand out, even with your elbow locked. If you don't hear or feel any ruminations over a minute or two, there's a problem. If the rumen doesn't churn, microbes die. And that's a really big problem.

There are a number of factors that could contribute to slowing and stopping ruminations. In dairy cattle, one of the most common contributing conditions is a *displaced abomasum (DA)*. We'll discuss that in a little while. For now, understand that it is a major digestive

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disorder that halts ruminations and leads to the death of microbes in the rumen. But if the microbes die, where do we find replacements? We get them from another healthy cow. How? Well, there are two methods. First, we can attempt to pass a tube the size of a garden hose through the mouth and esophagus into the rumen of a healthy cow. Then we can siphon some of the rumen contents out. Most cows object to this. The other, more popular method is through a ***rumenostomy*** [*rumen* + *-ostomy* creation of a "mouth", opening].

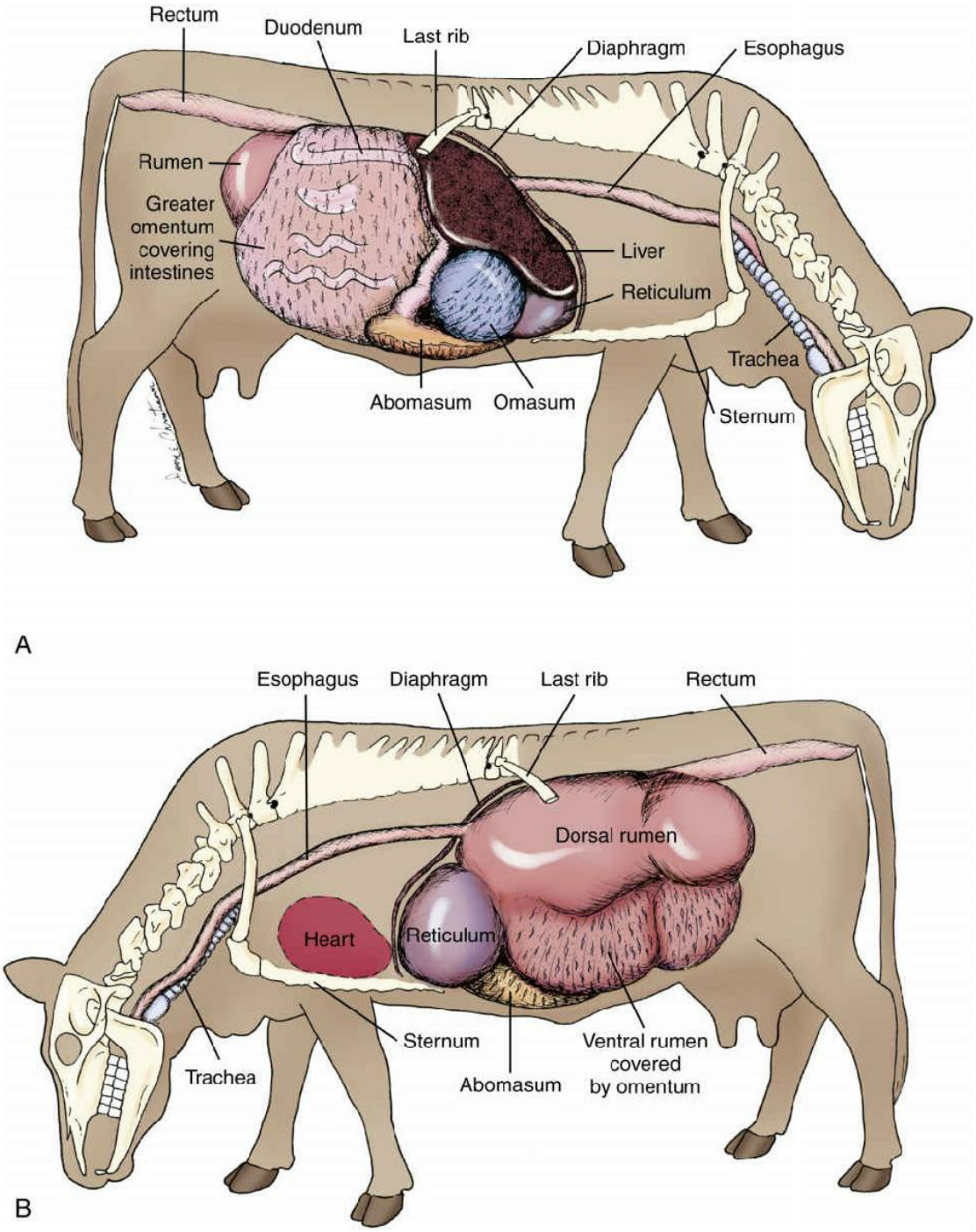

FIG. 7.21 Bovine abdominal viscera: (A) right lateral view; (B) left lateral view.

During the surgery for a *rumenostomy*, a flexible, plastic "porthole" is surgically placed through the left paralumbar fossa into the rumen. (This is done with the animal wide awake and standing. We anesthetize the surgical site with a regional block, so that she doesn't feel a thing. She usually just stands there chewing her cud. Amazing, huh?) The porthole has an outer ring that is affixed to the margins of the opening through the body wall and

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rumen. At the center of the porthole is a large, removable plug. Most dairy farmers keep one or more cows, depending on size of the total herd, with *rumenostomies*. Anytime, another cow needs to replenish her rumen contents with fresh microbes, we simply remove the plug from the rumenostomy porthole and collect some fresh juices from the healthy cow's rumen. (The rumenostomy opening is big enough to reach in with our whole hand and arm.) We take what we need and then give them to the sick cow. If you ever do this, wear gloves … that is, one of the long OB sleeves that cover your arm up to the shoulder. If you get rumen contents on you, you'll smell like rumen contents for days. (Been there; done that.)

Reticulum

The ***reticulum*** is a small chamber that freely communicates with the rumen. Its interior surface is rather honeycombed in appearance. That geometric pattern is common to many nets, such as fishing nets. As you may recall, fromChapter 2, the term *reticulum* means "net" or "network." Boom—That's how it got its name.

The reticulum is the most cranial of the chambers of a ruminant's *forestomach*. In fact, if you look at Fig. 7.21B, you can see that the reticulum lies right next to the diaphragm. (Hold that thought.) This is the part that was modified out of the digestive tracts of llamas and alpacas. They don't have a reticulum. Do they need one? Probably not. I mean really, the reticulum doesn't do much. Frankly, I think it's a bit of an unnecessary redundancy. So, for llamas and alpacas not to have a reticulum is probably just as well. You see, the reticulum tends to collect heavy objects. It lies on an angle below the esophageal opening. If heavy objects, such as nails, screws, bolts, and wire, are consumed, they will tend to drop straight down into the reticulum. And if they happen to fall into the rumen, ruminations, moving ingesta from the rumen to the reticulum, may push the metallic objects into the reticulum. And that can lead to "***hardware disease***."

Hardware Disease

Sharp metal objects (i.e., metal hardware) in the reticulum may cause ***reticulitis*** [*reticul(o)-* reticulum + *-itis* inflammation of]. This is

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one facet of ***hardware disease***. And this alone can be problematic. It's one of those factors that may contribute to stopping ruminations. But there are more serious consequences. Take another look at Fig. 7.21B. Look at what's on the opposite side of the diaphragm from the reticulum. That's right, the heart. Now consider this. Ruminations push rumen contents into the reticulum before the ingesta moves on to the next chamber (the omasum). If there are metallic objects in the reticulum, they will be pushed cranially. And if those objects are sharp like nails and wire, they may be pushed through the cranial wall of the reticulum. Another push and they may penetrate the diaphragm. Yet another push and they may penetrate the heart. If that happens, game over.

Ruminants do not have prehensile lips like horses. So, they cannot be discerning over what they take into their mouths. This is especially true for cattle. They can easily consume metal hardware. (Have you ever been around a barn, barn yard, or pasture? There's metal hardware laying around all over the place!) So, if ruminants are likely to ingest metal hardware, is there anything we can do to prevent them from getting hardware disease? Yes. We often place a magnet in the reticulum. We give the magnet just like we would any large capsule of medication. Metal objects stick to the magnet. That makes them less likely to be able to migrate and damage other organs like the heart. Is it foolproof? No. Not all metal objects are made of iron. So, not all metal objects will stick to a magnet (e.g., copper wire or aluminum nails). But the magnet reduces the chances of most objects causing significant damage.

Omasum

The ***omasum*** is a small, round chamber. It is the last in the series of forestomach chambers. Its interior reminds me of an automobile air filter, with many layers of leaflets or plies. Ingesta from the reticulum enters the omasum. As the ingesta moves through the omasum betwixt and between all of the many plies of tissue, it is squeezed and further pulverized by the muscular contractions of the omasum. Absorption of water and salts is probably the most important function of the omasum. The remaining ingesta is pushed onward to the abomasum.

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Abomasum

The ***abomasum*** [*ab-* away from + *omasum*] is often referred to as the "true stomach" of ruminants. As you can see in Fig. 7.21A and B, the abomasum is found along the ventral midline of the abdominal cavity. Its structure and function are most like the monogastric stomach compared to any other portion of the ruminant digestive tract. That said, it has less glandular tissue. Ruminants really don't need as much glandular tissue in their abomasums. Think back to the monogastric stomach. What did we need HCl and pepsin for?—to initiate protein digestion. Well, by the time ingesta gets to the abomasum of a ruminant, digestion is well underway. So, typical stomach secretions are simply not that important. Really, the abomasum becomes more of a temporary holding tank for the ingesta. And like the monogastric stomach, the abomasum will titrate ***abomasal*** [*abomas(o)-* abomasum + *-al* pertaining to] contents into the duodenum. But if anything disrupts and slows abomasal motility, it may fill with gas and become displaced from its normal position in the abdomen.

Displaced Abomasum

A ***displaced abomasum (DA)*** is one of the most common disease events in the life of a dairy cow. Yes, other ruminants can certainly develop a DA, but it is most common in dairy cows. Most commonly, a DA develops after the cow gives birth. There may be a number of factors contributing to displacement in these new mothers. Really, anything that disrupts and slows *abomasal* motility may promote filling of the abomasum with gas. Bubbles in a glass of soda rise to the top of the glass, right? Well, gas in the abomasum will make it rise in the abdominal cavity. The more gas filled the abomasum is, the higher it will rise in the abdomen, just like a balloon. And because its normal position in the abdomen is along the ventral midline, the abomasum may float up to the left (***left displaced abomasum***; ***LDA***) or to the right (***right displaced abomasum***; ***RDA***). The majority of cows develop an LDA. In worst-case scenarios, the abomasum may ***torse*** (twist) as it floats up. A torsed abomasum (***RTA*** or ***LTA***) is a true surgical emergency, just like a dog with GDV.

Now, we know that motility was already slowed, creating this

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problem. But now that the abomasum is filled with gas and abnormally positioned like a dog with bloat, the entrance to and the exit from the abomasum are blocked. If the abomasum is torsed, blood flow to the abomasum is also severely compromised. In either case, abomasal obstruction promotes even more distension with gas. Do you remember the "*ping*" that we could elicit by tapping on the abdominal wall over the distended stomach of a dog with bloat? Well, the same type of *tympany* can be elicited from the distended abomasum of a cow. In most LDAs, the abomasum will float up dorsally to the left paralumbar fossa. The fossa usually appears to have an abnormal bulge and, when we tap on it, it "pings."

How are cows with a DA treated? Surgery is the most common and successful treatment option. It is the only option with *abomasal* torsion. Surgery for a DA is done with the cow standing. Entry for the *laparotomy* will be a flank incision through the *paralumbar fossa*. A regional anesthesia block is used to desensitize the skin, muscle, and peritoneal wall. It is the surgeon's preference for either an ***ipsilateral*** [*ipsi-* same + *later(o)-* side + *-al* pertaining to] or ***contralateral*** [*contra-* opposite + *later(o)-* side + *-al* pertaining to] incision, in relation to the displacement. So, for an LDA, the surgeon may make the incision for the laparotomy on either the right or the left. If the surgeon enters on the left, the abomasum is at the incision site, making it easy for the surgeon to ***aseptically*** [*a-* without + *sept(o)-* microbes, infection + *-ically* pertaining to] *trocarize* the abomasum to evacuate all of the gas. (In the case of an LTA, the surgeon would also *detorse* the abomasum. For that, an *ipsilateral* laparotomy is absolutely necessary.)

Once the abomasum is decompressed, the surgeon can push the abomasum back to the ventral abdomen where it belongs and perform an ***abomasopexy*** [*abomas(o)-* abomasum + *-pexy* fixation of]. Yes, the suturing of the *abomasopexy* is somewhat "blind" and requires the surgeon to reach into the abdomen up to his or her shoulder. A sterile OB sleeve (i.e., a type of glove that covers the hand and arm up to the shoulder) is worn to maintain ***asepsis*** [*a-* without + *sep(o)-* infection + *-sis* state or condition of]. But a skilled surgeon can successfully perform the abomasopexy. And this is necessary to prevent repeated displacement. Her abomasal motility

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is still poor and probably worse than before it displaced.

If a *contralateral laparotomy* is performed for the LDA, the surgeon won't be able to access the abomasum directly. But there is *omentum* attached to the abomasum. And the surgeon can pull on the omentum to reposition the abomasum along the ventral abdomen. Often, someone will need to trocarize the abomasum through the left flank to relieve enough gas so that the surgeon can more easily pull the abomasum down to the ventral abdomen. The surgeon still won't be able to actually touch the abomasum. So, to help hold it in position, an ***omentopexy*** [*oment(o)-* omentum + *-pexy* fixation of] is performed.

DAs frequently cause *ruminations* to stop. You know that means microbes in the rumen die. So, transplanting fresh, live microbes from a healthy cow's rumen is part of the ***postoperative*** [*post-* after + *operat(o)-* surgery + *-ive* pertaining to] care for a DA cow. This is why most dairy farmers keep a few *rumenostomy* cows on the farm. Those girls are invaluable for their DA girlfriends. I said at the start of this discussion that DAs frequently occur in cows shortly after ***calving*** (giving birth to a calf). She probably started producing milk shortly before calving. ***Lactation*** [*lact(o)-* milk + *-tion* state of; i.e., milk production] can create electrolyte imbalances that may have contributed to the poor abomasal motility. So, we may need to treat her electrolyte disturbances. The other factor that commonly plays into a DA is an abrupt dietary change late in pregnancy. The dietary change is well intended to meet the growth and energy needs of the calf and the cow. Unfortunately, abrupt dietary changes alter microbial populations, especially in the rumen. And that can create a whole cascade of pH and electrolyte changes that alter gut motility. So, to try to prevent DAs in our patient, and in the herd, avoidance of abrupt dietary changes is important.

Small Intestine

Alrighty, let's put abnormal situations like DAs aside. In normal ruminants, the abomasum titrates its liquid contents into the duodenum. Remember, the microbes in the rumen have already made a significant impact on digestion. They have freed up numerous nutrients and produced wonderful by-products (volatile fatty acids and B vitamins). Many of these nutrients and by-

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products can be readily absorbed by the duodenum and jejunum. We'll still have *bile* and *pancreatic enzymes* secreted into the duodenum. Those secretions will finish off the digestive process that the microbes began. By the time the ingesta leaves the small intestine, most available nutrients that were available in the food are absorbed. There is the efficiency of the ruminant digestive tract. There is very little waste. What's left in the fecal matter is predominantly the woody parts of plants. Only termites can digest that.

Cecum, Colon, and Rectum

The *cecum* of ruminants is relatively small. We don't need a large cecum for microbial fermentation. That's what the rumen is for. The structure of the colon and rectum varies, depending on the ruminant. The function is the same as other animals, with a primary focus on absorption of water. How efficient that water absorption is depends on the actual structure of the colon and rectum. The longer the colon and the larger number of well-defined *haustrations*, the better the water absorption. So, the ovine (sheep) and caprine (goat) colon is arranged in a spiral of sorts. This increases its overall length. The small, well-defined haustrations throughout the colon and rectum give sheep and goats lots of surface area for tremendous absorption of water. The end result is little pellets of feces. Comparatively, the bovine (cattle) colon and rectum is not as long and does not have well-defined haustrations. So, water absorption is not as great from a cow's colon and rectum. (But that's okay, water absorption from the rumen, reticulum, and omasum compensates to maintain good hydration.) Of course, this means that the fecal matter of cattle tends to have a more liquid consistency—i.e., "cow pie."

Scours

Scours is a term that is used for diarrhea among ruminants. Many factors can cause *scours*, from parasites to infectious viral and bacterial diseases. For those ruminants who tend to have pelleted feces, it's easy to see when they develop scours, so that we can quickly treat them if needed. In cattle, it can be a little more challenging to recognize scours. I mean, how liquid is too liquid?

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Okay, this is going to be a little gross, but we're going to have this discussion anyway. Normal bovine fecal material forms a "cow pie," right? Well, if you've ever really looked at a cow pie, you know that it does pile up into a broad, rounded mass of feces. In any bovine with scours, the fecal matter is super watery. It forms a puddle, not a pile. By the way, some of the ruminant parasites that often cause scours are blood suckers. Many of the worst blood suckers reside in the abomasum. So, even ruminants can have *melenic* scours. It is an important observation to make because it will guide our treatment protocol.

There! You did it! You waded through an awful lot of digestive information. Hopefully, the repetition of those structures and functions that are common across species made this journey a little easier for you. Now, take a little break. You've earned it. Then, let's wade into our case study.

Case Study

Sadie is an 11-year-old, spayed female Basset Hound, presented on emergency by her pet sitter. Per the pet sitter, Sadie was normal when she went outside at 6:00 a.m. to urinate and defecate. She ate breakfast at approximately 6:15 a.m. Sadie went outside, with her house mate, to go potty around 9:30 a.m. for approximately 10 minutes. When the sitter called Sadie to come inside, Sadie refused to move. She exhibited an odd stance, with her head held low. The sitter, a veterinary technician student, quickly evaluated Sadie for injuries and vitals. She found no abnormalities, other than acute depression. Because the pet sitter would be gone the rest of the day, she thought it best to bring Sadie in to the ER. She phoned the owner (a credentialed veterinary technician) to alert her of a potential problem. In route (a 25-minute drive), Sadie began episodes of unproductive retching.

On presentation, Sadie's physical examination findings included abdominal distension with tympany, difficulty breathing, mucous membrane pallor, hypotension (mean BP 55 mm Hg), rapid heart rate (150 bpm) with weak and thready pulses, capillary refill time > 3 seconds, and unproductive retching. Body weight 55 pounds,

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BCS 3/5. During physical assessment of Sadie, two 18-gauge IV catheters were placed in peripheral veins, per shock protocol. A 1-liter bolus of Lactated Ringer's solution (LRS) was administered, while Sadie's stomach was trocarized. She remained hypotensive following the fluid bolus and trocarization. A second bolus of LRS was initiated in route to radiology. A 125-mL bolus of IV Hetastarch (a plasma expander) was also initiated. Abdominal radiographs confirmed gastric dilatation volvulus. Sadie's surgical prep for the emergency laparotomy was initiated, while the surgery and anesthesia teams were summoned, STAT.

The surgeon made a ventral midline incision from xiphoid to pubis. He quickly detorsed the stomach. Nonsterile personnel then passed an orogastric tube to evacuate gastric contents and perform gastric lavage, while the surgeon assessed the integrity of the stomach and spleen. Circulation was slow to be restored to portions of the gastric wall and spleen. However, there did not appear to be any obvious areas of necrosis. On restoration of gastric and splenic circulation, the surgeon determined that neither a splenectomy nor partial gastrectomy was warranted. He then proceeded to perform a gastropexy. After peritoneal lavage with warm, sterile saline, the abdomen was closed in a routine manner.

Sadie's postoperative recovery from anesthesia was uneventful. However, several of her vitals remained outside the normal range for several hours, including rapid breathing (36 to 40 bpm), rapid heart rate (115 to 120 bpm), hypotension (mean BP 75 to 80 mmHg), mucous membrane pallor, and capillary refill time >2. In addition to continued IV LRS at 100 mL per hour, Hetastarch was continued at 15 mL per hour, along with IV antibiotics, IV famotidine (an H_2 blocker), and IV hydromorphone (an opioid) for pain. She was to receive continual monitoring over the next 24 hours, including continuous ECG for any abnormal cardiac rhythms.

Sadie's vitals steadily improved over the next 12 hours. She was discharged 3 days postop. The owner was instructed to feed 4 to 6 small meals per day, of Sadie's regular kibble, presoaked in warm water for the first week she's home. Feeding volumes and frequency were to be gradually adjusted during the second week. By her 3rd week home, she could resume regular feedings two to

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three times per day. All postprandial exercise and play are to be avoided for at least 2 to 3 hours for the remainder of Sadie's life. Sadie fully recovered and lived to a ripe old age of 13 ½ years.

Case Study Questions

1. On presentation in the emergency room, Sadie had extremely low blood pressure, which is medically termed _____.
2. When examining Sadie's distended abdomen, tapping on the abdomen over the stomach created an audible "ping," also called gastric _____.
3. To relieve some of the pressure in Sadie's stomach, the clinician performed a procedure using a large needle to puncture through the abdominal and stomach walls. This procedure is called _____.
4. Sadie was diagnosed with extreme stomach expansion and twisting, which is medically termed _____ (3 words).
5. Emergency surgery, called a _____ (i.e., cutting into her abdomen), was necessary to save Sadie's life.
6. After untwisting the stomach, the surgeon determined that it was not necessary to remove a portion of the stomach, medically termed a partial _____.
7. Even though the spleen was twisted with the stomach, it was not necessary to perform a _____ (removal of the spleen) either.
8. The reason those organs were not removed is because the surgeon saw no evidence of tissue death, medically termed _____.

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9. To prevent future emergency situations like this for Sadie, the surgeon also sutured the stomach to the abdominal wall—a procedure termed a _____.
10. While the surgeon was evaluating the stomach and spleen during surgery, someone else passed a tube from the mouth to the stomach to evacuate stomach contents. That tube is called an _____ tube.
11. It is very important for patients like Sadie to avoid _____ (i.e., after meals) exercise and play.
12. Before closing Sadie's abdomen, the surgeon performed _____ (i.e., abdominal) lavage with sterile saline to remove any contaminants.

The Answer Key to these case study questions may be found in Appendix B.

[a] Dawn is a registered trademark of the Procter & Gamble Company.

[b] Couto CG and Nelson RW. *Small Animal Internal Medicine*, 2nd Ed., Mosby; 1998: 558.

[c] *Cujo*, a novel by Stephen King that became the basis for the movie *Cujo*, Paramount Pictures, 1983.

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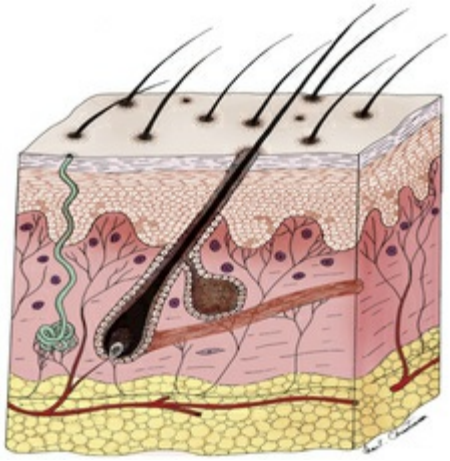
Applied Terminology for the Integumentary System



Skin and Integument,
- Epidermis,
- Common Forms of Skin Cancer,
- Dermis,
- Intradermal Vessels,
- Thermoregulation,
- Subcutis,

Accessory Structures,
- Sebaceous Glands,
- Anal and Perianal Glands,
- Interdigital Glands,
- Lanolin Glands,
- Circumoral Glands,
- Cornual Glands,
- Horns and Antlers,

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- Nails,
  - Onychectomy,
  - Hooves,
  - Laminitis,
  - Paw Pads and Noses,
- Hair and Fur,
  - Guard Hair,
  - Wool Hair,
  - Tactile Hairs,
  - Hair Growth,
  - Alopecia,
- Burns,
  - 1st Degree Burns,
  - 2nd Degree Burns,
  - 3rd Degree Burns,
- Wound Healing,
  - Inflammatory Stage,
  - Debridement Stage,
  - Repair Stage,
  - Maturation Stage,
  - Keloid,
  - Proud Flesh,
- Allergic Dermatitis,
  - Flea Allergy Dermatitis,
  - Food Allergy Dermatitis,
  - Atopic Dermatitis,
- Case Study,

GOALS AND OBJECTIVES

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By the conclusion of this chapter, the student will be able to:

1. Recognize common root words, prefixes, and suffixes related to the integumentary system.
2. Divide simple and compound words into their respective parts.
3. Recognize, correctly pronounce, and appropriately use common medical terms related to the integumentary system.
4. Demonstrate an understanding of skin anatomy, as well as accessory structures such as glands, nails, hooves, and horns.
5. Demonstrate familiarity with hair structure, growth, loss, and abnormal loss.
6. Demonstrate familiarity with wound healing.
7. Demonstrate familiarity with common diseases, such as allergic dermatitis and laminitis.

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Skin and Integument

What's so special about skin? I mean, okay, we have it. Big deal, you say? Yes, it's a big deal, a very big deal. Did you know that the skin is the largest organ of the body? The skin is an organ?! You bet it is! It is a very important organ—one that we so often overlook and take for granted. And let's face it, it's tough to overlook, since it covers the whole body. That's a lot of surface area! In spite of our taking the skin for granted, it provides protection from the hostile environment around us in many, many ways. Why, it even produces essential things, like vitamin D, for the body. Frankly, our survival depends on this remarkable organ and the whole of the ***integumentary*** [*in-* on + *tegument* from *L. tegere* to cover + *-ary* pertaining to] ***system*** (i.e., the skin and all its related structures). Let's take a "skin-deep" look at the structure and functions of the *integument*. That's right, more anatomy—but if you don't know the players, how can you understand the game? Refer to Fig. 8.1 for much of these discussions. As you can see, the skin is fundamentally made of two layers: the ***epidermis*** [*epi-* on, upon + *dermis* the skin] and the ***dermis***. We'll discuss each of them separately, beginning with the top layer, the *epidermis*.

Epidermis

The *epidermis* is the most superficial layer of the skin. It's composed of ***stratified*** (layered) ***squamous*** [*squam(o)-* scales + *-ous* pertaining to] ***epithelium*** (Fig. 8.2). As you can see, this type of epithelium is truly layered. Only the ***basal*** [*bas(o)-* base + *-al* pertaining to] cell layer, next to the basement membrane, undergoes mitosis. (Refer to Chapter 2 if you need to review mitosis.) From there, the new daughter cells get pushed up into the transitional layers. The closer to the surface the epithelium progresses, the flatter the cells become. They'll actually lose their nucleus. Eventually, at the very surface, the cells are completely flat like fish scales (hence the term "squamous"). Those superficial cells ultimately flake off. We've all experienced dry, flaky skin, right? Well, those flakes are squamous cells. Still, all of those ***keratinized*** [*kerat(o)-* "horn"; i.e., made tough

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like the stuff of horns] *squamous epithelial cells* of the ***stratum corneum*** can make up over half the thickness of the epidermis.

Now, it may seem odd that squamous epithelium ultimately flakes off. But that's by design. These cells are routinely being turned over and replaced with new ones. The old, crusty ones are expendable. Under normal circumstances, this provides us with fresh, new cells to maintain an appropriate thickness to the epidermis. And that's really important, because that's our first line of defense. Yep, the *epidermis* provides a physical barrier to potential ***pathogens*** [*path(o)-* disease + *gen(o)-* producers; i.e., disease organisms like bacteria]. In fact, when the most superficial, crusty squames of the *stratum corneum* are kept soft, supple, and flexible with natural oils of the skin, it's not only waterproof but virtually impenetrable to microbes and water-based chemicals. We'll talk about where those oils come from in a little bit. By the way, the waterproofing works two ways. First, it allows water to roll off like water off of a duck. Obviously, many animals (like ducks and other waterfowl, along with seals and otters, to name a few) have way better waterproofing than humans. Their feathers and fur are well oiled, making them buoyant and keeping the water away from the skin. That last part is really important for those animals swimming in cold water! Second, for all of us (animals and humans), the waterproof epidermis prevents evaporation of water from the body. Another factor making the epidermis a barrier against *pathogens* is its ***avascular*** [*a-* without + *vascul(o)-* vessels + *-ar* pertaining to] nature. Without vessels, pathogens can't gain quick and easy access to the rest of the body. But if the epidermis is *avascular*, how on earth does it survive? Well, the basal cell layer and transitional layers of epithelium are "fed and watered" from below by the ***vascular*** [*vascul(o)-* vessel + *-ar* pertaining to] *dermis*.

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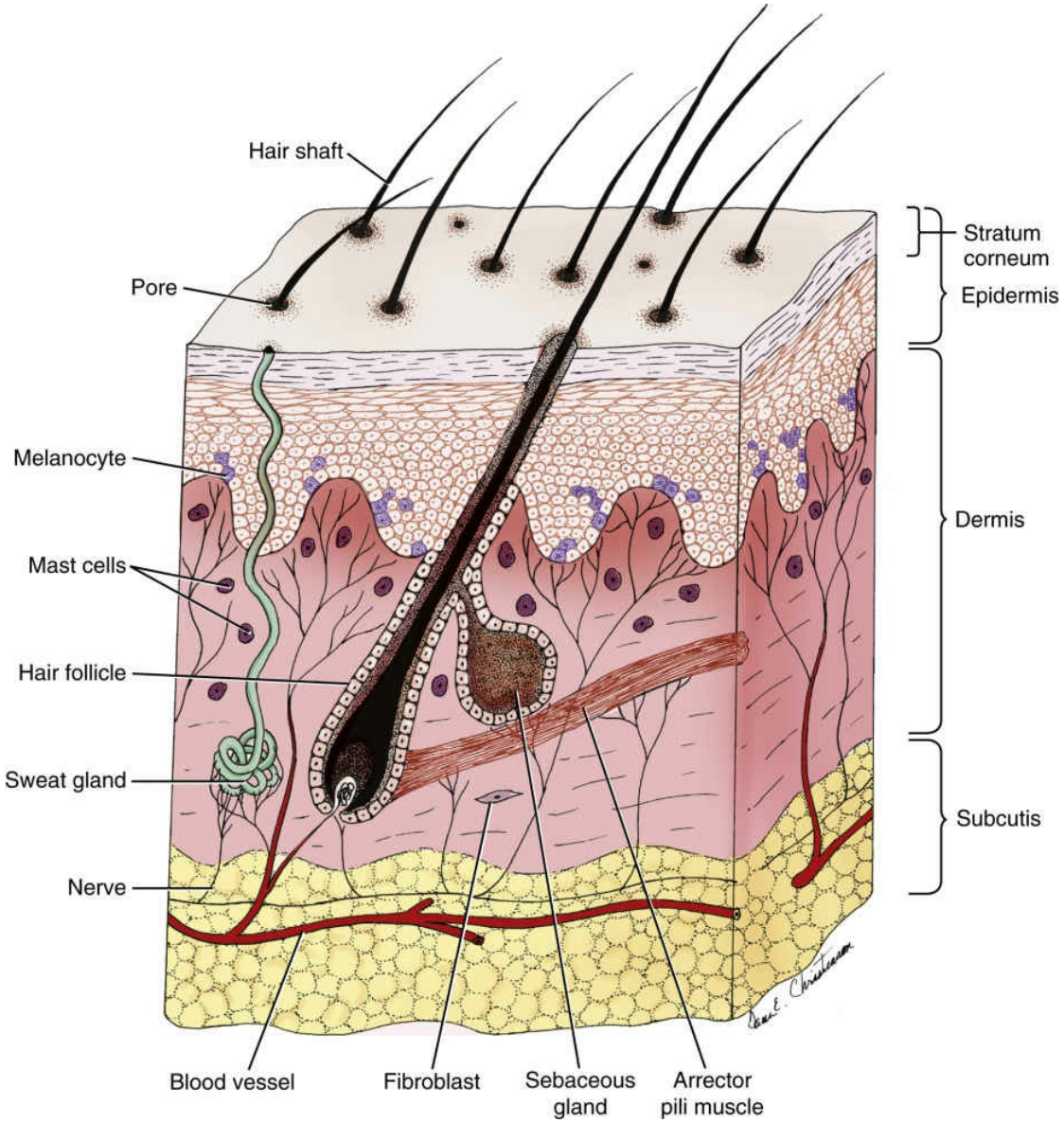

FIG. 8.1 Skin cross section.

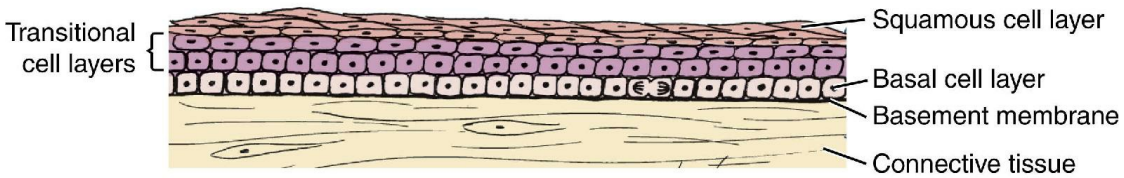

FIG. 8.2 Stratified squamous epithelium.

But wait, that's not all! There are other ways that the epidermis provides protection for the body. We all know that ultraviolet (UV) radiation can be very bad and can lead to skin cancer. That's why people wear sunscreen, right? Did you know that we also have a

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little built-in UV protection? That's right. ***Melanocytes*** [*melan(o)-* black + *cyt(o)-* cell] in the epidermis provide UV protection for the dermis and deeper tissue layers. The melanin in their cytoplasmic granules actually absorbs UV light. The more melanin in the epidermis, the better protection is provided for deeper skin and tissue layers. I liken it to pulling down a window shade to keep the sun out. But there aren't that many melanocytes in the epidermis. If that's the case, then how can the protective "shade" be drawn? As you can see in Fig. 8.3, a *melanocyte* is a rather odd-looking cell. It has some really weird cytoplasmic "tentacles," kind of like an octopus. Those tentacles allow the melanocyte to share portions of its cytoplasm and pigmented granules with other cells in the epidermis. Voila! We then have an abundance of pigmented epithelium in the epidermis—shade drawn! This is how and why people with naturally light-colored skin become tanned. Of course, the tan is only temporary. When no longer stimulated by exposure to the sun (UV light), the pigmented epithelial cells are eventually pushed to the surface and flake off. And that's how your tan is lost.

Common Forms of Skin Cancer

Now, before you think that your tanned skin is all the protection you need, think again. People should still use sunscreen, especially those who are fair-skinned and have few to no melanocytes. Failure to protect the basal cell layer of the epidermis could result in a very serious form of skin cancer: ***squamous cell carcinoma*** [*carcin(o)-* "crab", cancer + *-oma* tumor; carcinomas are derived from epithelium]. In fact, animals can develop *squamous cell carcinoma* too. I have seen numerous horses, cats, and dogs develop this form of skin cancer. It usually develops in nonpigmented areas of the body with very little or no fur like on the face and ears. For hairless breeds, it may develop anywhere on the body. And in some dominantly black dogs (like the Gordon Setter, Rottweiler, and Doberman Pinscher), they tend to develop this in their toes. I mentioned this in Chapter 4 with regard to our Gordon Setter Serena, who had a number of toes amputated due to squamous cell carcinoma. Regardless of who it affects or where on the body it is found, *squamous cell carcinoma* can be very aggressive and devastating. So sunscreen or other forms of protection from the sun

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are important for people and animals, especially for those who have little pigmentation and fur (speaking of animals, of course). I'm not a big advocate for dressing dogs or cats, but if it's for protecting the skin of a thin-coated or hairless beast, I'm all for it!

But even those with significant pigmentation like Rottweilers and similar breeds, they are not immune from cancer. In fact, those with pigmentation are more likely to develop a highly malignant and ***metastatic*** [*meta-* beyond + *stat(o)-* standing + *-ic* pertaining to; i.e., it spreads beyond its place of origin] form of skin cancer: ***malignant melanoma*** [*melan(o)-* black + *-oma* tumor]. Why is *malignant melanoma* more likely to ***metastasize*** (spread)? Well, think about the nature of melanocytes. Remember, they can easily and rapidly share portions of their cytoplasm and cytoplasmic granules with other cells. All we need is one rogue melanocyte sharing portions of its abnormal self and we've got the disease spreading like wildfire. What's the relationship between "going rogue" (i.e., becoming malignant) and sunshine? Remember, the melanin of these cells absorb UV light. UV radiation over time can alter cells, making them cancerous. That means that *all* people should protect themselves from the sun and UV radiation. As people, we can easily wear protective clothing and use sunscreen as well as minimize time in the sun during peak times of the day. Animals are a bit more challenging to protect. Their fur can provide a little protection, but it's not enough. Hairless and thinly coated areas are left at risk. So, minimizing sun exposure during peak periods of the day is very important. Sunscreen will only offer protection if it's in an area where the animal can't lick it off.

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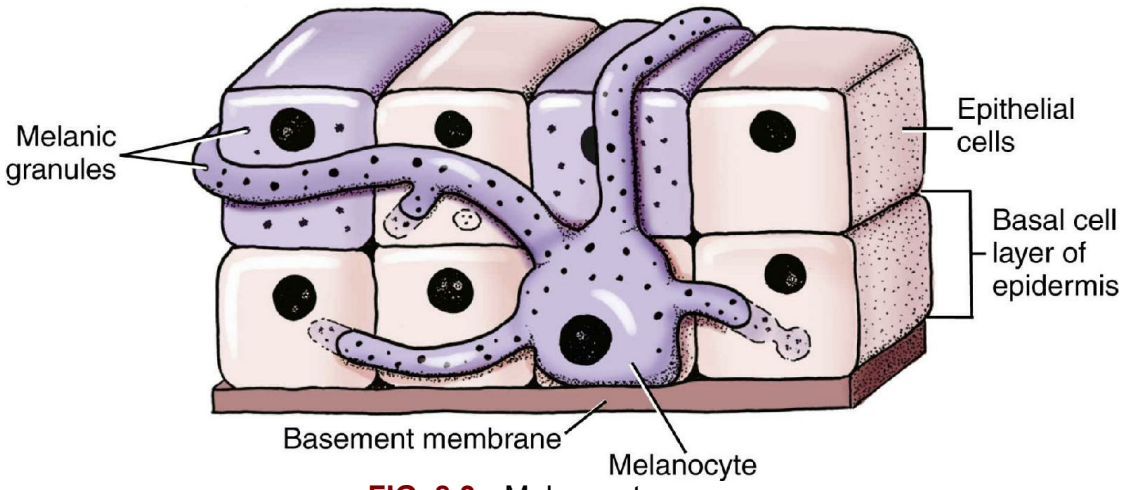

FIG. 8.3 Melanocyte.

That concludes our focus on the epidermis for now. Let's go deeper and take a look at the dermis.

Dermis

Again, looking at Fig. 8.1, you can see that the *dermis* is the thickest portion of the skin. It is composed of a ***fibroelastic*** [*fibr(o)-* fiber + *elastic*] ***connective tissue***. This makes it relatively tough and flexible at the same time. Notice, as you look at Fig. 8.1, that the intersection between the dermis and epidermis is not flat. The surface of the dermis has numerous bumps called ***papillae*** [L. *papilla* nipple; i.e., nipple-shaped projections]. This basic structure increases surface area between the dermis and epidermis for greater nutritional support of the epidermis. It also provides a more secure connection between the skin layers. Deeper into the dermis we find a host of supporting structures, such as vessels, glands, nerves, and such. These are the things that make the skin a living, breathing organ. More on those in a moment.

You'll notice, if you compare the dermis vs. the epidermis, that the dermis doesn't seem to have as many cells. You're right, it doesn't. It has a few ***fibroblasts*** [*fibr(o)-* fiber + *blast(o)-* germ, shoot]. If you remember from Chapter 3 any cell with "blast" in its name is some sort of building-block cell. For example, ***rubriblasts*** [*rubri(o)-* red + *blast(o)-* germ, shoot] ultimately create red blood cells. In Chapter 4 we learned that ***osteoblasts*** [*oste(o)-* bone + *blast(o)-* germ, shoot] actually produced the hard stuff that makes up bones. Well, *fibroblasts* produce the ***fibrous*** [*fibr(o)-* fiber + *-ous* pertaining to]

material of the skin and other connective tissues called ***collagen*** [*coll(o)-* glue + *gen(o)-* produce]. The *collagen* of *fibrous connective tissues* literally holds the body together. Please refer to Chapter 4 to review connective tissues. We'll talk about fibroblasts and collagen again, when we discuss wound healing, because this is the important stuff of scars. Wait. Didn't I say that the dermis was made up of fibroelastic connective tissue? Yes, I did. What about the elastic part? Well, that's irreplaceable. As we'll learn later, scar tissue is not as flexible as normal skin. That's why scars are roughly 15% weaker than normal skin. But let's talk about that later. For now, the important thing to remember about *fibroblasts* is that they are few and far between in healthy skin (and other connective tissues, for that matter).

Another cell type found in the dermis that is more abundant is the mast cell. We'll talk about ***mast cells*** again when we discuss ***allergic*** [*allerg(o)-* allergy + *-ic* pertaining to] ***dermatitis*** [*dermat(o)-* skin + *-itis* inflammation of]. To review immunology as it relates to allergies, please refer to Chapter 3. As discussed in Chapter 3 *mast cells* are found in abundance in the skin, along the respiratory tracts and the digestive tracts of animals. Their greatest abundance is in the skin. This is why animals tend to manifest allergies as *dermatitis*. A particular antibody (***IgE***) "arms" the mast cells. In an allergic reaction, the armed mast cells fire off the contents of their cytoplasmic granules. Those granules contain a number of inflammatory compounds, primarily histamine. And in the presence of histamine, inflammation develops—that means dermatitis, for the purpose of this discussion. If you've ever reacted to a mosquito bite or poison ivy, you know what *dermatitis* feels like. You know from experience that the principal symptoms are ***pruritus*** [*prurit(o)-* itching + *-us* state of] and ***erythema*** [Gr. *erythema* flush upon the skin; i.e., redness]. Speaking of *erythema*, let's talk about the vessels found in the dermis.

Intradermal Vessels

Now, we already mentioned that the dermis is ***vascular*** [*vascul(o)-* vessels + *-ar* pertaining to]. The dermal vessels are the smallest of all the vessels—arterioles, venules, and capillaries. If you need to review these tiny vessels, please refer to Chapter 5. It's primarily

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the ***intradermal*** [*intra-* within + *derm(o)-* skin + *-al* pertaining to] ***capillaries*** that provide nourishment directly for the dermis itself and, indirectly, for the epidermis. Obviously, supplying nutrients and oxygen is very important. These vessels also provide a way for the body to slowly absorb ***transdermal*** [*trans-* across, through + *derm(o)-* skin + *-al* pertaining to] medications. This and other routes of medication administration are discussed in Chapter 12. Equally important is the ***thermoregulatory*** [*therm(o)-* temperature + *regulat(o)-* control + *-ory* pertaining to] function of intradermal vessels, along with other structures.

Thermoregulation

Think about the amount of surface area on the skin. If we ***vasodilate*** [*vas(o)-* vessel + *dilate* to increase diameter] the arterioles leading to *intradermal capillaries*, we increase superficial blood flow and can rapidly dissipate heat from the body. This provides cooling by ***convection***—i.e., circulating warm blood from the core to the surface of the skin and returning the cooled blood to the core. That's much the same way the radiator cools the engine in your car. Of course, unlike your car, peripheral *vasodilation* and increased blood flow make the skin appear ***erythematous*** [*erythema* redness + *-tous* pertaining to]. Yet, *erythema* may be difficult to see in heavily coated areas of the body in animals. Areas where we may best visualize dermal vasodilation are perhaps the ears, ventral abdomen, and most certainly the mucous membranes like those in the mouth (gums and tongue). Now, you may recall that we mentioned erythema in Chapter 3. In that chapter, we mentioned other causes of erythema like inflammation. And when it comes to extremely ***hyperemic*** [*hyper-* excess + *em(o)-, hem(o)-* blood + *-ic* pertaining to] mucous membranes, we said in Chapter 3 that carbon monoxide toxicity (poisoning) could be a cause. I only point this out to remind you that there are many things that can affect dermal vessels to create vasodilation and superficial redness.

On the flip side, ***vasoconstriction*** [*vas(o)-* vessel + *constrict* make smaller] causes the decreased superficial blood flow to conserve heat for the body and regulate temperature. Depending on the degree of peripheral *vasoconstriction*, the affected skin may appear pale-pink to blanched (white). As with vasodilation, there are many

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other things besides cold that could result in peripheral vasoconstriction, including *anemia* (insufficient red blood cells), *hypotension* (low blood pressure), *hypoxia* (insufficient oxygen), pain, and even fear. So it is very important not to jump to conclusions about pallor or erythema. My goodness, fever (as we discussed in Chapter 3) can cause both, as the body attempts to maintain the new abnormal thermostatic setting. We've all experienced that—waxing and waning between hot flashes and icy chills, erythema, and pallor. As far as ***thermoregulation*** [*therm(o)-* temperature + *regulation* control] goes, suffice it to say that it is a very, very important function of the dermis. And that function doesn't end with intradermal vessels.

Do you see the ***arrector pili*** (uh-rek'tor pil-i) ***muscle*** that's attached to the base of the ***hair follicle***? (See Fig. 8.1.) Well, when animals are cold or exposed to cold conditions, these muscles throughout the dermis contract. This makes the fur stand up. (We don't have an abundance of fur, so we just get goosebumps.) With all of the fur fluffed up like that, dead-air space is created. This has an insulating effect. Of course, the more fur the animal has, the greater the insulation. We'll talk more about types of hair, hair growth, and various types of hair follicles a little later. Just recognize that ***piloerection*** [*pil(o)-* hair + *erect(o)-* to raise + *-tion, -ion* the act of; i.e., hair-raising] is yet another important way that the skin helps regulate body temperature for animals.

What about ***sweating***, you ask? After all, people sweat like pigs, right? Wrong. Pigs can't sweat. They don't have ***sweat glands***, like the one shown in Fig. 8.1. That's why they wallow in mud to cool off. In fact, most animals don't have many, if any, sweat glands. Animals like dogs and cats have only a few sweat glands in the pads of their feet. That's certainly not enough to help them cool off in hot weather. Cattle have some, enough to help keep them cool, but nowhere near as many as horses. Only horses have abundant sweat glands. So, if we're going to talk about sweating like any animal, we should say that we sweat like horses. And the way sweating helps horses and us cool off is by ***evaporation***. Evaporation (plus convection) is marginally useful for dogs and cats too … but only by moving air over the mucous membranes of their mouths when they pant. For that to be useful, the ambient

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temperature has to be cooler than body temperature with lower humidity. This is why, as we mentioned in Chapter 5, dogs and cats are at risk of ***heat stroke*** when left in hot environments like cars.

Of course, key elements that we've omitted from this *thermoregulatory* discussion thus far are nerves. Nerves will be discussed in detail in Chapter 11. For our purposes here, we need to understand that ***sensory nerves*** in the skin (and elsewhere in the body) are needed to sense both the environmental temperature and body temperature. The sensory input to thermoregulatory centers in the brain will result in appropriate physical responses. We've already discussed some of those responses like vasoconstriction, vasodilation, piloerection, panting, and sweating. But there are other responses too—like behaviorally moving toward or away from temperature differences (e.g., pigs to cool mud when it's hot outside and a cat seeking out a warm sunbeam on a cold winter's day).

Beyond thermoregulation, one of the most important abilities provided by nerves in the skin is ***tactile sensation***. Think about that for a moment. Without *tactile sensation* we and our animal kin could not physically feel anything. On one hand, that could be a good thing, because we wouldn't be annoyed by *pruritus*. The downside is that we could not feel the soft fur of animals as we pet them. Imagine a blind and deaf animal with no ***sense of touch***—unable to feel our touch or the touch of its animal companions, unable to feel the cool morning grass wet with dew, unable to feel the warmth of the sun, unable to feel its soft bedding, unable to feel its way to navigate through its environment safely, and maybe unable to find water and food. Such an animal would live a life of complete isolation—a miserable existence to be sure. Sensory nerves found throughout the dermis provide for these sensations and many more. In my opinion, the sense of touch alone makes the skin incredibly important.

Subcutis

As the old song says: "I've got you under my skin…".[a] It's a great tune. You should look it up sometime. Now, before you think I've gone completely off the deep end, that lyric and song title are right

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on the money when it comes to the ***subcutis*** [*sub-* under + *-cutis* the skin]. You see, technically the *subcutis* is truly under the skin, not part of it. It's really a transitional layer of tissue that helps connect the skin to underlying structures like muscle, fascia, and bone. But to provide ease of movement, we need a pretty loose and flexible yet supportive and cushioning type of ***subcutaneous*** [*sub-* under + *cutan(o)-* skin + *-ous* pertaining to] ***tissue***. And that is precisely what we have. The *subcutis* is made up of ***adipose*** (fat) and loose connective tissues. This is why you can push and pull on the skin and it glides freely over the underlying structures. Have you ever lifted up on the skin of your arm or the neck of an animal? It tents up easily and springs right back to its original location and shape when released, right? No harm done. You see, between the elasticity of the dermis, the slippery nature of fats, and the loose connective tissue attachments of the subcutis, the skin is less likely to tear when suffering light to moderate blunt trauma.

This flexibility also makes the subcutis an excellent location for injecting *subcutaneous* fluids, medications, or vaccinations. This route of medication administration is discussed in Chapter 12. In short, we poke a ***hypodermic*** [*hypo-* below + *derm(o)-* skin + *-ic* pertaining to] needle through the skin and deposit the medication into the subcutaneous tissue. Beyond a place to administer medications (as well as its innate cushioning and flexibility), there's another bonus to the subcutaneous adipose tissue—insulation. That's pretty important for animals like polar bears and seals who live in the Arctic, as well as other animals who experience icy winter weather. I always remind myself of this every winter when I put on a few extra pounds.

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Accessory Structures

There are so many wonderful and important accessory structures associated with the integument. We've already mentioned things like sweat glands and arrector pili muscles in the dermis. But we only barely mentioned oils that keep the epidermis soft and supple. So let's begin here by talking about the glands that produce those oils.

Sebaceous Glands

Sebaceous [from L. *sebaceus* pertaining to sebum] ***glands***, shown in Fig. 8.1, are found throughout the dermis. Most of these are found connected to hair follicles. They get their name from the ***sebum*** [L. *sebum* suet] they produce. *Sebum*, like suet, is a fatty, oily substance. It's relatively thick, semiliquid stuff, because it not only contains fats but also cellular debris from epithelial cells. The more cellular debris, the thicker it becomes. If you remember from our earlier discussion, these oils help provide that epidermal barrier against microbes. Under normal circumstances, these oils actually help prohibit colonization and overgrowth of bacteria that normally inhabit the skin. The other thing sebum does is make the skin and coat (or feathers) waterproof. As stated earlier, this function is extremely important for those animals that spend much of their time in water.

But these basic sebaceous glands are not the only ones. There are more isolated, specially adapted sebaceous glands in key locations on the body. The locations vary between species of animals. Let's investigate a few examples of these.

Anal and Perianal Glands

Who hasn't smelled a skunk? Potent, aren't they? Did you know that when skunks "spray," they're actually squirting out thick, foul-smelling sebaceous material from their anal glands? It is the eau de Cologne of the skunk world. Not that a dog's anal secretions smell much better . . . everything is relative, I guess. Back to the dog in a minute. Why on earth would a skunk's anal sac secretions smell so

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bad? Well, for one, it's a fantastic way to keep predators at bay. Second, it's a fantastic way to mark one's territory and recognize potential mates. You see, strategically located glands like these also contain something called ***pheromones*** (fār'uh-mōnz) in the sebaceous secretions. *Pheromones* are specialized scent markers that are recognized by others of the same species. And when recognized, those scents stimulate particular behaviors like giving a wide berth to an area already staked out by someone else or getting excited about a potential mate.

Because we're not skunks, we're simply repulsed by their scent markers. By the way, if you or your dog are ever sprayed by one of these stinky friends, I highly recommend the "recipe." There's actually science behind, it and it is the BEST way I've found to neutralize the odor. My beloved Corgi, Ellie, had a nasty habit of getting sprayed (even through a fence, many times!). So, I know this recipe by heart, and I always keep the following ingredients on hand: 3% hydrogen peroxide (i.e., the household kind you buy at the grocery store or pharmacy), lemon juice, Dawn dish soap,[b] and baking soda. Remember, the skunk's lovely sebaceous secretions are oily and rather alkaline. So, "Dawn cuts through grease," as they say in the ad. That's key. That's precisely why this dish soap is used for wildlife rescue and cleanup after major oil spills. The lemon juice helps alter the pH. Also, it creates one whale of a chemical reaction with the baking soda. Between the foaming of the baking soda and the bubbles from the peroxide, we get "Scrubbing Bubbles."[c] These scrubbing bubbles working with the soap help remove the sebaceous secretions from the fur and *exfoliate* (i.e., fall off like leaves) the superficial squamous epithelium.

Of course, all of this does pose a hazard to the eyes. To prevent damage to the cornea, protective eye lubricant should be applied to the eyes before using the recipe. Here's the process: in a clean bucket, mix one quart of household (3%) hydrogen peroxide with one tablespoon Dawn dish soap and two cups lemon juice. When eye lube is applied and the dog is in the tub (along with the bucket), mix 1 cup of baking soda to the liquid mixture in the bucket. It will foam, probably overflowing the bucket. Apply the mixture to the dog, working it through coat and onto the skin. Let the dog soak in the tub for 20 minutes. Then rinse with warm water and bathe the

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dog as you routinely would. You will be amazed at how well this removes most of the odor. Only a faint odor may remain, especially when the dog is wet. We need to wait for the epithelium to flake off for all of it to be completely gone. That takes time. By the way, I learned that the recipe also works on inanimate objects and surfaces too. Yeah, Ellie got sprayed in the mouth once and then proceeded to spit it all over my dining room carpet…at about 2:00 a.m. Ugh. But that was still better than an experience a friend of mine had. Not wearing his glasses one morning, he mistakenly let a skunk into his house, thinking it was his cat. When animal control arrived to remove the skunk from his home, the skunk proceeded to spray all over his kitchen. He used lots of the recipe that day!

Thank goodness dog and cat anal glands don't smell that bad. Still, they aren't nice. These glands (sacs) (shown in Fig. 8.4) are located at roughly 4 o'clock and 8 o'clock near the anal opening. Every time the animal has a bowel movement, the sacs are evacuated. This places their sebaceous scent markers with the feces. It's a "calling card" of sorts. There are also a bunch of tiny ***perianal*** [*peri-* around + *an(o)-* anus + *-al* pertaining to] ***glands*** on the surface of the anus that also contribute to the animal's scent. The scented "calling card" lets everyone else know he or she was there. It helps mark one's territory, stating clearly: "Cross this line at your own risk." And the scent from the anal sacs and the perianal glands is unique to the individual. This is why dogs typically greet each other by sniffing the other dog's anus. The *pheromones* provide recognition of the individual. "Is it you?" (sniff, sniff) "Oh, it is you! How ya doin?!"

I should note that problems can develop with perianal and anal glands. Most often, these problems develop in dogs. ***Perianal adenocarcinoma*** [*aden(o)-* gland + *carcinoma* an epithelial tumor of] is a form of cancer that arises from one or more of the perianal glands. These tumors need to be dealt with before the cancer invades deeper tissues. While I've seen a number of dogs with *perianal adenocarcinoma* in my career, fortunately it is not seen as frequently as other diseases. Anal sac inflammation and impaction occur with much greater frequency.

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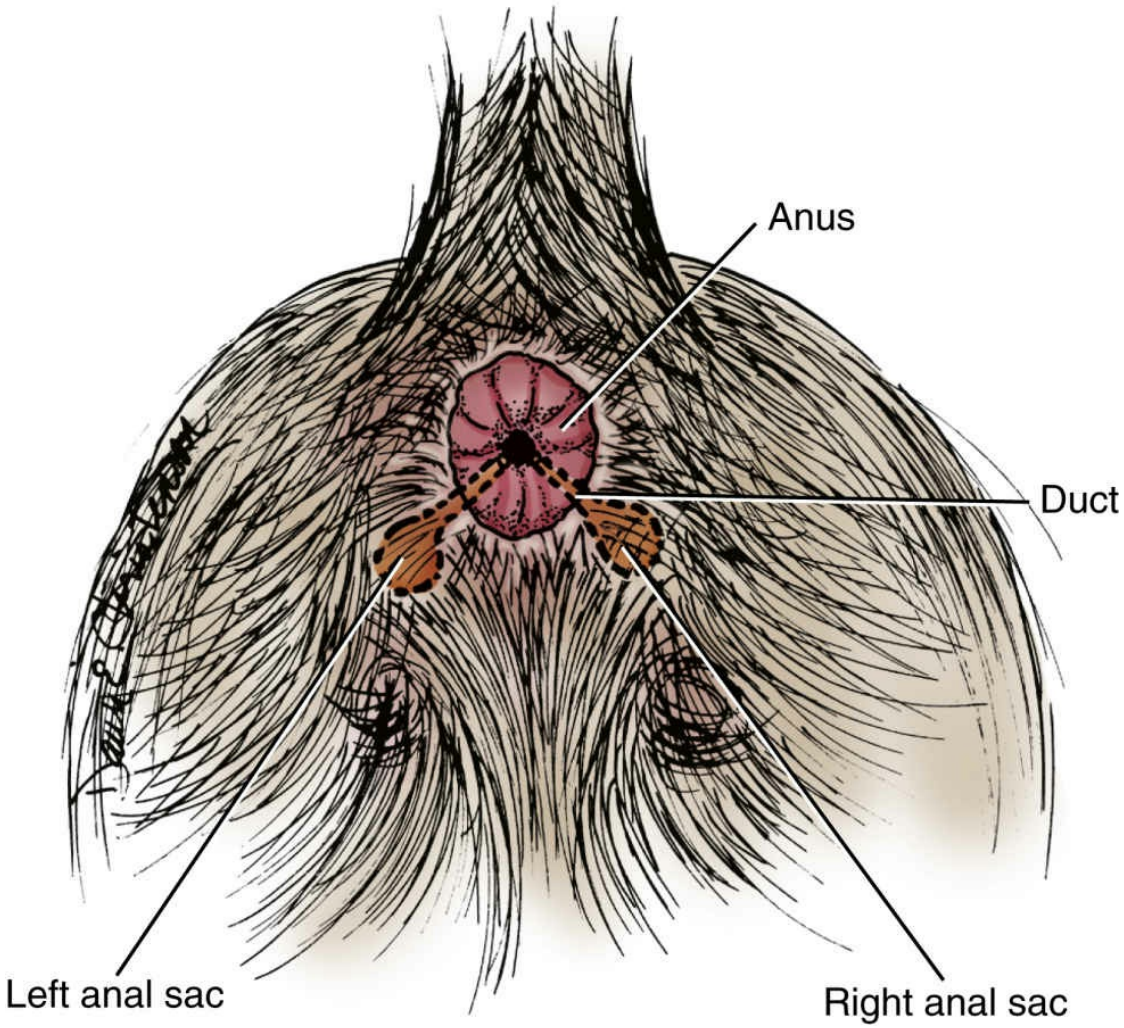

FIG. 8.4 Schematic of canine anal sacs.

Any dog with an underlying problem of ***seborrhea*** [*seb(o)-* sebum + *rrhea* flow of] is likely to develop anal sac issues. *Seborrhea* is often a symptom of ***chronic*** [*chron(o)-* time + *-ic* pertaining to; i.e., long-term] ***dermatitis*** [*dermat(o)-* skin + *-itis* inflammation of]. And with *seborrhea,* the character of sebaceous secretions changes. Superficial squamous epithelium *exfoliates* at an increased rate, increasing the thickness of the *sebum*. In terms of the anal sacs, this means that when the dog has a bowel movement, the anal sacs won't be emptied properly because their contents are simply too thick. The anal sac secretions can become thick like toothpaste (sometimes dried toothpaste). Of course, this leads to a catch-22, where the anal sacs become overly full, creating more inflammation, and then responding by producing thicker sebum. These dogs often scoot their bottoms on the floor in a futile attempt to empty their anal sacs to relieve the discomfort. Generally, all they really accomplish

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is leaving skid marks on the carpet. When the anal sacs become more inflamed, these dogs will often suddenly whip around as though something bit them in the butt. They may yelp and even avoid sitting. We need to intervene and empty those anal sacs before they ***abscess*** (become infected and filled with pus). This is best accomplished with sedation, adequate restraint, and perhaps a little pain control. Warning: most dogs object to this. The internal method is most effective, where we insert our gloved index finger into the anus. Then reaching beyond the anal sac with our thumb on the outside and index finger on the inside of the dog, we can gently and carefully "milk" the anal sac contents into something absorbent (like tissue or gauze). Some veterinarians will flush the anal sacs and infuse them with medication, depending on their condition.

Interdigital Glands

Perianal and anal glands are not the only important sebaceous glands. Most cloven-hoofed animals, such as goats and sheep, have ***interdigital*** [*inter-* between + *digit* toe + *-al* pertaining to] ***glands***. Yep, they are located strategically between the toes of each foot. Why on earth do they have them? Again, it's all about *pheromones*. Everywhere these animals walk, the interdigital glands leave a scent-trail of pheromones. It's better than bread crumbs to guide the herd to and fro.

Lanolin Glands

By the way, while we're thinking about sheep, I should mention their ***lanolin glands***. Yes, lanolin is a sebaceous secretion of sheep. That's why you'll probably never find a sheep farmer with dry, cracked hands. Sheep actually have a number of lanolin glands. Two of the more predominant glands are located bilaterally in the inguinal (groin) region. When you set a sheep up on its bum, you can easily locate these two glands. They're where you see the thick, brownish, waxy material caked in the inguinal (groin) area.

Circumoral Glands

If you look at Fig. 8.12, at the cat's face, you can't see them but there

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are ***circumoral*** [*circum-* around + *or(o)-* mouth + *-al* pertaining to] ***glands*** on that face. Even big cats like lions and tigers have them. *Circumoral glands* are concentrated on the chin and upper lips of cats. Like other sebaceous glands, the secretions of circumoral glands contain pheromones. Have you ever had a cat rub its chin and face on you? Not to break your bubble, but it wasn't being affectionate. It was marking you. By rubbing its circumoral secretions on you, you become marked with the cat's distinctive scent. And if you know anything at all about cats, you know that they are independent, possessive divas who train us quite well to serve their royal needs. I guess I'd rather have my cat rub her chin on me to claim me as her own, than express her anal sacs on me. Everything is relative.

Cornual Glands

Have you ever watched goats, especially bucks? They tend to head-butt and rub their heads next to their horns on things. Yes, it is in part a display of dominance. It is also a way of …wait for it … marking territory. I know, I sound like a broken record, don't I? You see, it's not just about the horns. It's also about the ***cornual*** [*cornu(o)-* horn + *-al* pertaining to] ***glands***. These are just another example of a specialized, species-specific sebaceous gland. And like all of the others we've discussed thus far, *cornual gland* secretions contain pheromones. So, when you see a buck rubbing his horned head on that fence post or trough, he's marked it and claimed it, just like the cat claimed you.

Horns and Antlers

Speaking of horns, have you ever wondered what the difference between horns and antlers is? There are actually very big structural and functional differences. You see ***antlers***, for animals like whitetail deer, are temporary. They grow from specific areas of the skin on the head. Antlers are basically modified epidermal tissue—highly keratinized, thick stuff that eventually becomes calcified. For animals like deer, the antlers grow rapidly in the fall at the beginning of their reproductive season. This gives all of the bucks weapons to battle other bucks for breeding rights with the does.

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After the breeding season, the growth area that's been supporting each antler recedes and becomes dormant. Then the antlers fall off. New ones will grow back the next fall. In some members of the deer family like caribou and reindeer, both males and females grow antlers.

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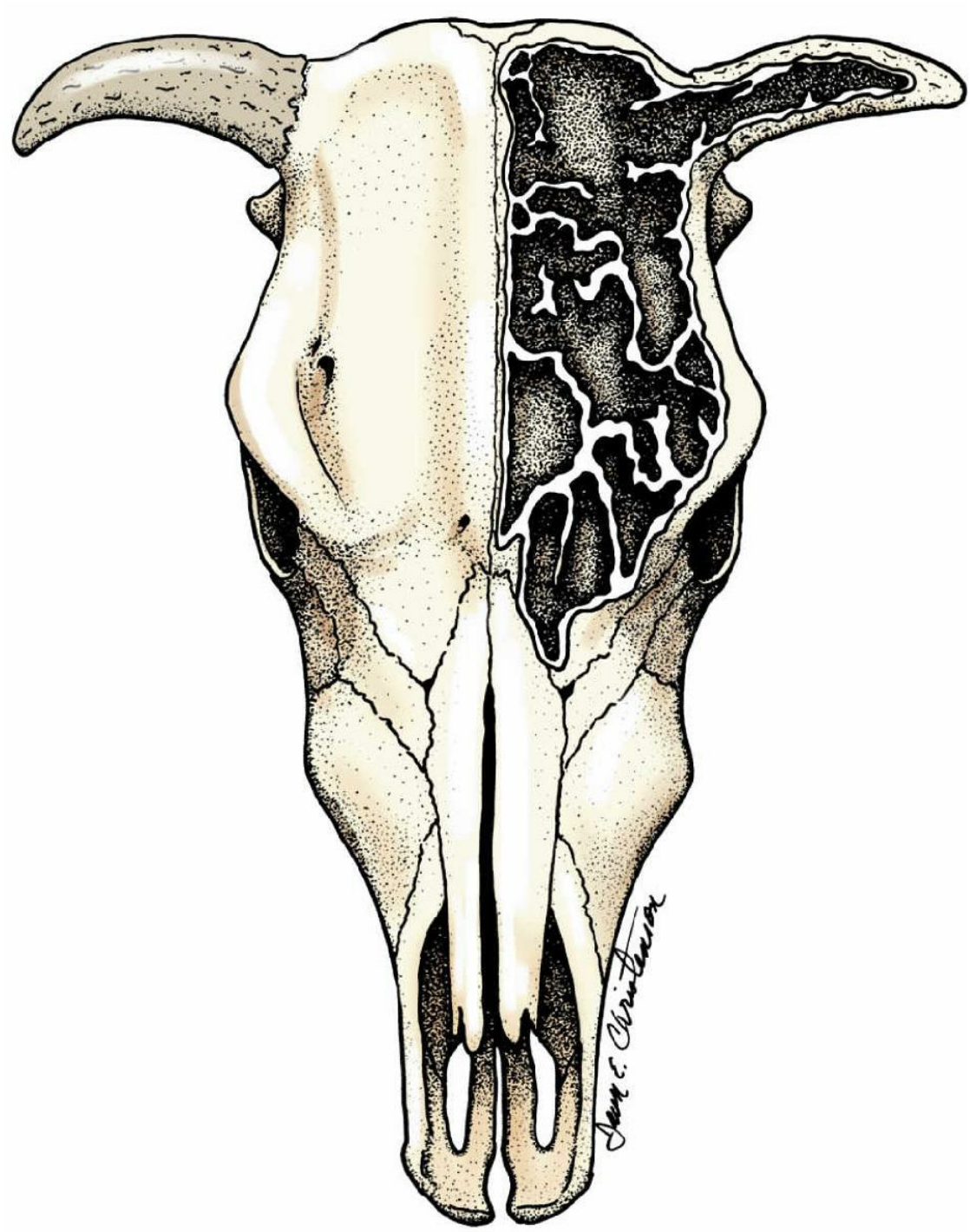

FIG. 8.5 Bovine horn and frontal sinus.

Horns are different. Horns are permanent, just like hooves and nails. In the mature animal, the keratinized horns are actually supported by the bone of the skull. As you can see in Fig. 8.5, this bovine skull shows not only how the bone of the skull grew out with the horn (keratinized tissue not shown), but also how the center of the horn actually communicates openly with the frontal sinus. Thinking of just domestic animals, this holds true for cattle,

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sheep, and goats. But they don't start out that way. Horns begin from a horn bud in the skin. As the horn grows from this bud, supportive bone of the skull begins to grow with it. Eventually, the animal's mature horn communicates openly with the sinus. Why point this out? It's very important, if we need to perform a ***cornuectomy*** [*cornu(o)-* horn + *-ectomy* to cut out, remove; i.e., dehorn]. Obviously, the least traumatic *cornuectomy* will be performed on a calf, kid, or lamb, before the horn bud begins to grow. (This is more accurately termed *disbudding*, rather than *dehorning*.) We can either surgically remove the bud (time-consuming and very bloody), or we can use an electric dehorner to burn the skin immediately around the horn bud. The burn (full thickness epidermis and dermis) destroys the growth area for the horn. The skin will heal. In older and mature animals, the *cornuectomy* will be much more difficult and traumatic. We can make the procedure itself painless by doing a *cornual block* (i.e., injecting a local anesthetic agent around the horn, or horn bud for disbuddin). This also helps prevent head-shyness. Unfortunately, it leaves the mature animal with an exposed frontal sinus. And this can lead to a host of other secondary problems like maggot infestations during fly season. Can you imagine the sinus headache an animal would get in the middle of winter? As they say, timing is everything.

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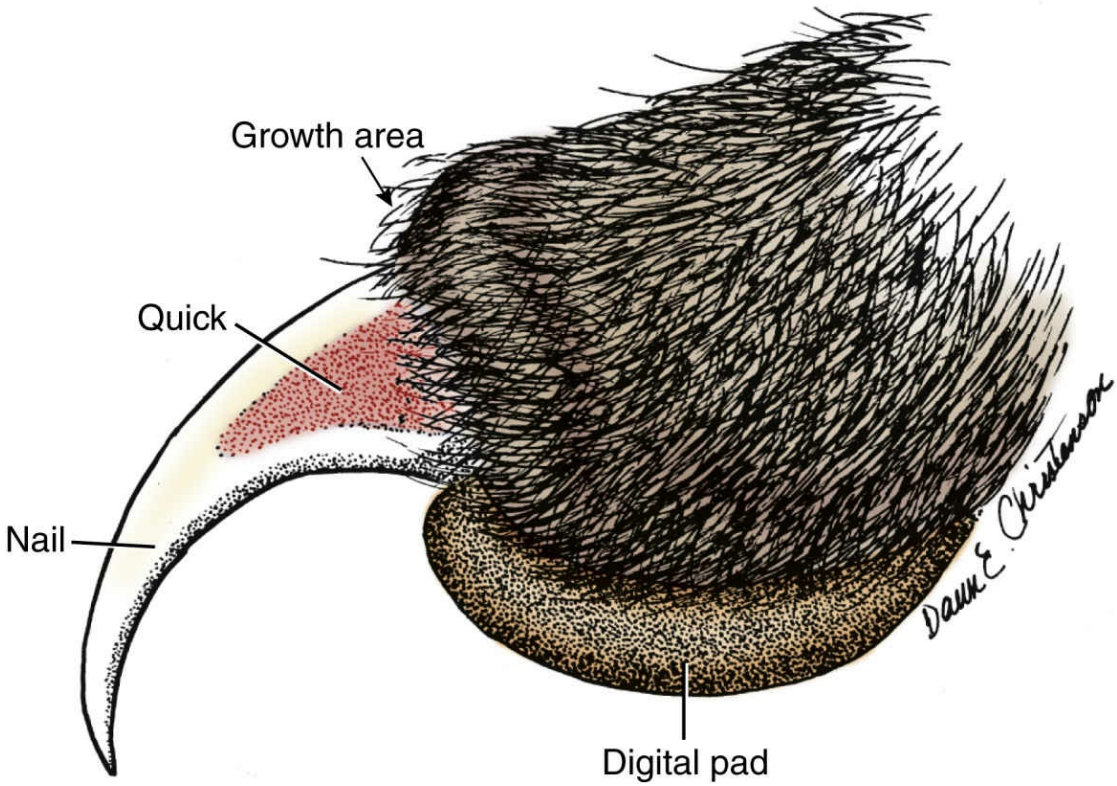

FIG. 8.6 Feline claw.

Nails

The hard nails (claws) of dogs, cats, and others are made of modified keratinized epidermal tissue (highly compact stratum corneum). Nails grow out from the base of the nail near the skin, much like the cuticle of our nails. Now, if you've ever trimmed nails and cut one a little too short, you know that they are not made exclusively of avascular epidermis. Nails bleed if we trim them too short. Bleeding comes from the "***quick***" (Fig. 8.6), which is merely dermal tissue. The quick is basically the dermal support and the attachment between the hardened nail and the bone of the toe. And as you already know, the dermis is very vascular tissue. Of course, the quick is easy to avoid when trimming white nails. Pigmented nails are another story. It's best to take your time with those, slowly trimming them back incrementally. When the center begins to look softer and translucent, stop because the quick is near. How often should nails be trimmed? That depends on the individual. Once a month is probably a ballpark average. The key is to keep the nails short enough so that the dog or cat walks on its pads. I've seen nails

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so overgrown that toes were horribly twisted, as well as some where the tips actually grew into the pads. Imagine how painful that must have been.

By the way, it's always good to see if there are other toes and nails, other than the ones the dog or cat walks on. There may be ***dewclaws*** (first digit) that need to be trimmed on the medial aspect of the limb, near the foot. Again, if we miss these, they may curl around and grow into the side of the foot. (Refer to Chapter 4 to review the location of the first digit.) And some ***polydactyl*** [*poly-* many + *dactyl(o)-* toes] animals will have not just five nails to trim, but six or more. I've seen *polydactyl* cats with seven or eight toes on a single paw! Time out for a fun story—did you know that polydactyl cats are frequently called "Hemingway" cats? The writer, Ernest Hemingway, received a white polydactyl cat named Snowball from a ship's captain. (Keeping cats on ships was quite common to reduce the mouse population aboard ship.) Well, Mr. Hemingway fell in love with that polydactyl feline. Many polydactyl cats today are believed to be descendants of Snowball. And that is why you may hear them called Hemingway cats. Okay, let's get back to business. I've always been very glad when the polydactyl cats I've cared for were easygoing. Maine Coon cats tend to be very easygoing. They experience ***polydactyly*** [the "y" like *-ia* indicates "condition" or "state of"] more than most other cat breeds. That's a very good thing. It gives them built in snowshoes if they go outside in the winter. Yet, I can't imagine having a huge cat like that, with *polydactyly*, put all of those weapons to use! Yikes!

Onychectomy

The common name for an ***onychectomy*** [*onych(o)-* nail + *-ectomy* to cut out, remove] is "***declaw.***" But it's not that simple. Look at your own hand for a moment. Each finger has three bones, right? The distal bone (phalanx) is found at the tip of each finger and supports the nail, right? Okay, picture an *onychectomy* on your own hand. The surgeon won't simply be removing your nail—painful enough. The surgeon will be amputating each finger at the distal joint. THAT is an *onychectomy*. If done correctly, the digital pad will remain completely intact to cushion the end of the second phalanx. Unfortunately, many times a portion of the sensitive pad is

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removed too. The term "declaw" is very misleading indeed. And think about it: you don't walk on your hands. So, as painful as an *onychectomy* would be, at least you could rest and keep your hands elevated to minimize the throbbing. Cats don't have that luxury. They have to get up and walk to go to the litter box, to the water bowl to drink, and to the food bowl to eat. Frankly, it doesn't matter that most often it's only a front declaw that is performed. At rest, 60% of the cat's body weight is on the front paws. In motion, that force is even higher. Suffice it to say that recovery from an onychectomy is very painful.

There are other consequences to onychectomies. Behaviorally, these cats have lost a very important defensive tool. In my experience, declawed cats are more likely to quickly try to bite. That's important for us as veterinary professionals, because we tend to unintentionally frighten and threaten them when they're in our care. They will defend themselves. You see, cats with their claws tend to strike with their claws first and bite as a last resort. Declawed cats are left with no other options to protect themselves. I kid you not, I would much rather be bitten by a dog than a cat any day. More importantly, we need to think about how defenseless declawed cats actually are, especially if all four feet are declawed. If such a cat somehow slips outside (and it happens), how will it protect itself from another cat or an aggressive dog? It has no claws to climb a tree to escape the threat. And it has no claws to physically fight off the aggressor. The defenseless cat may be seriously harmed or even killed.

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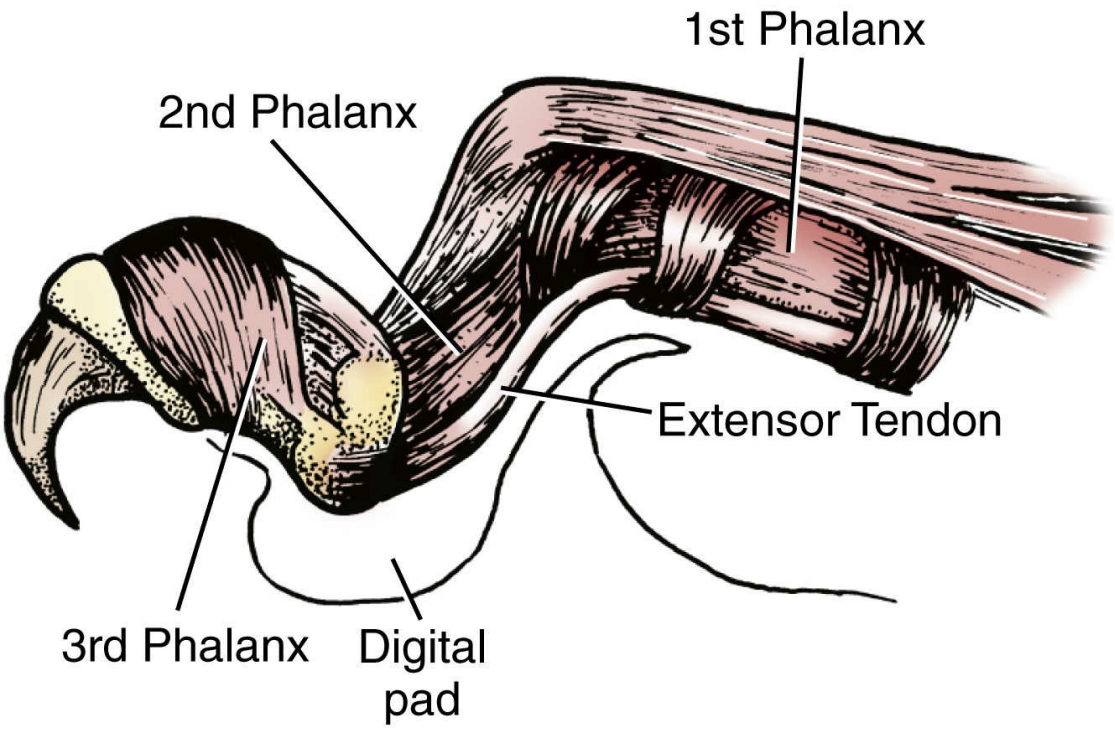

FIG. 8.7 Feline claw in extension (skin removed).

If it's so painful and traumatic, why do owners get their cats declawed? Usually, it's to prevent destructive behaviors, namely scratching. Scratching is a very natural, though undesirable, behavior for cats. It can become quite costly to have a cat destroy furniture, draperies, and other things in one's home. So, many owners opt for surgery. Now, in fairness to the owners, because we have so minimized the procedure by calling it a "declaw," they may not really know or understand the magnitude of the trauma to the cat. Perhaps no one has taken the time to really explain it. And perhaps no one has taken the time to explain alternatives to prevent destructive scratching behaviors. What alternatives are there? Well, there are ways to redirect where cats scratch. Training a kitten or cat to a scratching post can be very effective. Keeping the nails regularly trimmed is also very important. There are also soft synthetic nail covers that can be glued over the trimmed nails. (They do need to be replaced on a regular basis.) Keeping the cat active with appropriate play may also minimize destructive behaviors. There is also a less traumatic surgical procedure—a ***tenotomy*** [*ten(o)-* tendon + *-tomy* to cut]. By cutting the extensor tendon (Fig. 8.7), the cat (in theory) cannot extend its claws. Admittedly, there are some cats who, after a *tenotomy*, learn to exert

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just the right amount of pressure on the digital pads to extend their claws. So a determined cat may still be able to engage in destructive scratching behavior. Frankly, there are times when an owner is backed into a corner. They may have tried everything to no avail. Such owners may find themselves having to make a difficult choice between either an *onychectomy* or euthanasia. As veterinary professionals, we need to educate owners to help them make the optimal informed decisions for them and their cats. We should be quick to offer options and alternative solutions and slow to judge.

Hooves

If you need to, please refer to Chapter 4 to review foot anatomy of large animals (horses, cattle, sheep, goats, etc.). Now, think about how their distal extremities differ from dogs, cats, and us. They don't have four or five toes on each foot to distribute the body weight placed on each foot. In the case of ruminants, they focus their weight on two toes. Horses are the extreme. They focus their weight on one toe. With that in mind, they need foot construction that's up to the task. It needs to be tough and durable, not easily injured as they walk or run around on pasture or in the barn.

If you look at the horse's foot in Fig. 8.8, you'll find that kind of durable construction. The ***hoof wall*** is made of the same type of keratinized stratum corneum as the horns and nails we've already discussed. The growth area for the hoof wall is found in the region of the ***coronary*** [*coron(o)-* crown + *-ary* pertaining to] ***band***. If you think of how a crown, as the word infers, encircles the head of the wearer, you have a pretty good visual for how the *coronary band* encircles the foot. Turning the foot over to look at the palmar or plantar surface (Fig. 8.9A), you can see that the hoof wall is quite thick. It needs to be. Because that is really what's supporting the weight of the animal. The same is true for the bovine foot shown in Fig. 8.9B. These animals (sheep and goats too) bear weight predominantly on their hoof walls and heels, not the ***sole*** as you might think. The sole is actually rather concave. While the sole may contact uneven ground, it is not the principal point of contact or support for the foot. The sole is also made of thick, tough epidermal tissue, but it's not as hard as the hoof wall. It's a little flexible,

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enough so that underlying tissue can be bruised when stepping on hard objects.

We'll need to turn our attention to Fig. 8.10 to understand the rest of the structure of the foot. This is a horse's foot, but ruminants' toes are quite similar. First notice that the epidermal tissue of the hoof wall and sole are quite thick. Next, notice that there is a layer of dermal tissue right alongside the hoof wall and sole. But the intersection between these structures is just a little different from that of regular skin. In the skin, remember, there are dermal papillae that provide kind of a Velcro connection between the dermis and epidermis. But that won't work with the hoof wall, because it's chronically growing from the coronary band to the toe. The hoof wall needs to be secure, but it also must almost glide over the dermis as it grows. So the two tissues are laminated with interlocking "hills and valleys" of dermis (***sensitive lamina***) and epidermis (***insensitive lamina***) that run from near the coronet (or coronary band) to the distal margin of the hoof wall. Similar laminations are found between the dermis and sole. But the laminations between the hoof wall and dermis are most pronounced, providing a very strong bond. Right near that intersection is something called the ***white line*** (named because it appears white). If you ever trim hooves and begin to see the white line, stop. You're getting dangerously close to the dermal tissue in that hoof. Of course, on the opposite side of that dermal tissue is bone—the third phalanx. Notice how the dorsal surface of the third phalanx is parallel with the hoof wall. This is very important, along with the insertion of the deep digital flexor tendon on the 3rd phalanx. Remember this, when we talk about laminitis. You'll notice that the rest of the foot, especially near the heel, has a bunch of cushiony tissue. This is modified subcutaneous tissue that provides support and shock absorption.

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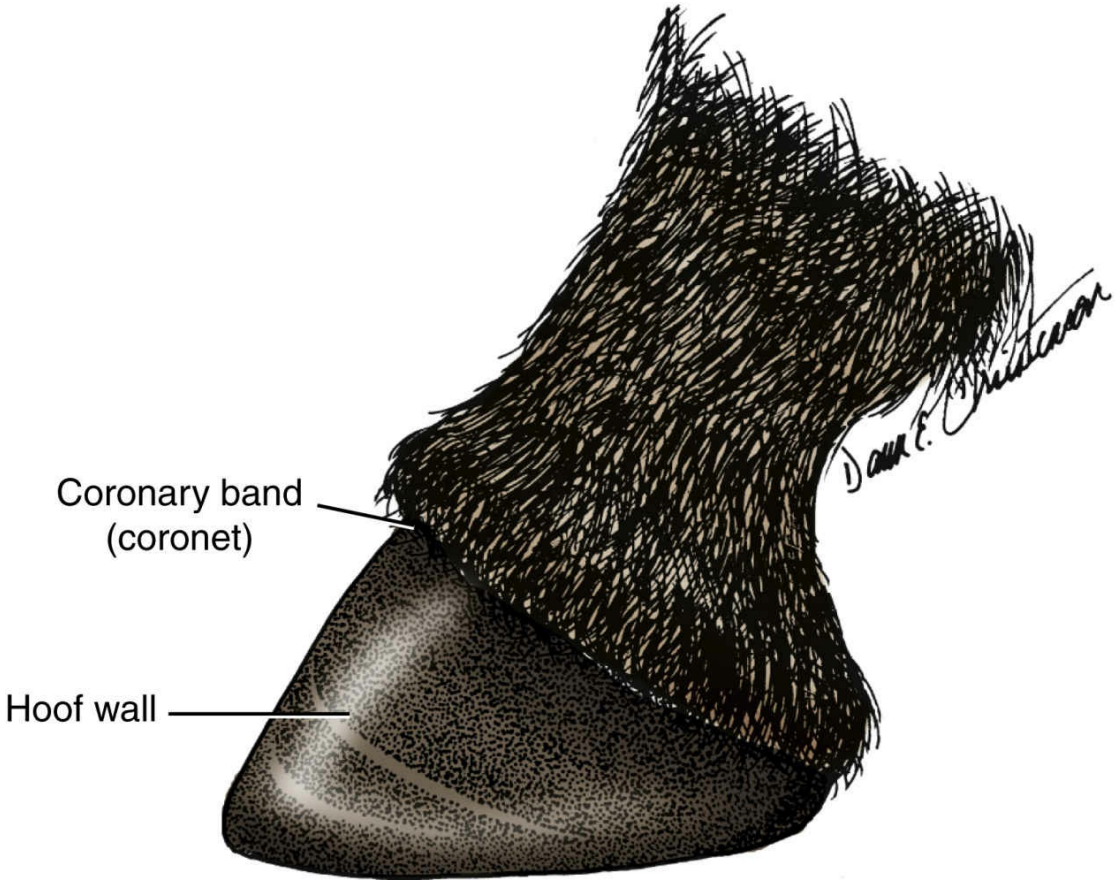

FIG. 8.8 Equine hoof, lateral view.

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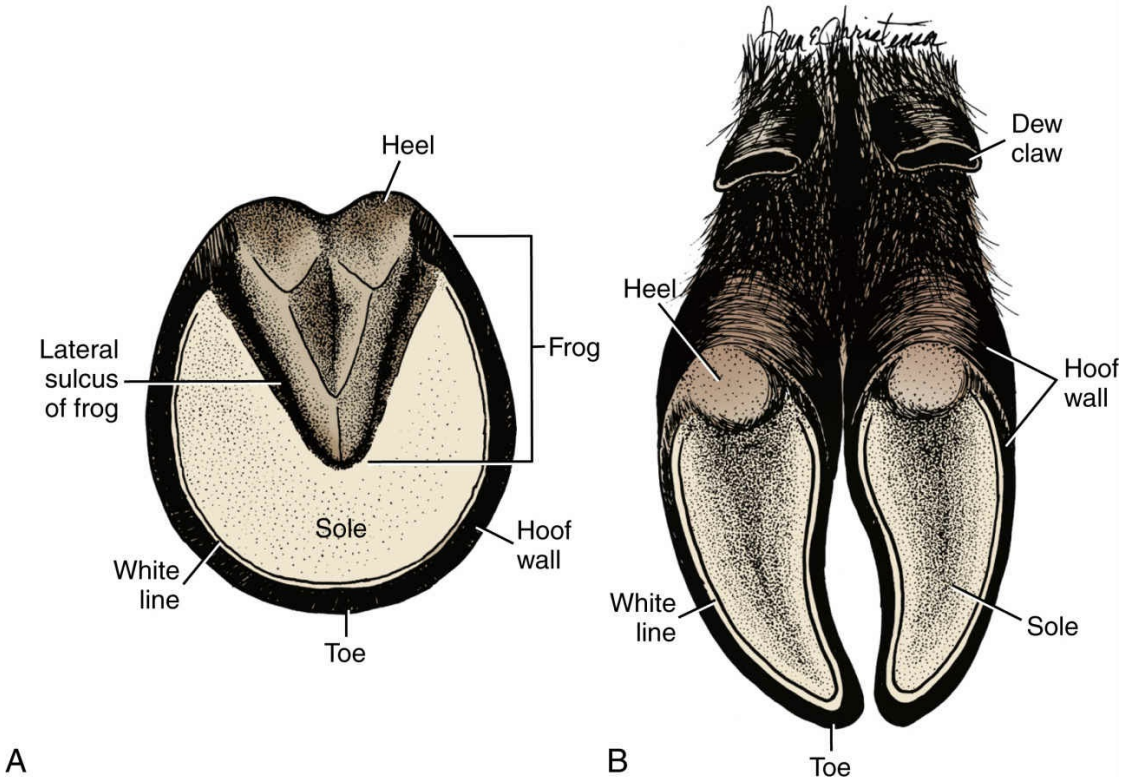

FIG. 8.9 (A) Equine foot, palmar/plantar view; (B) bovine foot, palmar/plantar view.

Laminitis

Laminitis [*lamin(o)-* lamina + *-itis* inflammation of] or ***founder,*** as many horse owners like to call it, is inflammation of the *sensitive lamina*. That's right—it's the dermal tissue that's inflamed. Now, any animals with hooves can develop *laminitis*. But horses develop it more frequently than any other animal, so that's where we will focus our attention. Just remember, anyone with hooves can develop laminitis.

For a moment, let's review the location and structure of dermal tissue in the hoof. Remember, this is highly vascular tissue, loaded with capillaries. The capillary blood flow sustains not only the dermal tissue itself, but the epidermal tissue as well (just like the skin). It is also important to remember that this vascular tissue layer covers the entire interior surfaces of the hoof wall and the sole. Why is this important? Well, think about our discussions of inflammation and ***edema*** [swelling] formation in prior chapters, such as Chapters 3 and 5. In the presence of inflammation, capillaries become more permeable (leaky), allowing fluid to pour

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into the ***interstitial*** [*inter-* between + *stiti(o)-* tissue + *-al* pertaining to] space. This results in excess interstitial fluid—edema. While uncomfortable, edema in the skin or other soft tissues can be tolerated. These tissues have room to swell. But now, put this in the context of the hoof. It is a confined space. The hoof wall is rigid and cannot expand when the dermal tissue becomes ***edematous*** [*edema* swelling + *-tous* pertaining to]. If the tissues become more and more *edematous* in that confined space, they will become strangulated. It would be no different than placing a rubber band around your finger. And if blood supply is cut off, tissues die.

Let's consider another consequence of inflammation. Inflamed tissues tend to become more fragile in the presence of inflammation. Forces, that under normal circumstance would create no problems whatsoever, may result in breakdown of those same tissues in the presence of inflammation. The breakdown will begin at the weakest link. Again, let's think about the intersection of the epidermis and dermis of the skin. Have you ever had a minor burn that blistered? Have you ever raked the lawn with poorly fitting gloves and developed blisters from the friction? In both cases, inflammation developed. And the epidermal-dermal interface was weakened by the inflammation created by the traumatic events (i.e., burn and friction). The edematous fluids that developed in response to the inflammation ultimately created separation at the weakest link (the connection between the epidermis and dermis), resulting in the blister formation.

Let's apply this same effect in the context of laminitis. Inflammation will weaken the connection between the sensitive and insensitive laminae. But because of the deep laminations, they will be able to hold together for a while. Where's the weakest link? Looking at Fig. 8.10 again, there are no deep laminations over the surface of the 3rd phalanx. There is a very weak link between the dermal tissue and the ***periosteum*** [*peri-* around + *oste(o)-* bone + *-um* the] of that bone. And we have tremendous tension pulling on the caudal aspect of that bone, by the deep digital flexor tendon. This adds to the potential for breakdown. Also, the sole is somewhat flexible to permit movement of the bone. Let's add this up: (1) inflammation making tissues fragile + (2) weak connections of dermis to dorsal surface of 3rd phalanx + (3) strong caudal tension

on the 3rd phalanx by the deep digital flexor tendon + flexibility of the sole = rotation of the 3rd phalanx. Because of the distal ***interphalangeal*** [*inter-* between + *phalang(o)-* phalanx + *-al* pertaining to] ***joint***, the bone pivots like a hinge. So, the toe of the bone pivots away from the hoof wall. The rotation of that bone is progressive. If we catch laminitis early, our goal is to prevent rotation. If rotation has already begun, our goal is to minimize or prevent further rotation. Why bother? Well, if the bone fully rotates, the point of its toe will press on and may eventually perforate the sole. By the way, the intersection between the sensitive and insensitive laminae eventually can completely break down too. I've seen horses with such severe laminitis that they literally walked out of their hooves.

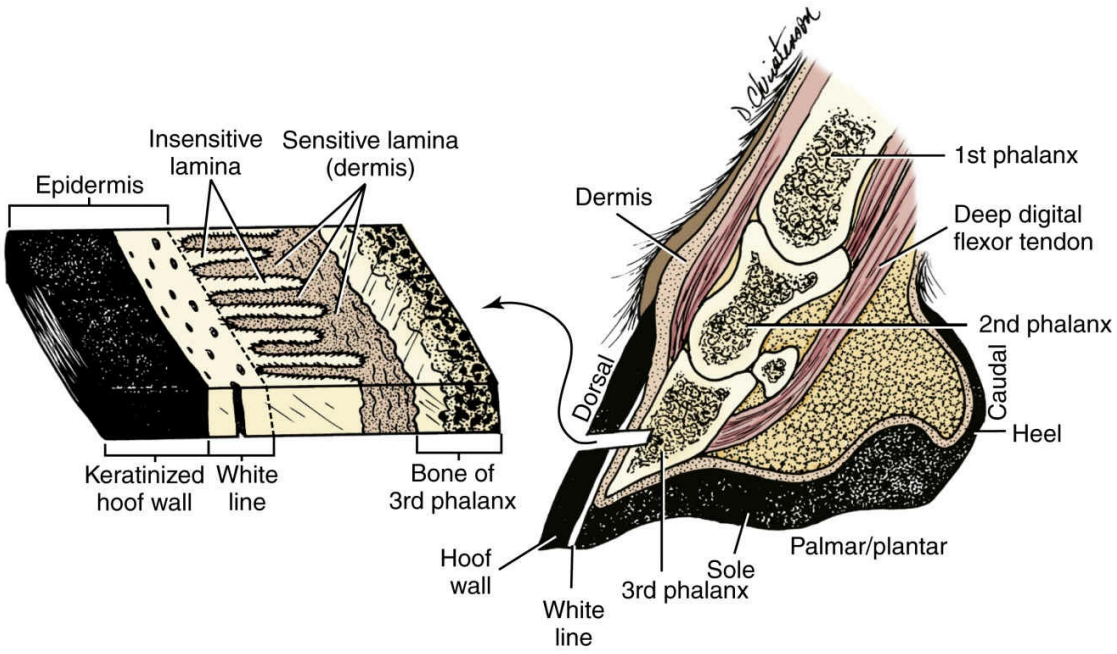

FIG. 8.10 Schematic of equine foot.

So, that's what happens in laminitis. But why does it happen in the first place? That is the million-dollar question. So many factors could precipitate laminitis in a horse. The list is seemingly endless. It could be a digestive disturbance like diarrhea. It could be an abrupt dietary change. It could be colic. Pain could precipitate it. Trauma to the foot like stepping on a stone and bruising the sole may cause it. Many, many, many things could precipitate laminitis.

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And once an animal has had laminitis, it is more likely to develop it again. Whatever the cause, the end result is altered blood flow in the foot and resultant profound inflammation. From there, the inflammation and vascular changes become a catch-22. Obviously, systemic factors could affect all four feet. A traumatic event may remain isolated in the affected foot. Most generally, laminitis occurs in the front feet, even when it stems from systemic causes. This is probably because there is always added stress placed on the front feet. Remember, at rest, roughly 60% of the body weight is on the front feet. In motion, that percentage goes up.

It's important to educate horse owners about laminitis. Some things they can prevent like abrupt dietary changes. Unfortunately, not all cases can be prevented. So, it is very important that we and horse owners recognize and act on early signs that may indicate the development of laminitis. Signs of painful feet may be subtle like slight changes in the horse's behavior—slightly diminished responsiveness or reluctance to do things that it generally loves to do. Perhaps the horse acts out its pain with vocalization or aggression that's out of character. Subtle shifting of weight may indicate discomfort—shifting repeatedly from one foot to another, unable to stand still, or leaning to shift weight off the affected feet. Some horses will sweat profusely when in pain, in spite of inactivity and cooler ambient temperatures. Lameness and limping could indicate pain anywhere along a limb. It is probably wise to investigate the feet first. Whatever subtle signs the horse is showing, the safe play is to promptly investigate it. Catching laminitis early, we may be able to prevent or minimize rotation of the 3rd phalanx. If left unchecked, severe laminitis and all of the consequences that it brings leaves us with one humane option—euthanasia. That's not a treatment plan that any of us want to face.

Paw Pads and Noses

We've all seen the paw pads of dogs and cats. Each paw has multiple pads. Each toe has a ***digital pad*** (including the dewclaws). Then there is a larger, principal weight-bearing pad named for its location (i.e., ***metacarpal*** [*meta-* after, beyond + *carp(o)-* wrist, carpus + *-al* pertaining to] ***pad*** or ***metatarsal*** [*meta-* after, beyond + *tars(o)-*

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ankle, tarsus + *-al* pertaining to] ***pad***). On the caudal aspect of the carpus is the ***accessory carpal pad*** (associated with the accessory carpal bone). Refer to Chapter 4 to review foot anatomy.

Structurally, these pads are designed for weight-bearing, so they need to be adequately padded. Their thickened structure (especially epidermis) and abundant, dense subcutaneous tissue provide excellent padding and shock absorption to protect the bones of the foot. And because these creatures walk around in their bare feet, the surface of the pads needs to be extremely thick and tough to resist wear and tear. The stratum corneum is extremely thick here, as well as papillated (for traction). The paw pads are pretty much the only place on dogs and cats that they have *sweat glands*. There aren't that many—certainly not enough to cool them off, as we discussed earlier. But they may provide just enough moisture to give a little more traction on slippery linoleum.

The ***noses*** of dogs, cats, cattle, and pigs may look quite different at first glance. But if you get up close and personal, you'll see that the tissue there is very similar. You'll notice if you look closely that the surfaces of those noses have individual interlocking plates or plaques. This is just a little different arrangement of the epidermal tissue. Compared to normal skin, the epidermis is much thicker on the nose (though not as thick as on the paw pads). The stratum corneum of the plaques is particularly thick. Why would it need to be so thick? Well, these animals are always sticking their noses into things. This is especially true for pigs, who always root around in dirt and mud with their noses. You never know what delectable treats a pig might find in the dirt (e.g., grubs, earthworms, etc. – yum!).

Hair and Fur

If there's one thing most domestic animals have, it's fur. Hair coats vary tremendously from species to species and breed to breed. Why, even parasites recognize this! That's right—***lice*** tend to be very host-specific. And a major factor playing into that host-specificity is hair type. Their claws are sized and designed to grasp a particular type and size of hair shaft. That's why a louse like *Haematopinus suis* (he-mat'o-pi"nus soo'is; the exclusive louse of

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pigs) cannot survive on another type of animal. It can't hang on! No one else has hairs as coarse and large in diameter as a pig.

Oh, my goodness, the variety of hair coats animals may have! Sure, there are color differences. But I'm talking about quality or character differences. Some are short and prickly like the Shar-Pei. Some are silky, soft, and wavy like the Rex cat. Some are curly like the Poodle and Rambouillet sheep. Others are really thick like the Samoyed and Malamute. Some have very little hair like the Chinese Crested. Some have extraordinary long thick "feathers" (i.e., long thick hair) around the feet like the Clydesdale. Each species and each breed within a given species is unique. But what's the scientific basis for all of these differences? Well, there are different types of hairs and different types of hair follicles. Follicles may be simple or compound. If it's a ***simple follicle***, it has only one hair growing from it like the one shown in Fig. 8.1. Single-coated animals, such as pigs, have the types of follicles that support one type of hair. A ***compound follicle*** (Fig. 8.11) really is a cluster of multiple follicles. All of the hairs from these follicles pass through a single ***follicular*** [*follicul(o)-* follicle + *-ar* pertaining to] opening in the skin. This is the follicular arrangement for double-coated animals, such as Corgis, German Shepherds, and Huskies. This leads us to a discussion of hair types.

Guard Hair

In Fig. 8.11, you'll notice that there is one large, long ***guard hair***, also called a ***primary hair***. Guard hairs tend to be relatively long and stiff. The stiffness is due to the hair's structure. As you can see, there is an outer ***cuticle*** that has overlapping sheets like roofing shingles. Then there is the ***cortex*** [L. "bark, rind, shell"]. At the center is the stiffened material of the ***medulla*** [L. "inward part"]. It's the medulla that makes guard hairs rather rigid.

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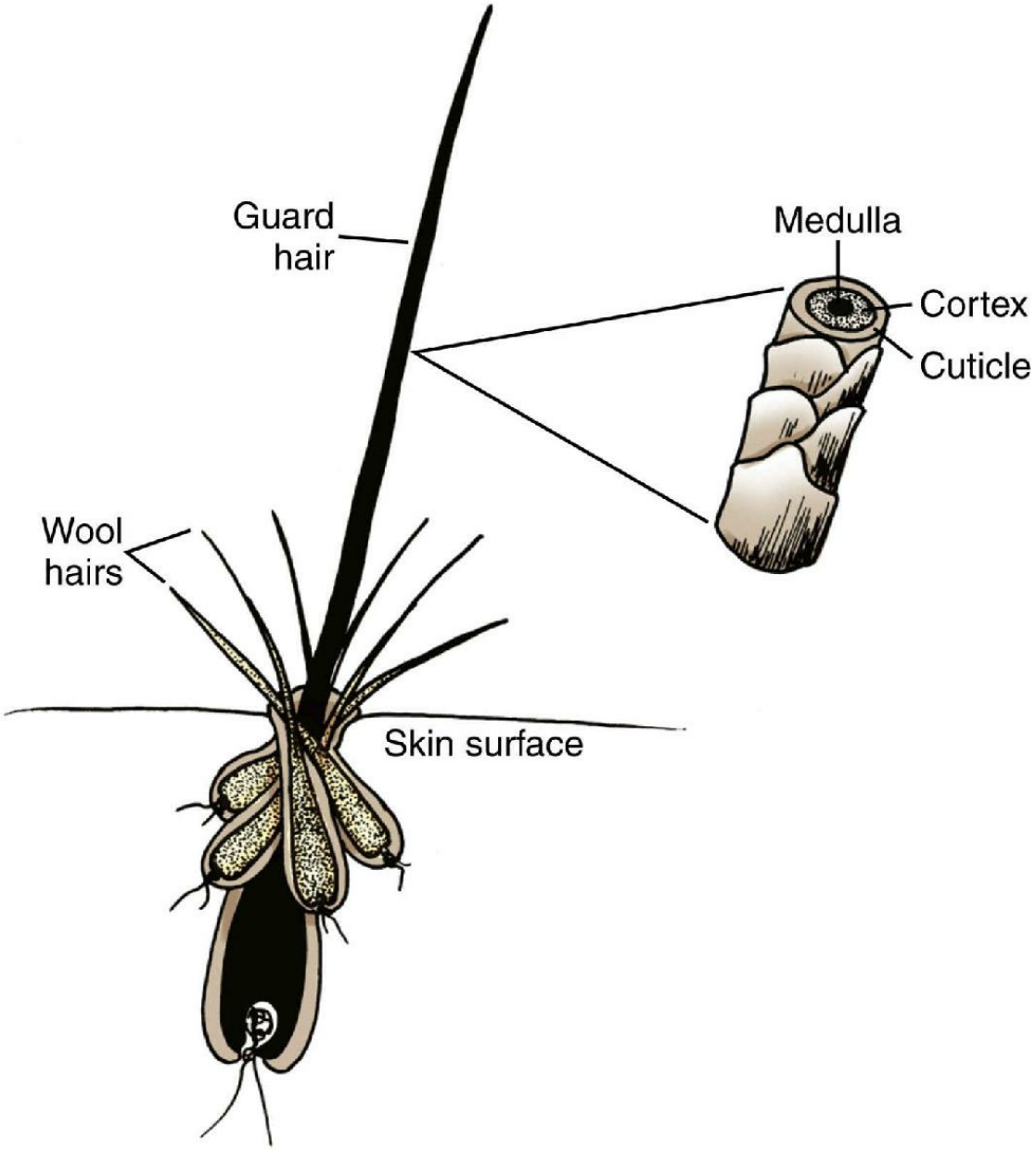

FIG. 8.11 Compound follicle.

Wool Hair

Wool hairs or secondary hairs in compound follicles tend to be shorter and softer than guard hairs. The softness is due to their different structure. Wool hairs do not have a *medulla*. Therefore they are thinner and do not have the rigidity of their guard hair counterparts. Wool hairs form the ***undercoat*** of double-coated breeds. This is the thick, fluffy stuff that comes out by the handfuls when these animals "blow their coats" for seasonal shedding. Obviously, the undercoat provides a marvelous dead–air space for

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insulation, especially with *piloerection*. This is why Arctic breeds, such as Malamutes, Huskies, and Samoyeds, all have thick undercoats. Of course, wool hairs are the only type of hair over the bodies of sheep. That's why it's called … wait for it … ***wool***. Other breeds like the Poodle and Rex cat are covered in wool hairs. Even horses and cattle in temperate regions produce a lush, thick undercoat of wool hairs to get them through the winter months.

Tactile Hairs

Tactile hairs are located strategically to assist the animal with *tactile sensation* (touch). ***Whiskers*** like those shown in Fig. 8.12 are good examples of tactile hairs. For dogs and cats, these whiskers help them navigate through their environment and even locate prey. Horses use whiskers on their muzzles and chins when foraging for food. If you look closely at the faces of many animals, you'll notice that they also have tactile hairs dorsal to the eyes. Now you might think that they just look like really long guard hairs. But tactile hairs are much, much stiffer. And the part that makes them so beneficial for tactile sensation is actually surrounding the hair follicle. You see, a basic hair follicle only has major vessels in the hair bulb, at the base of the root. But tactile hairs have a highly integrated network of capillaries and nerves surrounding the whole follicle. The slightest movement of the tactile hair (by a puff of air or by lightly brushing up against something) is magnified by the capillary network. It's the magnification that results in such tremendous sensory nerve stimulation. That's why they are so sensitive to tactile sensations. Compared to other hair types, tactile hairs have the least amount of turnover.

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FIG. 8.12 Tactile hairs.

Hair Growth

That's a good segue to discuss hair growth (and loss). Hair is grown and lost in phases. Fortunately, not all follicles are on the exact same schedule or we'd all experience periodic baldness! Wow, that's quite a visual, isn't it? The same is true for animals. Different types of hairs and various follicles are on different schedules. We already mentioned that tactile hairs tend to be in place the longest. Guard hairs are probably in second place for longevity. Wool hairs are shed the most. But even among wool hairs there are differences in rates of growth and shedding. Think about sheep and Poodles. They really don't shed their wooly coats at all. But the wool hairs of Arctic breeds are shed seasonally. The same is true for horses at the end of winter. Eventually, even guard and tactile hairs will be shed. But as we said a moment ago, they won't all be shed at once, or as frequently.

Regardless of the hair type, each will go through the cycle of hair growth, shown in Fig. 8.13. The hairs and follicles shown in this schematic are quite simplified. (Far more details like the epithelial lining of the follicle and associated sebaceous gland are shown in

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Fig. 8.1.) For our purposes here, we'll focus on the larger elements, including the ***hair shaft*** (above the skin), the ***hair root*** (below the surface of the skin), the *follicle*, and ***hair bulb***. The hair bulb is covered in specialized epithelium from which the hair grows. It also has a rich blood supply, especially during the growth and sustaining of each hair.

We find three distinct phases in the life cycle of hair growth and loss. Let's begin by talking about the ***anagen*** [*ana-* up, again + *gen(o)-* production] ***phase***. You'll notice that I have this phase shown as bookends for the whole cycle. There's a reason for that, because the *anagen* phase is the longest phase within this cycle. And for some hair types like tactile hairs, it is a very long phase. Looking at the far right of Fig. 8.13, we find the very beginning of the *anagen* phase. The hair bulb is invigorated with a rich new blood supply to support active growth of the new hair. Ah, now the name—*anagen*—makes sense. Growth is renewed [*ana-* up, again] production [*gen(o)*-] of the hair. That growth ultimately pushes what's left of the old hair out of the follicle. Now looking at the hair and follicle on the far left of Fig. 8.13, we find a hair during the bulk of the anagen phase. The hair may have reached its full length. Now it simply needs to be sustained. A rich blood supply is still present in the hair bulb.

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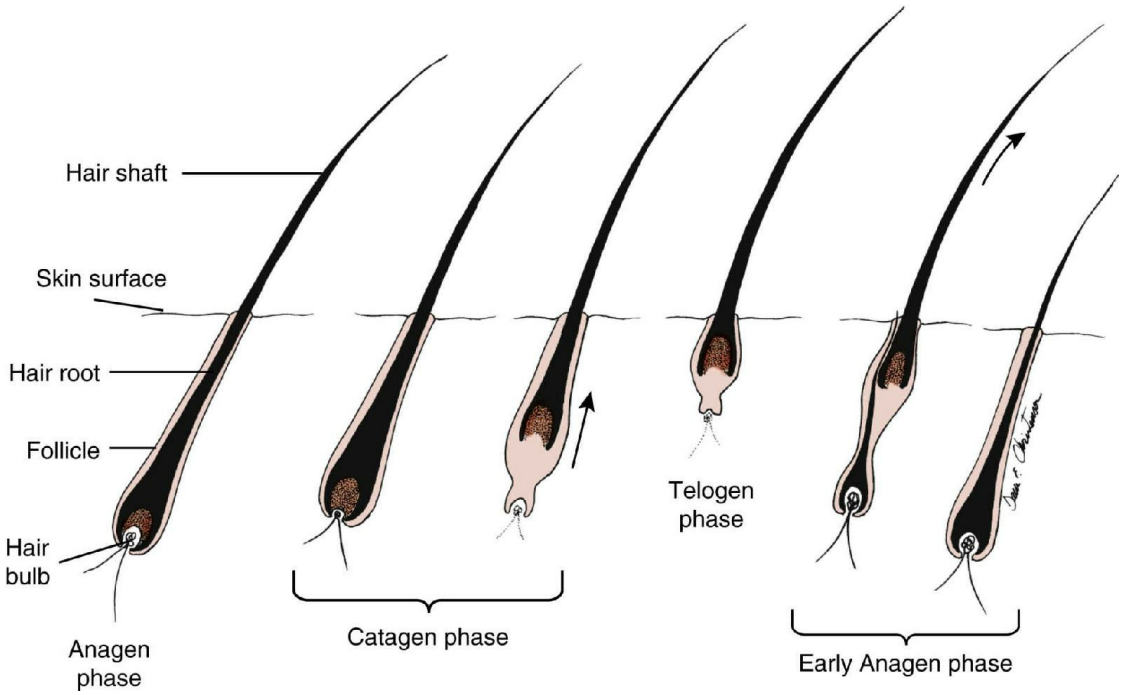

FIG. 8.13 Hair growth.

All good things must come to an end. We need to begin preparations for rest. We need to transition from the late anagen phase to the ***catagen*** [*cata-* down, against + *gen(o)-* production] ***phase***. To make sense of the prefix, *cata-*, in this context, I like to think of this as a "slow-down" so that the follicle can rest. To initiate this resting period, the hair bulb's blood supply must diminish. In fact, the hair bulb itself shrinks. No longer sustained, the root of the hair becomes detached from the shriveled-up bulb. Finally, the hair follicle itself begins to shrink and shorten. This helps push the old hair closer to the skin surface. Finally, when the follicle has completely shortened and the old hair is just loosely hanging out there, we have reached the ***telogen*** [*telo-* end + *gen(o)-* production] ***phase***—literally, the end. Now, all of our preparations for rest come to fruition. This is the true resting phase. Ah, sleep, glorious sleep! How long will the *telogen* phase last? That's variable. Often, it's weeks or months, but it could be longer. Aren't you glad that all hair follicles are not on the same schedule?!

What influences this whole cycle? Many things, including hormones, daylight (photoperiod), and temperature, all contribute. As you will learn in Chapter 10, growth hormone and testosterone are key hormones for growth and development. They are also important for hair growth. Think about a male lion, for example.

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It's testosterone that promotes growth of his magnificent mane, distinguishing him from female lions. Photoperiod and environmental temperature play a larger role when it comes to animals gaining their "winter coats" and "blowing the coat." (Obviously, this applies mainly to those animals who have some degree of undercoat for insulation.) And of the two factors, the photoperiod is most influential. As daylight periods grow shorter, animals enter into the early anagen phase. In the dead of winter, when the days are the shortest, these animals should have a thick, luxurious undercoat (wool hairs). As the days progressively get longer, those same follicles will begin their transition through the catagen phase. When they reach the telogen phase (longer and warmer days), they are visibly blowing their coats, with tufts sticking out all over. Grooming will help remove much of that. Many of these follicles will remain at rest during the long days of summer, when the weather is really warm.

By the way, birds experience a similar cycle of events for feather growth. ***Molting*** for birds and shedding and blowing coat for mammals are normal parts of the cycle for hair/feather growth.

Alopecia

Alopecia (al"o-pe'shuh [Gr. *alopecia*, a disease of hair falling out]) is different from normal shedding. *Alopecia* is abnormal hair loss. It usually results in abnormally thin or bald areas, where there should be an abundance of hair. Why does it happen? Alopecia is usually a symptom of something else. As veterinary professionals, we need to investigate thoroughly to determine the cause—and there are many causes. Certain hormonal diseases commonly result in *alopecia*, especially those that affect thyroid and reproductive hormones. We'll save hormonal disease discussions for Chapter 10. For our purposes here, simply remember that hormonal disorders usually cause very symmetric alopecia. This is helpful for shortening our disease rule-out list. *Dermatitis* (from many causes) can result in alopecia. Obviously, the skin inflammation can directly affect the follicles. But scratching, in response to the *pruritus* associated with dermatitis, will also contribute.

Parasites, especially parasitic mites, frequently cause alopecia. For example, there is a particular parasitic mite, *Demodex sp*.

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(de'muh-deks), that lives in the hair follicles. They are long and slender and look like a cigar with stumpy legs to fit comfortably in the follicle. Each host species has its own species of *Demodex*. Yes, even humans have them. They're usually transferred from Mom, when a youngster is nursing, so they're most abundant in the follicles of the face (yes, even yours). Of course, most of the time we get along just fine, sharing our follicles. But if the host's immunity drops, *Demodex* can reproduce out of control. It's not rocket science to figure out that if hair follicles become packed with mites, there's no room for hair. Localized ***demodicosis*** [*demodic(o)- Demodex* + *-sis* condition of] that creates focal areas of alopecia on the face and head may resolve on its own, when the host's immunity improves. But if it spreads over the body, ***pyoderma*** [*py(o)-* pus + *derma* skin] often develops from secondary bacterial infection. These infections can be very serious, even fatal.

Among animals, another common cause of alopecia is ***dermatophytosis*** [*dermat(o)-* skin + *phyt(o)-* plant + *-sis* condition of], also called ***dermatomycosis*** [*dermat(o)-* skin + *myc(o)-* fungus + *-sis* condition of]. Obviously, the "plant" in the first word is actually a fungus. There are a number of fungal organisms that cause *dermatophytosis*. Some of them may invade deep into the skin. The most common form of the disease tends to stay rather superficial. This type is commonly referred to as "***ringworm,***" because the alopecia appears as well-defined circles. The organism prefers to grow on stratum corneum and (its favorite) growing hairs. Unfortunately, it invades the hair shaft, weakening it. That's how it creates the patchy alopecia. Just remember, ringworm is not a worm. It's a fungus among us. And by the way, it's also ***zoonotic*** [*zo(o)-* animal + *nos(o)-* disease + *-tic* pertaining to; i.e., a disease transmitted between animals and people].

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Burns

Burns are a unique type of dermal injury that may be caused by a number of things. There are thermal burns, chemical burns, and even electrical burns. And while the immediate first aid, depending on cause, may differ, the end result is still a burn. And I believe it is important to understand how burns are classified. The depth of the burn determines, in part, our management of the patient. For further clarification of burns, watch the Evolve animation entitled *Burns*.

1st Degree Burns

1st degree burns involve injury to the epidermis. The area of the burn will still experience a mild inflammatory response that most notably reveals itself with erythema. This type of burn usually resolves in a matter of days. There is no scarring. If you've ever had a mild sunburn, you know the warm feeling and redness in the area. In a few days, the redness and warmth probably resolved and you experienced a bit of flaky skin in the area.

2nd Degree Burns

2nd degree burns involve injury to the epidermis and partial thickness of the dermis. This is obviously more serious, because there are numerous structures that have been damaged or destroyed by this burn. These burns are much more painful. Blisters form in the area. Much more time will be required for healing of 2nd degree burns.

3rd Degree Burns

3rd degree burns destroy the full thickness of the skin (epidermis and dermis) and may extend into the subcutis. Obviously, this is a very serious burn. The very center of the burn on the surface will be insensitive. Only at the margins will nerve endings be intact to elicit pain. In addition to pain, this deep tissue injury will promote a

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huge inflammatory response in all of the adjoining tissue. That means that large volumes of fluids (water and plasma proteins) will be lost through the injury. Burn victims with a large percentage of body surface area involved in the burn can literally lose gallons of fluid through those injuries per day. That is very problematic. Another facet of 3rd degree burns that is equally important is the fact that we have lost our natural barrier. The same is true for 2nd degree burns, because we've lost the epidermis there too. But in 3rd degree burns, the depth and breadth of the injury, coupled with all of the plasma proteins weeping into the area, provide an excellent environment for bacterial growth and invasion of the body. This is why burn victims, especially those with 3rd degree burns, must be managed with the utmost in ***aseptic*** [*a-* without + *sept(o)-* bacteria + *-ic* pertaining to; cf. sterile] technique. We need to ensure that we do not introduce any bacteria to the injury. Everything that comes in contact with the burned area must be sterile. Failure to do this could put the patient at risk of ***sepsis*** [Gr. *sepsis* decay, i.e., a systemic bacterial infection], and that could be fatal. If the patient survives, it will experience significant scarring.

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Wound Healing

Obviously, there are many types of wounds. We just finished describing the various types of burns. For the purpose of this discussion, we'll simplify the injury by talking about a simple accidental laceration (cut) or surgical incision. Still, keep in mind that whether the injury is a small laceration or a 3rd degree burn, all of the following stages will occur to heal the injury. The larger the injury, the longer the healing process will take.

Inflammatory Stage

Imagine a deep paper cut on your finger. We've all experienced those. As soon as the injury occurred, it entered into the *inflammatory stage* of wound healing (Fig. 8.14). You knew it immediately when it happened because it hurt. And as soon as you looked at it, you could see the blood. Remember all of those intradermal capillaries that we talked about earlier? Well, you just severed some of them. They bleed. Bleeding is important for two reasons. First, it helps cleanse the wound of contaminants. Second, the fibrin in the clot that forms will provide temporary (weak) support to hold the wound edges together. It will also provide some scaffolding to begin repairs on later. (If you need to review information about blood and clotting, please refer to Chapter 3.)

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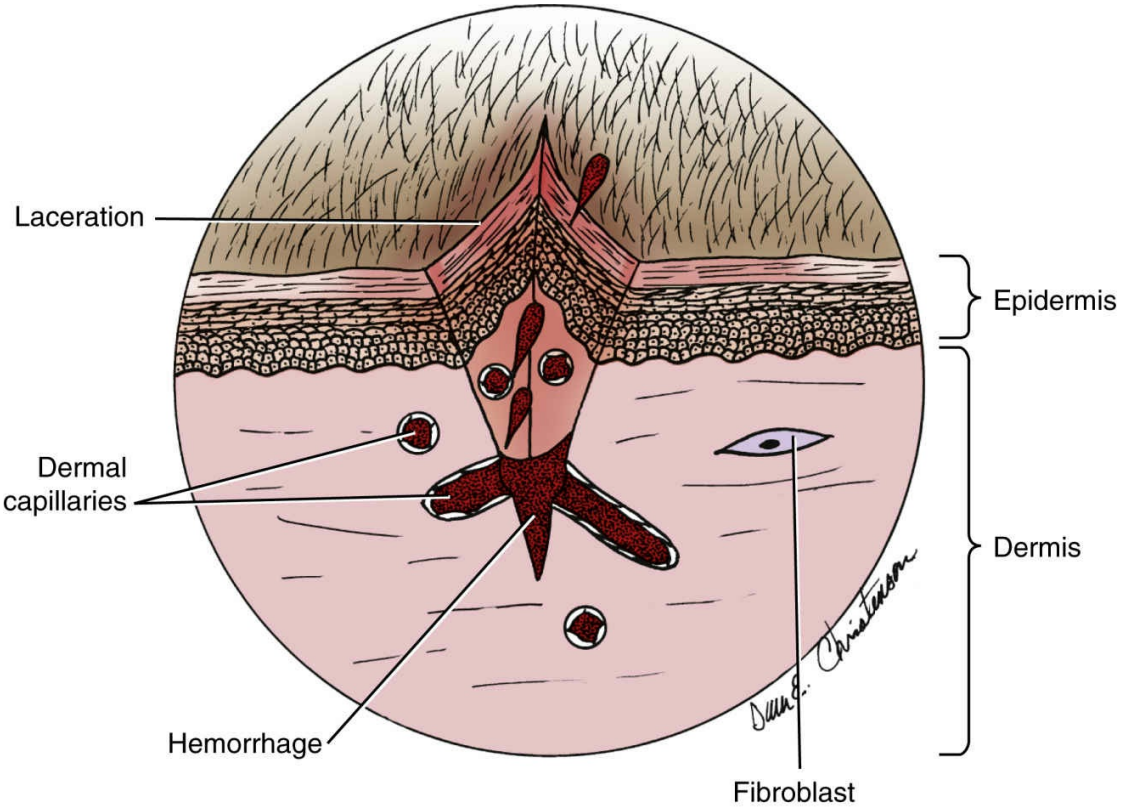

FIG. 8.14 Wounding and the inflammatory stage of healing.

In addition to damaging vessels, you also damage tissue, cells, and other structures (of particular importance in the dermis). Inevitably that damage results in the release of inflammatory mediators. (Again, refer to Chapter 3 if you need to review inflammation.) Inflammation is not fun. It increases our pain. (Remember the throbbing?) Believe it or not, even that pain is beneficial—it forces us to protect and rest the injured area. Of course, other intact vessels in the area dilate in response to inflammation, making the area *erythematous* and warm. *Edema* from a paper cut was probably pretty minimal. A larger laceration or a surgical incision will have noticeable edema formation. More importantly, inflammation sets us up for the next stage of healing—debridement. If you remember anything at all, remember that *inflammation is absolutely essential for the rest of wound healing*. Without inflammation wound healing will be delayed or prevented. Period.

As you recall from Chapter 3, inflammatory mediators attract various white blood cells to the area. We need them for *debridement*.

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Debridement Stage

So, inflammatory mediators attract white blood cells to the injury (Fig. 8.15). First responders are the *neutrophils*. Neutrophils, as you'll recall from Chapter 3, are ***phagocytic*** [*phag(o)-* eat + *cyt(o)-* cell + *-ic* pertaining to]. This is important for *debriding* (removing) cellular debris and other contaminants like bacteria. They begin *debridement* as soon as they arrive on the scene. They continue working in the wound, even after a scab forms over the surface of the clot. Here's the thing about neutrophils—they're messy. And their messiness increases inflammation. Catch-22? Yes, temporarily. But this shows you the importance of inflammation in the healing process. As inflammation increases, even more neutrophils are called to the area.

By the way, all of this activity, especially in larger wounds, contributes to the ***purulent*** (pu'roo-lent) ***exudate*** (eks'u-dāt) that oozes from the wound (i.e., pus). What's an exudate? It's a fluid that contains lots of proteins. If the exudate contains mostly water and plasma proteins, it will be transparent. We call this a ***serous*** [*ser(o)-* serum + *-ous* pertaining to] ***exudate*** because it looks like serum. A *purulent exudate*, in addition to the water and proteins, contains a large amount of cellular debris and white blood cells, making it appear opaque and whitish or yellowish. The larger and more contaminated the wound, the more debridement, inflammation, and exudation of pus will occur. While we're on the topic, I must address something. You may call this material pus. You may call it a purulent exudate. To use the term "pussy" is inappropriate for numerous reasons. Don't use that term, please.

Eventually, *monocytes* migrate to the wound in response to inflammation and the *neutrophilic* [*neutrophil* + *-ic* pertaining to] activity. Once monocytes leave the bloodstream and enter the wound, we call them ***macrophages*** [*macr(o)-* large + *phag(o)-* eater]. Okay, so they help debride the wound. True, they are important for debridement. They clean up the mess the neutrophils leave behind. But there is something equally, if not more, important—these macrophages secrete *growth factors*. And those growth factors are necessary to attract *fibroblasts*. If you remember, we said that there were very few fibroblasts in any isolated area of the skin. We're going to need them and lots of them to heal this dermal tissue.

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That's going to take time.

Do you see the importance of each step and each cell? Each one sets up the next in the series of events. We need inflammation to attract neutrophils for debridement. We need neutrophils to increase inflammation and attract more neutrophils and monocytes. We need monocytes (macrophages) to attract fibroblasts. And fibroblasts are necessary to repair the dermis.

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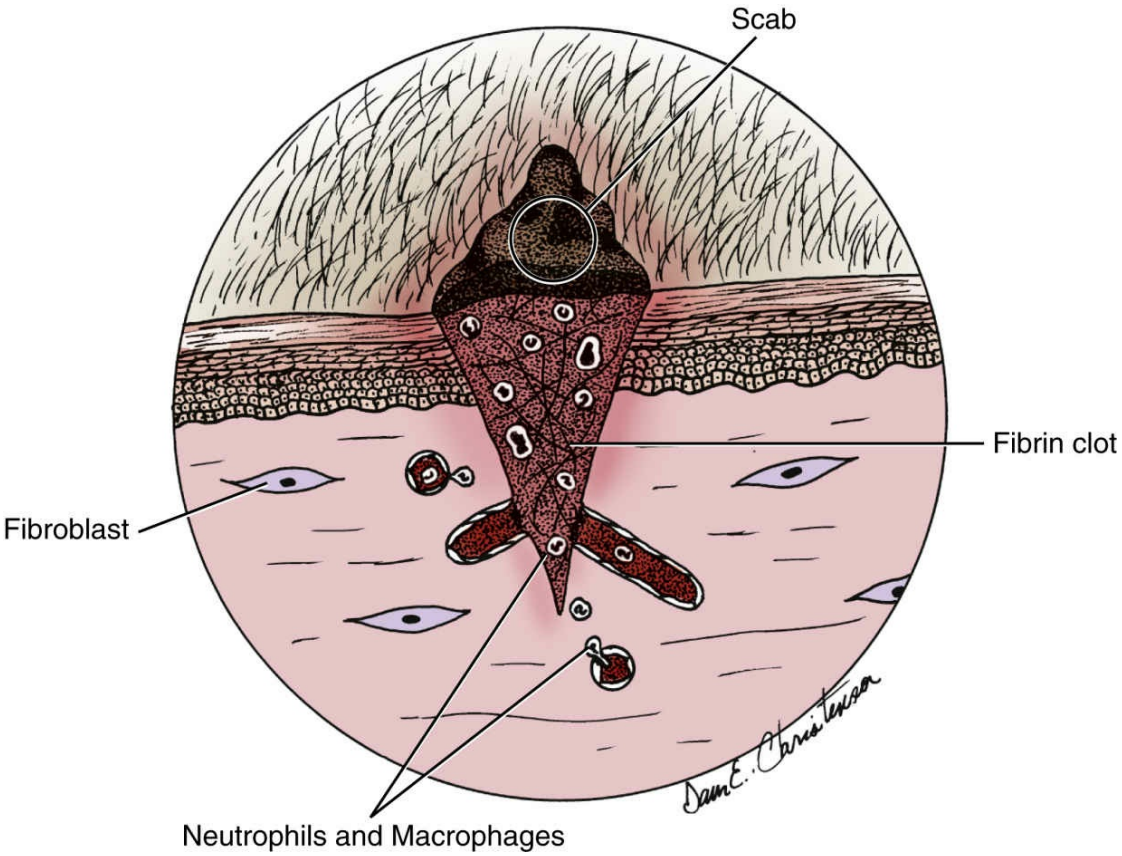

FIG. 8.15 Debridement stage of wound healing.

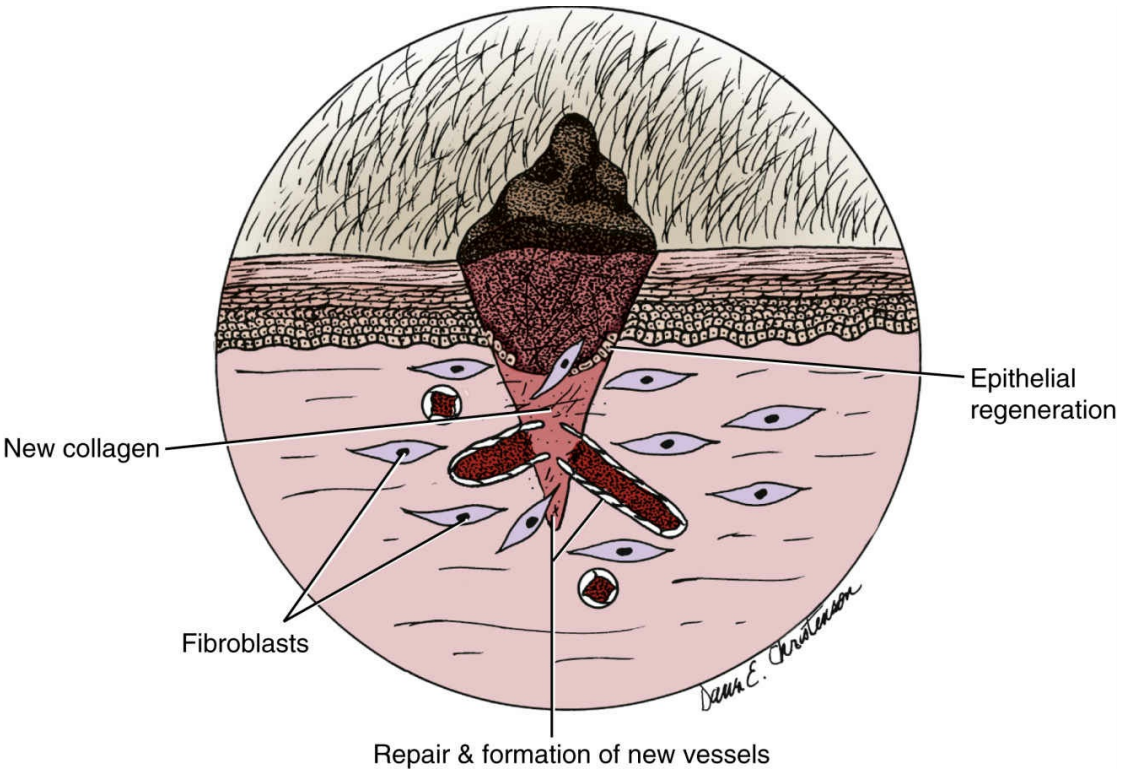

FIG. 8.16 Repair stage.

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Repair Stage

Spoiler alert: because the epidermis and dermis are made of two distinctly different types of tissue, we need to focus the discussions of those skin layers separately. Please recognize that repair of the epidermis and dermis is simultaneous events. In fact, in Figs. 8.16 and 8.17, you can see repair of both skin layers happening concurrently.

Let's focus first on the *dermis*. As I said, it takes time for *fibroblasts* to migrate to the wound. We may have a few beginning the repair process. But especially for a full-thickness surgical incision, we won't have peak numbers of fibroblasts for repair until between 5 and 21 days after the wound was created. This allows time for debridement. We'll arrive at the numbers needed to repair our paper cut sooner than that. When fibroblasts migrate into the wound, they use the *fibrin* strands of the clot as scaffolding. They actually begin to deposit *collagen* onto those fibrin strands. Of course, the fibrin strands have no organization. So, as the fibroblasts continue with the repair process, they deposit more collagen and begin to reorganize what was already created. They begin aligning the new collagen strands into a parallel horizontal arrangement (i.e., oriented much like the surrounding dermis). They also begin contracting (shortening) these fibers. The whole point of this is to improve strength in the area. There is virtually no strength early on. You know this if you've bumped, rubbed, or scratched your paper cut within a couple days of the injury. In a surgical incision, we don't begin gaining good strength until about 10 days after surgery. That's really the earliest time we should consider removing any external support devices like skin sutures or staples. Maximum strength of an incision is usually achieved by about 15 days after surgery. If we remove support too soon, the incision could ***dehisce*** (de-his' [L. *dehiscere* to gape]). Imagine ***dehiscence*** (splitting open) of an abdominal incision. That could be disastrous.

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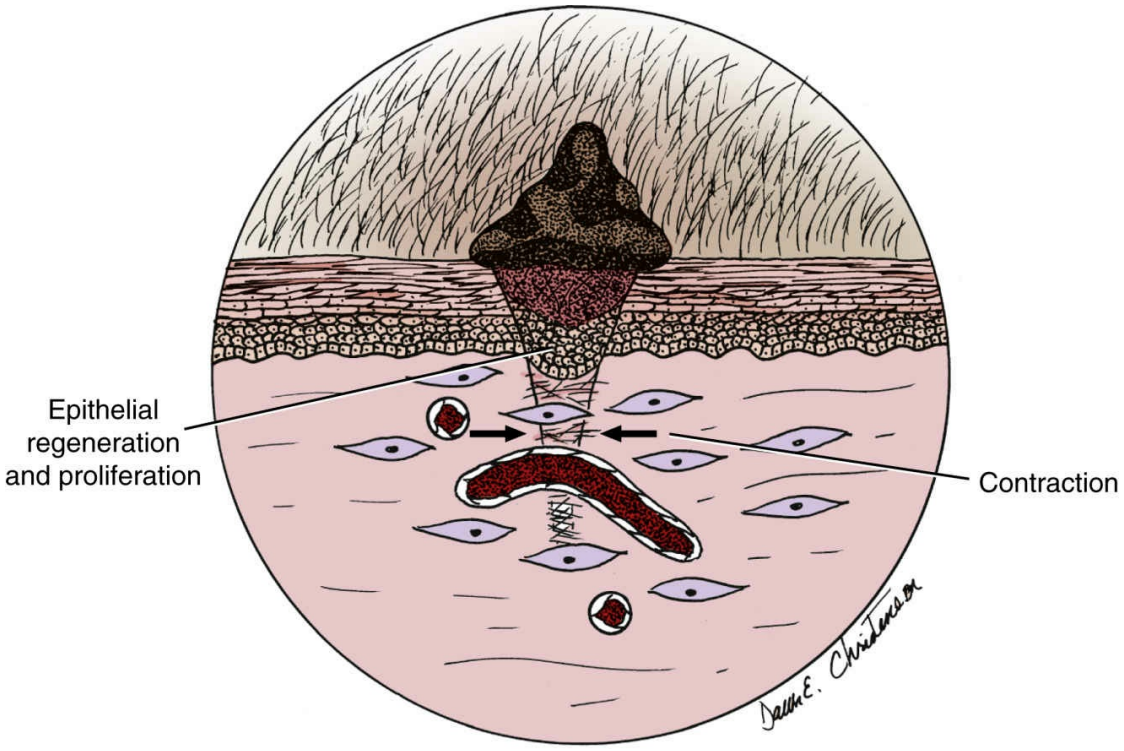

FIG. 8.17 Late repair stage.

Other activity is happening down in the dermis during the repair stage too. We need to repair vessels. The outer walls of vessels are made of collagen. In larger wounds (larger than our paper cut), we may need to actually create new vessels. And by the way, vascular repair and creation is a joint endeavor between fibroblasts laying down collagen for the outer vessel wall and ***endothelium*** [*endo-* within; endothelium is a type of epithelium on the inside of vessels] lining the interior surface. It's the combination of new collagen and vessels in large open wounds that we refer to as ***granulation tissue***. *Granulation tissue* is beautiful pink, moist, granular (sort of a bumpy surface) tissue. In a large open wound, this is the stuff that provides a foundation for all of the epithelial repairs. And reorganization and contraction of that granulation tissue is really important for reducing the surface area that the epithelium needs to cover.

Speaking of *epithelium*, let's talk about the *epidermis*. You can see in Fig. 8.16 that epithelium from the basal cell layer on either side of the wound begins to bridge the divide. As soon as a single layer of epithelium bridges the divide, it will begin to proliferate to fill the epidermal defect. Obviously, we need a good foundation of granulation tissue to support that epithelial regeneration. And here is a really cool phenomenon: as soon as the new dermal tissue is

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covered by epithelium, *pruritus* often develops. In our paper cut or even a surgical incision, this is often at about 2 days following wounding. Is our wound strong enough two days later to withstand digging and scratching? No! So it is important when sending home surgical patients to warn the owners that itching may develop 2 days later and persist during the healing process. They must prevent the animal from disturbing the incision. Yes, this may require "the cone of shame" (i.e., an Elizabethan collar).

Once we have complete epithelial regeneration and proliferation (see Fig. 8.17), the epithelium will begin to break down the last fibrin attachments for the scab. Eventually, it will fall off on its own. Have you ever scratched at or picked at scabs? You have, haven't you? And I'll bet that healing wound bled, didn't it? Do you realize that when you prematurely pick off a scab, you are ripping off much of the new epithelium and some of the granulation tissue? Now what? Now, we have to start all over again! The more you persist in ripping off those scabs and all of the new tissue with it, the more likely you will create greater amounts of scar tissue. So don't do it!

Another thing that adversely impacts repair is steroids. Remember, anything that significantly reduces inflammation in a wound/incision will significantly slow the healing process. Steroids, whether produced by the body in chronic disease or given as medication, significantly reduce inflammation. Inflammation is necessary to attract neutrophils and monocytes for debridement. We can't begin repair until the wound is debrided. Steroids significantly reduce inflammation and thereby reduce attraction of those important white blood cells. Plus, in the presence of steroids, neutrophils aren't able to exit the bloodstream easily, if at all. We need them, and we need monocytes for their growth factors. Those growth factors are needed to attract fibroblasts. Even the epithelial activity is slowed by steroids. Slow down the numbers and activity of all these cells in the wound and we significantly slow down repair. That means any surgical incision will not achieve needed strength to hold the wound edges together at a normal rate. In a patient like this, if we remove skin sutures or staples at 10 days or less, that incision is at risk of dehiscence. We may need to wait much longer than usual before removal of support.

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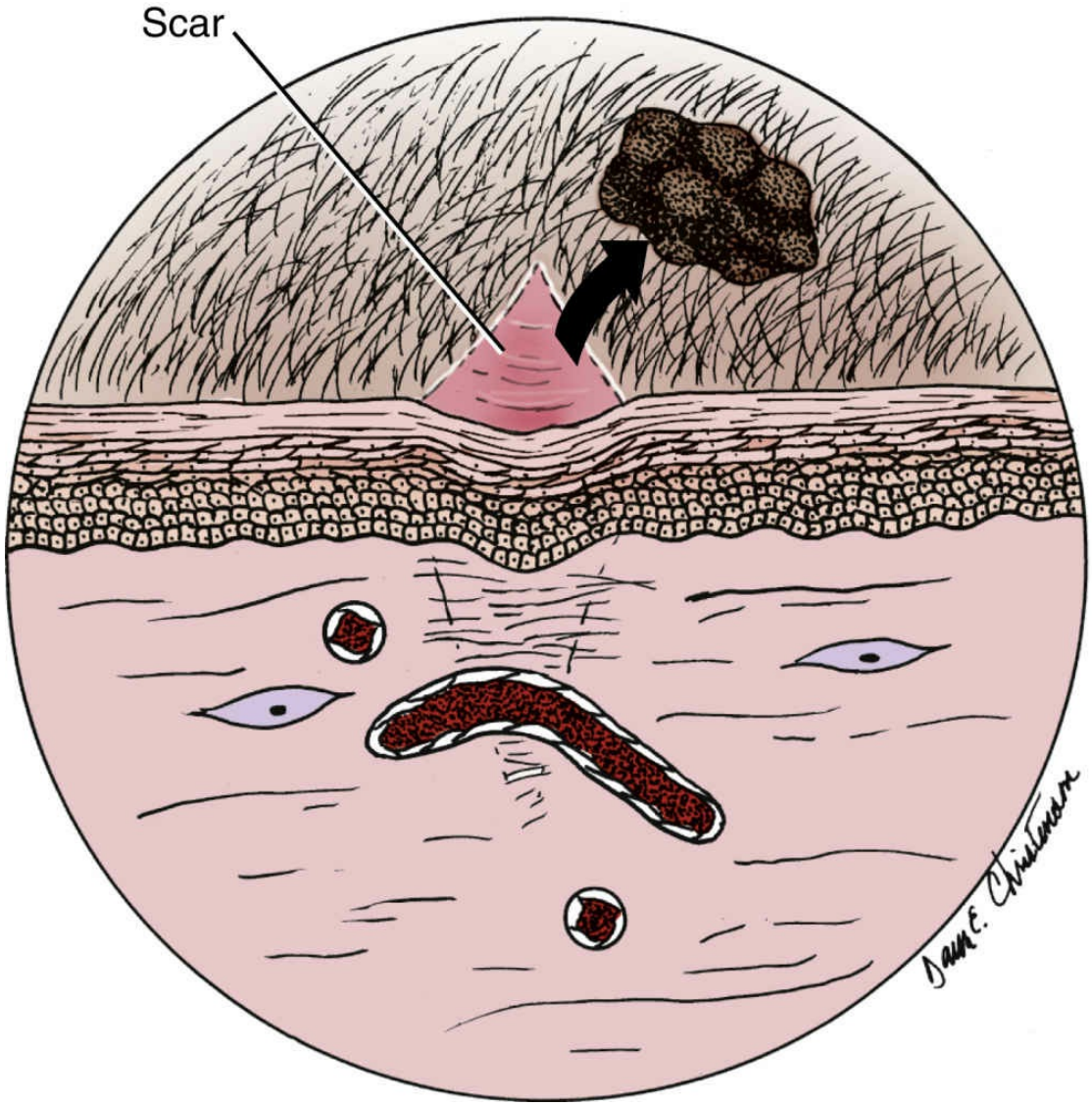

FIG. 8.18 Early maturation stage.

Finally, healing of large open wounds is always delayed and prolonged. Yes, there is a larger area to repair. But it's not just the size of territory. Epithelial regeneration will always be delayed in an open wound. We cannot build a bridge across the divide until we have a strong foundation. Remember, we won't have peak numbers of fibroblasts in the area for dermal repair until between 5 and 21 days after wounding. It may easily be a week or more before we have a good granulation bed to support the epithelium. As to the pruritus that we mentioned earlier, it will begin at the margins of the large open wound, so it is very important to prevent scratching at those wound edges. We'll never be able to cover that wound if the animal keeps scratching off all of the new epithelium at the margins. Even after a wound appears healed, there is still a

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long maturation phase ahead of it.

Maturation Stage

In the *maturation stage* of wound healing, we've lost the scab and we are left with a *scar* (Fig. 8.18). The scar from our paper cut is probably not noticeable. A larger wound like a surgical incision will have a visible scar. Over time, many of the fibroblasts will disburse out into the surrounding dermis. But a handful of workhorses will stay behind. Why? We still have to reorient those collagen fibers. They still need to be shortened. So, over time, a visible scar should become narrower. This activity may go on for nearly a year (in surgical wounds). Even once a scar is fully mature, that scar tissue will always be 15% to 20% weaker than normal skin. Why? It's made primarily of *collagen*. There is no *elastin*. So scar tissue can't flex and stretch like normal skin. Subjected to enough force, a scar will break before the surrounding skin does.

Keloid

A ***keloid*** (ke'loid [Gr. *kelis* blemish + *-oid* resembling]) is a very large, raised scar. Some people are prone to *keloid* formation. That's the exception rather than the rule. Keloids result from excessive collagen deposited in a maturing scar. In most people and certainly in animals, this is most likely to occur in an area where the scar is subjected to lots of stress and tension as well as forces chronically pulling and tugging at it like over a joint. Because of the unusual forces placed on the scarred area, more fibroblasts stay in the area. They continue to remodel and deposit more and more collagen. The goal is to strengthen the collagen fibers by bulking them up. Unfortunately, this only serves to create a really big, ugly scar that, despite its size, is still no stronger than the average scar. Bigger is not necessarily better.

Proud Flesh

Proud flesh occurs commonly in horses. What is it? It is ***exuberant granulation tissue***. Uh, what on earth does that mean? To be exuberant is to be really enthusiastic, right? And you know what

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granulation tissue is. In horses prone to *proud flesh* formation, they have accelerated—extremely accelerated—repair of the dermal tissue. This most commonly occurs in wounds over distal extremities, where the skin is under a great deal of tension. Okay, how is this different from a keloid? Simple—proud flesh is not covered by epithelium. This is not a scar in the maturation stage. Exuberant granulation tissue develops extremely early in the healing process, long before any epithelium can possibly cover it.

We said that in a normal healing process, peak numbers and activity of fibroblasts happen between 5 and 21 days after wounding. Well, in horses prone to proud flesh, the exuberant granulation tissue may develop in less than 24 hours. It's like those early-bird fibroblasts are racing around on caffeine, already bulking up collagen and creating new vessels long before they should be. And they just keep going and going! The epithelium doesn't stand a chance of covering the granulation tissue. The granulation tissue within a couple of days may already be protruding from the wound like a tumor. In fact, I've seen some horses where the proud flesh looks like a giant tumor. We'll never reach the maturation stage, because we'll never cover the granulation tissue with epithelium. When it gets like that, we need to remove the excess tissue and try to close the wound. In a laceration that is sutured closed, the wound edges are close together, right? So it shouldn't take as long for epithelium to bridge the gap. That's our goal. That's why in horses prone to proud flesh, even minor lacerations should receive prompt veterinary attention to close the injury. We may also have to employ other things, such as gentle pressure, to slightly diminish blood flow and slow down those fibroblasts, without slowing the epithelium. That's a delicate dance that we don't always win. Our best chance of winning is if we close the wound right away.

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Allergic Dermatitis

We discussed ***allergic*** [*allerg(o)-* allergy + *-ic* pertaining to] responses in Chapter 3. If you need to, please take the time to review that information in Chapter 3. In brief, *allergic* or ***hypersensitivity*** [*hyper-* excess + *sensitiv(o)-* sensitive + *-ity* state of being] reactions are exaggerated responses to ***allergens*** [*allerg(o)-* allergy + *gen(o)-* producer]. In animals, regardless of type of *allergen* (inhaled, ingested, or contact), *hypersensitivity* reactions most often result in *dermatitis*. This is because the lion's share of *mast cells* in the body is found in the dermis. So when the body goes overboard in its production of *IgE* and arms a bunch of mast cells, inflammatory mediators like histamine will be "fired off" in the skin. These animals predisposed to developing allergies usually do so early in life (often by 6 months of age). Dogs with allergies are usually easy to spot because of the ***pododermatitis*** [*pod(o)-* foot + *dermat(o)-* skin + *-itis* inflammation of] that they chronically have. They tend to lick their pruritic feet, and the saliva creates characteristic brown staining there.

Determining problematic allergens may be accomplished in a number of ways. In *intradermal* skin testing, we actually inject a very small volume of specific allergen into the dermis. There is a whole grid of allergens injected in plotted-out locations on the side of the animal. A *positive control* (histamine) and *negative control* (sterile saline) are also injected. Then the sites are timed, observed, and compared to the positive and negative controls. Positive reactions create round, erythematous, raised *wheals* at the injection sites. A ***wheal*** is similar to the red, raised areas seen with ***urticaria*** [*urticar(o)-* "stinging nettles" + *-ia* condition of; i.e. hives]. But comparatively, wheals are round and isolated to the injection site. *Urticaria* (hives) usually develop over the whole skin surface. Another form of testing involves testing the blood for antibodies for specific allergens. Both forms of testing have advantages and disadvantages. But our goal is to determine specific allergens so that we can avoid these allergens or desensitize the animal to them. Flea saliva is one of the most common allergens to be identified in dogs and cats.

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Flea Allergy Dermatitis

Flea allergy dermatitis is a very, very common problem in dogs and cats. But this is a complicated allergic reaction. These animals are allergic to the flea's saliva—usually the cat flea (yes, even for dogs). So, when bitten by a single flea, an immediate allergic reaction develops. The dermatitis is body-wide. But it doesn't end there. This over-the-top allergic reaction will persist from a single flea bite for up to 90 days! Fleas and flea bites are annoying enough in a normal animal. In an animal who's allergic to them, we have intense and prolonged dermatitis.

So, in flea-allergic animals, it is important to prevent even a single bite. And with all of the products available today, that's relatively easy. We can easily target adult fleas as well as their eggs and developing offspring. And it's really important to wage war against fleas on multiple fronts. We need to treat the allergic animal, killing any adults. Hopefully, our treatment will help prevent fleas from getting back on the patient. We also need to treat all other animals in the household to reduce the flea population threat to the allergic animal. Finally, we need to treat the environment to eliminate fleas in all their life stages from the environment. I cannot emphasize enough how important a multifaceted flea control protocol is for a flea-allergic animal. I've seen some extreme dermatitis and pyoderma in flea-allergic animals. For us, as veterinary professionals, it is extremely important that we understand the flea life cycle and all of the available products to provide the best management of flea-allergic patients.

Food Allergy Dermatitis

When dealing with year-round allergic dermatitis, food allergies have to be suspect. My basset hound, Sadie, had food allergies. Corn was a primary allergen for her. Corn in any form (even in corn tortilla chips) would result in a profound allergic reaction. If she ate even a small piece of a corn chip, within the hour one of her ears would develop ***otitis*** [*ot(o)-* ear + *-itis* inflammation of]. Within a couple hours, the other ear would flare, along with the rest of her skin. (Food allergies are often the underlying problem in roughly

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85% of chronic, recurrent *otitis*.) Sometimes Sadie developed *urticaria*, usually if she ate a large amount of corn. She was so predictable and extreme in her allergic reaction. Once the hypersensitivity reaction was underway, all we could do is treat her with ***antihistamines*** [*anti-* against + *histamine*]—with marginal success in reducing the reaction. If we saw her eat the chip and immediately gave the antihistamine, we could more successfully combat the dermatitis. But the best treatment was to prevent her from eating it in the first place.

This is the goal in food allergies—avoidance. But to avoid an allergen, we need to know which allergens are problematic. Skin and blood tests may help us determine this. But probably the best and most difficult method of isolation is a feeding elimination trial. Done right, a feeding elimination trial places the animal on a novel, limited ingredient diet. What's a novel, limited ingredient diet? It contains limited ingredients of a "novel" (never ever been consumed by the animal) protein and carbohydrate. This may be found in a commercially prepared diet or the owner may cook for the pet. For Sadie, I cooked lamb and sweet potatoes. Yes, it was labor-intensive for me, but she loved it! The novel diet is fed until the animal is no longer showing allergic signs. That means nothing else may pass the lips of that animal—no treats, no chew toys, only the novel diet! This could take up to 8 or 12 weeks, before the dermatitis is gone. Once gone, specific individual foods are added to the diet. Because reactions could be immediate or delayed, each new food may need to be fed for up to a week, while carefully observing for any signs of an allergic reaction (usually dermatitis, sometimes vomiting or diarrhea—the digestive tract also has moderate numbers of mast cells). If no reaction occurs during the week, that food is stopped and another specific food is added to the novel diet. If a reaction occurs, that food is removed and only the novel diet is fed until the allergic reaction is gone. Then, on to the next. Most owners are not willing to go to these lengths. And any specific foods that are identified as allergens should be avoided at all costs. We hope, by determining carbohydrate and protein sources that the animal is not allergic to, we'll be able to find a ***hypoallergenic*** [*hypo-* low, reduced + *allerg(o)-* allergy + *gen(o)-* producing + *-ic* pertaining to] commercial diet for the pet. In

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addition to novel ingredients, some hypoallergenic diets are hydrolyzed [from *hydr(o)-* water + *-lysis* process of breaking]. In hydrolyzed diets, the molecular structure of ingredients is altered such that it is no longer recognizable as an allergen.

Atopic Dermatitis

Atopic (allergic) ***dermatitis*** is caused by environmental allergens, such as pollen. Inhaled pollens usually give us ***pruritic*** [*prurit(o)-* itchy + *-ic* pertaining to], watery eyes and nasal congestion. Animals with ***atopy*** (*-y* state of) develop dermatitis. Did I mention that when I adopted Sadie at 6 months of age from the Humane Society, I asked why she was surrendered? She was surrendered with the complaint of allergies. I assumed that someone in the family was allergic to Sadie. Wrong! We've already talked about her food allergies. In terms of atopy, she was allergic to ragweed pollen. Well, that's mighty hard to avoid in Michigan. Fortunately, it's seasonal. In fact, when animals have seasonal allergic dermatitis, environmental allergens should be suspect. Either form of testing (intradermal skin testing or blood testing) can be useful in determining specific allergens. The thing to remember with intradermal skin testing is that it cannot be done during the animal's peak allergy season—because it will cause overreaction to most of the injected allergens. And it should not be done well into the off-season—then it may not produce a sufficient reaction. Shortly after the pet's allergy season is the best.

If we can't avoid the allergens, why figure out which ones the animal is allergic to? Well, it offers us the opportunity to desensitize the animal. We talked about this in Chapter 3. To desensitize, we inject really low doses of the allergen, low enough that we do not elicit an allergic reaction. We're trying to teach the body not to overreact by producing excess amounts of IgE. Without IgE, mast cells won't fire off their inflammatory mediators. Over time we gradually increase the quantity of the allergen in each injection. By the time we're done, the animal no longer reacts. Because Sadie's allergic condition was not that severe, and ragweed pollen was only around for a matter of weeks, I did not try to desensitize her. I merely reduced the dermatitis with *antihistamines*.

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Case Study

Sadie is a 2-year-old spayed female Basset Hound. Since the owner acquired Sadie, when she was 6 months old, she has experienced chronic problems with dermatitis and otitis. She has been treated for multiple ear infections. Twice she required treatment for pyoderma. Treatment with antihistamines has been tried, but seems to be insufficient. Her pruritus drives her and her owner nuts. She has evidence of pododermatitis. Sadie does not have fleas or any other parasites. Atopy is suspected.

To determine potential allergens, the veterinarian ordered intradermal skin testing. Because her condition seems to be worse in August and September, the intradermal skin test is scheduled for November. That test reveals ragweed pollen as a principal allergen. During the next ragweed season, the owner treats Sadie with antihistamines. The antihistamines seem to help, but the dermatitis does not resolve completely. In fact, she seems to have low-grade dermatitis year-round. And over the course of the year, she requires treatment for bacterial otitis two more times. Once, when she developed urticaria and pyoderma, she was treated with antihistamines, steroids, and antibiotics.

Due to her extreme, chronic problems with dermatitis and otitis, the veterinarian strongly suspects food allergies in addition to atopy. A feeding elimination trial is recommended to the owner. A limited-ingredient diet, with a novel protein and carbohydrate, is fed. Over the course of four months, the owner engages Sadie in the elimination trial. When complete, it is discovered that Sadie is allergic to corn and chicken. She is placed on a limited-ingredient fish and potato diet. By avoiding any foods containing corn and chicken, Sadie's dermatitis and otitis episodes are dramatically reduced. When dietary indiscretions occur, the owner is able to minimize the severity of the reactions with antihistamines.

Case Study Questions

1. Sadie is experiencing inflammation of the skin,

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medically termed _____.

2. Sadie also periodically experiences inflammation of her ears, medically termed _____.
3. Sadie has a condition of very itchy skin, which is termed _____.
4. She has also required treatment for _____, a condition in which, due to infection, she had pus on her skin.
5. Sadie has evidence of skin inflammation on her feet or _____, demonstrated by the brown salivary staining on her paws.
6. Another way to refer to pus is to refer to it as a _____exudate.
7. On rare occasions, Sadie has also developed _____ or hives.
8. Due to the seasonal nature of some of her skin inflammation, _____ (a type of allergic condition that involves environmental things like pollen) was suspected.
9. We performed _____ skin testing, in which substances are injected into the dermis.
10. Ragweed pollen was found to be a principal _____ (allergy producer) for Sadie.
11. Sadie was treated with an _____ (i.e., a medication against histamine) during peak ragweed season.
12. Due to the _____ (i.e., long-term), year-round nature of her skin inflammation, food allergies are also suspected.

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The Answer Key to these case study questions may be found in Appendix B.

[a] Porter, Cole – composer/arranger of the song *I've Got You Under My Skin*, from the 1936 MGM musical *Born to Dance*; the song was made famous by Frank Sinatra on his 1956 album *Songs for Swingin' Lovers*, Capitol Records.

[b] *Dawn* is a registered trademark of the Proctor & Gamble Company.

[c] *Scrubbing Bubbles* is a registered trademark for a line of products made by SC Johnson & Son, Inc.

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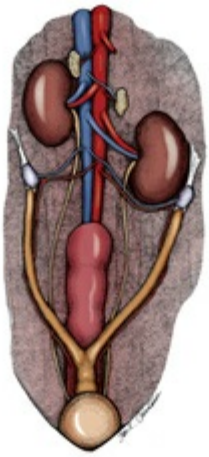

Applied Reproductive Terminology

Female,
- Ovaries,
- Uterus and Reproductive Tract,
- Reproductive Cycle,
  - Proestrus,
  - Estrus,
  - Metestrus,
  - Anestrus/Diestrus,
  - Canine Vaginal Cytology,
- Pregnancy,
  - Birth,
- Mammary Glands and Lactation,
  - Mastitis,
  - Hypocalcemia, Eclampsia, and “Milk Fever”,
- Pseudocyesis,
- Pyometra,

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Male,
- Testicles,
  - Spermatogenesis,
  - Spermatozoa,
  - Semen,
- Accessory Glands and Structures,
- Penis and Prepuce,
- Erection and Ejaculation,
- Paraphimosis,
- Prostatic Hyperplasia and Prostatitis,

Common Reproductive Surgeries,
- Ovariohysterectomy Versus Oophorectomy,
- Orchiectomy/Orchidectomy,
- Episioplasty and Episiorrhaphy,
- Mastectomy,

Case Study,

GOALS AND OBJECTIVES

By the conclusion of this chapter, the student will be able to:

1. Recognize common root words, prefixes, and suffixes related to the reproductive system.
2. Divide simple and compound words into their respective parts.
3. Recognize, correctly pronounce, and appropriately use common medical terms related to the reproductive system.
4. Demonstrate an understanding of male and female reproductive anatomies.
5. Demonstrate an understanding of reproductive anatomy and physiology of female animals, as it relates to the estrous cycle, pregnancy, birth, and lactation.

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6. Demonstrate an understanding of reproductive anatomy and physiology of male animals, as it relates to the production of sperm, semen, and breeding.
7. Demonstrate familiarity with common species differences in naming females, males, and offspring.
8. Demonstrate familiarity with the relationship between the reproductive and urinary systems.
9. Demonstrate familiarity with common medical conditions and surgeries for the reproductive system.

Please note, as we begin our discussions of female and male anatomy and physiology, that this is by no means endorsing this binary as exclusively "normal." There are sexual differences that fall outside of this male-female binary, such as ***hermaphroditism*** [*hermaphrodit(o)-* having both male and female sex organs + *-ism* state of] for example. The term hermaphrodite was originally derived from Greek mythology. According to myth, the son of the gods Hermēs and Aphroditē had his body merge with a nymph who was in love with him. Hence, his body had both male and female sexual characteristics. This phenomenon frequently occurs throughout nature. Among parasitic worms, both flukes and tapeworms are ***hermaphroditic*** [*hermaphrodit(o)-* having both male and female sex organs + *-ic* pertaining to]. Still, other helminths [L. *helminth* worm], such as *Strongyloides* (stron"jil-oid'ēz), are not hermaphroditic, yet sexually unique. *Strongyloides* may reproduce via sexual activity between male and female worms or by the female worms alone. You see, in the environment (e.g., on pasture), reproductive activity between male and female worms may produce multiple generations of offspring. But in a host animal, only the females reside and are parasitic in the host. This exclusive female population of Strongyloides reproduce via ***parthenogenesis*** [*parthen(o)-* virgin + *gen(o)-* produce + *-sis* process of]. And if you think that's unique, there are actually some animals that can shift gender, when warranted by the reproductive needs of the group. So, while we will be focusing our discussion on mammalian male and female attributes and how reproduction occurs to produce

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offspring, please remember that there are other gender and reproductive possibilities that are beyond the scope of this discussion.

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Female

Ovaries

Among domestic animals, it is the female that carries and gives birth to young. In order for her to reproduce, she requires ***ovaries***. Each female has two ovaries, which are located in the dorsal abdomen (Figs. 9.1 and 9.2). They are firmly attached to the abdominal wall by bands of fibrous connective tissue called the ***suspensory ligaments***. The ovaries contain ***oocytes*** [*oo-* from Gr. *oon* egg + *cyt(o)-* cell] in various stages of development. Now, these cells are very unique. You see, they are formed via a special process. If you recall from Chapter 2, *mitosis* is the process of cellular reproduction that results in two new duplicate, "daughter" cells, with complete sets of chromosomes and organelles of the original. If you're rusty on this, please review this information in Chapter 2. Oocytes are different from other "daughter" cells of the body, because they contain only half of the chromosomes of the "parent" cell. How does this happen? Well, oocytes are not formed via the typical mitosis. It is the process of ***meiosis*** (mi-o'sis [Gr. *meiosis* to diminish, decrease]). *Meiosis* is a very special method of cellular division that only occurs in sex cells. Through this process, each new cell contains only half of the original chromosomal information. This is by design. These incomplete oocytes will be made complete, with regard to DNA, when they are fertilized by sperm. We'll talk about that later. First let's finish our discussion of female anatomy.

The ovaries are located near a funnel-shaped structure, called the ***infundibulum*** [L. *infundibula* funnel]. The *infundibulum* acts like a "catcher's mitt" to receive the oocytes during ***ovulation*** [*ovul(o)-* egg + *-tion* process/state of; i.e., release of eggs]. Because the *infundibulum* is funnel-shaped, it is very effective in catching and directing the eggs through the ***oviduct*** [*ov(o)-* egg + *duct(o)-* passage] to the uterus. And to make sure that things don't shift, the ovaries are held in close proximity to the uterine horns and *infundibula* by the ***proper ligaments***.

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Uterus and Reproductive Tract

Did you notice a moment ago that there was a reference to the ***uterine horns***? You see, the uterus of most domestic animals is "Y" shaped, as shown in Fig. 9.2. The actual structure and shape of uterine horns vary from species to species. Regardless of variations, because many of these animals may produce multiple offspring, the uterine horns provide adequate space for their development during pregnancy. Now, most cows and mares tend to produce individual young. However, they may produce twins (probably one from each horn), and their uterine horns accommodate that. Twins are much more common in sheep and goats. Dogs, cats, and pigs, on the other hand, tend to produce litters. Obviously, if a female is going to carry multiple young in the uterine horns during pregnancy, there needs to be some sort of support. We don't want the uterine horns just sitting on the floor of the abdomen. So, the ***broad ligaments*** provide this needed support. The uterine vessels are also suspended by these connective tissues. Needless to say, the connective tissue of the broad ligaments does stretch due to the weight of the young in the uterus. Still, the heavier the horns become, the closer to the ventral abdomen they become.

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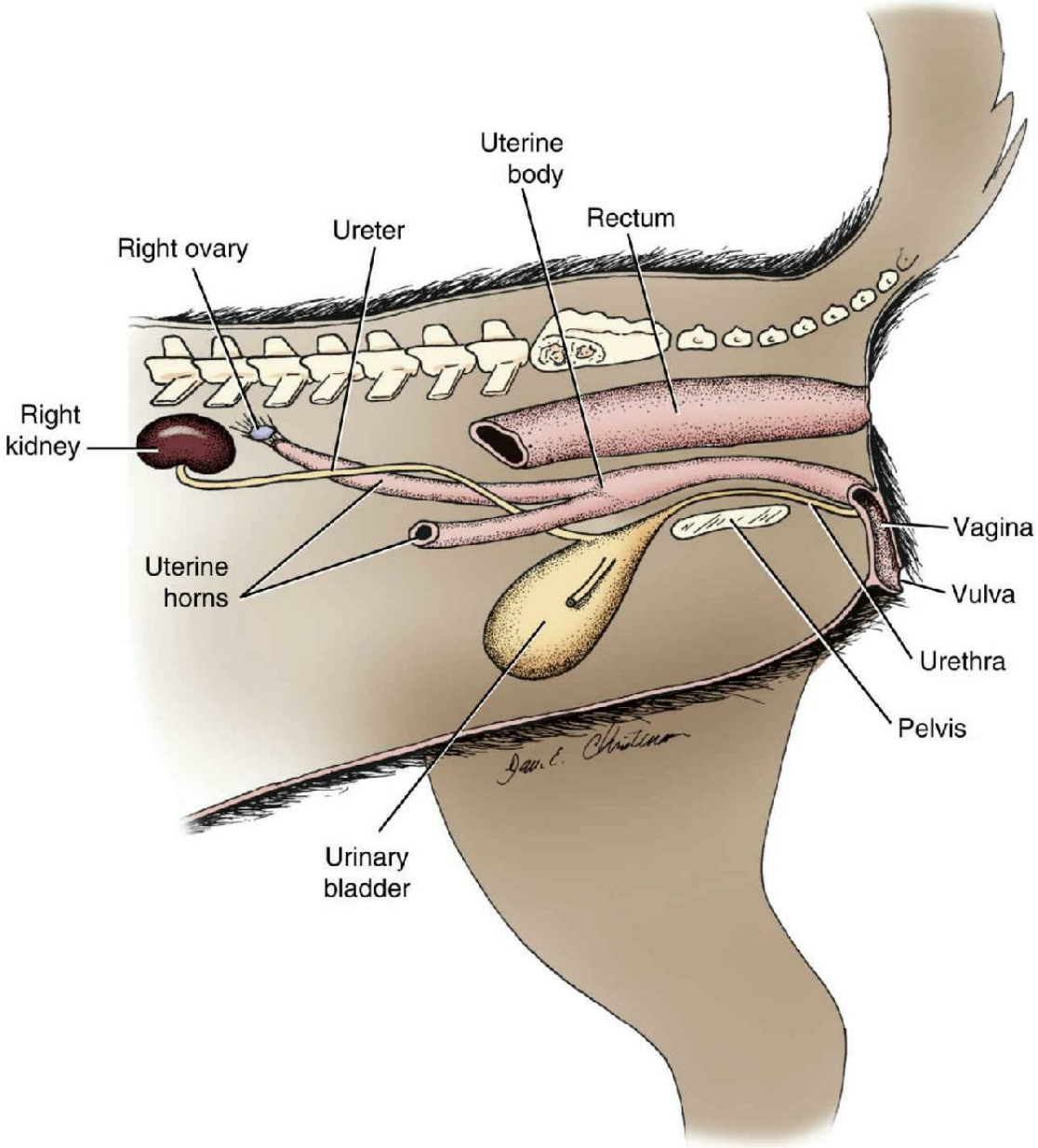

FIG. 9.1 Canine female urogenital tract (lateral view).

As we said, each uterine horn has the potential, depending on the species, to hold multiple young. But there is only one path out of the uterus for those young. So the caudal end of each uterine horn opens into the ***uterine body***. The ***cervix*** (ser'viks [L. "neck"]) is the strong muscular ***sphincter*** (sfingk'ter [Gr. *sphinktēr* that which binds tight]) near the caudal end of the uterine body. The cervix should be tightly closed during pregnancy and during quiet, non-active periods of the reproductive cycle. This helps keep ***pathogens*** [*path(o)-* disease + *gen(o)-* producers; i.e., disease-causing organisms] out of the uterus. Obviously, the cervix needs to relax during birth,

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which allows passage of the young through the rest of the "birth canal." The "birth canal" per se is the ***vagina***, which lies between the cervix and the ***vulva***. The urethra enters the caudal part of this reproductive tract too—near the caudal vagina and proximal ***vestibule*** [L. "chamber"] of the ***vulva***. This dual purpose is one reason why, when discussing structure of reproductive and urinary systems, many folks refer to ***genitourinary*** [*genit(o)-* reproductive + *urin(o)-* urine + *-ary* pertaining to] or ***urogenital*** [*ur(o)-* urine + *genit(o)-* reproductive + *-al* pertaining to] anatomy. When we study male anatomy a little later, you'll discover that they too have similar dual-purpose portions of their *urogenital* tracts.

Reproductive Cycle

Talk about variations on a theme! There is so much variability in the ***estrous*** [*estrus* + *-ous* pertaining to] ***cycles*** of female domestic animals. They all experience the four phases of the estrous cycle, which we're about to discuss. But when, how long, and how frequently these phases occur varies from species to species. Even within a given species, there may be variability. Take the dog for instance: small dogs (bitches) may cycle twice a year (i.e., ***diestrous*** [*bi-* two + *estr(o)-* estrus + *-ous* pertaining to]), while large breed bitches often cycle only once a year (i.e., ***monestrous*** [*mono-* one + *estr(o)-* estrus + *-ous* pertaining to]). Cattle (cows), on the other hand, cycle monthly. Because they cycle many times during the year, cows are considered ***polyestrous*** [*poly-* many + *estr(o)-* estrus + *-ous* pertaining to]. Pigs (sows) are *polyestrous* too. Horses (mares), sheep (ewes), goats (does), and cats (queens), on the other hand, are ***seasonally polyestrous***, because they cycle many times during a particular season of the year. By the way, when queens are "in heat," they are obnoxious, to say the least. Don't be surprised to field telephone calls from cat owners (often in late winter) who are concerned because their quiet, cuddly, demure little feline girls are "going crazy," screaming their heads off and rubbing and rolling all over the place. Suffice it to say that owners of breeding animals, regardless of species, expect (and rightly so) veterinary professionals to be knowledgeable about the female reproductive cycle, especially with regard to their particular species of animal.

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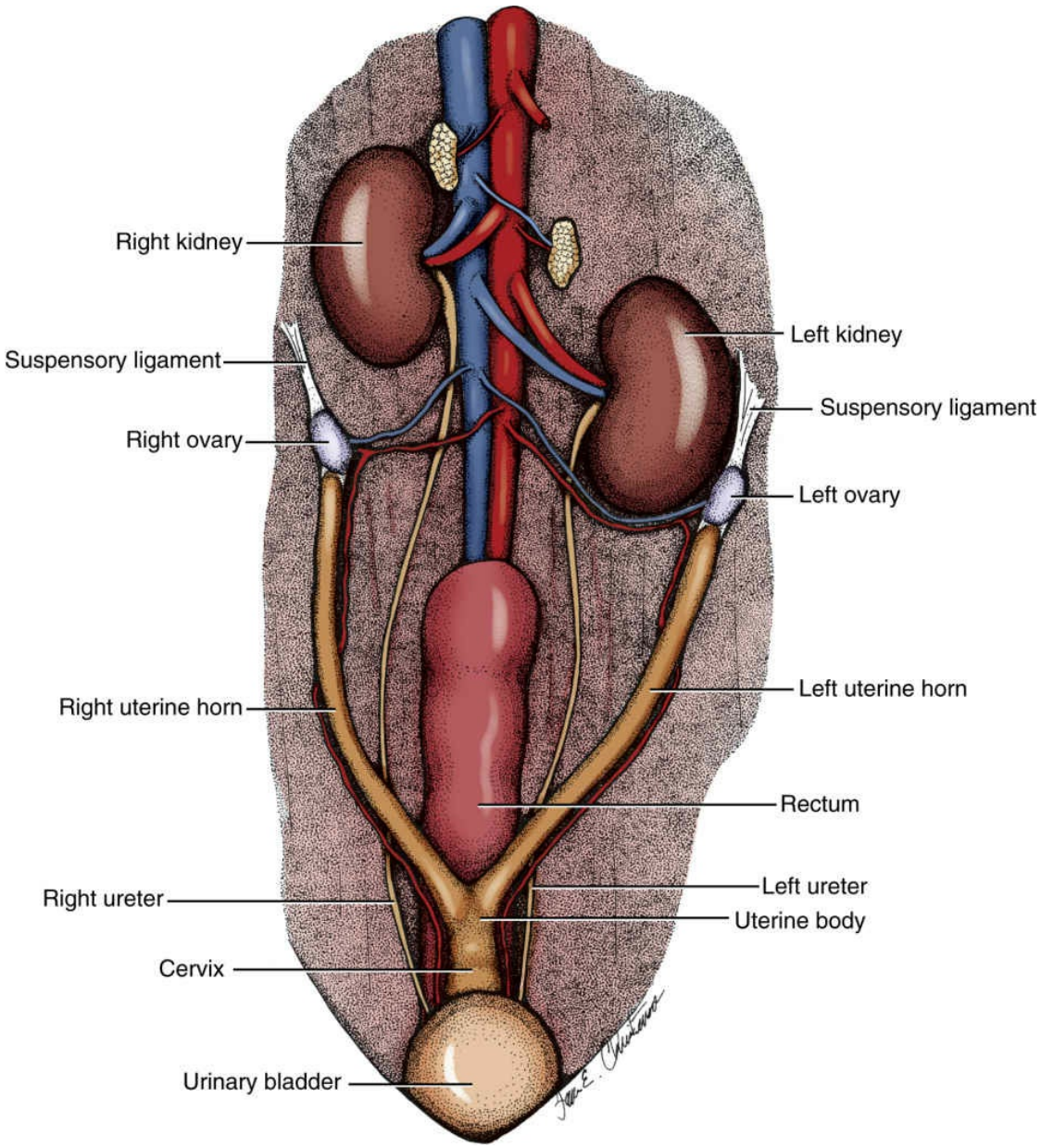

FIG. 9.2 Canine female urogenital tract (ventrodorsal view).

Proestrus

Proestrus [*pro-* for, before + *estrus*], as its name implies, is that phase of the reproductive cycle that precedes estrus. Most females will begin to exhibit behavioral changes during proestrus. What kind of behavioral changes? Well, they become more flirtatious. Some may flag their tails. Others become more vocal. Still others, like cats, may playfully roll around on the floor or ground (and vocalize—LOUDLY). It's hormones driving these behaviors. You see, the ovaries, in response to follicle-stimulating hormone (FSH) from the pituitary, secrete ***estrogens*** [*estr(o)-* estrus + *gen(o)-* producing].

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These *estrogens* alter both physical and behavioral characteristics of the female, preparing her for breeding. The ***endometrium*** [*endo-* within, inside + *metr(o)-* uterus + *-um* the] thickens and has an increased blood supply. This preparation of the ***endometrial*** [*endo-* inside, within + *metr(o)-* uterus + *-al* pertaining to] lining is necessary so that it is ready for implantation of fertilized eggs.

Estrus

Estrus [Gr. *oistros* anything that drives mad; vehement desire] is the phase of the reproductive cycle when the female is behaviorally and physically receptive for breeding. This is what people commonly refer to as being "in heat." These animals are "hot" with passion. Behaviorally, everything mentioned earlier becomes exaggerated. Cows may actually exhibit "mounting" behavior. Oh my goodness, the actual length of *estrus* is SO variable among animals. Most cows are in estrus for only about 12 hours. Many bitches are in estrus for between 1 and 3 days. It's one thing to miss estrus in a cow. She'll be in heat again in another month, able to breed. But if we miss our breeding window in a champion dog, it could be another 6 months to a year before she's in heat again. That wastes valuable time, money and other resources. Don't worry about missing estrus in a queen; she will literally TELL you when she's in heat.

Hormonally, by the time a female is in estrus, estrogen levels are peaking. This corresponds to her behavioral receptivity. It also corresponds to ovulation. Another hormonal shift occurs surrounding ovulation—follicle stimulating hormone (FSH) from the pituitary gland decreases while luteinizing [*lutein* from L. *luteus* yellow + *-izing* causing] hormone (LH) increases. By the time ovulation occurs, the LH level is actually peaking. Why? Because if the egg is fertilized, we will need a corpus luteum (literally, "a yellow body") in order to maintain the pregnancy. More on that later.

Metestrus

Metestrus [*met-, meta-* after, beyond + *estrus*] is typically a very brief transition from estrus to reproductive quiescence. Behaviorally, the females let any potential suitors know that they're beyond estrus. They're no longer playfully receptive to male suitors. In fact,

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females during this phase and beyond can become quite grumpy. Hormonal changes during metestrus depend on whether or not the female has been bred and conceived. We'll talk about hormones associated with pregnancy later.

Anestrus/Diestrus

Anestrus [*an-* without + *estrus*], sometimes called ***diestrus*** [*di-* two + *estrus*; i.e., the phase between two periods of estrus], is the period of reproductive quiescence. Following our previous examples, in the cow, *anestrus* lasts a little less than a month. Obviously in the dog, *diestrus* could last 6 months to a year, depending on the dog. And while we're on the subject, let's see how the ***vaginal*** [*vagin(o)-* vagina + *-al* pertaining to] epithelium in the dog corresponds to the phases of the reproductive cycle. Late in diestrus, hormonal changes (i.e., increasing levels of estrogens) will trigger changes in the epithelium.

Canine Vaginal Cytology

Vaginal ***cytology*** [*cyt(o)-* cell + *-logy* study of] can be very valuable, especially when it comes to a female dog who has a very short period of estrus. Yes, we need to pay attention to her behavior. That's helpful. And yes, we can check hormonal levels too. But vaginal cytology, in addition to her behavior, is a simple, noninvasive, and cost-effective means of figuring out her unique cycle and to determine the optimal time for breeding.

As you may recall from Chapter 6, we said that the urinary bladder was lined with stratified squamous epithelium. The same is true for the uterine and vaginal linings (Fig. 9.3). And that epithelium (along with other cellular populations) changes dramatically during certain phases of the reproductive cycle. While the combination of all of the cellular information can be very useful, it is important to focus our attention on the epithelium. If the other cellular populations, including white blood cells (WBCs), red blood cells (RBCs), and perhaps bacteria, provide supportive information to corroborate our conclusion, great. If not, so be it. It's really the epithelium that's the the star here. Everyone else is nothing more than a "supporting actor."

Before we talk about what we might see during each stage of the

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reproductive cycle, let's talk about the epithelium and its cellular as well as tissue structure. If you look at Fig. 9.3, you'll see that the tissue layers are arranged with the youngest cells at the bottom and the oldest cells at the surface. I love to use the analogy of chicken eggs, to help describe the appearance of the cells in each of the epithelial layers. I liken the yolk of the egg to the nucleus of the cell, and the white of the egg I liken to the cytoplasm of the cell. So for me, looking at ***parabasal*** [*para-* near + *bas(o)-* base + *-al* pertaining to] cells is kind of like looking at a hard-boiled chicken egg. If you look down on the end of a hard-boiled egg, it is relatively small and somewhat round. The egg yolk seems to take up a large amount of space in the egg. *Parabasal* cells are much like that. This is why we often call them "small rounds." And because they are so plump, the light from the microscope doesn't penetrate them very well. So, they appear rather dark.

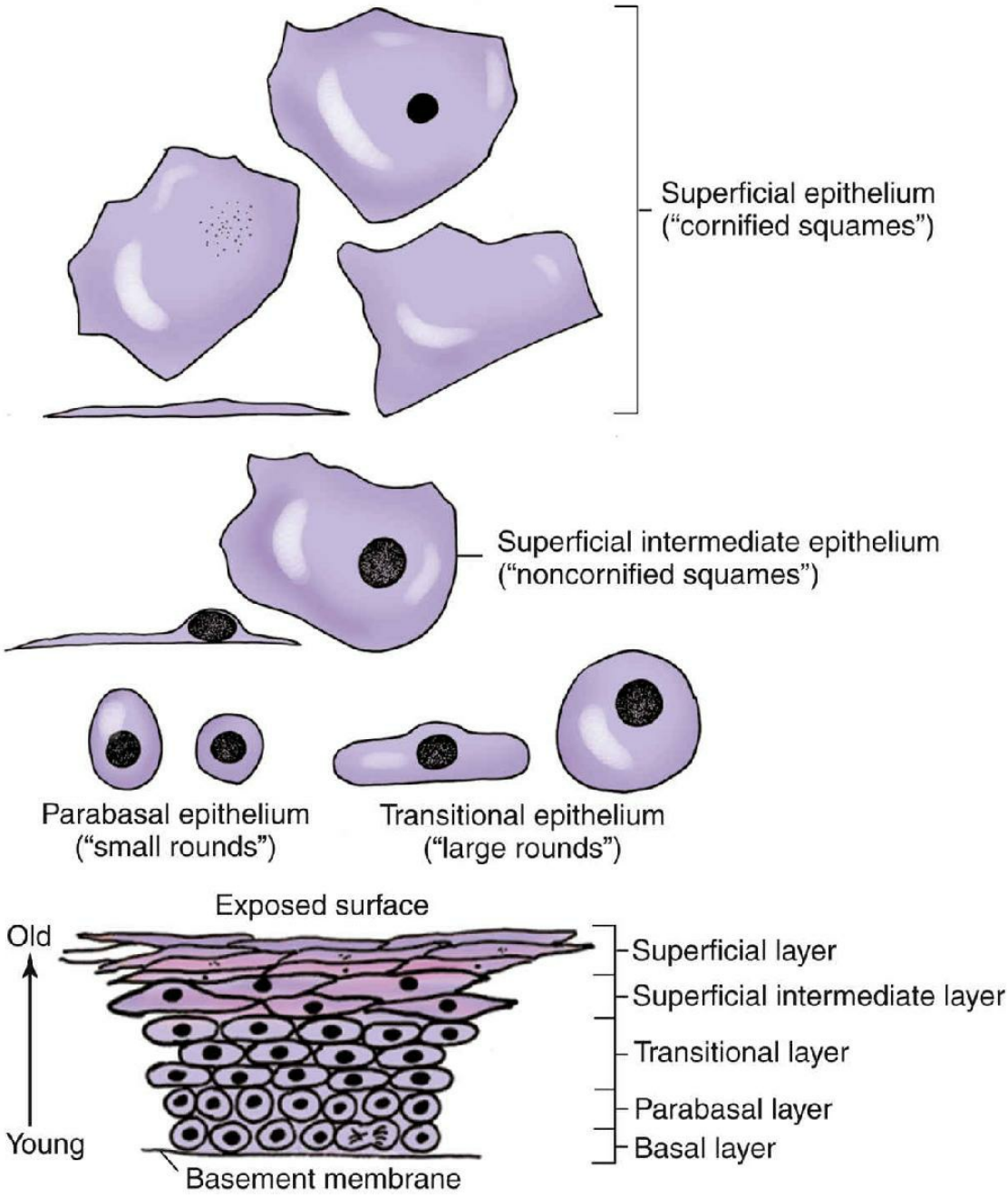

FIG. 9.3 Canine vaginal epithelium for cytology.

As these cells move up in the stratification, they gain a little more "elbow room," allowing them to spread out a bit. So, in the transitional layer, our "large rounds" might be likened to poached eggs. Transitional cells, like poached eggs, are still somewhat plump. But, because the cytoplasm can spread out a bit, they appear larger with more cytoplasm. As the cells progress even closer to the surface, they spread out even more, taking on the appearance of sunny-side-up eggs. These are the superficial intermediate epithelial cells or noncornified squames. Notice that

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they still have a plump nucleus, but the cytoplasm is spread out and very thin. The most superficial layer contains the superficial epithelium. (Go figure.) Superficial epithelial cells are also called "cornified squames." They are thin, flat, jagged, ragged cells. If a nucleus is present, it's all shriveled up. Most cornified squames have lost any discernable nucleus, making them resemble fried eggs with a broken yolk spread out all over. These ragged cornified squames are the most important, when it comes to determining estrus. Okay, so now that we know what the various layers of the epithelium should look like, let's talk about which ones will be seen in each phase of the estrous cycle.

Proestrus: During proestrus we expect to see a few WBCs that naturally inhabit the vaginal tract to keep it free of ***pathogens*** [*path(o)-* disease + *gen(o)-* producer], especially during anestrus. The presence of WBCs should diminish as we approach estrus. By the time estrus arrives, WBCs should be long gone. RBCs may also be seen during proestrus. It really depends on the individual dog, as to how much bleeding she'll actually experience. This is related to changes in the uterine lining in preparation for possible pregnancy. Most of the bleeding happens early in proestrus and tapers off as she approaches estrus. Both WBCs and RBCs are variable, but the epithelium tells the true story. At the beginning of proestrus, because she's transitioning out of diestrus, we expect to see many plump epithelial cells. These come from the *parabasal* and *transitional* layers. As she approaches estrus, the epithelium should transition away from small and large rounds (hard-boiled and poached eggs) to noncornified and cornified squames (sunny-side up and broken fried eggs). Remember, this is a continual progression. I would never estimate a dog's reproductive stage from one vaginal cytology swab. Multiple swabs are needed to see the progression of cells, especially the epithelial cells.

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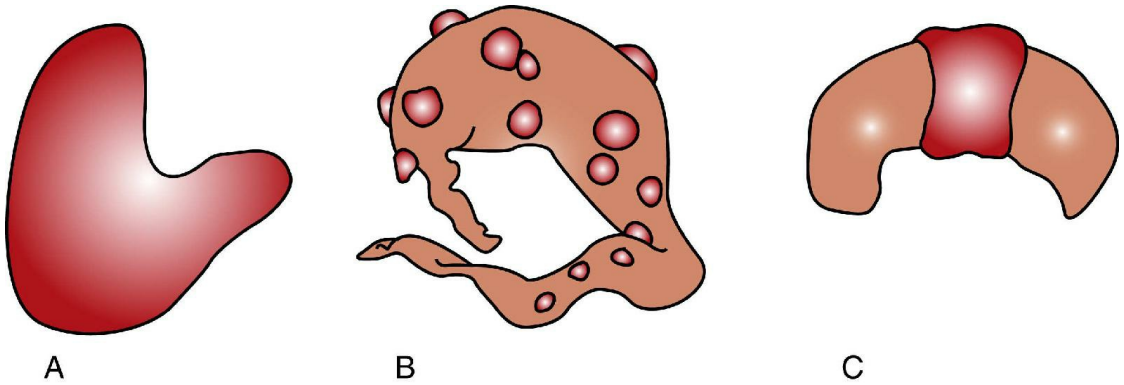

FIG. 9.4 Placental differences: (A) diffuse (horse and pig), (B) cotyledonary (ruminants), (C) zonary (dog and cat).
From Colville T, Bassert JM. *Clinical Anatomy and Physiology for Veterinary Technicians.* 2nd ed. St Louis: Mosby; 2008.

Estrus: When she is in estrus, we should see predominantly cornified squames. Most of these cells will be anuclear, with a ragged and jagged "shells" of their former selves. And these cornified cells almost always tend to clump together. We shouldn't see any WBCs. We don't want them there. They would view sperm as potential pathogens and try to destroy them. A few females may have small numbers of RBCs present when they are in estrus. Not knowing how long this gal will be in estrus, if we're seeing 95% to 100% of cornified squames, she should probably be bred today. If she continues to be receptive to the male, the breeder may want to repeat breeding over the next couple of days.

Metestrus: Metestrus is a very brief period of time. Often, when we do repeated cytology every other day or so, we miss metestrus. Classic ***metestrual*** [*metestr(o)-* metestrus + *-al* pertaining to] cells are epithelial cells with numerous vacuoles. These cells are rarely seen. Does it matter if we miss metestrus? No. Whether we swab her on the day of or on the day after metestrus, we are still going to see a shift in the cytology that points us away from estrus. Either way, if she has not been bred by now, it is simply too late. And whether she is in metestrus or early diestrus, we should see reduced numbers of cornified squames and increased numbers of noncornified and younger cells.

Diestrus: As one moves further away from estrus, younger and younger epithelium will be seen. These cells show up, in somewhat reverse order of the way they appeared leading up to estrus. So, we begin seeing more noncornified squames and transitional

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epithelium. When fully in diestrus, we expect to see an abundance of transitional and parabasal cells. WBCs show up in large numbers too. Not only do they have a lot of crusty cornified squames to clean up, but throughout diestrus, WBCs also need to be the first line of defense against pathogens.

Pregnancy

When the estrous cycle was discussed earlier, I mentioned hormonal changes surrounding ovulation. I said that by the time ovulation occurred, LH would be peaking, to sustain the corpus luteum. So what? Well, the corpus luteum secretes ***progesterone*** [*pro-* for + *gester(o)-* bearing + *-one* a hormone; i.e., the hormone that maintains pregnancy]. Progesterone is absolutely essential for ***gestation*** [*gest(o)-* bearing + *-tion* state of; i.e., pregnancy] It prepares the ***endometrium*** [*endo-* inside, within + *metr(o)-* uterus + *-um* the; i.e., the interior lining of the uterus] for implantation of the fertilized egg(s) and sustains pregnancy through to term.

A placenta develops at the implantation site. This is a highly vascular structure that is attached to the endometrium and provides for delivery of nutrient-rich blood to the embryo/fetus as well as elimination of wastes from the embryo/fetus. Umbilical veins and arteries connect the placenta to the growing fetus. It's interesting to note that the placenta actually grows with the growing fetus to keep up with demand. However, not all placentas are alike. Different species have different types of placentas (Fig. 9.4). Dogs and cats have ***zonary*** [*zon(o)-* zone + *-ary* pertaining to] placentas, shaped in belt-like bands. Ruminants (cattle, sheep, and goats) have ***cotyledonary*** (kot"uh-le'dun-ar-e [Gr. *kotylēdōn* seed-shaped + *-ary* pertaining to]) placentas, with multiple placental attachments. Horses and pigs have large, ***diffuse*** placental attachments.

There are also two fluid-filled connective tissue sacs that surround the developing fetus and are connected to the placenta. The ***amniotic*** [*amni(o)-* Gr. *amnion* "bowl" + *-tic* pertaining to] ***sac*** directly surrounds the fetus and is filled with *amniotic fluid*. Surrounding the amniotic sac is the ***allantoic*** [*allant(o)-* Gr. *allantos* "sausage" + *-ic* pertaining to] ***sac***. It is also filled with fluid;

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however, this fluid also contains urinary waste from the fetus. You see, there is something called the ***urachus*** (u'ra-kus [Gr. *ourachos* "tail"]), which is a tubular structure running through the umbilical cord that connects the cranial border of the bladder to the allantoic sac. Shortly after birth, the urachal opening from the bladder should close. If it remains open (patent; i.e., patent urachus), the newborn will dribble urine from its umbilicus.

How long does gestation last? Well, that depends on the species. Table 9.1 provides approximate gestation periods for most domestic animal species. Please note that these are truly approximate times. I know very few mothers who have been on time every time, with each pregnancy. There is always variability. So, these are rough approximations. For instance, the average gestation period for a dog is 63 days, but it may be a little more or a little less. So, I've approximated by saying 2 months. I've also included the average number of young produced from each pregnancy. Here again, these are averages. So, while a cow will average one calf per pregnancy, she may also have twins. Goats, on the other hand frequently have twins. And those litters of 8 to 10 or 10 to 12 that I've listed? Well, let's just say that there are remarkable litters that fall well outside these averages. You know, one or two pups for a Chihuahua might be just right, especially if she's a petite female; however, at the other end of the spectrum, I know of one Cardigan Welsh Corgi bitch that actually gave birth to a litter of 15 puppies! That poor girl must have been dragging bottom by the end of her pregnancy. By the way, all of those pups survived, as did mom. Can you imagine poor mamma being mauled by all those hungry mouths?! I'll bet she weaned them really fast.

TABLE 9.1

Approximate Gestation Periods and Offspring Numbers

Species	Approximate Gestation Period	Average Number of Young
Cat	2 months	8 to 10 kittens
Cattle	9 months	1 calf
Dog	2 months	8 to 10 puppies
Goat	5 months	2 kids
Horse	11 months	1 foal

Pig	4 months	10 to 12 piglets
Sheep	5 months	1 to 2 lambs

Birth

Well, after mentioning that large litter of puppies, this seems like an opportune time to discuss ***parturition*** (pahr"tu-ri'shun [L. *parturitio* "childbirth"]). By the way, there is wide variation in common names for parturition: in dogs, it's commonly called whelping; queening in cats; foaling in horses; calving in cattle; kidding in goats; lambing in sheep; and farrowing in pigs. Regardless of what we call it, when the pregnancy is at full-term, hormones will shift. As you'll learn in Chapter 10, hormones are always a moving target. Typically near the end of gestation, ***preparturient*** [*pre-* before + *parturi(o)-* birth + *-ent* pertaining to] progesterone levels will drop. Perhaps this is what stimulates changes in the dam's behavior. Many dams (mothers) will begin to exhibit "nesting" behaviors, sometimes even a week or more prior to parturition. As birth becomes imminent, most females will become quite restless. This is probably related to effects of the hormone ***oxytocin*** [*oxy(o)-* quick + *toc(o)-* birth + *-in* a, the]. As will be discussed in Chapter 10, oxytocin is secreted by posterior pituitary gland. It has a two-fold effect. First, it promotes "milk letdown." More on that later. But before we worry about milk, we need youngsters to suckle that milk. So, most important right now is oxytocin's other effect. Oxytocin stimulates muscular contractions of the uterus, to initiate the birthing process and keep it going. It literally promotes "quick birth" by stimulating powerful contractions. Whether the dam has one or multiple offspring, those rhythmic uterine contractions progressively move the youngsters toward the end of the uterine body. Simultaneously, the cervix is relaxing. By the time the first (or only) youngster approaches the cervix, it is fully relaxed to permit passage into the rest of the "birth canal" (i.e., the vagina). By now, the contractions are reaching their "crescendo," becoming extremely intense, forcing the ***neonate*** [*ne(o)-* new + *nat(o)-* born] out into the cold environment. Compared to the mother's body temperature (avg. 99°F to 101°F), the environment is cold, especially when you consider that the babe is soaking wet from the

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womb. And for those lambs born in temperate regions in January and February, it can be *really* cold! Boy, that'll make you gasp and take your first breath!

Generally, parturition goes smoothly. The "presentation" of young is usually front feet and head first, while lying in sternal recumbency. Yep, that means if mom is standing, the neonate lands on its head. But it's only a huge drop (about 6 feet) for a giraffe. Immediately ***postpartum*** [*post-* after + *partum* birth] the mother will begin licking the youngster to remove the amniotic and allantoic sacs, to stimulate breathing and activity in the newborn, chew through the umbilical cord, and begin drying the youngster off. If there are more to be born, she'll repeat the process multiple times. After each neonate, the "afterbirth" must be passed (i.e., the placenta). It is extremely important to monitor, to make sure each placenta is passed. Retained placentas can lead to ***metritis*** [*metr(o)-* uterus + *-itis* inflammation of], and even serious systemic illness for the dam. But we need to watch closely, because many times the dam will eat the placenta. Yeah, that's sounds pretty gross. But it's important, especially for animals born outside the safety of a barn or home. Eating the afterbirth out on pasture "disposes" of the evidence. This is important where predators are concerned.

Since we're talking about parturition, we need to discuss the possibility of ***dystocia*** [*dys-* difficult + *toc(o)-* birth + *-ia* a condition or process of]. There are a number of factors that may result in *dystocia*. First, the presentation of the youngster may be poor, for example, a breech (butt first with the rear legs folded up toward the head), or head first with either the head or the front legs folded caudally. Either way, this increases the diameter of the youngster's body and makes it less flexible. So, it may become wedged and unable to pass over the floor of the pelvis. Sometimes the fetus is simply huge. Here again, it is simply too big to effectively pass through the birth canal, especially over the pelvis. And sometimes, for a variety of reasons, the uterine muscles fatigue and cannot force the young out. In this last circumstance, we can actually give an injection of oxytocin to the dam to promote stronger contractions. Of course, we need to first ensure that no one is stuck in the birth canal. If someone is stuck, we need to act quickly. The longer he or she is wedged in the birth canal, the more likely it will

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become oxygen deprived and not survive. So, we either need to try to rapidly reposition a malpositioned [*mal-* bad, badly] fetus or take the young via a C-section.

In some animals that are prone to dystocia (like English Bulldogs), C-sections are frequently scheduled to avoid dystocia and all of its risks for the dam and young, as well as avoid the emergency C-section in the middle of the night. If a C-section is performed, ***neonatal*** [*ne(o)-* new + *nat(o)-* born + *-al* pertaining to] care falls to us, until the dam has recovered from surgery and is able to safely suckle and care for the neonates herself. Until she is able, we are responsible as each neonate is taken from the uterus to clear its airway, stimulate breathing, dry it off, provide umbilical care, stimulate urination and defecation, and keep *all* of them warm. This is a big job, especially with a large litter. Ideally, we'll have numerous personnel present to assist with the neonates, during the C-section. This is why we prefer to schedule C-sections, if at all possible. As soon as the dam is ready, we need to make sure the neonates nurse.

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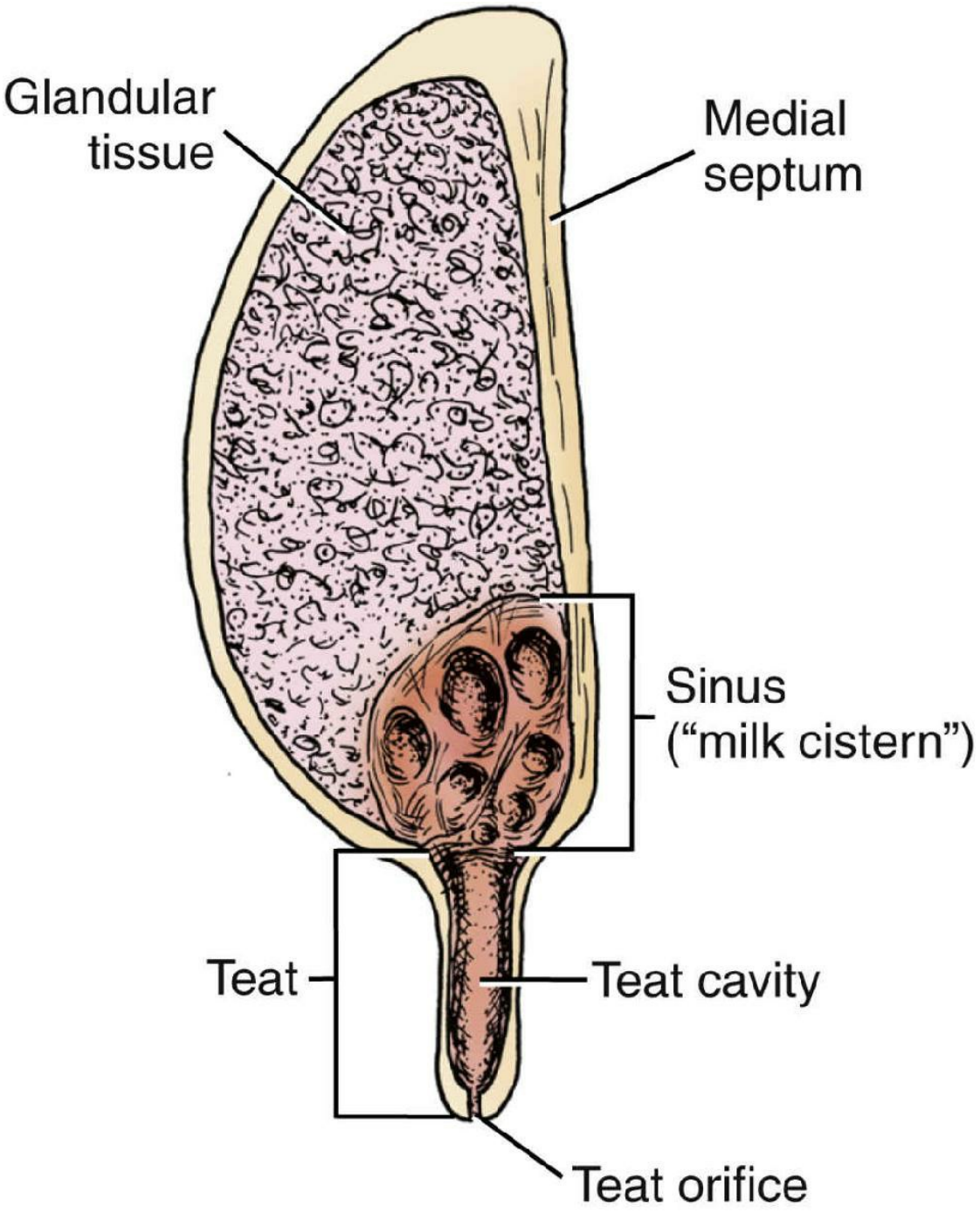

FIG. 9.5 Bovine udder: individual quarter.

Mammary Glands and Lactation

As I said earlier, preparturient hormonal changes promote the birthing process and changes in the ***mammary*** [*mamm(o)-* breast + *-ary* pertaining to] glands. ***Prolactin*** [*pro-* for + *lact(o)-* milk + *-in* the; i.e., the "for" milk hormone], secreted by the anterior pituitary gland, promotes ***lactation*** [*lact(o)-* milk + -tion state of; i.e., milk

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production]. Prolactin will continue to promote milk production throughout the lactation period. Previously, I mentioned that oxytocin promotes "***milk letdown.***" You might want to refer to the bovine quarter in Fig. 9.5 for this discussion. Why is it a quarter? Well, cows have four separate mammary glands making up their udders. Other animal's mammary glands are merely variations on this theme. Mares and ewes have only two. Those animals that tend to have litters of offspring have "chains" of mammary glands, strung along the ventral abdomen and chest. This provides an individual teat and gland for each neonate. Most neonates in a litter "claim" one favorite teat as theirs and theirs–sort of like reserved seating at a fine-dining restaurant–for every meal when they nurse. Because the cow's udder is so large and well defined structurally, we'll use it to show and explain how mammary glands work. You'll simply have to "down-size" this structure in your mind when you think about mammary glands of dogs and cats (or mice!).

So, *prolactin* stimulates milk production in the glandular tissue. That milk is then stored well up in the glandular tissue and small ducts. In the presence of oxytocin, the tiny muscles surrounding the little ducts in the glandular tissue contract, forcing the milk down into the milk cistern (chamber) and teat cavity. This squeezing milk down into the cistern and teat makes the milk readily available for the first neonate's first meal. For subsequent meals or milking (in the case of dairy cows or goats), stimulation at the base of the teat by the neonate or by us preparing the udder for milking promotes secretion of oxytocin and then milk letdown. Continued stimulation, by the youngster, us, or the milking machine, promotes further oxytocin release and milk letdown. When stimulation stops, so does secretion of oxytocin and ultimately milk letdown.

Remember, if you're trying to collect a milk sample for cow-side or laboratory testing, milk letdown in response to oxytocin is a temporary effect. If we fail to collect the milk sample quickly enough after stimulating milk letdown, we will miss the window of opportunity when milk is readily available in the milk cistern and teat cavity. I have seen many students, when learning how to collect milk samples, take way too long cleaning and stimulating the udder and then become frustrated by their inability to collect milk from the cow. Calves (and all other mammalian offspring) don't nurse by

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starting and stopping multiple times. They don't pause to contemplate their meal. They simply latch onto the teat, bumping and pressing on the mammary gland to stimulate milk letdown, and then continue to suckle until they are full. If they snooze, they lose; this is especially true for littermates. Failure to hold on and eat all they can in the moment opens the door for a littermate to push them out of the way and eat more than their fair share. So, the next time you need to collect a milk sample from a cow, do it like your life depends on it. Be the calf. Once you get started, don't stop until you've got what you need.

Did you know that there are different types of milk produced by the mother at different times? No, I'm not talking about chocolate versus plain or vanilla. I'm talking about the very first milk produced. That first milk is different from the rest, because it is incredibly rich and it contains numerous maternal antibodies. Those maternal antibodies in the ***colostrum*** (kuh-los'trum [L. *colostrum* "beastlings"]) are very important to provide passive immunity (protection) for the neonates against infectious diseases. This is the only immunity those youngsters have, until their own immune systems begin to develop. And that takes a while. However, those antibodies can only be absorbed by the neonates for a very brief period of time—within the first 12 hours of the ***postparturient*** [*post-* after + *parturi(o)-* birth + *-ent* pertaining to; cf. postpartum] period. The neonates may still be able to absorb *some* antibodies between 12 to 24 hours postpartum. But absorption rapidly decreases beyond 12 hours. Failure to receive colostrum during this critical postparturient period leaves the young completely unprotected and at grave risk of dying from infectious disease. This is why many breeders and farmers store frozen colostrum. If the dam doesn't produce milk or something happens that prevents her from nursing her young immediately postpartum, the frozen colostrum can be thawed, warmed, and bottle fed to the neonates. It's that important.

Mastitis

Since we're on the topic of lactation, this seems like a good time to talk about ***mastitis*** [*mast(o)-* breast + *-itis* inflammation of]. This can afflict any mother. Sometimes, structure of the mammary gland and

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teats predispose the dam to *mastitis*. For example, a dairy cow with a poor suspensory apparatus may be predisposed. A strong suspensory apparatus should suspend the cow's udder tight to the abdomen, making it appear ... well ... "perky." When looking at the udder from the rear, the teats of the rear quarters should sit above the cow's hocks. In cows with poor udder conformation, often the rear quarters sag below the level of the hocks, for example. This is often due to aging (we all tend to sag here and there the older we get). Unfortunately for dairy cows, a sagging udder makes it more likely that the udder will be injured (e.g., stepped on or kicked) or contaminated with things like manure. Much of the time, mastitis is a husbandry issue, especially with regard to milking procedures (for dairy animals), housing, sanitation, and hygiene. Any lactating animal needs clean, dry bedding. Yes, this can be challenging in a barn or a whelping box. But failure to remove bedding that is soiled with excrement (urine and feces) provides a breeding ground for bacteria. Milk provides an even better medium for bacterial growth. This is why you should never ever squirt or "strip" milk from an udder into the bedding. You'll set up the "perfect storm" for mastitis to develop.

In the dairy industry, mastitis can deliver a devastating economic blow to already-struggling farmers. Not only will mastitis reduce milk production, but the milk from those cows must be discarded. This is just the farmer's side of it. For the cow (or anyone for that matter) with mastitis, it is a very painful condition. It can make her systemically ill. And if it is too costly and time-consuming to treat, she may be culled (killed) from the herd. Any time we, as veterinary professionals, are evaluating or collecting samples from dairy animals, we need to take care that we don't contaminate their teats and udders. Even the normal bacteria present on our hands could serve to contaminate the teat orifice (opening), precipitating mastitis. So, the udder, teats, and our hands should be appropriately cleansed. Ideally, we should wear disposable examination gloves. And any suspicious or known mastitis quarters should be handled last for evaluation and sample collection, to avoid cross-contamination. Following collection, it is wise to apply a teat dip to coat, seal, and provide ***antibacterial*** [*anti-* against + *bacter(o)-* bacteria + *-al* pertaining to] protection for the teats. Really,

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our care and handling of any lactating dam should be done with the utmost care. Prevention is key.

Over the years, I've seen numerous animals with mastitis. For the individual suffering with this painful condition, it is not fun at all. It can take a long time to treat. I've seen some ***mastitic*** [*mast(o)-* breast + *-itic* pertaining to inflammation] animals with mammary glands so diseased that the infection literally ruptured through the skin. Ouch! For those still nursing their young, we need to remember that mastitis doesn't simply affect the dam; it affects the young too. They may be receiving inadequate nutrition, due to reduced milk production and/or the mother not permitting the young to nurse, due to pain. It may be necessary to wean the youngsters early. For those too young to wean, they will need to be cared for as if orphans. Someone will need to feed milk replacer (i.e., "formula") and tend to all of the care that the mother would routinely do. The young need to be separated from the mother and kept warm by artificial means (with inherent hazards). She needs to "dry off." Chronic [*chron(o)-* time + *-ic* pertaining to; i.e., long-term] stimulation by the young will simply promote continued lactation and that's counter-productive for treatment of the mastitis. If the dam is very painful, she may harm the young when they attempt to nurse. And if they are able to suckle some of the mastitic milk, the pathogens may make them ill, potentially costing them their lives. So, the dam and her young need to be separated. This is all very stressful for everyone. Suffice it to say that mastitis is a big deal. It is treatable. But the best treatment is prevention. As the old saying goes, "An ounce of prevention is worth a pound of cure."

Hypocalcemia, Eclampsia, and "Milk Fever"

Isn't it amazing how often electrolytes, like calcium, seem to keep resurfacing, chapter after chapter and body system after body system? I mean, my gosh, we've talked about electrolytes in Chapters 2, 3, 4, 5, 6, and 7, to name a few. They'll be discussed again in Chapters 10 and 11. Do you get the idea that electrolytes are rather important? Well, they are! Calcium is one of several key electrolytes in the body. It is necessary for the clotting of blood, muscular contraction, strong bones, neuronal transmission, and much more. We and our animal kin cannot survive without it. Each

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time calcium is discussed in this text, it is addressed from the perspective of the given chapter. It is up to the student to integrate all of that information, not for the sake of pure knowledge, but for the sake of your patients.

In the context of this chapter, our focus is reproduction. We just finished discussing lactation. Let's see, what is the key electrolyte needed for lactation? Yep, it's calcium! Now, without getting into all of the details of hormonal controls and specific functions of calcium in other body systems, let's think about how lactation can affect calcium in the body. You see, in order to produce milk, the mammary glands need calcium. From where do the mammary glands get calcium? That's right, the bloodstream. And if we think about the ***periparturient*** [*peri-* around + *parturi(o)-* birth + *-ent* pertaining to; i.e., the time immediately surrounding birth] period, when milk is suddenly being produced, we can deplete blood calcium levels pretty rapidly. In fact, ***hypocalcemia*** [*hypo-* low + *calc(o)-* calcium + *-emia* a blood condition of] can develop so rapidly that the body's ***homeostatic*** [*home(o)-* unchanged + *-static* pertaining to standing; i.e., equilibrium] mechanisms don't have time to correct the imbalance.

In other circumstances, if we progressively begin running a bit low on blood calcium levels, we can begin to take withdrawals from our calcium "savings" that's "banked" (stored) away in bones. Away in bones. But that withdrawal process takes time. There's a little more readily available source of calcium in muscle tissue. Unfortunately, if we rob muscles of calcium, they can't contract as well and eventually not at all. This is why hypocalcemia or "***milk fever***," as it is commonly called in cows, usually presents as a cow suddenly unable to get up (i.e., a "downer cow"). But this cow is not a "downer." She's down because her muscles can't contract to get her up and keep her up. Her mammary glands have successfully depleted needed calcium from the blood and muscles. Now, what may have been missed before she went down are all of the muscle twitches that usually accompany a developing calcium deficiency. Muscle twitching, tremors, and weakness tend to be noticed more in dogs and cats suffering from lactation-related hypocalcemia, commonly called ***eclampsia*** (e-klamp'se-uh). Why the twitching? It's related to nerve function. You see calcium also

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controls the gates for ionic exchange in nerve transmission. With too much calcium, nerve transmission is suppressed, because the gates are held tightly closed. With too little calcium, the gates fly wide open, resulting in excessive and wild nerve transmission. But this can become much more serious than just muscle twitching and tremors. Because nerves in the brain can also be affected by the hypocalcemia, the animal can experience full-blown seizures. That alone could kill the new mother. And looming silently beneath the surface of any of these ***hypocalcemic*** [*hypo-* low + *calc(o)-* calcium + *em(o)-, hem(o)-* blood + *-ic* pertaining to] mothers is abnormal heart activity. Both the electrical activity of the heart and the heart muscle itself are adversely affected. Suffice it to say that hypocalcemia in any lactating female can easily be life-threatening.

How do we treat it? We need to give ***intravenous*** [*intra-* within + *ven(o)-* vein + *-ous* pertaining to; i.e., IV] calcium. This has its own risks. Infusing calcium too rapidly can result in a life-threatening drop in blood pressure or even ***cardiac*** [*cardi(o)-* heart + *-ac* pertaining to] arrest. We have to intervene with calcium; her life depends on it. Yet, we need to be sure that we monitor her very, very closely during the infusion. At the first signs of irregular heart rhythms or decreased blood pressure, we should slow the infusion before she develops deadly consequences. Failure to monitor closely and respond accordingly during a calcium infusion is a death-sentence.

Pseudocyesis

Pseudocyesis [*pseud(o)-* false + *-cyesis* pregnancy] is a relatively common condition, especially in dogs. What?! How can a dog have a false pregnancy? Either she's pregnant or she's not. Well, yes and no. As you may recall, when we discussed the estrous cycle, we said that around the time of ovulation luteinizing hormone (LH) levels peak and promote corpus luteum development. The corpus luteum secretes progesterone for maintenance of the pregnancy. If she successfully breeds and conceives, the corpus luteum does just what it's supposed to do—maintains the pregnancy. If she is not bred or does not conceive, the corpus luteum should shrivel up and disappear. For whatever reason, in some unbred or unsuccessfully

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bred females, the corpus luteum remains. The progesterone that it produces creates the same effects, as though she's pregnant. In fact, not only will she look pregnant, including mammary development, she'll act pregnant. She'll even go through nesting behaviors. I've known many dogs who, in the absence of offspring, gathered stuffed toys to nurture. I've also known of a few dogs who snatched someone else's puppies or kittens to nurture. Most of the time, pseudocyesis resolves on its own, without intervention. Sometimes hormonal therapy is required.

No harm done, right? For the most part, that's true. However, because of the abnormal physiologic effects of pseudocyesis, the cervix in these dogs may be more relaxed. That, coupled with the warm, moist endometrial lining, could leave her at risk of developing *metritis* or ***pyometra*** [*py(o)-* pus + *metra* uterus; i.e., a uterine infection]. Thus, while the behavioral changes of pseudocyesis may be cute and endearing, owners need to watch these ladies very closely. As we'll learn in a moment, conditions like pyometra can be deadly.

Pyometra

Obviously, pseudocyesis develops shortly after the female is "in heat" (estrus). That places her in diestrus. So, if pyometra is a possible consequence of pseudocyesis, it too must develop shortly after she's in heat, right? Right. It is important to note that pyometra also occurs in females who do not experience pseudocyesis. Regardless, in most cases of pyometra, the dogs will typically be presented for medical care within a month of their last heat. If we're lucky, she'll have an "open" pyometra. This means the cervix remains open, allowing the foul-smelling pus to be discharged through the vulva. Owners quickly see and smell it, prompting them to quickly seek medical attention. "Closed" pyometras, on the other hand, are silent killers.

Owners of dogs with closed pyometras are completely unaware of the serious condition developing in her. In these cases, the cervix remains tightly closed, trapping the infection. As the infection rages, the uterus fills with more and more pus. The uterine wall itself may become friable (i.e., very fragile). It can easily burst,

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resulting in ***peritonitis*** [*periton(o)-* peritoneum + *-itis* inflammation of; i.e., inflammation/infection of the abdominal cavity]. If she wasn't already ***septic*** [*sept(o)-* decay, infection + *-ic* pertaining to; i.e., systemic infection] with the closed pyometra, she will be with the secondary bacterial *peritonitis*. These girls are gravely ill. I have seen far too many dogs with closed pyometra who have ***acutely*** (suddenly) collapsed and are near death when presented in the emergency room. Emergency surgery to remove the infected uterus is absolutely necessary; but it is not without profound risk. With ***sepsis*** [L. *sepsis* decay; i.e., condition of systemic infection], these dogs have significant electrolyte disturbances and dehydration. They often have significant ***hypoglycemia*** [*hypo-* low + *glyc(o)-* glucose, sugar + *-emia* a blood condition of], ***hypoproteinemia*** [*hypo-* low + *protein* + *-emia* a blood condition of], and ***hypotension*** [*hypo-* low + *tens(o)-* pressure + *-ion* state of; i.e., low blood pressure]. And *IF* they can survive the anesthesia and surgery, they are in critical condition and at risk of dying for days after surgery.

Do you see why, when we discussed pseudocyesis, I said that those dogs need to be watched closely? In fact, any unbred dog should be watched very closely after being in heat. Remember, pyometra can develop without pseudocyesis. Yet, pyometra almost always develops soon after estrus, usually within a month after. So, any subtle changes following her heat cycle, like decreased activity, decreased appetite, ***polydipsia*** [*poly-* much + *dips(o)-* thirst + *-ia* condition of; i.e., increased thirst], and ***polyuria*** [*poly-* much + *ur(o)-* urine + *-ia* condition of; i.e., increased urine volume], should prompt a veterinary visit. Waiting a little too long can put her life in jeopardy.

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Male

In the previous section about females, we learned that eggs released during ovulation contain only half of the needed DNA to produce offspring. Males provide the other half, completing the chromosomal puzzle for producing new living beings. Refer to Figs. 9.6 and 9.7 for basic urogenital anatomy of the dog and cat. For most males among domestic animals, their basic urogenital arrangement is very similar to the dog. Male cats are quite unique, with the penis positioned caudally. Also unique in the male cat is the location of the scrotum and testes, found between the anus and prepuce, rather than in the groin. We'll touch on these and other unique male features, as we move along through this section.

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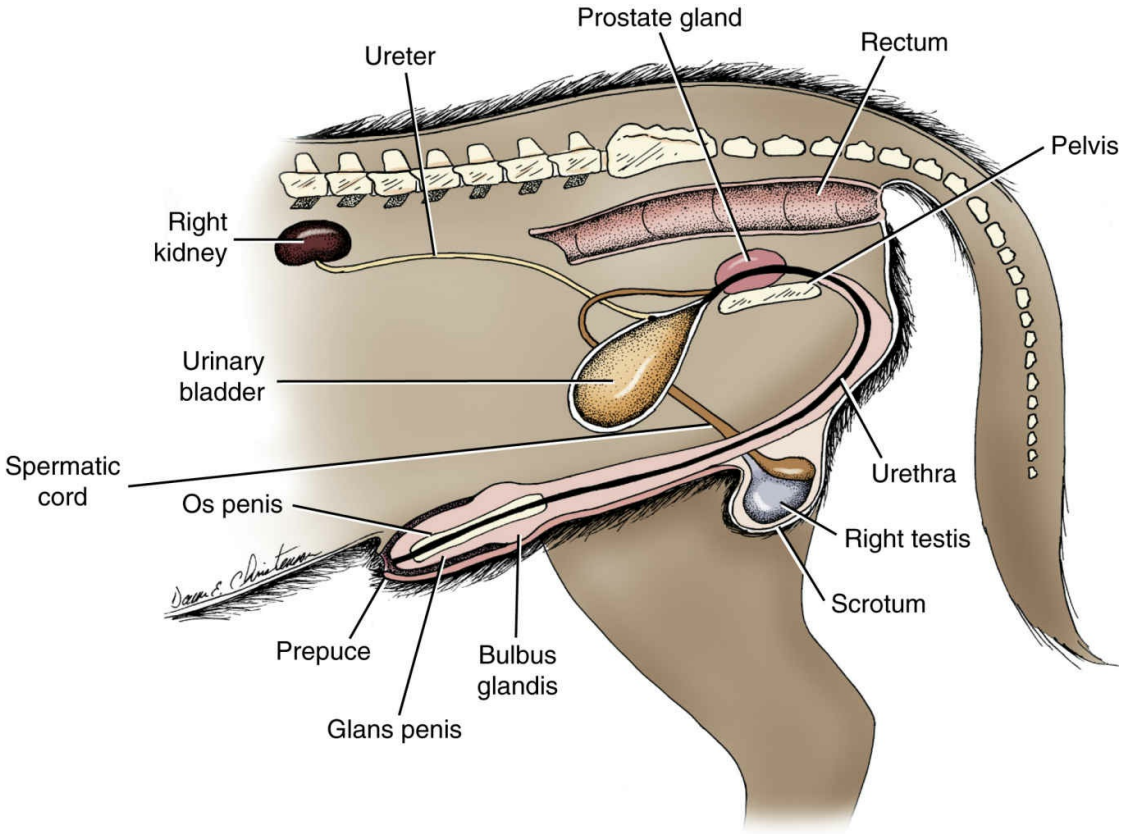

FIG. 9.6 Canine male urogenital tract (lateral view).

Testicles

The ***testes*** (tes'tēz) or ***testicles*** (tes'tĭ-kulz) are the male counterparts to the female's ovaries. The testes (see Figs. 9.6 and 9.7) of an adult male are found in the ***scrotum*** (skro'tum [L. *scrotum* "bag"]). They don't begin there. For most male animals, the testes begin in the abdomen, very near to where we expect to find ovaries in females. As the young males grow, the testes descend from the abdomen, through the ***inguinal*** [*inguin(o)-* groin + *-al* pertaining to] ***ring*** into the ***scrotal*** [*scrot(o)-* scrotum + *-al* pertaining to] ***sac***. Further development of the testicles takes place in the scrotum, until the male is fully mature. If one or both testicles fail to descend into the scrotum, his condition is called unilateral or bilateral ***cryptorchidism*** [*crypt(o)-* hidden + *orchid(o)-* testes + *-ism* state of] respectively. We'll talk about this again later. The testes of most male animals are located in groin, between the rear legs, as shown for the dog in Fig. 9.6. The cat is unique, with its testes located between the anus and prepuce (see Fig. 9.7).

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Why are the testicles outside the body in the scrotum? Well, this helps maintain the testes at an optimal temperature. Normal core body temperature is simply a bit too warm for the testes. So, in the scrotal sac, they are kept just a little cooler than the rest of the body. What about marked fluctuations in environmental temperatures? Well, there is a ***cremaster*** (kre-mas'ter [Gr. *kremasthai* to suspend]) ***muscle*** attached to the scrotum near each testicle. In really warm weather, the *cremaster muscles* relax, allowing the testicles to rest further away from the body, keeping them cooler. In really cold weather, the *cremaster muscles* contract, holding the testicles closer to the body to keep them warmer. In some species, like rats, the cremaster muscles are extremely powerful and can retract the testicles into the abdomen. Male rats have been known to do this even when frightened. So, if you ever have to do a reproductive exam on a male rat, don't frighten him!

The testes are responsible for both secretion of ***testosterone*** [*test(o)-* testes + *sterone* a steroid; i.e., hormone] and production of ***sperm***. Hormones will be discussed in Chapter 10 in greater detail. For our purposes here, we need to understand that *testosterone* is an ***androgenic*** [*andr(o)-* male + *gen(o)-* producing + *-ic* pertaining to] hormone. It is produced by cells that lie between the tubules in the testicle. ***Androgens*** [*andr(o)-* male + *gen(o)-* producer] literally produce what are perceived to be male characteristics, like greater body hair (think of the mane on a male lion), heavier muscling, and more massive bones. We frequently hear of "doping" with steroids, among athletes (human and performance animals, like racehorses). Those allegations surround the inappropriate use of ***exogenous*** [*ex-* out, outside + *gen(o)-* produced + *-ous* pertaining to; i.e., those produced outside the body] ***androgenous*** [*andr(o)-* male + *gen(o)-* produce + *-ous* pertaining to] steroids, like testosterone. Obviously, if testosterone promotes greater muscle development, strength, and stamina, giving *exogenous* testosterone to someone gives that individual an unfair advantage in competition. From a purely reproductive perspective, testosterone gives males their sexual drive. It can also promote such a competitive sexual drive that it sometimes leads to ***intermale*** [*inter-* between + *male*] aggression, to earn the right to breed.

If you look at Fig. 9.8, you can see an individual testicle in cross

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section. As you can see, it is subdivided into the main, ovoid portion of the testicle where sperm are produced and the ***epididymis*** [*epi-* on, upon + *didymis* from Gr. *didymos* testis]. The epididymis is a rather flattened, linear structure. The ***head of the epididymis*** is a bit thicker and more prominent near the proximal border of the testicle. The ***tail of the epididymis*** is near the distal border of the testicle. The ***body of the epididymis*** lies between the head and the tail. You'll notice in Fig. 9.8 that both the epididymis and main testicle are loaded with squiggly tubules. Sperm are produced in the ***seminiferous*** [*semin(o)-* semen, seed + *-ferous* pertaining to producing] ***tubules*** of the testicle. (More on that in a moment.) The tubules of the epididymis provide a place for storage and maturation of sperm. Sperm enter at the head of the epididymis. Eventually, they move toward the tail. From there, they will eventually make their exit through the ***vas*** (vahz [L. "vessel"]) ***deferens*** (def'er-enz [L. "carrying away"]). The *vas deferens* are also called the ***ductus*** (duk'tus [L. "duct," "passageway"]) ***deferens***.

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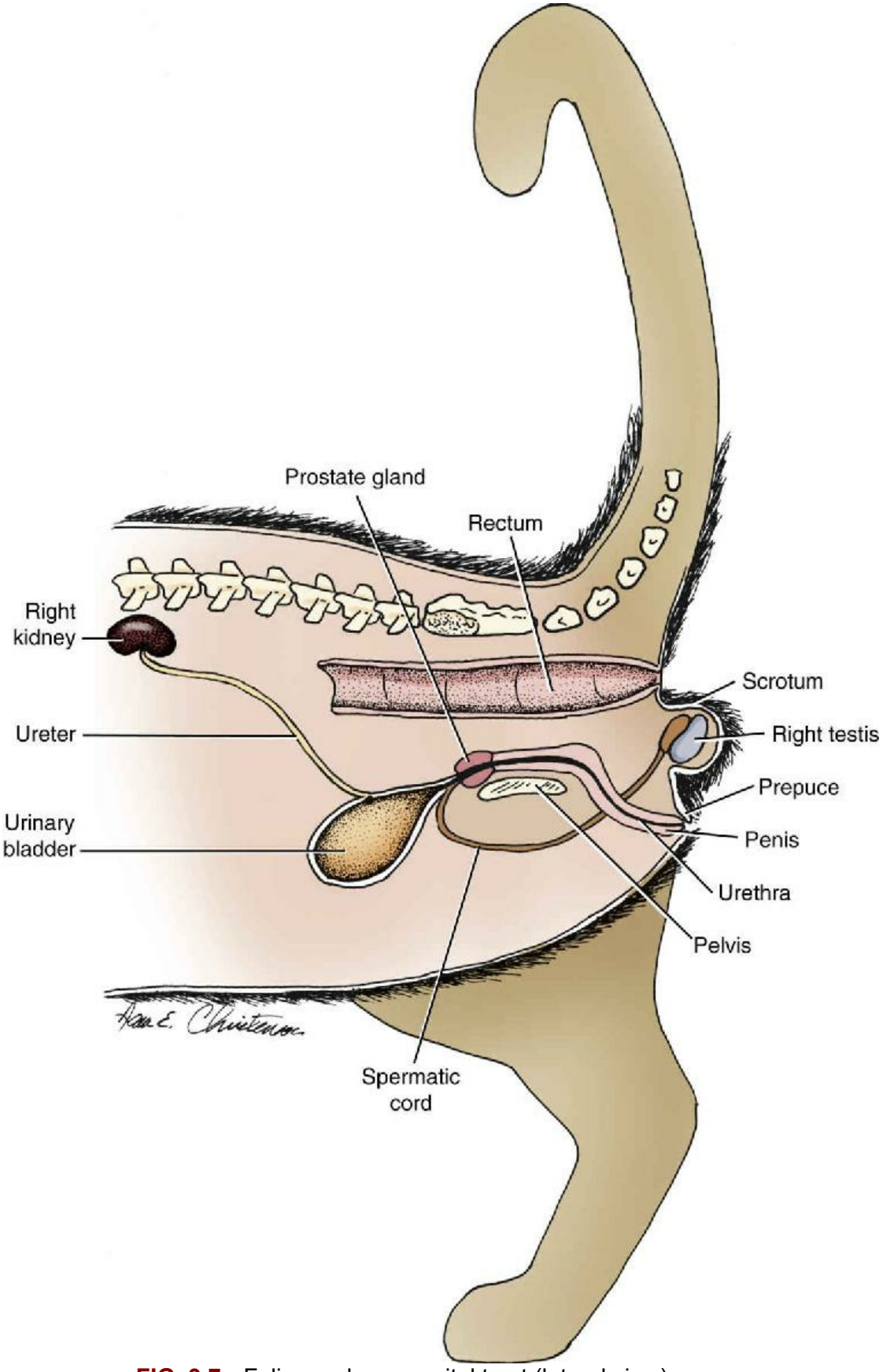

FIG. 9.7 Feline male urogenital tract (lateral view).

The *ductus deferens* become incorporated into the ***spermatic***

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[*spermat(o)-* sperm + *-ic* pertaining to] ***cord***, along with the ***testicular*** [*testicul(o)-* testis + *-ar* pertaining to] artery and vein. Both the testes and spermatic cords are enveloped in a layer of tough connective tissue, called (oddly enough) the ***vaginal tunic*** (too'nik [L. "coat"]). The *vaginal tunic* is actually the same connective tissue that lines the ***peritoneal*** [*peritone(o)-* peritoneum, abdomen + *-al* pertaining to] cavity. Remember, we said that the testicles originated in the abdomen. When the testes descend from the abdomen through the inguinal ring, the ***peritoneum*** stretches with them. Ultimately, that peritoneal tissue layer envelopes the spermatic cord and testicle. As I'm sure you've already gathered, the *vaginal tunic* has nothing to do with the female's vagina. But it is something to remember, when we discuss castrations later on.

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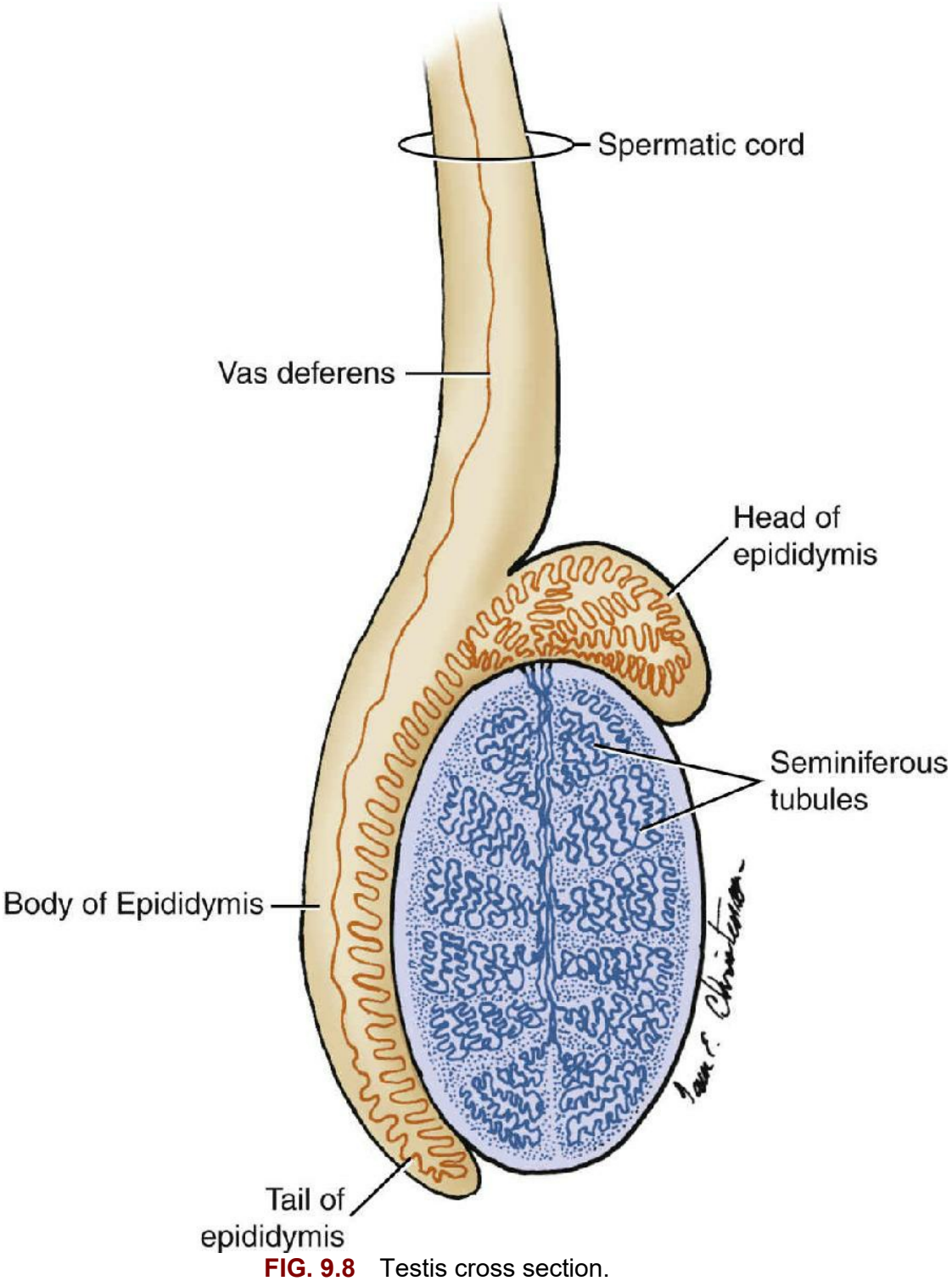

FIG. 9.8 Testis cross section.

Spermatogenesis

Under the influence of testosterone, cells along the seminiferous tubules undergo *meiosis*. As you'll recall from our earlier discussion of the ovaries, meiosis is different from mitosis. In meiosis, the new "daughter" cells (oocytes) contain only half of the original cell's

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chromosomes. The same is true in ***spermatogenesis*** [*spermat(o)-* sperm + *gen(o)-* production + *-sis* process of]. But the ***spermatocytes*** [*spermat(o)-* sperm + *cyt(o)-* cell] produced through meiosis are not ready to fertilize the female's eggs straight out of the gate. These little guys are like toddlers at first. They need to go through a great deal of development and maturation, before they are ready to to go to work. In fact, before the spermatocytes even leave the seminiferous tubules, they become attached to ***Sertoli*** (ser-to'le) ***cells***. Sertoli cells in effect nurse them along and protect them from the body's immune system. Under the "care" of the Sertoli cells, the spermatocytes eventually develop into ***spermatozoa*** [*spermat(o)-* sperm + *zo(o)-* animal]. Now, they're finally ready to leave the protection and care of the Sertoli cells and move on to the epididymis.

In the epididymis, the spermatozoa (aka. sperm) are still not ready for to go to work (fertilization). Let's consider them prepubescent or preteen at this point. They need to "hang out" in the epididymis for a week or so to fully mature. Once fully mature, they are ready to go to work to fertilize an egg. During breeding, mature sperm are passed from the epididymis via the vas deferens to the urethra during ***ejaculation*** [L. *ejaculatus* to throw out + *-ion* the act of] of the semen. We'll talk about some of the fluids that contribute to semen. For now, let's stay focused on the sperm.

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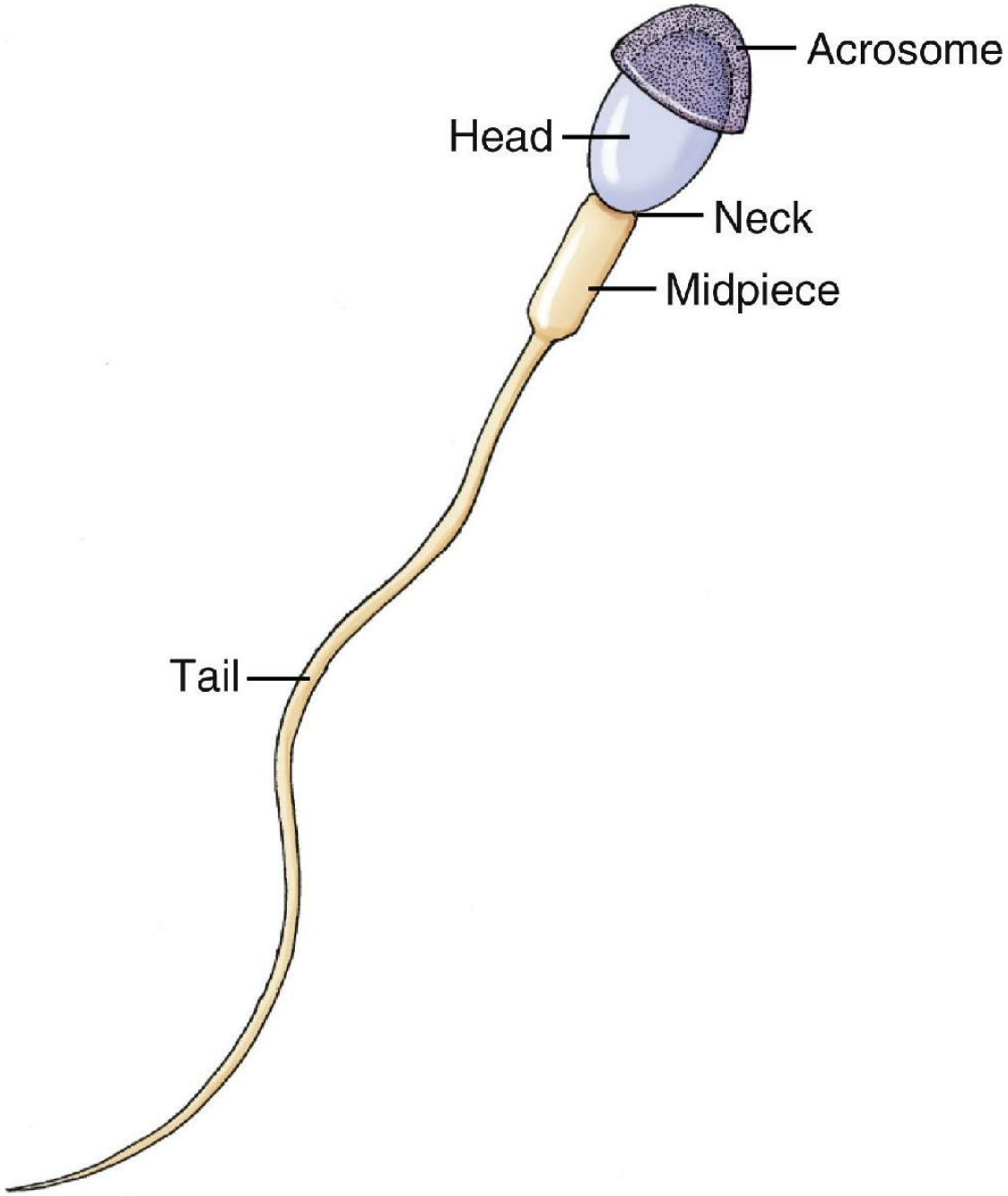

FIG. 9.9 Spermatozoon.

Spermatozoa

Structurally, ***mature sperm*** are designed to be fully self-propelled entities. If you look at the individual spermatozoon in Fig. 9.9, you'll notice that it has quite a few pieces and parts. The spermatic ***head*** contains the partial complement of chromosomes. Interestingly, roughly half of the sperm contain an X chromosome and the other half contain a Y chromosome. So, whichever lucky sperm (with the X or Y chromosome) that succeeds in fertilizing the

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egg determines the genetic sex of the offspring. If the fertilized egg has XX chromosomes, the neonate will be genetically female, while those with XY chromosomes are genetically male. Ah, but our sperm can't fertilize the egg without enzymes to penetrate the wall of the oocyte. That's where the tiny cap that it's "wearing," the ***acrosome*** [*acr(o)-* top, summit + *som(o)-* body] comes into play. The *acrosome* contains the needed enzymes to penetrate the wall of the oocyte. The spermatic ***neck*** connects the head to the midpiece, just like our neck connects our heads to our torso. The ***midpiece*** provides power for the sperm's propulsion system, because in the midpiece are mitochondria. If you need to review intracellular organelles, like mitochondria, please refer to Chapter 2. Finally, the spermatic ***tail*** not only whips back and forth to propel the spermatozoon, but it also contains stored fuel. After all, even when the semen is deposited near the cervix of the female, the sperm have a long way to swim to get to the oocyte(s) in the oviduct.

They don't all make it. Those that do "gang up" on the oocyte(s). Talk about competition! An individual egg will be completely surrounded by sperm, all wildly whipping their tails and pressing their acrosomes against the oocyte's cell membrane. This is really a race to the finish. The first sperm to penetrate the wall of the egg successfully fertilizes it. In that moment, only the head enters. The neck, midpiece, and tail are left outside in the cold, so to speak. Immediately, the fertilized egg puts "shields up!," creating a barrier so that no other sperm can penetrate. Thus, new life begins.

Semen

Obviously, in order for the sperm to swim, they need fluid, right? If all we had in semen were spermatozoa, they would probably never be able to make their way the eggs for fertilization. The ***seminal*** [*semin(o)-* semen + *-al* pertaining to] fluids make up the bulk of seminal volume. Sperm make up only a small fraction of the total volume. Most importantly, seminal fluids provide a suitable environment in which the sperm may propel themselves. Those fluids are produced by a number of accessory glands along the male urogenital tract. (More on that in a moment.) Seminal fluids tend to be somewhat alkaline, in contrast to the more acidic environment of the female urogenital tract. By neutralizing the pH

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of the environment, the seminal fluid makes it more likely for the sperm to survive. The seminal fluids also contain some simple sugars. This readily available fuel gives the sperm the energy they need to keep going. Finally, the seminal fluids contain substances that actually stimulate contractions along the female reproductive tract. Those contractions help push the semen with the sperm toward the oviducts.

Accessory Glands and Structures

All males have a ***prostate*** [Gr. *prostates* "one who stands before"] ***gland***, found near the neck of the urinary bladder. In most males, the prostate nearly, if not completely surrounds the proximal urethra. In dogs (see Fig. 9.6), the prostate is quite large. And it needs to be, because the prostate is the only accessory gland to produce seminal fluids for the dog. ***Prostatic*** [*prostat(o)-* prostate + *-ic* pertaining to] ducts enter multiple points along the proximal urethra. The vas deferens also enters the proximal urethra. Seminal vesicles (or vesicular glands) are found near the prostate, in many domestic animal males, except in the cat and dog. Fluids produced by the seminal vesicles enter the urethra in the same region as the vas deferens. ***Bulbourethral*** (bul"bo-u-re'thrul [*bulb(o)-* bulb + *urethr(o)-* urethra + *-al* pertaining to] ***glands*** are found in all domestic animal males, except the dog. Technically, the *bulbourethral glands* don't contribute to the semen. Actually, they produce a thick, ***mucoid*** [*muc(o)-* mucus + *-oid* resembling] substance that lubricates the urethra before discharge of the semen. The bulbourethral glands vary in size, shape, and location, depending on the species. Generally, they will be found somewhere along-side to the urethra, near the proximal portion of the penis.

Penis and Prepuce

The prepuce is the protective skin that covers the nonerect penis. ***Penile*** [*pen(o)-* penis + *-ile* pertaining to] shape varies among different species. For instance, the nonerect penis, among cattle (bulls), sheep (rams), goats (bucks), and pigs (boars), is S-shaped. These animals actually have an accessory muscle that keeps the

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nonerect penis retracted in this S-shape. Boars also have a spiral, cork-screw shape at the distal tip of the penis. Male cats (toms) have a caudally directed penis that is covered in barbs. The dog has a bone (the *os penis*) within his penis. As we discussed in Chapter 6, the urethra passes through the os penis. Despite species differences, there are basic structural components present in every male. The ***body*** of the penis is the largest portion. The penile body is composed of bundles of spongy, erectile tissue. The ***glans penis***, at the distal end, has variable amounts of erectile tissue, depending on the species. Horses (stallions) have the most highly-developed glans, with large amounts of erectile tissue. The glans also has numerous sensory nerve endings. (That's something to remember when you're trying to place a urethral catheter. His objection to catheter placement may be in response to stimulation of those sensory nerves.) Another unique feature of the dog's penis is the ***bulbus glandis*** or bulb of the glans. This is an enlarged area of erectile tissue near the proximal part of the glans. During breeding, the bulbus glandis is responsible for creating the "tie." In the tie, the dog's penis becomes locked in the female's vagina. It may take 20 minutes or so for the bulb to shrink enough for the dogs to separate. Most of the time, neither dog seems to mind. Anyone unfamiliar with this facet of canine reproduction may find the tie rather troubling. If you receive a call from a panicking owner, reassure them that the tie is normal. And tell them not to try to separate the dogs prematurely. One or both dogs could be injured in the process.

Erection and Ejaculation

An erection is a reflex event, triggered by sexual stimuli (e.g., sights, smells, and physical touch). The reflex activity results in the erectile tissue of the body and glans becoming engorged with blood. This engorgement enlarges and stiffens the penis. For bulls, rams, and boars, the penis will also straighten out of its relaxed S-shape, exposing it from the prepuce. The whole point in the erection is to permit penetration of the female genitalia.

Physical sensations of breeding will ultimately result in ejaculation of semen. Technically, ejaculation is the reflex expulsion

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of semen from the penis. But recognize that this is a highly coordinated event, involving seminal fluids from accessory reproductive glands AND spermatozoa from the epididymis. Plus, there are actually rhythmic contractions of the penile urethra that help push the semen into the female reproductive tract or into a collection receptacle. Why collect semen? Well, if we suspect an infertility problem, we may need to evaluate it. Semen is also collected and stored for artificial insemination (AI). Please remember, in the world of reproduction, "AI" does not refer to artificial intelligence.

Paraphimosis

Paraphimosis [*para-* beside, near + *phimosis* muzzling or closure] is most common in dogs and can be a painful consequence of an erection after breeding. That is probably the most frequent precipitating event. Yet, I have seen a number of dogs, usually younger dogs, with an abnormal ***preputial*** [*preput(o)*- prepuce + *-al* pertaining to] opening. I have also seen dogs with long hair coats get a band of hair caught across the preputial opening. In all of these cases, the preputial opening creates a point of stricture around the penis, creating and complicating engorgement of the erectile tissue. Plus, the more time that the penis spends extruded from the prepuce, the dryer it becomes. All of this makes it very difficult to slide the penis back into the prepuce. Needless to say, this condition can be uncomfortable in mild cases to extremely painful in severe cases. And if the blood supply to the glans becomes completely blocked, the tissue could actually become ***necrotic*** [*necr(o)*- death + *-tic* pertaining to]. Because of this (i.e., possible tissue death), paraphimosis is always an emergency.

One's goal should be to intervene as quickly as possible, to prevent serious penile trauma and ***necrosis*** [*necr(o)*- death + *-sis* condition of]. Often times lubricants, along with therapeutics to reduce swelling and engorgement, will resolve the paraphimosis. Let's just say that these fellas don't appreciate cold compresses, in spite of the benefits. Sometimes surgery is required. This is certainly the case for those with abnormal preputial openings. If necrosis has occurred, penile amputation is necessary. But remember, the penis

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is not simply a reproductive organ. It's a *urogenital* organ. So, if amputation is necessary, we will also have to perform a ***urethrostomy*** [*urethr(o)-* urethra + *-ostomy* creation of a stoma ("mouth")] so that he can urinate. Refer to Chapter 6 to review urethrostomies.

Prostatic Hyperplasia and Prostatitis

The prostate is another part of male anatomy that can create urogenital problems. Remember, this gland surrounds the urethra near the neck of the bladder. So, in any male that develops prostatic enlargement (most frequently dogs), due to swelling or ***hyperplasia*** [*hyper-* excessive + *plas(o)-* formation + *-ia*], his ability to urinate will be affected. That is a big concern, especially if he can't evacuate enough urine or urinate at all. So, whenever we see a male animal having difficulty urinating, especially if he's only able to dribble a small stream of urine, the prostate should be one of the things we evaluate. In dogs, the prostate is easily palpated through the rectum. It's obvious upon palpation, if the prostate is enlarged. If it's not, then we turn our attention to rule out other urinary problems (discussed in Chapter 6).

Prostatitis [*prostat(o)-* prostate + *-itis* inflammation of] is one possible cause of prostatic enlargement. As we discussed in Chapter 3, one of the cardinal signs of inflammation is swelling. So, with inflammation of the prostate, whatever its cause, the gland may enlarge purely due to swelling. ***Prostatic hyperplasia*** is different. With hyperplasia of any tissue, the tissue itself is actually bulking up. Most of the time in dogs, prostatic hyperplasia is ***benign*** (buh-nīn', not malignant). Benign prostatic hyperplasia (BPH) is also common in human males. In dogs, this hyperplasia appears to be driven by testosterone. So, castration often resolves the problem. Fortunately, unlike human males, malignant prostatic cancer in animals is rare.

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Common Reproductive Surgeries

Neutering of animals is quite common in veterinary medicine. (Note: the term "neutering" [from L. *neuter* neither] applies to both male and female animals.) Sometimes neutering is done to resolve disease problems, as previously mentioned with prostatic hyperplasia in males. Much of the time, it is done to prevent unwanted reproduction and overpopulation. Let's face it; there are way too many unwanted dogs and cats overwhelming animal shelters and humane societies. But this is not purely an animal reproductive problem. The glut of unwanted animals in shelters is also due to human error and negligence. So, to focus exclusively on reproduction alone will not solve the whole problem.

Also, it is important to note that neutering is not appropriate for everyone. Prime breeding stock cannot and should not be neutered, unless absolutely necessary due to life-threatening disease, like pyometra. Reputable breeders work to maintain and improve the best qualities of the breed according to well established standards. In zoo and other wildlife situations with endangered species, conservation breeding programs are essential to prevent extinction of the species. There are other birth-control measures beyond irreversible surgery. Finally, current research indicates that neutering later in life may prevent debilitating disease conditions, like ***arthritis*** [*arthr(o)-* joint + *-itis* inflammation of]. Neutering too young has been shown to create numerous physical developmental problems. There are many, many factors that should be considered for any individual or group of animals, to choose an appropriate course of action. To say that all animals should be neutered is inappropriate, just as permitting any animal to breed is inappropriate. Those in the veterinary profession need to do our due-diligence, reading all of the current research and weighing all of the benefits and consequences of neutering for a given individual.

Times have changed. Years ago, when I first started my veterinary career, the prevailing thought was that by spaying dogs and cats early, before the first heat cycle, we would prevent mammary cancer. Still today, spaying before the first heat can

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reduce risk of mammary cancer. But we also know today that not everyone is predisposed to breast cancer. We also know that by spaying (and castrating) later, perhaps after 1.5 to 2 years of age, we can reduce the risk of debilitating arthritis and ligament injuries (e.g., cruciate ligament tears). This is all related to joint development. Recent research shows that by maintaining gonadal hormonal influences for longer periods, the joints are able to fully develop and mature. I own a Cardigan Welsh Corgi, a breed like other ***chondrodystrophic*** [*chondr(o)-* cartilage + *dys-* bad + *troph(o)-* development + *-ic* pertaining to] canine breeds that is structurally predisposed to developing arthritic problems. In weighing health benefits over consequences, I am delaying neutering her, to minimize her risk of severe arthritis. I also know that if I monitor her closely, I can discover potential mammary changes early. Early detection of mammary cancer, in animals and people, has been shown to reduce risk of of the cancer spreading, thereby increasing survival.

All of this is a very long way for me to make a point. But it is a very important point to make: Everyone is different, with different physical conditions, different predispositions to disease, different life circumstances, and different purposes. We as veterinary professionals need to recommend and do what is best for each individual. There, I'll get off my soapbox now.

Ovariohysterectomy Versus Oophorectomy

It is common in dogs and cats to "spay." I really wish that we wouldn't use such a simple name for this surgery. It's very deceiving. It gives the impression that this is a simple, routine surgery. Yes, it is routine. And it is also major abdominal surgery. Talk to any woman who has had an ***ovariohysterectomy*** [*ovari(o)-* ovary + *hyster(o)-* uterus + *-ectomy* to cut out, surgical removal of]. She will tell you: It. Is. Major. Surgery. Because of that, there are risks associated with it, such as severe bleeding or ***ligation*** (li-ga'shun [L. *ligatio* to tie or bind; ligation is the application of suture material tightly tied to cut off blood supply to a body part]) of a ureter. As you'll recall from Chapter 6, the ureter is a tube that connects the kidney to the urinary bladder. There are other risks

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too, not the least of which is general anesthesia. Also, if the female is in heat or pregnant at the time of the *ovariohysterectomy*, her risk of life-threatening bleeding during and after the surgery increases exponentially, due to the increased blood supply to the uterus.

That said, ovariohysterectomy is a very common and routine surgery, especially in dogs and cats. The primary purpose for performing this surgery is to prevent unwanted pregnancy. Most commonly, it is performed as a ***laparotomy*** (lap"uh-rot'uh-me [*lapar(o)-* flank + *-otomy* to cut, incise; i.e., abdominal surgery via a large incision through the abdominal wall]). However, with more veterinary surgeons becoming skilled in ***laparoscopic*** (lap"uh-ro-skop'ik [*lapar(o)-* flank + *scop(o)-* to view + *-ic* pertaining to; i.e., surgery using a laparoscope]) surgery, less traumatic surgical options are available. In *laparoscopic* surgery, there is no large abdominal incision. There are typically only three or four small (perhaps 1 centimeter) incisions through the abdominal wall for insertion of various laparoscopic components. Because there is no large abdominal incision, recovery from laparoscopic surgery is much quicker. This is true in veterinary and human medicine. Still, we must not forget that this is major surgery. The uterus and ovaries are still being removed in a laparoscopic ovariohysterectomy.

There are other surgical birth control options for females. An ***oophorectomy*** [*oophor(o)-* ovary + *-ectomy* surgical removal of] is another possibility. This is a less traumatic procedure, especially if it is laparoscopic. This method of "sterilization" of dogs and cats has been used for decades in Europe. And with the advent of laparoscopic surgery, it is growing in popularity in the U.S. For mares, beyond their reproductive years, this is probably the most logical surgical option. Of course, we need to remember that the uterus remains intact. So, there is still the potential of her developing pyometra. However, generally, without the hormonal influence, this risk is low.

Orchiectomy/Orchidectomy

Castration of male animals is commonplace in veterinary medicine. Which term should we use in reference to this surgery? Is it

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orchiectomy [*orchi(o)-* testis + *-ectomy* surgical removal of], or is it ***orchidectomy*** [*orchid(o)-* testis + *-ectomy* surgical removal of]? Both are correct and acceptable medical terms for this surgical procedure. This is certainly a much less invasive surgical procedure, compared to a laparotomy for an oophorectomy or an ovariohysterectomy in the female. However, even beyond the risks associated with anesthesia, there are risks of complications, such as infection and bleeding.

There are significant differences in surgical approach, depending on the species. In most farm animals (i.e., bulls, stallions, rams, and boars), a scrotal approach is most common. With this approach, an incision is made through the scrotum over each testicle. If the size of the spermatic cord and testicular vessels is not great, a "closed" orchiectomy may be performed. In a closed orchiectomy, the vaginal tunic is left intact. In the presence of larger blood vessels, an "open" orchiectomy may be performed. In an open castration, the vaginal tunic is incised to allow full retraction of the testicle, and to provide good visualization and access to the vessels, cremaster muscle, and the rest of the spermatic cord. One disadvantage to an open castration is that it provides a direct pathway for bacterial contaminants to enter the abdomen. In very young males, an ***emasculator*** [*emascul(o)-* L. *emasculare* to castrate + *-tor* the; a hinged castration instrument with both crushing and cutting parts] may be used. An emasculator may be used for either closed or open castrations. The crushing action of the instrument facilitates occlusion and clotting of blood vessels to minimize bleeding. In males with larger, well-developed vessels most surgeons will also ligate (tie a suture) around the vessels. It is interesting to note that the scrotal incisions are not sutured closed. This allows for drainage from the surgical site, which is of particular importance should an infection develop. It also prevents the development of a scrotal ***hematoma*** [*hemat(o)-* blood + *-oma* swelling; an accumulation of blood]. If significant bleeding does occur, because it is allowed to drain from the scrotal incisions, it will be very noticeable to the owner/farmer, permitting them to seek prompt medical intervention. A scrotal approach is also used in tomcats; however, rather than using suture to ligate the spermatic cord, the spermatic cord is often tied in a knot on itself.

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The most common surgical approach for orchiectomy in dogs is ***prescrotal*** [*pre-* before + *scrot(o)-* scrotum + *-al* pertaining to]. In this procedure, a single incision is made in the prescrotal skin (i.e., the skin between the scrotum and prepuce). Each testicle is then pushed from the scrotum through the incision, ligated and removed. Again, either an open or a closed orchiectomy may be used, depending on surgeon preference and development of the structures (especially vessels). Why use a prescrotal approach? First, the scrotal skin is typically very thin. So, surgical closure would be difficult. Second, a prescrotal incision is less annoying for the dog. Scrotal skin is very sensitive, and anything that would cause irritation will be very noticeable for the dog. And where there is irritation and discomfort, he WILL lick. That is a big problem for a surgical site. But won't there be discomfort at the prescrotal incision too? Yes. But comparatively, the prescrotal discomfort is far less. Many dogs don't pay any attention to their prescrotal incisions. If we surgically clip and prep the scrotum, I guarantee that we'll cause tremendous irritation. So, scrotal orchiectomies are simply not done for dogs. That said, there is a potential drawback to the prescrotal castration: if bleeding occurs, the blood has nowhere to go. Blood will accumulate in the scrotum. I have seen many extremely large, painful scrotal hematomas in dogs. It is very important for us to monitor orchiectomy patients closely to catch signs of bleeding early. It is also very important for owners to monitor and to limit the dogs' activities (no running or jumping), after they go home. Failure to do so may put him at risk of significant bleeding, hematoma formation, and emergency surgery to either stop the bleeding and/or to relieve the engorged scrotum.

Now, in the case of *cryptorchidism*, orchiectomies become much more challenging. As noted earlier, cryptorchidism is a male condition in which one or both testicles fail to descend into the scrotum. Often, in a unilateral cryptorchid, the retained testicle may be at or very near to the inguinal ring. If that's the case, the surgeon may be able to force the testicle into the scrotum to complete a "normal" castration. But this is not always the case. If one or both testicles are retained well within the abdominal cavity, a *laparotomy* will be required remove them. This increases both the surgical risk and the time needed for recovery. What difference does it make if

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we leave a retained testicle in him? First, a retained testicle still secretes testosterone, which can contribute to unwanted behaviors such as roaming and intermale aggression. Also, a retained testicle can still enable him to breed. Finally, a retained testicle can develop ***neoplasia*** [*ne(o)-* new + *plas(o)-* formation + *-ia* a condition of; i.e., cancer]. Yes, descended testicles can also become ***neoplastic*** [*ne(o)-* new + *plas(o)-* formation +-*tic* pertaining to]. The problem with a retained testicle is that, because it is hidden in the abdomen, we may not discover the neoplasia until it is quite advanced. I remember a Schnauzer whose retained testicle became neoplastic. It was the size of a large grapefruit by the time it was found and removed. Unfortunately, the cancer had ***metastasized*** [*meta-* beyond + *stasis* standing + *-ize* act of; i.e., spread to other areas of the body], significantly shortening his lifespan.

Episioplasty and Episiorrhaphy

Episiorrhaphy [*episi(o)-* vulva + *-rrhaphy* suturing of] is most commonly performed in mares. It is commonly called ***Caslick surgery***, named for the veterinarian who developed it. Why on earth would we want to partially suture her vulva closed? Well, sometimes a mare's conformation leaves her at risk for reproductive tract infections. These mares usually have significant angulation of the vulva. When they have a bowel movement, the feces contaminates the vulvar opening and vestibule. Those fecal contaminants can lead to ***vaginitis*** [*vagin(o)-* vagina + *-itis* inflammation of] and even *metritis*. So, by partially suturing the vulva closed, leaving enough of the ventral segment open for urination, contamination of the vestibule is greatly reduced. For breeding and parturition, the sutures are removed. Now, think about this for a moment. In order to suture the vulva, some poor soul needs to stand directly behind the mare. Well, that's not a safe place to be period, especially when she's being poked multiple times in a very sensitive area with that suture needle. It won't be appreciated to say the least. In order to safely accomplish the task, without any major objections from the mare, we'll need to be sure to provide good local or regional anesthesia. If we don't she may kick the person suturing into the next county. Yikes! Seriously,

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veterinary work like this can be very dangerous.

Episioplasty [*episi(o)-* vulva + *-plasty* repair, reconstruction of] is most commonly performed on dogs, although it may be warranted in any female. Over the span of my long career, I have only seen episioplasties in dogs. Most often these dogs had inverted vulvas. That conformation, with the deep crevices in the skin in and around the vulva provides an excellent, chronically (long-term) moist environment that promotes bacterial growth. Bacterial growth promotes ***dermatitis*** [*dermat(o)-* skin + *-itis* inflammation of], ***vulvitis*** [*vulv(o)-* vulva + *-itis* inflammation of], ***vaginitis*** [*vagin(o)-* vagina + *-itis* inflammation of], and even ***retrograde*** [*retro-* backward + *grade* to step, move] urinary tract infections (UTI). My Basset Hound had a bit of an inverted vulva, and as a result, she also had numerous UTIs. However, I was able to minimize the number of retrograde UTIs, with daily cleansing and drying of all of the folds and crevices surrounding her vulva. But her vulvar inversion was not severe. So, medical management was possible and relatively easy. I have seen many other dogs with significant inversion. By the way, obesity complicates the already poor structure. So, weight-loss is beneficial on a number of levels.

If it is determined that *episioplasty* is in the dog's best interest, this is no minor surgery. Usually, it is necessary to remove a large amount of skin and tissue to reconstruct her vulva. One (Think of this as a major "butt-lift.") That means significant surgical trauma and discomfort. The surgeon wants to take enough but not too much skin and tissue. Taking too much may place too much tension on the suture-line, making it possible for the surgical site to dehisce (break open). If insufficient tissue is taken, subsequent surgery may be necessary.

Mastectomy

Most often a ***mastectomy*** [*mast(o)-* breast + *-ectomy* surgical removal of] is performed in dogs, cats, and people due to mammary cancer. While breast cancer most frequently occurs in females of the species, it is important to note that males have breast tissue too. Therefore even males within the species can develop breast cancer, though fortunately, it is rare. Believe it or not, I've known men men

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who have lost both male and female family members to breast cancer. For our discussion of mastectomies here, I won't cover all of the ancillary care that may be needed in addition to surgery (e.g., chemotherapy or radiation therapy). At this time we will only discuss *mastectomy* as a relatively common reproductive surgery.

One big difference between mastectomies in people versus animals is the extent of the surgical site. Remember, dogs and cats (male and female) have mammary tissue that extends bilaterally over the ventral abdomen and over much of the ventral chest. So, if we need to remove a complete mammary chain, the surgical site will be huge. If the procedure is bilateral, the animal will be subjected to extreme surgical trauma. Many times, partial mastectomies are performed, i.e., removing the affected gland(s) and perhaps an adjoining gland. Often, wide margins of skin in the area will also be removed. Mastectomies can be brutal. For those predisposed to breast cancer (i.e., family history of the disease), research has shown that estrogen often plays a role in its development. So, an oophorectomy could be viewed as a preventive measure. Unfortunately, "family history" is usually a big unknown for most dogs and cats. If it is known, removal of the ovaries via an oophorectomy or ovariohysterectomy before the animal's first "heat" could significantly reduce her risk of developing mammary cancer.

Case Study

Rosie is a 4-year old, female, white, Toy Poodle. She became a Grand Champion in conformation at 2 years of age, and she has also earned titles in obedience and is certified as a Therapy Dog. Her open, friendly, gentle demeanor makes her a favorite visitor for nursing home residents and hospital patients. To date, she has produced one litter of six puppies. Because of dystocia with that litter and postparturient health concerns (namely eclampsia), her owner/breeder skipped her last two heat cycles. She was in estrus a little over a month ago. Though she was not bred, her owner reported that Rosie was acting much like she did when she was pregnant with her first litter. Even Rosie's abdomen seemed larger.

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As a result, Rosie was brought in last week for an abdominal ultrasound, to see if she was actually pregnant. Unplanned breeding, while a slim chance, is always a possibility. The ultrasound showed that Rosie was not gestational. The veterinarian diagnosed her with pseudocyesis and told the owner to simply keep an eye on Rosie.

Over a period of 2 days, Rosie became rather lethargic, and her appetite decreased. In fact, on the morning she was presented to us, she refused to eat anything at all. She was also running a fever. So, her owner brought Rosie in again to have her evaluated. Rosie's blood work showed evidence in keeping with a profound infection somewhere in her body. Many of her electrolytes were abnormal. Rosie was also somewhat hypoglycemic. She was dehydrated, despite the fact that the owner indicated Rosie had polyuria and polydipsia. Her blood pressure was marginally low. The nature of her infection was confirmed with radiographs and another abdominal ultrasound. Rosie was diagnosed with a closed pyometra. Based on her rapidly deteriorating physical condition and the profound distention of her uterine horns, the doctor's best recommendation was an emergency ovariohysterectomy. At first the owner was reluctant to approve the surgery. She was hoping for another litter or two from this Grand Champion. However, once the gravity of Rosie's condition was explained, she gave her consent for the surgery.

The decision for surgery was made none too soon. Had Rosie's surgery been delayed, her uterus likely would have ruptured, resulting in peritonitis. Even so, following surgery, Rosie required hospitalization in ICU for over 48 hours. She was eventually discharged 4 days after her surgery. Once she fully recovered, Rosie continued her service as a Certified Therapy dog.

Case Study Questions

1. What is the medical term for pregnancy? _____
2. Rosie experienced _____ (i.e., a difficult birth) when she delivered her first litter of puppies.

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3. Rosie also experienced postparturient eclampsia. The medical term for this calcium deficiency is _____.
4. Approximately a month after her latest heat cycle, Rosie was diagnosed with a false pregnancy, which is medically termed _____.
5. Roughly a week after Rosie was diagnosed with her false pregnancy, she became very ill. Probably due to her fever, infection, and poor appetite, she presented to us with low blood glucose levels, medically termed _____.
6. Rosie's condition made her very thirsty. The medical term for much thirst is _____.
7. Rosie was also urinating large amounts. The medical term for much urination is _____.
8. The infection was determined to be in her uterus, filling it with pus. What is the medical term for this condition? _____.
9. If Rosie's uterus had ruptured, she would have developed _____ or inflammation/an infection of her abdominal cavity.
10. To save Rosie's life, the owner consented to an emergency surgery to remove her ovaries and uterus. This "spay" procedure is medically called an _____.

The Answer Key to these case study questions may be found in Appendix B.

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10

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Applied Endocrine Terminology

Invisible Hormones,
- Negative Feedback—A Positive Thing,

Endocrine Organs and Hormones,
- Pituitary Gland,
- Adenohypophysis—Anterior Pituitary,
  - Adrenocorticotropic Hormone,
  - Follicle-Stimulating Hormone,
  - Growth Hormone,
  - Luteinizing Hormone,
  - Prolactin,
  - Thyroid-Stimulating Hormone,
- Neurohypophysis—Posterior Pituitary,
  - Antidiuretic Hormone,
  - Oxytocin,
- Thyroid Gland,
  - Triiodothyronine (T_3) and Thyroxine

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GOALS AND OBJECTIVES

By the conclusion of this chapter, the student will be able to:

1. Recognize common root words, prefixes, and suffixes related to the endocrine system.
2. Divide simple and compound words into their respective parts.
3. Recognize, correctly pronounce, and appropriately use common medical terms related to the endocrine system.
4. Demonstrate an understanding of endocrinology with regard to anatomy.

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5. Demonstrate an understanding of endocrinology with regard to specific hormones, correlating them to their secretory organs.
6. Demonstrate an understanding of endocrinology with regard to negative feedback.
7. Demonstrate an understanding of endocrinology with regard to health, homeostasis, and common diseases.

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Invisible Hormones

I think the most difficult thing about ***endocrinology*** [*endo-* inside + *crin(o)-* secretion + *-logy* study of; i.e., "the study of hormones"] is the fact that we cannot see or touch hormones. They're just there, secretly going about the work that they do—invisible to the naked eye. Ah, but we can see the end result of ***hormonal*** [*hormon(o)-* hormone + *-al* pertaining to] influence. You see, hormones [Gr. *hormaein* to set in motion] are chemicals that subtly and profoundly influence many, many activities in the body. And the end result of what a hormone "sets in motion"—an increased heart rate, dilated pupils, growth of youngsters and much more—we often can see or feel.

To quote Sir Isaac Newton: "For every action there is an equal and opposite reaction." That's the way the ***endocrine*** [*endo-* inside, within + *crin(o)-* secretion] ***system*** works—give-and-take—to maintain ***homeostasis*** [*home(o)-* sameness + *-stasis* a state of standing; i.e., equilibrium]. We talked a little bit about *homeostasis* in Chapter 2, as well as numerous other chapters. The information in our current chapter will really expand your understanding of ***homeostatic*** [*home(o)-* sameness + *-static* pertaining to standing] mechanisms. The Evolve animation, *Endocrine Overview*, will support your understanding, as we begin. But before we can talk about specific *endocrine* organs and hormones, we need to gain a basic understanding of how the give-and-take of the system works.

Negative Feedback—A Positive Thing

I like to think of the endocrine system as a covert, wireless communication system for the body. It's wireless because the hormonal messages are chemical. And it's covert because, as we said, we can't see the hormones themselves, only some of their effects. Now, good communication always involves some sort of feedback, right? No one wants to listen to someone go on and on about the same story that they've told dozens of times before. So the person listening might verbally interrupt, or make a hand gesture to communicate "time out" or "talk to the hand." By

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providing the feedback, the listener hopes that the rambling person will stop. That may happen in any casual relationship. In more important relationships, open communication and feedback are essential for the stability of the relationship. There has to be feedback, be it positive or negative, to maintain a healthy relationship. The feedback mechanisms in endocrinology are a little bit like that.

Hypothetically, let's say we need hormone A to increase blood pressure. Great. Gland A begins secreting hormone A, and, as a result, the blood pressure goes up. But we don't want it to keep going up, because extremely high blood pressure is dangerous. So once we reach a reasonable blood pressure, gland A will be told either to decrease production of hormone A or to stop it all together. This is what's known as ***negative feedback***. It's negative because the feedback is against overproduction of the hormone. Whatever you do, don't think of "negative" as "bad." In endocrinology, negative feedback is very good because it prevents endocrine organs from going overboard with their hormone production. And that's how we maintain equilibrium in the body. As we move along, I will try to point out some of the feedback loops for you, to reinforce the concept of negative feedback.

Without further ado, let's get into the nitty-gritty of endocrine organs and hormones.

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Endocrine Organs and Hormones

Our focus in this chapter will be on the major endocrine organs of the body. Understand that there are other tissues of the body that also secrete hormones. Major endocrine organs and hormones can be confusing as it is. So we'll stick to the "majors", like those shown in the *Endocrine Overview* animation, and leave the rest alone.

Pituitary Gland

In endocrinology, the pituitary gland is truly the "command and control center." This tiny, little gland is the "big boss" of hormone production. It is so important that it actually has multiple names: pituitary and ***hypophysis*** [*hypo-* below + *-physis* growth, growing]. It has names for its tiny subdivisions too. We'll talk about those momentarily. First, let's talk about its location in the body. Because of its importance, the pituitary has a penthouse office. That's right. The hypophysis is found in the ventral cranium, beneath the brain, just rostral to the brainstem (Fig. 10.1). Technically, the pituitary is ventral to the ***hypothalamus*** [*hypo-* below + *thalamus* Gr. *thalamos* inner chamber]. You'll learn much more about the brain in Chapter 11. For now, recognize that when it comes to controlling many automatic, life-sustaining activities of the body (e.g., body temperature, heart and breathing rates, etc.), the hypothalamus and brainstem are key players. In essence, we have clustered all of our most important executives (hypothalamus, brainstem, and pituitary) in this penthouse suite.

Adenohypophysis—Anterior Pituitary

We said that the pituitary is subdivided. The ***anterior pituitary*** or ***adenohypophysis*** [*aden(o)-* gland + *hypo-* below + *physis* growth, growing] is responsible for many, many things. Comparatively, the anterior pituitary does way more than the posterior pituitary. We'll take a brief look at each of the command and control hormones secreted by the adenohypophysis first. (By the way, we'll be taking a look at each of these hormones in alphabetical order; a hormone's

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place in the order of presentation in no way indicates its importance. Each one is important in its own right. We'll reinforce negative feedback mechanisms when we talk about the other endocrine organs. Then we can put the whole cause-and-effect loop together. It may seem that we're stopping short in our anterior pituitary hormone discussions here. But we'll get to the full story eventually. I promise.)

Adrenocorticotropic Hormone

First on our list of anterior pituitary hormones is the ***adrenocorticotropic*** [*adren(o)-* adrenal gland + *cortic(o)-* cortex + *trop(o)-* turning, changing, influencing + *-ic* pertaining to] ***hormone*** (***ACTH***). Holy cow, that's a big word! No wonder we abbreviate it! But don't be intimidated by the size of the word. When it's broken down, it makes sense, doesn't it? *ACTH* is an anterior pituitary hormone that is secreted to tell the cortex (i.e., the outer "crust" portion) of the ***adrenal*** [*ad-* toward, near + *ren(o)-* kidney + *-al* pertaining to] ***gland*** what to do. By the way, there's nothing really special about the name of the adrenal gland. It got its name based solely on its location—it's really close to the kidney. Anyway, ACTH is merely a chemical message to the adrenal cortex: "Yo! Get to work!" In response, the adrenal cortex will secrete its hormones. Rather than muddy the water here, we'll talk about adrenal cortex hormones in a little bit.

Follicle-Stimulating Hormone

Well, the name ***follicle-stimulating hormone*** (***FSH***) says it all, doesn't it? Obviously, it is a hormone that stimulates follicles. Ah, but where are these follicles? They are found in a female's ovaries. If you've already studied the Chapter 9, you've learned about the estrus cycle and pregnancy. Let's quickly review some of this, as it relates to FSH. Remember, there are a whole bunch of tiny *follicles* [from L. *folliculus*, dim. of *follis* a leather bag] with immature eggs in the ovary. When the anterior pituitary secretes FSH, one or several of these follicles will be stimulated to develop, along with the eggs within them. The developing follicle does more than just surround and protect the developing egg. It also begins to secrete its own hormones to prepare the uterus for potential conception and

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pregnancy. The female's behavior and genitals (e.g., vulva) are also influenced by these follicular hormones. All of this activity happens because the adenohypophysis secreted FSH. We can't release the egg yet. (More on that in a moment.)

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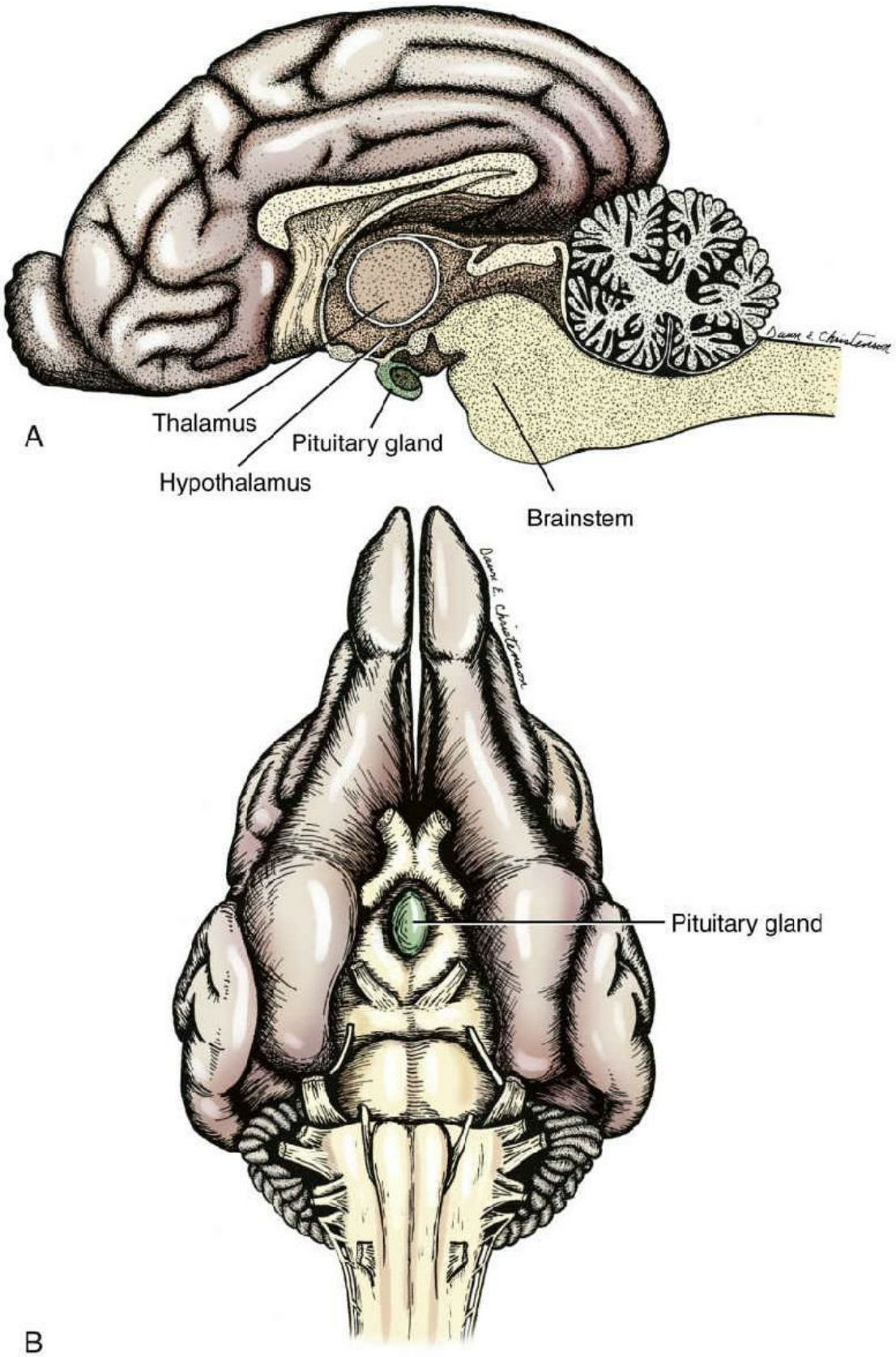

FIG. 10.1 Pituitary gland. (A) Midsagittal view. (B) Ventrodorsal view.

When is FSH secreted? Well, that depends on the species.

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Remember, cows cycle every month. Other animals, such as dogs, may experience an estrus cycle every 6 to 9 months or so. There's a great deal of variability. But when the time is right, FSH will be secreted.

Growth Hormone

Growth hormone (***GH***) is obviously secreted in young, growing animals. The powerful ***somatotropic*** [*somat(o)-* body + *trop(o)-* changing, influencing + *-ic* pertaining to] effects are easy to see when we watch youngsters grow into adulthood. Now, you might think that once an animal reaches puberty or an adult age, there is no longer a need for GH. That would be a very wrong assumption. Sure, the amount of GH secreted in a mature animal is far less than what was secreted in its youth. But we still need it to stimulate growth of cells, especially in those tissues that experience regular turnover of cells. For example, stratified (layered) epithelium that lines the urinary bladder wall and composes the ***epidermis*** [*epi-* upon + *dermis* the skin], the most superficial layer of the skin, is constantly losing the most superficial cells. The epithelial cells must constantly be replenished from the bottom layer of cells. GH is needed for that. In injuries, in whatever the tissue may be, GH is needed to stimulate activity (especially mitosis) of cells for repair of the injured area. Refer to Chapter 2 if you need to review cellular anatomy and reproduction (mitosis). So, growth hormone is needed in all life-stages.

Luteinizing Hormone

Earlier in our discussion of follicle-stimulating hormone (FSH) we said that follicles respond to the hormone with development of the follicle and the immature egg within it. By the time the egg is fully developed, the follicle looks like a blister. As we approach the end of this follicle development, the anterior pituitary also gradually begins and increases its secretion of ***luteinizing*** [*lutein* from L. *luteus* yellow + *-izing* causing] ***hormone*** (***LH***). By the time the follicle ruptures, releasing the egg, LH production is peaking. Why? Because if the egg is fertilized, we need a ***corpus luteum*** (literally, "a yellow body") to maintain the pregnancy. The reason they called it that is because corpus luteum is literally a yellow body (blob) that

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is grossly visible on the ovary. LH stimulates transformation of the now-defunct follicle into the corpus luteum. See? LH causes the development of this yellow body, just like the term *luteinizing* suggests.

Prolactin

Oh, this is an easy one. The name of this hormone says it all. ***Prolactin*** [*pro-* for + *lact(o)-* milk + *-in* the] (PRL) is a hormone that is needed to stimulate milk production. It is "for" milk production. Get it? Obviously, the ***mammary*** [*mamm(o)-* breast + *-ary* pertaining to] tissue is the target of this hormone, so prolactin is typically only secreted in nursing mothers. However, as we discussed in Chapter 9, mammary development and milk production may also occur in "false pregnancy." That is a situation of pure chaos with female reproductive hormones that's best left for Chapter 9.

Thyroid-Stimulating Hormone

Here's another no-brainer. ***Thyroid-stimulating hormone*** (***TSH***) is produced by the adenohypophysis to stimulate the thyroid gland. I love it when hormones are named for their function. There's no major effort required by us to figure out the basic purpose of a hormone when it's named like this. We'll discuss the thyroid's response to TSH later.

Neurohypophysis—Posterior Pituitary

The ***neurohypophysis*** [*neur(o)-* nerve + *hypo-* below + *physis* growth, growing] or ***posterior pituitary*** is not as prolific in its hormone production. Still, the hormones produced here are equally important to many of those secreted by the adenohypophysis. Again, we'll take them in alphabetical order. (That's easy. There are only two.)

Antidiuretic Hormone

Antidiuretic [*anti-* against + *diure(o)-* urine, urination + *-tic* pertaining to] ***hormone*** (***ADH***) does just what its name implies. It targets the kidneys to decrease urine production. Why on earth

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would we want to do that, you ask? Well, we need a means to conserve water for the body. This is necessary to prevent dehydration. Remember, the body is roughly 70% water. As we begin to dehydrate, the hypothalamus is triggered to (a) increase thirst and (b) stimulate the posterior pituitary to secrete *anti*diuretic hormone (ADH). When ADH reaches the kidneys, rather than lose valuable water in urine, the kidneys actually reabsorb water for the body. The end result is decreased urine production. Urine may still be produced but in smaller, very concentrated volumes. If we replenish body fluids, the hypothalamus is no longer triggered, so thirst dissipates and neurohypophysis is no longer stimulated to produce ADH. Without ADH, the kidneys remove any excess water from the body in the urine. That is a great example of a negative feedback loop.

ADH goes by another name: ***vasopressin*** [*vas(o)-* vessel + *press(o)-* pressure + *-in* the]. You see, this hormone has multiple functions in the body. If we focus exclusively on its ***renal*** [*ren(o)-* kidney + *-al* pertaining to] effect, with regard to urine production, the name ADH seems befitting for the task. But this hormone also causes contraction of vessel walls to increase blood pressure. Then, the name *vasopressin* seems befitting for the task. In fact, maintenance of adequate blood pressure is a very delicate dance performed by the heart, vessels, and kidneys. Hormonally, ADH/vasopressin targets two of the three dancers. As you may recall, in Chapter 5, we discussed blood pressure from a ***cardiovascular*** [*cardi(o)-* heart + *vascul(o)-* vessels + *-ar* pertaining to] perspective. We'll discuss the complete hormonal story of blood pressure near the end of this chapter. It's that important.

Diabetes Insipidus

In ***diabetes*** [Gr. *diabētēs* siphon] ***insipidus*** [L. *insĭpĭdus* tasteless], we have an animal with excess thirst and production of large volumes of dilute urine. There are a number of disorders that could result in these symptoms, including ***diabetes mellitus*** [L. *mellĭtus* honeyed, sweet]. Both forms of diabetes produce ***polyuria*** [*poly-* much + *ur(o)-* urine + *-ia* condition of] and ***polydipsia*** [*poly-* much + *dips(o)-* thirst + *-ia* condition of] (***PU/PD***). And that's what the term *diabetes* refers to: water being siphoned—literally flowing through the body,

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seemingly unchanged, as if it were flowing through a siphon hose. It's the second part of each name that distinguishes the disease disorders. In diabetes insipidus, it's purely a water-management issue. Water is tasteless, and—this is going to sound gross—if we were to taste the urine of a patient with diabetes insipidus, it would be virtually tasteless (insipidus). The urine from a patient with diabetes mellitus, on the other hand, would taste sweet because it contains sugar (glucose). (We'll talk more about diabetes mellitus when we discuss the pancreas. I bring it up here only to point out that there is a big difference between the disorders.) So we should never just broadly talk about diabetes as a single disease. It's not. Each type is different, as are the mechanisms that cause them.

For now, let's focus on *diabetes insipidus*. In diabetes insipidus, there is a problem somewhere among the three players involved with water management for thirst and urine production (i.e., the hypothalamus, posterior pituitary, and kidneys). Break any one of the three players or interfere with their communication and a patient may develop diabetes insipidus. It would be a rare circumstance for there to be an issue with the hypothalamus. If that is the origin of the problem, there may be bigger concerns for the patient than just diabetes insipidus. A far more likely cause of diabetes insipidus would be the kidneys. They may simply become less responsive to ADH. This can often be resolved if we can figure out the underlying source of the renal disease. Yet most often, diabetes insipidus results from too little production of ADH. Trauma, tumors, or even cyst formation in or in the vicinity of the posterior pituitary may adversely affect its secretion of ADH. Sometimes we have no idea why the pituitary isn't producing enough ADH. This is what we would term an ***idiopathic*** [*idi(o)-* one's own + *path(o)-* disease + *-ic* pertaining to; i.e., a disease of unknown origin] disorder. It's unique to the individual, and we have no idea how or why. Regardless, too little ADH means we can't concentrate urine to conserve water for the body. Fortunately, we can make up for the deficiency by giving synthetic ADH to the patient.

Oxytocin

Oxytocin [*oxy-* fast, quick + *toc(o)-* birth + *-in* a, the] *(OT)* is a

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hormone with a two-fold purpose. First, for a pregnant female, at the end of ***gestation*** [*gest(o)-* to bear + *-tion* state of; i.e., pregnancy], oxytocin is needed to stimulate contraction of uterine muscles. Typically, it stimulates powerful contractions to literally accelerate the birthing process. If the contractions are weak, provided there is nothing physically preventing passage of the newborn through the birth canal, we may actually administer OT by injection to strengthen the contractions. In human medicine, this is what is referred to as *inducing labor*. The administration of oxytocin can get the ball rolling and keep things moving.

Very soon after birth, ***neonates*** [*ne(o)-* new + *nat(o)-* born] must nurse (suckle). As you may recall from Chapter 9, ***colostrum*** (ko-los'trum [L. *colostrum* beastings; i.e., first milk]) contains maternal antibodies, essential to protect the little newborn beast until its own immunity develops. For optimal absorption of those antibodies, the colostrum should be consumed within the first 12 hours after birthing. To coin a phrase from the dairy industry: "Got milk?" This milk needs to be available as soon as the neonate is able to nurse, so, even during the birthing process, OT is also stimulating ***milk letdown.*** "Letdown" sounds passive doesn't it? Well, this process is anything but passive. There is actual muscle contraction to push the milk "down" from the mammary tissue, where it is produced and stored, to the collection cisterns (chambers) near the teat/nipple. As soon as the neonate is able to "belly up to the bar," it can suckle this all-important milk. After this, any time there is stimulation of a teat/nipple, the hypothalamus stimulates the posterior pituitary to secrete oxytocin for milk letdown for all subsequent meals. The effect of OT is temporary: it lasts only a minute or so. Remember this if you're trying to collect a milk sample. If you hesitate or take too long, you may miss the window of opportunity for available milk.

Thyroid Gland

The ***thyroid gland*** got its name from its shape. In ancient Greece, a *thyreos* was an oblong shield; the name *thyroid* literally means "*resembles* [*-oid*] a *shield*." In dogs (Fig. 10.2), the thyroid is actually two separate lobes on the ventrolateral aspect of the trachea, just

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caudal to the larynx. In other species, such as horses and cattle, there is an ***isthmus*** [Gr. *isthmos* narrow band] connecting the lobes. This is another small gland with a big job. Let's take a look at the thyroid hormones to learn their importance in homeostasis. Watch the Evolve animation, *Thyroid Secretion*, to help with your understanding of thyroid function and negative feedback loops.

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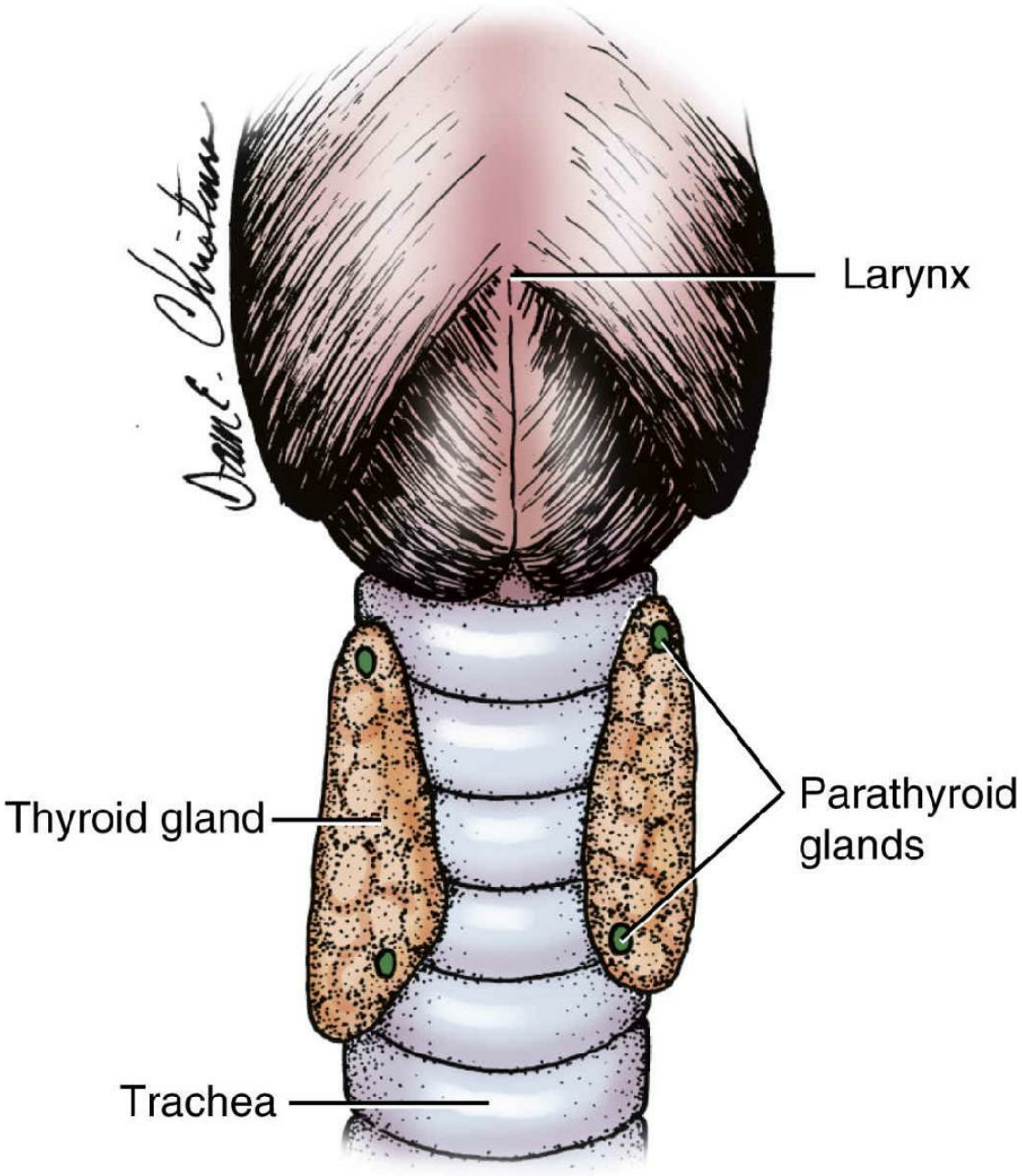

FIG. 10.2 Canine thyroid and parathyroid glands.

Triiodothyronine (T_3) and Thyroxine (Tetraiodothyronine, T_4)

Triiodothyronine [*tri-* three + *iod(o)-* iodine + *thyr(o)-* thyroid + *-ine* pertaining to, a substance] (T_3) is a very big way of saying that this thyroid hormone contains three iodine atoms. In the name ***tetraiodothyronine*** [*tetra-* four] (T_4) we've merely changed the prefix, telling us that there are four iodine atoms in the hormone.

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Iodine? Yep, iodine. Just a little bit of dietary iodine is necessary for normal thyroid function. Obviously, it's an essential part of the molecular structure of triiodothyronine and tetraiodothyronine, as the names imply. Now, I'll admit, these are really cumbersome words. I think most ***endocrinologists*** [*endocrin(o)-* hormone + *log(o)-* study, knowledge + *-ist* one who specializes in] would agree. That's probably why most folks simply abbreviate and refer to them as T_3 and T_4. Just so you know, T_4 is also called ***thyroxine*** (thi-roks'in).

Both T_3 and T_4 are important for ***metabolism*** [*metabol(o)-* to change + *-ism* state or process of]. Metabolism is the process of turning usable fuel (carbohydrates, proteins, and fats) into energy for cellular activity. Important cellular activities include rebuilding and repairing tissues, protein synthesis, and so much more. All of those activities require an awful lot of energy.

The ***metabolic*** [*metabol(o)-* to change + *-ic* pertaining to] ***rate*** is the set point, if you will, for an individual's caloric needs. We're all just a little bit different. A higher metabolic rate means that the individual will need and burn more fuel. A lower metabolic rate means that the individual will need and burn less fuel. It's all about supply and demand. This is probably best demonstrated by comparing a young, growing animal to a ***geriatric*** [*ger(o)-* age + *-iatric* pertaining to; i.e., old] animal. The youngster has a much higher metabolic rate to support all of its cellular activity for growth. The geriatric adult has a much lower metabolic rate. After all, it takes very little energy to sleep 18 hours a day, as geriatric animals often do. What controls this rate? Well, it's a cooperative arrangement between the hypothalamus and the anterior pituitary. The hypothalamus, which also controls the body's temperature setting, tells the anterior pituitary to secrete *thyroid stimulating hormone (TSH)*. TSH then tells the thyroid to secrete T_3 and T_4. In the presence of these thyroid hormones, cells of the body can take in and burn fuel for energy for all of their important activities. A by-product of this activity is heat. Aha! Do you see why I mentioned that the hypothalamus controls body temperature? Metabolism and body temperature are closely linked.

Think about it: a person or animal living in a cold climate needs to burn a lot of fuel to produce enough heat to stay warm. So, the hypothalamus, receiving all of the sensory information perceiving

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that it's cold outside, increases the metabolic rate (and hunger). Burning more fuel will maintain a stable core body temperature, in spite of the environmental conditions (to a point of course).

In terms of a negative feedback loop, there's a lot going on here, isn't there? First, we have the sensory input to the hypothalamus (core and environmental temperatures, etc.). In response to that input, the hypothalamus increases hunger and tells the anterior pituitary to secrete more TSH. The thyroid responds to the TSH by secreting T_3 and T_4. Metabolic rate increases. The body produces more energy to maintain the thermostatic setting for the core body temperature. When the perfect balance of energy and body temperature is achieved, the hypothalamus may tell the anterior pituitary to back off a little on TSH secretion. We don't want to burn too much fuel. After all, if we're going to remain in a hostile, cold environment (like a seal, a polar bear, or cattle on wintry pasture), we'd like to keep an insulating layer of fat and a thicker coat of fur. Less TSH translates to less secretion of T_3 and T_4. If this same animal is taken to a warmer environment, the negative feedback mechanisms will result in more dramatic reductions of TSH and, thereby, thyroid hormones. That's how negative feedback works. The whole point is to maintain a given set point. In this case, the set point of the metabolic rate goes hand in hand with the set point for body temperature.

Calcitonin

Another very important thyroid hormone is ***calcitonin***. The *calc(o)-* root in the name gives a clue to what the hormone does. It helps control calcium (*calc(o)-*) homeostasis. We need just the right amount of calcium readily available in the bloodstream. We have talked about the importance of calcium in a number of other chapters, for things like clotting, maintaining healthy bones, bone growth and repair, nerve transmission, muscle contraction, and milk production. Too much or too little calcium in the blood and tissues can create devastating disease problems. Calcitonin is secreted when there is too much calcium in circulation. It reduces blood calcium in two very important ways. First, it causes calcium to be deposited into bone. This is like putting money in the bank. (We'll talk later about making a withdrawal from the bank, when

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we discuss the parathyroid glands.) The other way calcitonin helps reduce blood calcium is by stimulating greater excretion of calcium by the kidneys. I think with this hormone, it's a little bit easier to see how negative feedback works. It's all about maintaining a narrow range of blood calcium levels. In ***hypercalcemia*** [*hyper-* excess + *calc(o)-* calcium + *-emia* a blood condition of], the thyroid will be stimulated to secrete more calcitonin. If there's too little calcium, secretion of calcitonin will be decreased or stopped altogether.

Goiter

The term ***goiter*** comes from the Latin word *guttur*, meaning "throat." I guess this makes sense since the thyroid gland is located in the throat region. Generally, we can't see or easily feel the thyroid gland when performing a physical exam. But in *goiter*, the gland becomes enlarged. It usually results from insufficient dietary iodine. This gives us a great demonstration of how hormonal feedback loops work. If we don't have enough dietary iodine, we can't produce enough T_3 or T_4, right? Iodine is absolutely necessary to build those hormonal molecules. With deficient T_3 and T_4, our metabolic needs cannot be met. So the hypothalamus stimulates the anterior pituitary to secrete more TSH. With the excessive stimulation by TSH, the thyroid gland actually becomes ***hyperplastic*** [*hyper-* excess + *plas(o)-* formation, development + *-tic* pertaining to]. The ***hyperplasia*** [*hyper-* excessive + *plas(o)-* formation, development + *-ia* condition of] eventually becomes visible and palpable. Yes, there are other things that could cause thyroid enlargement, like cancer. Only a ***biopsy*** [*bi(o)-* life + *-opsy* viewing; i.e., surgical removal of tissue for microscopic examination] or a fine-needle aspirate will distinguish hyperplasia from cancer. Of course, all the hyperplasia in the world won't increase production of the needed hormones if we don't have the necessary building blocks (iodine). Goiter is more common in people than in animals. Regardless, it is easily treated with a balanced diet that contains sufficient dietary iodine. Once the thyroid has all of the essential building blocks to create T_3 and T_4, and it secretes enough to meet the body's metabolic needs, the

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anterior pituitary will reduce its secretion of TSH. With less stimulation of the thyroid, the hyperplasia will resolve.

Hyperthyroidism

Hyperthyroidism [*hyper-* excess + *thyroid* + *-ism* state or condition of] is most common in middle-aged to older cats (usually over 8 years of age). For whatever reason, the thyroid of a hyperthyroid cat secretes way too much T_3 and T_4. The question is: why? There are many suspect ***etiologies*** [*eti(o)-* cause + *-logy* study/knowledge of; i.e., causes], some proven, such as benign and malignant forms of cancer. Most of the time, hyperthyroidism is an idiopathic disease. Whether we know the cause or not, the end result is the same: the metabolic rate is exponentially increased. Consequently, these cats can become extremely skinny. They may have ravenous appetites, but no matter how much they eat, the food can't provide enough fuel to burn. These cats may also be unusually restless, agitated, or even aggressive (my cat was SO grumpy!). They may experience polyuria-polydipsia (PU/PD), as well as vomiting and diarrhea. Hyperthyroidism can also create serious, life-threatening problems like ***hypertrophic*** [*hyper-* excess + *troph(o)-* development + *-ic* pertaining to] ***cardiomyopathy*** [*cardi(o)-* heart + *my(o)-* muscle + *-pathy* disease of]. Left unchecked, *hypertrophic cardiomyopathy* can lead to heart failure and ***thromboembolism*** [*thromb(o)-* clot + *embol(o)-* to throw in + *-ism* process, state of; i.e., throwing a clot]. As you can see, hyperthyroidism creates a host of problems for the cat. This is not just a matter of having a skinny cat. The condition needs to be treated.

Some endocrinologists would recommend ***thyroidectomy*** [*thyroid* + *-ectomy* to cut out; i.e., surgical removal], especially in cases of unilateral thyroid changes. Removing one thyroid lobe may return thyroid function to normal. However, bilateral thyroidectomy dramatically impacts not only metabolism but also calcium homeostasis. You see, the ***parathyroid*** [*para-* near + *thyroid*] ***glands*** are nestled on the thyroid. If we remove both thyroid lobes, the parathyroid glands come along for the ride. This would leave us with no calcitonin or parathyroid hormone (PTH)—a problem indeed when it comes to managing calcium. Aside from that, sometimes surgery and anesthesia are simply too risky. After all,

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we are often dealing with geriatric cats with this condition. Fortunately, we can treat these cats medically. They are given ***antithyroid*** [*anti-* against + *thyroid*] drugs such as methimazole. How do they work? They basically inhibit the secretion of T_3 and T_4 by blocking the thyroid's ability to incorporate iodine into the hormonal molecules. Regular physical examination and laboratory evaluation of T_3 and T_4 levels are important for these cats. The medication is adjusted, based on clinical and laboratory data, to establish and maintain optimal T_3 and T_4 secretion. Once she was on methimazole, my cat was much happier and healthier.

Hypothyroidism

In ***hypothyroidism*** [*hypo-* below, insufficient + *thyroid* + *-ism* state or condition of], we have too little T_3 and T_4. Hypothyroidism is most common in dogs. Most often, this ***endocrinopathy*** [*endocrin(o)-* hormones + *-pathy* a disease of] results from ***autoimmune*** [*auto-* self + *immune* protection] ***thyroiditis*** [*thyroid* + *-itis* inflammation of] or *idiopathic* thyroid ***atrophy*** [*a-* not, without + *troph(o)-* development]. In the autoimmune disorder, for whatever reason, the body's immune system targets and progressively destroys thyroid tissue. In the idiopathic ***atrophic*** [*a-* not, without + *troph(o)-* development + *-ic* pertaining to] disorder, functional thyroid tissue, for all practical purposes, shrinks. In either case, the thyroid simply cannot produce enough T_3 and T_4. The anterior pituitary may be secreting tons (figuratively, of course) of TSH, but to no avail. It's trying to communicate, but its words are falling on deaf ears. Talk about a breakdown in communication! The end result is too little T_3 and T_4.

Now, whenever the body has a deficiency like this, it tries to be selective in supporting essential organs and tissues. A luxurious, shiny hair coat is probably not a top priority. So, we tend to see problems with the skin and hair coat. Protein synthesis and mitosis to provide for routine turnover and growth of hair simply can't be supported, so the coat is often dull and thin, especially over the trunk of the body. Other skin issues, including ***dermatitis*** [*dermat(o)-* skin + *-itis* inflammation of] may occur. And because the body can't burn fuel as efficiently, any excess calories are tucked away in fat storage. Weight gain happens in spite of significantly

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reduced caloric intake. The fat provides some needed insulation in animals whose ability to generate body heat is impaired. In fact, dogs with hypothyroidism often seek ***exogenous*** [*ex-, exo-* out, outside + *gen(o)-* produce + *-ous* pertaining to; i.e., outside the body] heat sources such as lying in the sunshine or next to heat ducts. And because metabolism at the cellular level is slow, overall activity of the animal is greatly reduced. They become very ***lethargic*** [*letharg(o)-* drowsy, *lethargy* drowsiness + *-ic* pertaining to].

Diagnosis of hypothyroidism depends on patient history, clinical evidence, and laboratory analysis of TSH and thyroid hormones. If possible, the etiology is treated (e.g., with immunosuppressants for autoimmune thyroiditis). Beyond that, we need to do something about the T_3 and T_4 deficiencies. Fortunately, for any animal with hypothyroidism, we can replace what the thyroid is not producing. We administer oral synthetic thyroxine (T_4). What about T_3? Well, much of T_4 is converted to T_3, even in normal animals, so thyroxine replacement therapy covers both. With exogenous thyroxine, metabolic homeostasis can be reestablished, so we can transform a rough-looking, lazy, fat slug of an dog into a healthy-looking, active one. This is what I call better living through chemistry!

Is it possible for cats to develop hypothyroidism? You betcha! However, this is usually an ***iatrogenic*** [*iatr(o)-* physician + *gen(o)-* produced + *-ic* pertaining to] problem. Remember, we said that hyperthyroid cats can be treated surgically or medically. Either one of those could result in hypothyroidism. Now, after a bilateral thyroidectomy, we can't put the thyroid back in, but we can give exogenous thyroxine to make up for the deficit we created. If the hypothyroidism was caused by an antithyroid drug like methimazole, reducing the dose of the drug should resolve the problem. This is why regular monitoring is so important. We don't want iatrogenic disease to develop by fixing one problem that in turn creates another.

Parathyroid Glands

As you can see in Fig. 10.2, the parathyroid glands are tiny structures found on top of the thyroid gland. There are four parathyroid glands. Because calcium homeostasis is so important,

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the redundancy is a good thing. What do parathyroid glands secrete? ***parathyroid hormone (PTH)***. There's a no-brainer, eh?

Parathyroid Hormone (PTH)

Do you remember from our thyroid discussion of calcitonin that we said calcitonin reduced blood calcium levels by putting calcium in the "bank" (i.e., bone)? Well, parathyroid hormone (PTH) permits us to take withdrawals of calcium from that "savings account." Remember, we need just the right amount of calcium in the blood and body tissues. Calcium is needed for clotting, muscle contraction, nerve transmission, and milk production, to name a few. So, if for whatever reason, ***hypocalcemia*** [*hypo-* low, insufficient + *calc(o)-* calcium + *-emia* a blood condition of] develops, the parathyroid gland will be stimulated to secrete PTH. In the presence of PTH, calcium can be extracted from bone. PTH also increases absorption of calcium from the digestive tract in the presence of vitamin D and increases reabsorption of Ca^{++} from the kidneys.

The feedback loop is a complicated one that involves both the thyroid and parathyroid glands. It's complicated because there are so many factors that influence it, such as diet and vitamin D. From a purely endocrine perspective, there is a yin-yang relationship between calcitonin from the thyroid and PTH from the parathyroid glands. So, you already know that calcitonin facilitates calcium deposits in bone to reduce blood calcium levels. Vitamin D is necessary for this to happen. Calcitonin also decreases Ca^{++} absorption from the gut and increases renal excretion of Ca^{++}. PTH creates the opposite effects. Which hormone wins the day all depends on blood calcium concentrations. The whole point here is to maintain blood calcium within an optimal range. Generally speaking, the give-and-take between calcitonin and PTH results in only minor fluctuations of blood calcium, maintaining it within a normal range. This is another great example of negative feedback loops.

Hypocalcemia

So, if these endocrine mechanisms are so efficient, how could

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hypocalcemia [*hypo-* low + *calc(o)-* calcium + *-emia* a blood condition of] possibly develop? Well, we could have a diet with insufficient calcium and/or vitamin D. Too little exposure to sunshine decreases production of vitamin D by the body. This is often a problem in birds and especially in reptiles. (That's why it's important to provide an artificial UV light source for pet reptiles.) Now, the parathyroid glands might be able to maintain adequate blood calcium levels for a while. In fact, PTH can promote excellent resorption of calcium from bone. Unfortunately, this can leave the bones so weak and brittle that normal movement of the animal results in fractures. When there's nothing left in the bank—account closed (death).

Among mammals, the most common cause of ***acute*** (sudden) hypocalcemia is ***lactation*** [*lact(o)-* milk + *-tion* process, state of milk; i.e., milk production]. We learned earlier that prolactin promotes milk production. Where does the mammary gland get the calcium? From the bloodstream. So, shortly after giving birth, a female animal needs to produce enough milk to provide for its offspring. This sudden calcium demand for milk production can rapidly deplete calcium from the blood. Will the parathyroid glands respond by stimulating ***osteoclastic*** [*oste(o)-* bone + *clast(o)-* to break + *-ic* pertaining to] activity to resorb bone? Yes. But that takes time. In the meantime, we have a clinically ill animal. This could be life-threatening. We can get calcium more rapidly from other ***extracellular*** [*extra-* outside + *cellul(o)-* cells + *-ar* pertaining to] and ***interstitial*** [*inter-* between + *stiti(o)-* tissue + *-al* pertaining to] fluids.

In fact, we can draw on calcium that's in muscle tissue. That's certainly easier than breaking down bone. But what happens if we rob the muscle of calcium? It can't contract as well. It becomes weak. This is why cows often go down ("downer cows") in "milk fever" (hypocalcemia). Their muscles are simply too weak to support their body weight. How might the hypocalcemia affect nerve function? Well, we need enough calcium to keep the lid on ionic exchange for nerve transmission. Too little calcium may allow those channels to fly wide open and cause excessive nerve stimulation of muscles. This is why in ***eclampsia*** (e-klamp'se-uh), as we call it in dogs and cats, we often see muscle twitching and tremors. If the hypocalcemia is bad enough, we can see full-blown

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seizures (nerve pathways in the brain fly open, too). Imagine how this is affecting the heart, electrically as well with regard to heart muscle functionally. That's why hypocalcemia can be life-threatening.

Obviously, severe hypocalcemia like this is beyond anything the endocrine system can manage on its own. We need to intervene by administering ***intravenous*** [*intra-* within + *ven(o)-* vein + *-ous* pertaining to] calcium. This is not without risk either. If we infuse calcium too rapidly, we can cause an extreme drop in blood pressure, irregular heartbeats, or even ***cardiac*** [*cardi(o)-* heart + *-ac* pertaining to] ***arrest***. So, cardiovascular function should be monitored very closely during the administration.

Hypercalcemia

Why bring up hypercalcemia in a section about the parathyroid glands? Shouldn't we have talked about that along with calcitonin from the thyroid? Perhaps. But ***hyperparathyroidism*** [*hyper-* excess + *parathyroid* + *-ism* state or condition of] can cause hypercalcemia, most often in older dogs. There is usually ***pathology*** [*path(o)-* disease + *-logy* knowledge, study of] of one or more of the parathyroid glands. It may simply be hyperplasia, or it could be cancer. Regardless, the abnormal parathyroid glands secrete too much parathyroid hormone. This in turn stimulates excessive osteoclastic activity, releasing calcium from the bone. This is such an abnormal situation that it's not something that the thyroid can fix with calcitonin.

Of course, we need to rule out other causes of hypercalcemia, like ***osteosarcoma*** [*oste(o)-* bone + *sarc(o)-* flesh + *-oma* tumor; i.e., an aggressive form of bone cancer]. If other causes are ruled out, then our suspicion of hyperparathyroidism is even greater. Diagnostic imaging, like ultrasound or CT scans, may help us identify abnormal parathyroid glands. Checking PTH levels may also help confirm a diagnosis of hyperparathyroidism. Ultimately, we can perform a ***parathyroidectomy*** [*parathyroid* + *-ectomy* to cut out; i.e., surgically remove]. When I was working in surgery, most of the time we could actually grossly (i.e., with the naked eye) distinguish between normal and abnormal parathyroid glands. Any that appeared suspicious were ***excised*** [*ex-* out + *cis(o)-* cut; i.e., cut out].

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Usually one or two parathyroid glands were removed. The glands were always sent out for ***histopathology*** [*hist(o)-* tissue + *path(o)-* disease + *-logy* study of; i.e., microscopic study of tissue for disease]. Histopath findings would help determine ***prognosis*** (forecast of outcome) and any follow-up care necessary, beyond routine supportive care. ***Postoperatively*** [*post-* after + *operat(o)-* surgery + *-ive* pertaining to], these patients were carefully monitored. Calcium levels were checked frequently. We were looking and hoping for a gradual reduction in blood calcium. But we always needed to be vigilant, to catch signs of hypocalcemia early. Once stable blood calcium levels were achieved, the patients were discharged. Most of these patients recovered very well.

Adrenal Glands

Here is another pair of endocrine glands that are really small but have a huge impact. The ***adrenal*** [*ad-* toward, near + *ren(o)-* kidney + *-al* pertaining to] ***glands*** are located, as their name implies, near the kidneys (Fig. 10.3). Each adrenal gland is subdivided into the ***adrenal cortex*** [L. *cortex* bark, rind, shell] at its outer borders and the ***adrenal medulla*** [L. *medulla* inward part] at its center. The whole gland is enveloped by a connective-tissue capsule. ***Adrenocortical*** [*adren(o)-* adrenal gland + *cortic(o)-* cortex + *-al* pertaining to] hormones are a collection of ***endogenous*** [*endo-* inside, within + *gen(o)-* produced + *-ous* pertaining to] steroids. The principal ***adrenomedullary*** [*adren(o)-* adrenal gland + *medull(o)-* medulla + *-ary* pertaining to] hormone is epinephrine (a.k.a. adrenaline). We'll discuss the implications for each of these separately.

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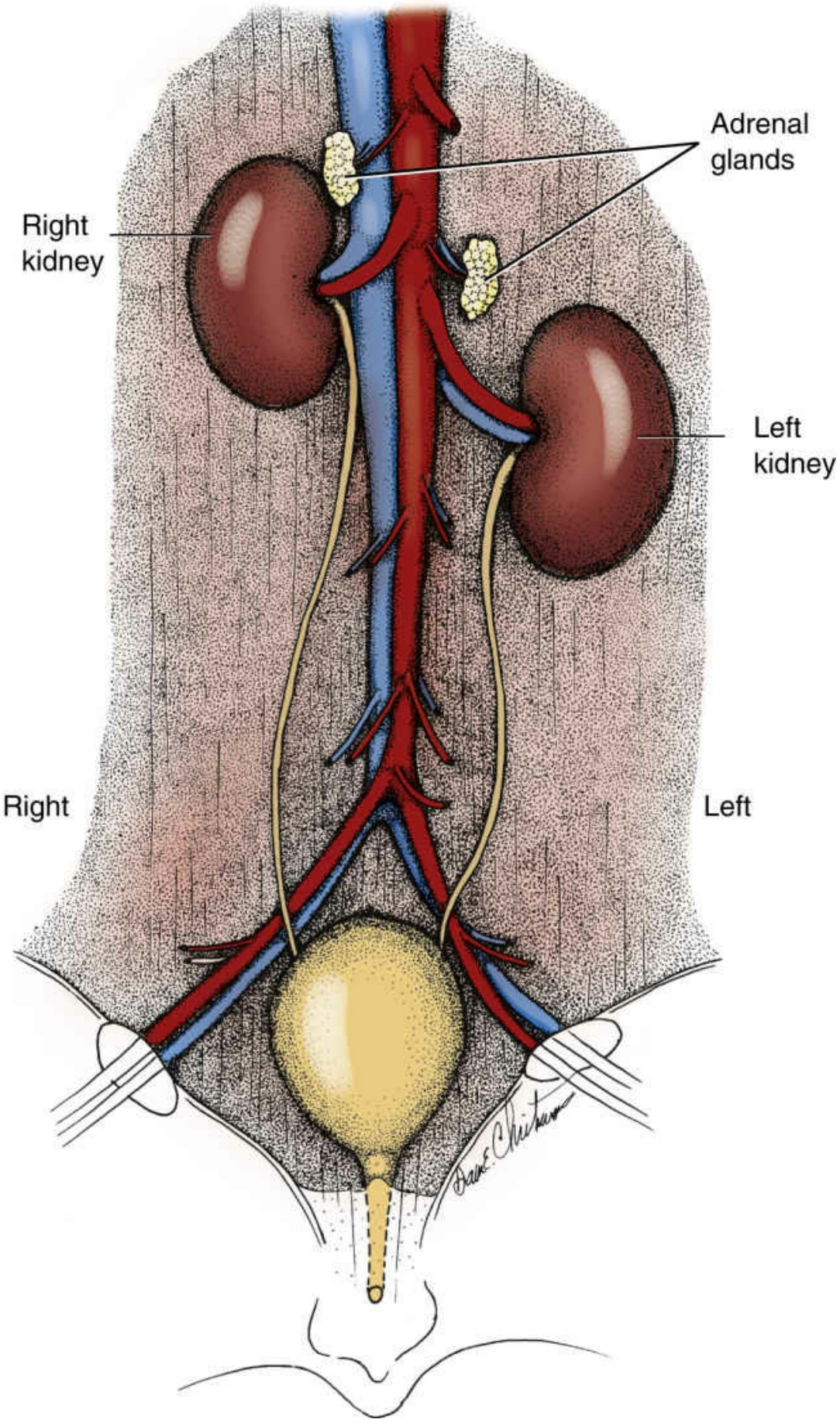

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FIG. 10.3 Adrenal glands.

Adrenocortical Hormones

Earlier, when we talked about the anterior pituitary, we mentioned ***adrenocorticotropic hormone (ACTH)***. ACTH is the principal driving force for hormone secretion from the adrenal cortex. Adrenocortical hormones secreted by the adrenal glands are types of ***steroids***. I would love to break down the word *steroid* for you, but that's not possible. What I *can* tell you is that that steroids are huge ***lipid*** [*lip(o)-* fat] molecules (sterols) with long chains of carbon, hydrogen, and oxygen atoms. There are many steroids produced in the body. The adrenal glands don't have a corner on the market, by any means. Believe it or not, even cholesterol produced by the liver is considered a steroid. There are numerous ***corticosteroids*** [*cortic(o)-* cortex + *steroid*] produced by the adrenal cortex. Rather than enumerate each one, we'll discuss them based on function.

Mineralocorticoids

Mineralocorticoids [*mineral* + *corticoids* adrenal cortex steroids] help regulate electrolytes in the body. (If you need to review key electrolytes of the body, go back to Chapter 2.) Recognize that electrolyte concentrations and movements into and out of the body are largely controlled by mineralocorticoids. ***Aldosterone*** is a key mineralocorticoid. Aldosterone is most important for homeostasis of sodium, potassium, and hydrogen ions. Its principal target to manage these electrolytes is the kidney. That's handy since the adrenal glands are so close to the kidneys. In response to aldosterone, the kidney will resorb sodium. Where sodium goes, water follows, so aldosterone also affects hydration. Aldosterone also increases excretion of potassium and hydrogen ions in the urine. And because hydrogen ions are linked to acid-base balance in the body, aldosterone also impacts acid-base homeostasis. Do you see what a complicated web is hormonally woven into the functional fabric of the body?

Glucocorticoids and Stress

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Glucocorticosteroids [*gluc(o)-* glucose, sugar + *corticosteroid*] include hormones like cortisone and cortisol. You've probably heard of cortisone especially by the name commonly used for pharmaceutical products containing it: hydrocortisone. This is what we classically think of when we think of steroids. But you might be wondering: what does glucose have to do with these steroids? Well, most glucocorticoids increase blood glucose. How do they do this? They actually promote ***gluconeogenesis*** [*gluc(o)-* glucose + *ne(o)-* new + *gen(o)-* production + *-sis* process of] by the liver. In the presence of glucocorticoids, the liver can break down complex molecules, like proteins and fats, and convert the by-products to useable glucose. Remember, glucose is the principal fuel for most cells of the body. This is especially true for nerves. Most cells burn glucose in the presence of oxygen for energy. We call this ***aerobic*** [*aerob(o)-* air, oxygen + *-ic* pertaining to] ***glycolysis*** [*glyc(o)-* glucose + *-lysis* process of breaking]. In situations where there is inadequate intake of easily burned fuel (from carbohydrates), gluconeogenesis provides another way to produce energy. In starvation, it's about the only means for producing energy. Unfortunately, proteins are broken down as well as fats. So, in starvation, we not only lose fat but also break down proteins from muscle and other tissues. Remember that the next time you go on a fad diet.

Glucocorticoids have other protective functions for the body, too. For example, they reduce inflammation. Especially in ***chronic*** [*chron(o)-* time + *-ic* pertaining to; i.e., long-term] disease, a phenomenon called a ***glucocorticoid stress response*** occurs. Stress, emotional or physical, can activate this glucocorticoid response. The intent of it is strictly protective, providing energy from gluconeogenesis and antiinflammatory effects. Unfortunately, there is a dark side to this phenomenon: ***immunosuppression*** [*immun(o)-* immunity, protection + *suppress* depressed, reduced + *-ion* state of]. Glucocorticoids, whether endogenous or exogenous, have this impact. That means less antibody production. White blood cells (principally neutrophils) can't exit the bloodstream to fight ***pathogens*** [*path(o)-* disease + *gen(o)-* producer] on the front lines in the tissues. (See Chapter 3 if you need to review blood and immunology.) Risk of serious infection increases in the presence of glucocorticoids. Wound healing is significantly impaired by

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glucocorticoids. And from chronic exogenous glucocorticoids, especially at higher doses than the body produces, the breakdown of proteins for gluconeogenesis can be profound. This can produce really thin skin, hair loss, and loss of muscle mass. This happens in response to endogenous glucocorticoids, too, but usually not as dramatically. These are consequences to remember when we administer long-term steroidal therapy. Exogenous corticosteroids, like prednisone, prednisolone, and dexamethasone, are frequently administered to patients with allergies and autoimmune disorders.

Whether endogenous or exogenous, these consequences of glucocorticoids can put our patients on shaky ground. Imagine a dog with a chronic systemic disease of some sort (heart disease, kidney disease, etc.). If such a patient required surgery, the surgical site could take much, much longer to heal. That means removal of skin sutures or staples should be delayed so that the site does not break open. (Imagine that with abdominal surgery.) Because of the immunosuppressive effects, the surgical site is at risk of infection. In fact, if the patient is hospitalized and receiving intravenous (IV) fluids, the IV catheter site is at risk of infection. So, because we know the risks created by glucocorticosteroids, we need to protect these patients from secondary infections.

Hypoadrenocorticism (Addison's Disease)

Hypoadrenocorticism [*hypo-* below, low, insufficient + *adren(o)-* adrenal + *cortic(o)-* cortex + *-ism* state or condition of], also called ***Addison's disease***, may be primary or secondary. Secondary hypoadrenocorticism is usually iatrogenic. By administering exogenous glucocorticoids, we interrupt the normal feedback mechanisms. In a normal situation, the hypothalamus will detect low levels of glucocorticoids and tell the anterior pituitary to secrete adrenocorticotropic hormone (ACTH). In turn, the adrenal cortex will secrete more glucocorticoids. But when we administer exogenous glucocorticoids, high levels of glucocorticoids are detected in the blood. The perception by the hypothalamus is that there is no need to tell the anterior pituitary to secrete ACTH. "Why bother?" the hypothalamus says. "We're fine! We have plenty of glucocorticoids." Plus, with chronic administration of exogenous glucocorticoids, especially at higher doses, the adrenal cortex may

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actually atrophy (shrink). Now, if the exogenous steroidal therapy is suddenly withdrawn, the patient is left with a severe deficiency of glucocorticoids. This is why we never want to abruptly stop corticosteroid therapy. Doses should always be tapered. With gradual reduction of the exogenous steroids, the adrenal cortex is given a chance to recover, ultimately resuming its normal responsiveness to ACTH. Afterward, the adrenal cortex will typically resume normal function, but that may take a month or so. In the meantime, we'll have an ill animal that is temporarily at risk of low blood sugar—a big problem if the animal feels so ill that it doesn't want to eat.

In primary hypoadrenocorticism, it's a different story. The etiology is usually classified as idiopathic, likely from progressive autoimmune destruction of the adrenal cortex. This affects both glucocorticoid and mineralocorticoid production. Often, we don't see these patients until they present in an Addisonian crisis. That's a serious, life-threatening problem.

It's one thing to have a deficiency of glucocorticoids. With that, inflammation of any kind will be poorly controlled, and we won't have gluconeogenesis to provide energy. And if the animal has not been eating, which is typical, they may develop ***hypoglycemia*** [*hypo-* low + *glyc(o)-* glucose + *-emia* a blood condition of]. Sometimes the hypoglycemia is severe. Couple that with deficient mineralocorticoids and we have really big problems. Electrolyte disturbances create havoc in the body. First, remember that aldosterone is responsible for resorption of sodium by the kidneys. Without aldosterone, we lose sodium by the truckload in the urine. ***Hyponatremia*** [*hypo-* low + *natr(o)-* sodium + *-emia* a blood condition of] has a significant impact on the nervous system. It can severely depress mental acuity and level of consciousness. While working in emergency, I saw a number of animals in Addisonian crisis that were either barely responsive or unconscious. Those who were responsive were extremely weak—another symptom of hyponatremia. Second, you may recall that we said, earlier, "Wherever sodium goes, water follows." So if we lose lots of sodium in the urine, we'll lose lots of water, too. This can lead to severe ***dehydration*** [*de-* reduced + *hydr(o)-* water + *-tion* state of]. And if the patient is vomiting, as they often are, the dehydration

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can be significant and lead to dangerously low blood pressure. Third, aldosterone is responsible for renal excretion of potassium. Without aldosterone, the kidneys cannot remove potassium. ***Hyperkalemia*** [*hyper-* excess + *kal(o)-* potassium + *-emia* a blood condition of] can significantly slow and even stop the heart. A slow heart rate with low blood pressure is a very bad thing. Finally, remember that aldosterone is responsible for hydrogen ion excretion in the urine. Without aldosterone, the animal retains excess hydrogen ions, producing ***acidosis*** [*acid(o)-* acid + *-sis* condition of]. Suffice it to say that these patients are critical for a multitude of reasons.

Mind you, patients don't develop Addisonian crises all of a sudden. Their hypoadrenocorticism has a progressive onset (unless it is iatrogenic, as we mentioned earlier). But the signs tend to be so gradual and so subtle that many owners are unaware of the serious problem that is brewing. It's often not until the animal collapses or becomes so depressed and unresponsive that they rush it in to the emergency room. Sadly, not all patients in an Addisonian crisis will survive. That's how critical they are. If they do survive, they will require intensive care to stabilize them. The period of hospitalization may be long. With persistent adrenocortical insufficiencies, these patients require replacement therapy of both mineralocorticoids and glucocorticoids for the rest of their lives.

Hyperadrenocorticism (Cushing's Disease)

The etiology of ***hyperadrenocorticism*** [*hyper-* excess + *adren(o)-* adrenal gland + *cortic(o)-* cortex + *-ism* state or condition of], or ***Cushing's disease***, may also be iatrogenic. Here again, if we administer long-term glucocorticoid therapy at higher doses, the exogenous steroids can create signs of hyperadrenocorticism. What might those signs be? Well, typical side effects of steroidal therapy include panting, polyuria-polydipsia (PU/PD), ravenous appetite, and immunosuppression. Owners will often complain about the panting, PU/PD, and ravenous appetite, but immunosuppression may go unnoticed unless the patient experiences chronic, recurrent infections. Muscle wasting may go unnoticed, especially in thick-coated animals. But the hair loss (primarily over the trunk) and thin skin are obvious. The skin can become paper-thin, making it tear

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extremely easily. This could happen when an animal is being groomed—no fault of the groomer! And because of the steroids, healing of even the smallest skin injury will take seemingly forever. The other noticeable change is the large, distended (pot-belly) abdomen. The animal looks fat in the belly, but when you palpate the rest of the body, the animal may feel skinny elsewhere. We can fix this by *gradually* reducing the dosage of glucocorticoids and eventually discontinuing therapy. Remember, after chronic, higher doses, therapy should never be abruptly stopped.

In patients who develop hyperadrenocorticism and have never received exogenous glucocorticoids, we need to investigate the only two players that could cause this: the anterior pituitary and the adrenal glands. Acute (sudden) onset of Cushing's disease often points to ***neoplasia*** [*ne(o)-* new + *plas(o)-* formation + *-ia* condition of; i.e., tumors, cancer]. Pituitary and adrenal tumors can result in hyperadrenocorticism. If the overall condition of the patient is good and the location of the tumor is accessible, surgery may remedy the hyperadrenocorticism. If surgery is not an option, chemotherapy may reduce tumor size and adrenocortical hormone production. Once reduced, maintenance therapy may be required to prevent regrowth of the tumor.

Adrenomedullary Hormones

The adrenal medulla is the central portion of the adrenal glands. The hormones produced here include ***epinephrine*** (adrenaline) and ***norepinephrine***. Norepinephrine is also produced by nerves for ***neurotransmission*** [*neur(o)-* nerve + *transmission* act of transmitting] along the ***sympathetic*** [*sympath(o)-* sympathy + *-tic* pertaining to] branch of the ***autonomic*** [*auto-* self + *nom(o)-* control + *-ic* pertaining to; i.e., automatic] ***nervous system***. It is used as a neurotransmitter at the motor end plates of the sympathetic pathway. (If you need a refresher on neurotransmission at a motor endplate, you may refer to Chapters 4 and 11. Chapter 11 also contains detailed information on the autonomic nervous system.)

In brief, there are two branches of the autonomic nervous system: the sympathetic branch (fight or flight) and the parasympathetic branch (rest and repose). Most major organs and organ systems receive nerve input from both branches. One acts as an accelerator

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for activity and the other acts as a brake. The adrenal gland is the only organ exclusively controlled by the sympathetic branch. The purpose of the sympathetic branch is self-preservation, in response to an immediate threat. All of the activity in the body stimulated by the sympathetic branch gives us the ability either to run like crazy to get away from a threat or to stand and fight for our lives. These activities stimulated by the nerve impulses are immediate. But we have a limited supply of neurotransmitters at each motor end plate. We need a mechanism to sustain this response so that we can either get far away from or neutralize the threat. That's where the ***adrenomedullary*** [*adren(o)-* adrenal gland + *medull(o)-* medulla + *-ary* pertaining to] hormones come into the picture.

Epinephrine, Norepinephrine, and Fight or Flight

Both epinephrine and norepinephrine are secreted by the adrenal medulla. Of the two, epinephrine is the predominant hormone produced. When a threat is perceived and the sympathetic system is activated, the adrenal medulla will secrete tons of epinephrine. And epinephrine has a ***sympathomimetic*** [*sympath(o)-* sympathy + *mime(o)-* imitation + *-tic* pertaining to] effect, literally imitating what the sympathetic nerves do. Okay, fine: so epinephrine will sustain what the sympathetic nervous system started. But what did those nerves start?

First, in fight or flight, we need to see the threat, right? So one effect is dilation of the pupils. Second, whether we are going to run or fight, we need to engage large muscle masses, especially in our legs and arms, so we increase blood flow to those muscles. And third, because we have a limited volume of blood, in order to maintain adequate blood pressure to keep our brain crystal clear while we divert blood to our muscles, we increase the rate and contraction strength of the heart (making it feel like it will pound out of our chest) and shut down blood flow to nonessential areas. One of those nonessential areas is the digestive tract. Now is not the time to be digesting a meal. In fact, to lighten the load, we might even "fill our britches." The other nonessential area is the skin, so we experience peripheral ***vasoconstriction*** [*vas(o)-* vessel + *constrict* make smaller, contract + *-ion* act, state or process of]. This is why when someone is frightened they "turn white as a sheet." We can't

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see this, but the spleen contracts, too. With potentially 30% of our available blood volume stored in the spleen, squeezing it out of there can really improve our circulating blood volume. Finally, we need lots of oxygen to support a clear mind and heightened awareness, as well as all of our muscle activity, so our airways dilate, allowing us to easily take deep, rapid breaths. All of this happens in a split second, stimulated by the sympathetic nerve fibers. And because the adrenal medulla is stimulated by sympathetic nerve fibers at the same time, the body is simultaneously bathed in large quantities of epinephrine. But epinephrine does not have access to the motor end plates. It needs different receptors.

α-Adrenergic and β-Adrenergic Receptors

We have two different types of receptors that respond to epinephrine: ***α-adrenergic*** [*adren(o)-* adrenal gland + *erg(o)-* work, working + *-ic* pertaining to] and ***β-adrenergic***. (The "α " and the "β" are characters of the Greek alphabet; spoken "alpha" and "beta" respectively.) Why have two different types of receptors? Well, if the fight-or-flight response is really going to have sympathy for us and save our lives, we need all of this sympathomimetic activity to be well orchestrated. We can't afford to have every vessel in the body dilate at once—there's not enough blood volume to go around. So by placing primarily *α-adrenergic* receptors on vessels in the skin and digestive tract we can cause vasoconstriction there. With β-adrenergic receptors on the heart and vessels supplying the heart, brain, and major muscles, we can dilate those vessels simultaneously. β-adrenergic receptors are found along the airways, too. To keep these receptors straight, I've always thought to myself, "Beta is better," because it provides for better blood flow and breathing.

We can use these receptors to treat patients too. Let's say we have a cat whose small airways are constricted because of an asthmatic crisis; we can give an injection of a β-adrenergic drug to dilate the airways. Believe it or not, epinephrine is the A-number-1 sympathomimetic drug that we use in cardiac arrest. Even if the patient hasn't arrested yet, but its heart is beating weakly and blood pressure is extremely low, both the α- and β-adrenergic effects of

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epinephrine may stabilize the patient until we can fully assess and treat the underlying condition.

Pancreas

The pancreas is a switch-hitter in the body. It has both ***exocrine*** [*ex-, exo-* out, outside + *crin(o)-* secrete; i.e., secreted and sent out of the body, like digestive enzymes] and endocrine duties. I guess this makes sense. It gives the body follow-through, from ingestion, digestion, and absorption of nutrients to final use by cells throughout the body. We talked about the pancreas's exocrine function (i.e., digestive enzymes) in Chapter 7. If you've forgotten where the pancreas is located, please refer to Chapter 7. Our focus here will be on ***pancreatic*** [*pancreat(o)-* pancreas + *-ic* pertaining to] hormones. There are only two: ***insulin*** (in'suh-lin) and ***glucagon*** (gloo'kuh-gon).

Glucagon

Glucagon [*gluc(o)-* sugar + Gr. *agōn* leading, bringing], as its name implies, is another hormone to help us raise blood glucose levels when needed. Like glucocorticoids, glucagon stimulates the liver to produce glucose for the body through gluconeogenesis. Remember, that process is a complicated one because the liver needs to convert complex protein and fat molecules into smaller and simpler glucose molecules. Obviously, this won't cause an immediate, dramatic increase in blood glucose levels. So, in the meantime, glucagon also stimulates conversion of ***glycogen*** [*glyc(o)-* glucose + *gen(o)-* produce] to glucose. You may recall from Chapter 4 that glycogen is the storage form of glucose. Glycogen is stored in muscle and the liver. Muscle tissue hoards its glycogen for its own immediate use, but the liver will share with the rest of the body. When glucagon is secreted and bathes the liver, we can easily and quickly elevate blood glucose levels. The gluconeogenesis that was also stimulated will help sustain that increase.

Insulin

As you may recall from Chapter 2, glucose is the principal fuel preferred by cells. It is the A-number-1 choice of fuel for ***neurons***

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[*neur(o)-* nerve + *-on* a, the; i.e., a nerve cell]. But even this simple sugar is a pretty big molecule, so it can't simply diffuse across the cellular membrane. Active transport (facilitated diffusion) is required. Insulin gives the green light to active transport of glucose into cells. Watch the Evolve animation, *Insulin Function*, to see how this works. In addition to insulin, the animation also contains structural information about the pancreas, which may help you understand how the pancreas can secrete so many different things. For instance, after eating a meal, food is digested. This raises blood glucose. But we need to maintain just the right amount of glucose in the blood—not too much, not too little. Neurons, especially in the brain, don't function well outside the set range. So, as the blood glucose begins to rise, the pancreas secretes insulin. In the presence of insulin, glucose can be actively transported into cells. The end result is the lowering of blood glucose and maintaining it in a set range. As the animation explained, any excess glucose (beyond cellular needs) will be moved into the liver and muscle tissue to be stored as glycogen. Some may also be transformed for storage as fat in adipose tissues. Obviously, glucose homeostasis can go haywire, as it does in diabetes mellitus.

Diabetes Mellitus

We mentioned diabetes mellitus earlier, with our discussion of diabetes insipidus. We said that both can produce PU/PD. Certainly, the polyuria is why both have diabetes in their names, because water flows through these patients like water through a siphon. But *mellitus,* in Latin, means "honeyed or sweet"; and in diabetes mellitus (DM), the urine is literally sweet as a result of ***glycosuria*** [*glycos(o)-* glucose + *ur(o)-* urine + *-ia* condition of]. No, I've never tasted it. But I've measured the glucose in the urine of diabetic patients, so I know it's there. By the way, glucose hangs onto water like a sponge. So, with high concentrations of glucose in the urine, water follows. And that is why these animals are ***polyuric*** [*poly-* much + *ur(o)-* urine + *-ic* pertaining to]. Now you know how diabetes mellitus got its name.

In DM, ***hyperglycemia*** [*hyper-* excess + *glyc(o)-* glucose + *-emia* a blood condition of] is present. Why? Well, either the pancreas is not secreting enough insulin or we have a problem with insulin

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receptors. Remember, insulin is necessary to move glucose into cells. Most of the time, in dogs and cats, the pancreas is simply not producing enough insulin. A smaller percentage of cats have insulin receptors that are unresponsive to insulin. Obesity is thought to cause this. Either way, in DM, active transport of glucose into cells is disabled. That leaves us with an abundance of glucose in the blood. Some will be removed by being let to spill over into the urine. Hence the *glycosuria*. But this is only after blood glucose levels have surpassed the renal threshold. Even then, the kidneys can't possibly remove enough.

Why does diabetes mellitus (DM) develop in the first place? Well, there may be many factors. We've already mentioned obesity. Obesity predisposes animals and people to the development of DM. Many dogs and cats who develop DM have experienced ***pancreatitis*** [*pancreat(o)-* pancreas + *-itis* inflammation of]. That inflammation can be very damaging to the pancreas. I've seen numerous patients return time and time again with episodes of pancreatitis. It seems that once they have pancreatitis, it takes very little to make the pancreas "angry" with inflammation again and again and again. With full-blown pancreatitis, these animals are extremely ill and in pain. Recovery is usually long. Because high fats in the diet can often trigger subsequent episodes of pancreatitis, a low-fat diet is usually recommended during and after recovery. Each episode damages more pancreatic cells. Low-grade pancreatitis may not require hospitalization, but cells are still damaged. Bottom line, eventually we don't have enough insulin-secreting cells. We're not worried about glucagon. We've got glucocorticoids if we need to raise blood glucose. But insulin is the only hormone that can lower blood glucose. Without it, we have hyperglycemia due to diabetes mellitus. Just imagine how the hyperglycemia from DM would be complicated by hyperadrenocorticism. Yikes! But let's stick to purely insulin-dependent DM for this discussion.

Most DM can be effectively managed through dietary controls and exogenous insulin. Dietarily, our goal is to reduce the glucose load by reducing carbohydrates. Carbohydrates can be rapidly digested into glucose. Too many carbs and we cause a major spike in blood glucose. We want stable blood glucose levels. With a diet

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high in protein, low in fat, and really low in carbohydrates, we create a gradual release of glucose through the digestive process.

Most diabetic patients have ravenous appetites. That makes sense since the body's perception is that the animal is starving because it can't get glucose into cells. So to satisfy their appetites by giving them a "full" feeling, many commercial diets will include some indigestible fiber, too. For obese animals, this will also facilitate some weight loss. Animal owners need to be very compliant and regimented. Once we get the patient regulated with steady blood glucose levels, the owner needs to follow through to maintain that. They need to feed a prescribed amount of the special diet at prescribed times. After each meal, a prescribed amount of insulin must be given. Yes, the owners need to learn how to give injections. Why is it important to give the insulin AFTER each meal? What if we give it and the animal doesn't eat? That could create life-threatening hypoglycemia (low blood-sugar). So insulin should always be given *after* meals. The more regimented owners are, the better the DM will be controlled.

If owners fail to do this, they may put the patient at risk of ***diabetic ketoacidosis*** [*ket(o)-* ketones + *acid(o)-* acid + *-sis* condition of] *(DKA)*. While working in ICU, we saw a number of patients with **DKA**. For some, their diabetes mellitus had not been diagnosed yet. Owners missed all of the subtle symptoms, until the animal was critical with DKA. Some diabetic patients "live on the edge," and even if the owners are very compliant, any stressor could put them into a diabetic crisis like this. Many are presented with multiple problems; pancreatitis is common. An infection could precipitate diabetic ketoacidosis. And diabetic animals, because of their chronic disease and glucocorticoid production, are at higher risk of infection. A small percentage of diabetic cats develop insulin resistance. All of this creates a complicated scenario of uncontrolled DM.

You see, in untreated or uncontrolled diabetes mellitus, the body perceives that it needs glucose. The cells can't access glucose, in spite of its abundance. So various hormones will promote the breakdown of proteins and fats. The liver uses fats to produce ketones, and ketones can provide an alternative, albeit inefficient, fuel for cells. Are ketones produced in anyone with poorly

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regulated DM? Yes. But in diabetic ketoacidosis, the production of ketones goes through the roof. Too many ketones in the blood will compound the animal's already developing acidosis. Ketones, along with glucose, will drag even more water from the body into the urine.

As you can see, there are a multitude of factors that contribute to hyperglycemia, ***ketosis*** [*ket(o)-* ketones + *-sis* condition of], acidosis, dehydration, and electrolyte disturbances. That's why DKA is life-threatening. Aggressive, intensive care is required to treat all facets of disease in these patients. It's not easy and, sadly, not everyone will survive.

Reproductive Organs

The ***glandular*** [*glandul(o)-* gland + *-ar* pertaining to] reproductive organs, or ***gonads*** [L. *gonas* procreation], produce hormones. Female gonads are the ovaries and male gonads are the testes. For more information on these structures and reproduction, please refer to Chapter 9. Our focus here is on endocrinology, so we'll focus on gonadal [*gonad* procreation + *-al* pertaining to] hormones and the roles they play in reproduction.

Ovaries

As discussed and shown in Chapter 9, the ovaries are located in the abdomen of a female. The ovaries respond to ***gonadotropic*** [*gonad* + *trop(o)-* changing, influencing + *-ic* pertaining to] ***hormones*** from the anterior pituitary. Those gonadotropic hormones include follicle-stimulating hormone (FSH) and luteinizing hormone (LH).

Estrogens

Estrogens [*estr(o)-* estrus, heat + *gen(o)-* producing] are produced by the ovaries in response to FSH. When we discussed FSH earlier, we learned that it stimulates follicle growth and egg development. The follicle produces estrogens. The more the follicle grows, the more estrogens it produces. Those estrogens alter physical and behavioral characteristics, in preparation for breeding. For instance, in a female dog, the vulva swells. The interior wall of the uterus thickens and increases its blood supply to prepare for fertilized eggs.

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Behaviorally, the female becomes more receptive to breeding. Her receptivity peaks with ***ovulation*** [*ovul(o)-* egg + *-tion* act or process of; i.e., release of the egg from the follicle]. As all of this is happening, feedback to the anterior pituitary causes it to reduce its production of FSH and begin increasing production of LH. Luteinizing hormone, remember, is necessary for the development of the corpus luteum.

Progesterone

In the presence of LH the corpus luteum develops, and the corpus luteum is responsible for the production of ***progesterone*** [*pro-* for + *gest(o)-, gester(o)-* gestation, pregnancy + *-one* a, the, one that is]. Progesterone is literally a hormone *for* pregnancy. Once implantation of a fertilized egg takes place, there is actually hormonal feedback between the uterus, placenta, and corpus luteum, causing continued secretion of progesterone for maintenance of the pregnancy.

Testes

In male animals, the testes or testicles are responsible for the production of ***androgenous*** [*andr(o)-* male + *gen(o)-* producing + *-ous* pertaining to] hormones. The principal ***androgen*** [*andr(o)-* male + *gen(o)-* producer] is ***testosterone*** [*test(o)-* testes + *ster(o)-, steron(o)-* steroid; i.e., a steroidal hormone of the testes].

Testosterone

Yes, testosterone is actually a ***steroidal*** [*steroid* + *-al* pertaining to] hormone. So when you hear about athletes and *doping* with the use of *steroids*, they're not talking about glucocorticoids; they're talking about *androgenous* steroids, like testosterone.

Gonadotropic hormones from the anterior pituitary stimulate the interstitial cells of the testes to produce testosterone. This hormone is responsible for creating all of the physical male characteristics—for example, greater muscle, bone, and hair development. Testosterone is also responsible for stimulating development of accessory reproductive organs, such as the prostate, as well as sperm production. Behaviorally, it is the driving force behind the

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male desire to breed. It can also contribute to ***intermale*** [*inter-* between + *male*] competition and aggression. The purpose of this is to ensure that the strongest males do the breeding. If they're strong, genetically they are more likely to produce offspring that are stronger and have a greater chance of survival.

Miscellaneous Hormones

Through all of our discussions so far, most of the hormones we've discussed are produced by endocrine glands. Some are produced by other tissues, like those produced by the uterus to maintain the corpus luteum. The placenta contributes to this, too. In Chapter 3, we talked about ***erythropoietin*** [*erythr(o)-* red + *poie(o)-* producer + *-tin* pertaining to, the; i.e., a hormone that stimulates the production of red blood cells]. As you may recall, erythropoietin is produced by the kidneys. In Chapter 7, we talked about ***gastrin*** [*gastr(o)-* stomach + *-in* a, the], a hormone produced by the stomach wall, which stimulates the production of hydrochloric acid and digestive enzymes like pepsin. There are many others. Believe it or not, the kidneys produce other hormones that are very important for homeostasis of blood pressure. Hormonal control of blood pressure is complex. But it is a great way to demonstrate how sensory input and hormonal feedback coordinate homeostasis.

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Hormonal Regulation of Blood Pressure

As I said, this is complex. So let's start with what we already know. In Chapter 6, we talked in detail about the kidneys and renal function. We know from those discussions that adequate blood pressure is essential for appropriate renal function. So it stands to reason that the kidneys would respond to and produce hormones that contribute to blood pressure homeostasis. Earlier, we discussed the posterior pituitary hormone: antidiuretic hormone (ADH). We learned that in the presence of ADH, the kidneys will conserve water for the body by absorbing water from the renal tubules. This in and of itself is important for blood volume. And what's good for blood volume is good for blood pressure. But that's not all ADH does. We said that it also has another name: vasopressin. Vasopressin, as its name implies, causes vasoconstriction. If vessels, especially peripheral vessels, are smaller, then less blood is required to fill them. So blood pressure goes up. These events happen, even as a simple and common condition of dehydration develops. Both dehydration and low blood pressure will promote secretion of vasopressin (ADH) from the neurohypophysis. But ***hypotension*** [*hypo-* low + *tens(o)-* pressure + *-ion* state of i.e., low blood pressure] can occur for many reasons, beyond dehydration—heart failure, for example. And because adequate blood pressure is absolutely essential for all body tissues, especially major organs like the brain, kidneys, liver, and heart, we need lots of mechanisms to help us raise and maintain blood pressure.

We have pressure receptors in key areas throughout the body. In the presence of hypotension, the neurohypophysis will be stimulated to release vasopressin. ACTH, from the adenohypophysis, will stimulate secretion of aldosterone from the adrenal cortex, to promote resorption of both sodium and water from the kidneys, to improve circulating blood volume. Obviously, the sympathetic nervous system will be engaged, stimulating the release of adrenergic hormones. Remember, epinephrine (*epi*) and norepinephrine (*norepi*) stimulate α-adrenergic receptors, creating

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vasoconstriction in the periphery and other nonessential areas. This helps increase blood pressure. *Epi* and *norepi* also stimulate β-adrenergic receptors, especially on the heart, increasing heart rate and strength of contraction to help improve blood pressure.

But wait, there's more!

Adrenergic activity also stimulates the kidneys to secrete ***renin*** [*ren(o)-* kidney + *-in* a, the]. Decreased renal blood flow and other factors also stimulate the production of renin. Renin is needed to produce the active form of ***angiotensin*** [*angi(o)-* vessel + *tens(o)-* pressure + *-in* a, the]: ***angiotensin II***. But it's not as simple as it sounds. Renin must first cause angiotensinogen [*angi(o)-* vessel + *tensin(o)-* pressure + *gen(o)-* producer] to be transformed into ***angiotensin I***, (the inactive precursor to active angiotensin II). Then ***angiotensin-converting enzyme*** (***ACE***) transforms angiotensin I to active *angiotensin II*. The body's response to angiotensin II is significant. Angiotensin II increases thirst, increases aldosterone secretion, increases secretion of vasopressin (ADH), increases secretion of norepinephrine, increases sodium retention by the kidneys, and causes vasoconstriction. When pressure receptors detect adequate blood pressure, this hormonal activity will be reduced or disengaged. And that is a fabulous example of negative feedback. Do you see how important this renin-angiotensin hormonal system is for blood pressure? It's kind of self-serving, by the kidneys. But the rest of the body benefits, too. It's a win-win, most of the time.

Unfortunately, in heart failure, all of these compensatory mechanisms contribute to problems for the patient. As we discussed in Chapter 5, in heart failure, the heart is weak, so cardiac output is poor, reducing blood pressure. But this weakened heart will have difficulty pumping blood against the increased resistance produced by vasoconstriction. Combine the cardiovascular [*cardi(o)-* heart + *vascul(o)-* vessels + *-ar* pertaining to] problems with the hormonal influences that promote fluid retention and these patients are at risk of edema formation. As you may recall from Chapter 3, edema is increased interstitial fluid. In patients with heart failure, that excess fluid accumulation is often in the lungs and/or chest cavity. That's bad for oxygenation and removal of carbon dioxide. This is why heart failure patients should eat low-sodium diets.

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Sodium retention simply attracts water, and that contributes to edema. Many of these patients are also medicated with ACE-inhibitors. By inhibiting the conversion of angiotensin I to angiotensin II, we minimize the catch-22 cycle of events that the weakened heart simply can't cope with. It is a delicate dance for us to manage heart-failure patients. And as you can see, it involves more than just managing the heart. There are numerous hormonal influences that also must be managed to optimize blood pressure and cardiac function.

There! You did it! You successfully waded through endocrinology. Take a deep breath before we see how well you can apply what you've learned to the case study for this chapter.

Case Study

Fifi is a 9-year old, spayed female toy poodle. Fifi has been a "frequent flyer" in our hospital. She has been treated chronically for environmental allergies with corticosteroids. The owners blame her obesity and diabetes mellitus (DM) on the corticosteroid therapy. So, last fall, after many of the pollens causing her allergies were gone, the owners simply stopped giving her prednisolone. On the morning she was admitted to the hospital, Fifi stopped eating. Also, the owners reported that she vomited a few times that morning. They knew better than to give her any insulin that morning.

When Fifi was presented to us, she was unresponsive. Our STAT blood work revealed that Fifi was hypoglycemic, hyponatremic, and hyperkalemic, with acidosis. On physical examination, her heart rate was slow, in spite of the hypotension that was present. She was quite dehydrated, probably from the profound polyuria and vomiting that she was experiencing. Based on history, physical examination, and laboratory findings, the veterinarian was highly suspicious of an Addisonian crisis. The ACTH stimulation test and the small adrenal glands seen on abdominal ultrasound helped support the diagnosis of hypoadrenocorticism.

Treatment included the administration of intravenous fluids (0.9% sodium chloride solution). This restored her fluid volume

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and raised her blood pressure. Dextrose was added to the IV fluids to counter the hypoglycemia. Both glucocorticoids and mineralocorticoids were administered to make up for the adrenocortical insufficiency. After several hours of fluid therapy, a quick recheck of her electrolytes showed that the hyponatremia and hyperkalemia were resolving. After a couple of days, she seemed to be improving. Her appetite returned, and she was brighter and more responsive. The dextrose in her fluids was discontinued. Then she began to deteriorate.

Once again, her appetite disappeared. She was becoming progressively lethargic and depressed. She began to vomit, and her abdomen was extremely painful. Another abdominal ultrasound revealed pancreatic changes consistent with pancreatitis. Blood work revealed that now she was hyperglycemic. Fortunately, her electrolytes were normal. The Addisonian crisis was over, but now we needed to manage both her DM and the pancreatitis.

Finally, after two weeks of hospitalization, Fifi was well enough to be discharged. She was discharged with new dosing of her insulin. She was also given a prescription for glucocorticoid therapy. And she was placed on a low-fat, low-carbohydrate, high-fiber diet to help manage the DM and promote weight loss.

Case Study Questions

1. On initial presentation, Fifi had extremely low blood glucose. The medical term for this condition is _____.
2. Laboratory data also revealed that Fifi had numerous electrolyte disturbances. Of particular concern was the blood condition of excess potassium, which is medically called_____.
3. A blood condition of low sodium, called _____, was also present.
4. The owners reported that Fifi had been vomiting and was urinating a great deal. This urinary condition is

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medically termed _____.

5. Both Fifi's vomiting and excessive urination caused profound dehydration. The dehydration was so severe that Fifi was in a state of low blood pressure, termed _____.
6. In addition to all of these problems, Fifi was not able to excrete hydrogen ions in her urine. As this built up in her system, Fifi developed _____, or a condition of acid.
7. An ACTH stimulation test was performed. What does the abbreviation *ACTH* stand for? _____.
8. With results from the ACTH stimulation test and the abdominal ultrasound, the veterinarian made the diagnosis of _____, a state of low adrenal cortex hormones also called Addison disease.
9. The _____, or anterior pituitary gland, is responsible for secreting ACTH.
10. Fifi responded well to IV fluids and other therapeutics. Days later, she began to decline because of the development of _____, or inflammation of the pancreas.
11. Fifi became _____, pertaining to excessive amounts of glucose in her blood. This was due to both her inflamed pancreas and her chronic condition of diabetes mellitus.
12. The hormone _____ is responsible for lowering blood glucose concentrations by promoting active transport of glucose into cells.

The Answer Key to these case study questions may be found in Appendix B.

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11

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Applied Terminology for Neurology and Special Senses

The Nervous System,
- Neurons and Nerves,
  - Basic Structure,
  - Neurotransmission,

Central Nervous System,
- Brain,
  - Cerebrum,
  - Cerebellum,
  - Diencephalon,
  - Brain Stem,
  - Seizures,
  - Infectious Neurotropic Diseases,
- Spinal Cord,
  - Structure and Function,
- Meninges,
  - Cerebrospinal Fluid,

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Case Study,

GOALS AND OBJECTIVES

By the conclusion of this chapter, the student will be able to:

1. Recognize common root words, prefixes, and suffixes related to the neurologic system and special senses.
2. Divide simple and compound words into their respective parts.
3. Recognize, correctly pronounce, and appropriately use common medical terms related to the neurologic system and special senses.
4. Demonstrate a basic understanding of neurologic system, eye, and ear anatomy.
5. Demonstrate a basic understanding of nervous system divisions and their roles, nerve pathways, and nerve transmission.
6. Demonstrate a basic understanding of the five senses and their functions.
7. Demonstrate a basic understanding of normal eye structure and function, especially with regard to tears, aqueous production and flow, and the visual pathway.
8. Demonstrate a basic understanding of normal ear structure and function, especially with regard to the auditory pathway.
9. Demonstrate familiarity with integration of the nervous system and special senses for balance and equilibrium.
10. Demonstrate a basic understanding of common neurologic, eye, and ear diseases among domestic animals.

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The Nervous System

Most of us, whether we live in a house, apartment, or dormitory, have electricity at our finger tips. Our homes and classrooms are all wired with electrical conduit and connected to a larger power grid. All we need to do is plug into that power, and we can make appliances and computers run, illuminate light bulbs, and recharge batteries, cell phones, and even cars. I like to think of the ***nervous*** [*nerv(o)-* nerve + *-ous* pertaining to] system in a similar way. Nerves are the body's hard-wiring, connecting most of our tissues and organs. Between the *nervous system* and the *endocrine system* (Chapter 10), all tissues and organs are interconnected to maintain ***homeostasis*** [*home(o)-* unchanged, same + *-stasis* state of standing; i.e., balanced, stable state of the body]. The animation entitled *Nervous System Overview,* in the Evolve resources, provides an excellent introduction to the nervous system. We will discuss each of the major components mentioned in that animation in this chapter. But before we get into fine detail, I strongly recommend that you view that animation. The "bird's eye view" provided by this animation not only provides a great preview of "coming attractions," but it also captures the integration of components within the system and with the rest of the body. It might be wise to watch it now, as well as periodically as we move through the chapter.

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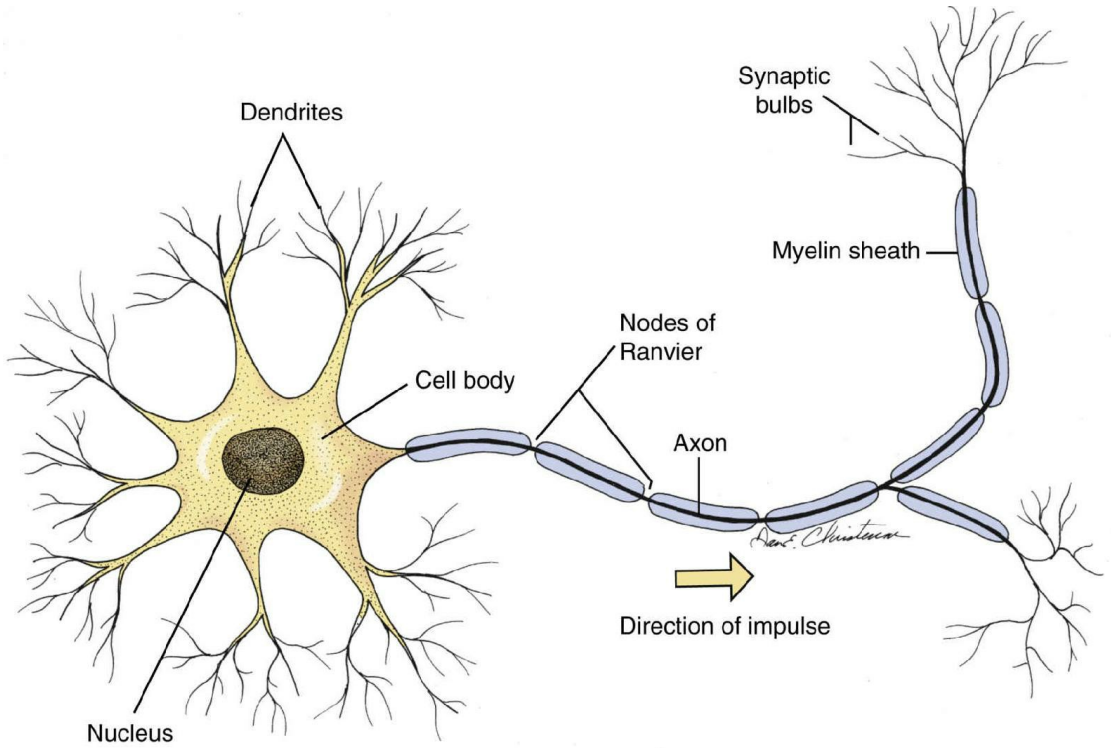

FIG. 11.1 Multipolar neuron.

Please note that I will be referring to bones, skeletal landmarks, and various muscles periodically in this chapter. So, if needed, please refer to Chapter 4 to review musculoskeletal anatomy. Now, let's get started and take a closer look at nerves.

Neurons and Nerves

Nerve cells or ***neurons*** are some of the most unique cells of the body. They are incredibly specialized and streamlined in their function. In fact, they are so specialized that they require an exclusive diet of glucose. And the glucose can be "burned" only in the presence of oxygen (i.e., ***aerobic*** [*aer(o)-* air, oxygen + *-bic* pertaining to] ***glycolysis*** [*glyc(o)-* glucose + *-lysis* breakdown of]). Yes, you may be remembering that in Chapter 10 we said that diabetic patients could use ketones as an alternate source of fuel. That is true. However, neurons do not function well in ***ketosis*** [*ket(o)-* ketones + *-sis* condition of]. In addition, most neurons do not have ***centrioles*** (refer to Chapter 2). That means they cannot reproduce. This is why trauma to nerves can be so devastating. Other cells of the body can undergo ***mitosis*** (cellular reproduction) to replace those lost to injury. Not so for neurons. Now, they do

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have limited capacity to repair themselves. But repair is very limited and takes a very, very long time. Remember that when dealing with ***neurology*** [*neur(o)-* nerve + *-logy* study of] patients.

Basic Structure

Let's look at one of the most abundant neurons of the body, a ***multipolar*** [*multi-* many + *pol(o)-* poles + *-ar* pertaining to] ***neuron*** (Fig. 11.1), to understand basic neuronal [*neur(o)-* nerve + *-al* pertaining to] structure. It probably looks pretty weird to you at first glance. But when you think of the need for providing "wiring" in the body, much of its weird structure actually makes sense. Let's begin with cellular components that you're familiar with from Chapter 2, such as the cell body. The cell body is where we find things like the nucleus and modified cytoplasmic organelles. But the cellular membrane makes it look more like Medusa[a] than a cell, with all of those projections, doesn't it? Not to worry. Unlike the Medusa of Greek mythology, you won't turn to stone by looking at this neuron. And those tiny projections from the cell body are not venomous snakes, but dendrites.

The term ***dendrite*** [from Gr. *dendron* tree] really does make this neuron look kind of like a tree. ***Dendritic*** [*dendr(o)-* tree + *-tic* pertaining to; i.e., pertaining to dendrites] endings serve as receptors for nerve impulses, typically received from other neurons. So, an initial nerve impulse will be received from the dendrites and then pass through the cell body. Then we come to the main "trunk"—the ***axon*** [Gr. *axon* axle]. Here is the true wiring of nerves. It's the axons that make up nerve fibers and rapidly transmit nerve impulses. Some axons can be very long. And because this is a *multipolar* neuron, the axon can have numerous "forks in the road," as you can see in the diagram. This provides for rapid transmission of nerve impulses to multiple places. And at the very ends, at what appear to be roots, we find the ***synaptic*** [*synap(o)-* connection + *-tic* pertaining to] ***bulbs***. We talked about the ***synapse*** [Gr. *synapsis* a connection] in Chapter 4, when we discussed muscle contraction. We'll review *synaptic* structure and function in a moment. First, let's finish our discussion of the axon. Notice that it's not a "bare wire." ***Axonal*** [*axon* + *-al* pertaining to] "insulation" is provided by a ***myelin sheath***.

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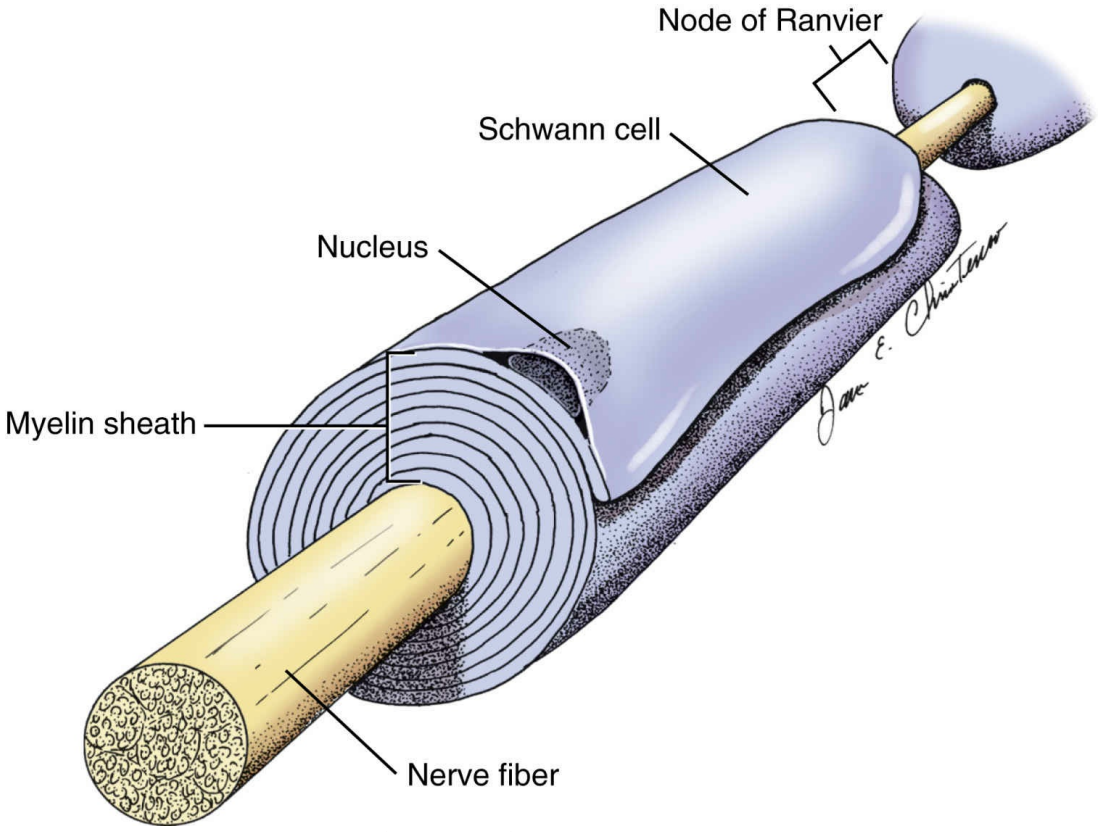

FIG. 11.2 Schwann cell.

Myelinated Versus Unmyelinated

Myelin is a fat-laden substance that insulates a neuron's axon. The *myelin sheath* is actually created by specialized support or ***glial*** [*gli(o)-* glue + *-al* pertaining to] ***cells***. There are numerous *glial cells* in the nervous system. We'll talk about others elsewhere in the chapter, as applicable. Here let's focus exclusively on those that provide myelin. In the ***central nervous system*** (***CNS***), it's the ***oligodendroglial*** [*olig(o)-* small + *dendr(o)-* tree + *gli(o)-* glue + *-al* pertaining to] cells that create the myelin sheath. In the ***peripheral nervous system*** (***PNS***), it's ***Schwann cells*** that create the myelin sheath. In both cases, these supportive cells wrap themselves around axonal nerve fibers. Fig. 11.2 shows a *Schwann cell* wrapped around the nerve fiber of a peripheral nerve. Notice that the myelin insulation doesn't cover the whole nerve fiber. There are gaps between the *glial* cells, called ***nodes of Ranvier*** (ron've-a). (The *nodes of Ranvier* were named after French anatomist and ***pathologist*** [*path(o)-* disease + *log(o)-* study + *-ist* specialist in], Louis Antoine Ranvier, who made their discovery.) At these gaps the nerve fiber is

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exposed. And along a ***myelinated*** [*myelin* + *-ated* that which has] nerve fiber, the nodes of Ranvier are the only places where ionic exchange takes place. Along the nerve fiber covered by the myelin sheath, the "electrical current" of a nerve impulse simply "jolts" through, like a bolt of lightning. And that's the whole point of the myelin, to insulate for faster nerve transmission. Because the electrical current seems to "jump" from one node of Ranvier to the next, this type of conduction is often called ***saltatory*** [L. *saltatio* to jump + *-ory* pertaining to] conduction.

You see along ***unmyelinated*** [*un-* without + *myelin*] nerve fibers there is no insulating myelin sheath. So, for ***neurotransmission*** [*neur(o)-* nerve + *transmission*] along unmyelinated fibers, ionic exchange must take place sequentially along the whole length of the fiber. That takes time. Okay, so that time is measured in milliseconds. But still, it takes longer for neurotransmission along unmyelinated nerve fibers than it does along myelinated nerve fibers. Fortunately, myelinated outnumber unmyelinated fibers. Plus, unmyelinated fibers are not innervating vital areas of the body. And just in case you're concerned about stray voltage from unmyelinated fibers, don't be. Unmyelinated nerve fibers are typically nestled in between bundles of myelinated fibers. So, surrounding tissues are not going to suffer adverse effects of stray voltage, like someone living directly under high-tension power lines might.

Neurotransmission

Before we begin this electrifying discussion, I suggest that you view the animation entitled *Nerve Impulse* in the Evolve resources. Whether you view it now or later in this discussion is entirely up to you. Frankly, viewing it multiple times for clarification is probably a wise plan. I believe that animation plus our discussion in this section of the chapter will provide an excellent understanding of neurotransmission along nerve fibers.

To begin, you need to understand that the whole process of neurotransmission truly creates electrical charge. And the literal voltage created occurs by virtue of diffusion of two key ions: ***sodium (Na+)*** and ***potassium (K+).*** Remember from our discussion in Chapter 2 that diffusion is the movement of ions across a

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semipermeable membrane, from an area of high concentration to an area of low concentration. We spoke about simple diffusion in Chapter 5, when we talked about gas exchange of oxygen and carbon dioxide. But the diffusion along neurons can't freely take place like that. It needs to be controlled, because the end result along motor neurons is some sort of action by a muscle or an organ. So, for neurotransmission we have gated channels. It's kind of like having a guard at the door. Until we reach a certain threshold (price-point?), the guard will not open the door. We'll talk about what it takes to get the guard to open the door momentarily.

Resting Potential

So, for diffusion to take place, we must have different concentrations of ions inside and outside of the neuron, right? At rest, there are higher concentrations of ***extracellular*** [*extra-* outside + *cellul(o)-* cell + *-ar* pertaining to] sodium (Na^+) and chloride (Cl^-) ions. Inside the neuron we have higher concentrations of potassium (K^+) and negative ions. (However, note that intracellular [*intra-* inside + *cellul(o)-* cell + *-ar* pertaining to] Cl^- concentrations are actually less that its extracellular concentrations.) At "rest" there is a positive charge outside and a negative charge inside the cell. Believe it or not, along the actual cell membrane we can literally measure a charge of approximately −70 millivolts (mV). This is the ***resting potential*** of the nerve cell. We call it *resting potential* because the neuron is at rest and has the potential for significant electrical changes, when diffusion takes place. And because in this state, with a net positive charge outside the cell and a net negative charge inside the cell, the neuron is said to be ***polarized*** [*-ize* state of]. Let's see if we can make better sense of the idea of a polarized neuron and resting potential. When you think of a battery, it has two opposite poles, right? One has a positive charge, and the other has a negative charge. And that *polarized* battery sitting in a drawer at *rest* has the *potential* to discharge a great deal of electrical power, when actively used. The same is true for neurons.

Action Potential

To make a neuron "fire" or discharge its electrical energy, we need

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sufficient stimulation. That stimulation is received at the dendrites. The dendritic end of a neuron is always the starting gate. And this holds true, whether it is receiving input from another neuron or dendritic sensory receptors are directly stimulated by something else. But each neuron has a set threshold to make it ***depolarize*** [*de-* away from, reduce], discharging its electrical energy. You see, for diffusion to take place for ***depolarization*** [*de-* away from + *polarize* + *-tion* act, process of], we need to actually pump sodium ions into the cell and pump potassium ions out of the cell. This mechanism is referred to as the ***sodium-potassium pump***. Sodium channels are always closed and locked when the cell is at rest. They are voltage-gated channels. So, unless there is a sufficient electrical shift along the cell membrane, our guard will not unlock the sodium gates. In contrast, there are always a few potassium gated channels open to allow a little potassium to freely diffuse. Still, sodium channels must be unlocked before all of the required potassium channels are unlocked.

Our voltage requirement to unlock the sodium channels is actually very little—perhaps only 15 mV. (By the way, calcium plays a role in controlling the voltage requirement. More on that later, when we discuss the synapse.) With stimulation of the dendrites, a few dendritic sodium channels are unlocked and sodium begins to get pumped into the dendrite and cell body. If this creates enough voltage, sodium channels along the axon are unlocked and the *sodium-potassium pump* is turned on. At this point, *depolarization* is ***all or nothing***. There is no such thing as half-hearted or partial *depolarization* of a neuron. Once we kick the *sodium-potassium pump* into gear, diffusion takes place sequentially and progressively along the neuron's axonal membrane. Progressively, each sodium channel will open and three sodium ions are pumped in. Then the adjoining potassium channel opens and two potassium ions are pumped out. It's like tipping over the first domino and then watching all of the rest of the dominoes fall, one by one in sequence. And because this is an ***active*** process, *depolarization* of a neuron is also referred to as ***action potential***. This creates an electrical current, like a battery being discharged. That electrical discharge is usually about 30 mV. That's a big change, from −70 to 30 mV!

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Refractory Period

What happens if a battery is completely discharged? Nothing, right? There's no resting potential left. Batteries need to be recharged. The same is true for neurons. Once a neuron has depolarized, it cannot "fire" again until it has ***repolarized*** [*re-* again + *polarized*]. Think of ***repolarization*** as recharging a battery. Or if you want to use the domino example, repolarization is standing all of the dominos back up in the order they fell. This restores the sodium and potassium ions to their original locations (sodium outside the cell and potassium inside the cell), making the neuron *polarized* once again. This restores its *resting potential*. Until that repolarization is complete, the neuron cannot "fire" again. This momentary delay is referred to as the ***refractory period***. During the *refractory period*, *depolarization* cannot occur until *repolarization* is complete. The only thing that might override this time-delay is a subsequent stimulus that greatly exceeds the minimal threshold stimulus. Then and only then could the *action potential* be produced at a more rapid rate.

Even with a more powerful stimulus, the rate of *action potential* is limited. Picture dominoes still falling near the distal end of the axon, with perhaps half of the dominoes standing again at the proximal end of the neuron. What might happen if the first domino is knocked down again with greater force than the first time? A new nerve impulse may or may not be created. The limiting factor here is how quickly the distal dominoes can be restored to a standing position, before the next wave of falling dominoes reaches that point. If the new wave of falling dominoes is moving faster than subsequent dominoes can be restored their standing positions, the new electrical impulse being generated stops. If the rates of falling and restored dominoes are synchronized, then the electrical impulse can reach the terminal end of the neuron. Hypothetically, if the *refractory period* for a neuron is 20 milliseconds (ms), with a stronger stimulus the *refractory period* might be reduced to 15 ms. Regardless, there will always be a *refractory period*. And remember, along myelinated nerve fibers, all of the *depolarization* and *repolarization* that we're talking about only happens at the *nodes of Ranvier*. But what happens when the electrical impulse reaches the terminal end of the axon?

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Synaptic Transmission

A ***synapse*** [L. *synapsis* connection] is literally a connection between two neurons or between a neuron and an organ. In Chapter 4, we discussed *synapses* with regard to the ***neuromuscular*** [*neur(o)-* nerve + *muscul(o)-* muscle + *-ar* pertaining to] ***junction***. The ***synaptic*** [*synap(o)-* connection + *-tic* pertaining to] ***bulbs*** (sometimes called synaptic knobs) shown in Fig. 4.7 are typical for most synapses. Synapses differ by virtue of the ***neurotransmitter*** [*neur(o)-* nerves + *transmit* to send across + *-er* one that] substances contained in the synaptic bulb's vesicles and by virtue of what lies on the other side of the ***synaptic cleft*** (e.g., another neuron, gland, etc.). The synaptic cleft, remember, is the gap between the ***presynaptic*** [*pre-* before + *synap(o)-* connection + *-tic* pertaining to] ***neuron*** and the ***postsynaptic*** [*post-* after + *synap(o)-* connection + *-tic* pertaining to] neuron or organ.

As the axon depolarizes and the electrical impulse nears the *presynaptic bulb*, the membrane of the bulb becomes more permeable to ***calcium (Ca^{++})*** ions. Ah, here we go again—another example of the importance of calcium in the body. We began discussing the importance of calcium way back in Chapter 3, when we talked about clotting. Calcium was discussed again in Chapter 4, for bone growth and repair, as well as muscle contraction. This electrolyte has been discussed in multiple chapters. By now, you should have a very good appreciation for the importance of Ca^{++} *homeostasis*. And in the case of *synaptic transmission*, calcium is absolutely necessary. You see, we need sufficient Ca^{++} diffusing into the *synaptic bulb* to activate enzymes within the bulb. These enzymes cause some of the *synaptic vesicles* to merge with the membrane and release their *neurotransmitter* substance into the *synaptic cleft*.

The neurotransmitter substance passes across the cleft and binds with specific receptors on the *postsynaptic* membrane. If that membrane is on a postsynaptic neuron, when the receptors are filled, the sodium channels are unlocked and action potential begins along that neuron. If the membrane is on a postsynaptic organ, the activity stimulated in the organ depends on the organ itself. If it's a salivary gland, it secretes saliva. If it's a muscle fiber, it will contract. You get the idea. But there's a catch. There is always

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more neurotransmitter substance released into the cleft than is necessary. We simply don't have that many receptors on the postsynaptic membrane. So, what do we do with all of the excess? Well, some of the excess neurotransmitter is reabsorbed and repacked by the presynaptic bulb. The rest will be removed by an enzyme. In our neuromuscular junction example in Chapter 4, we said that the most abundant neurotransmitter substance in the body, especially in the peripheral nervous system, is ***acetylcholine*** (uh-se'tul-ko"lēn). The enzyme that breaks down acetylcholine is ***acetylcholinesterase*** (uh-se'tul-ko-lin-es"ter-ās). Remember, the suffix *-ase* always indicates some sort of enzyme. We'll mention other synaptic enzymes when we talk about neurotransmitters in the next section. By the way, there is an excellent animation in the Evolve resources, entitled *The Synapse*. I strongly recommend that you watch it, as many times as necessary to functionally understand synaptic transmission. In fact, this animation and the *Nerve Impulse* animation are worth watching again and again.

Neurotransmitters

There are many ***neurotransmitter*** substances in the body. Neurotransmitters are chemicals produced by neurons for use in a synapse. As we said a moment ago, ***acetylcholine*** is the most abundant neurotransmitter in the body. It is the exclusive neurotransmitter used to stimulate skeletal muscle. We talked about that in Chapter 4. A little later, when we talk about the ***autonomic*** [*auto-* self + *nom(o)-* control + *-ic* pertaining to] ***nervous system***, you'll learn that acetylcholine is the exclusive neurotransmitter used by the ***parasympathetic*** branch. It's also used at particular synapses along the sympathetic branch. No matter where the ***cholinergic*** [*cholin(o)-* acetylcholine + *erg(o)-* working + *-ic* pertaining to] synapses reside, ***acetylcholinesterase*** [*-ase* enzyme] is the enzyme used to remove acetylcholine from the *synaptic cleft*.

Another very important neurotransmitter, in the brain and along the ***sympathetic*** branch of the *autonomic nervous system (ANS),* is ***norepinephrine***. We spoke of *norepinephrine* and the hormone *epinephrine* in Chapter 5. As you may recall from those discussions, we said that the *sympathetic* branch of the nervous system kicked

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into high gear for ***fight or flight***. Well, now you know that *norepinephrine* is the neurotransmitter that initiates the ***tachycardia*** [*tachy-* fast + *cardi(o)-* heart + *-ia* condition of], increased ***cardiac*** [*cardi(o)-* heart + *-ac* pertaining to] contractility, and ***bronchodilation*** [*bronch(o)-* bronchus + *dilation*]. You also now know that there is a limited amount of neurotransmitter released into the synaptic cleft. Some of the unused remainder is rapidly reabsorbed into the *presynaptic bulb,* and the rest is removed by an enzyme. In the case of *norepinephrine*, ***monoamine*** (mon'o-uh-mēn) ***oxidase (MAO)*** is the enzyme that removes it. I know that seems like a pretty odd name. You see, norepinephrine is in a group of neurotransmitter substances called *monoamines*. Other monoamines used in the brain include ***dopamine*** (do'puh-mēn) and ***serotonin*** (sur-uh-to'nin), two very important neurotransmitters in the brain. So, *MAO* will remove any of these from the synaptic cleft.

Now, especially when it comes to neurotransmitters in the ***brain,*** they are usually classified as either ***excitatory*** or ***inhibitory***. Remember, in the brain we're talking about ***interneuronal*** [*inter-* between + *neuron* nerve + *-al* pertaining to] synapses. So, if the neurotransmitter makes the *postsynaptic* neuron's membrane more positive, it is easier to activate the sodium channels, and therefore the neurotransmitter is considered *excitatory*. If on the other hand the neurotransmitter makes the *postsynaptic* neuron's membrane even more negative, it is much more difficult to activate sodium channels. Because these chemicals in essence inhibit activity, they are considered *inhibitory* neurotransmitters. So, as we think about the brain, neurotransmitters such as *dopamine*, *norepinephrine,* and *serotonin* are all *excitatory* neurotransmitters. (Note: depending on its location in the brain, dopamine may also be inhibitory.) The principal *inhibitory* neurotransmitter in the brain is ***gamma-aminobutyric*** (gam'uh-uh-me"no-bu-tir'-ik) ***acid*** (GABA). Another inhibitory neurotransmitter of the ***CNS*** (i.e., brain and spinal cord) is *beta-endorphin* (ba'tuh-en-dor'fin). ***Beta-endorphin*** is found predominantly along pain pathways. By inhibiting neurotransmission along those pathways, pain is reduced.

Electrolyte Imbalances and Neurotransmission

Now that you know the role of key electrolytes in the context of

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neurotransmission, let's see if you can figure out what might happen with abnormal electrolyte concentrations in the body. Let's start with calcium. How might neurotransmission be affected if a patient has ***hypocalcemia*** [*hypo-* insufficient + *calc(o)-* calcium + *-emia* a blood condition of]? Remember, it needs to diffuse into the presynaptic bulb in order for the neurotransmitter to be released. If you said that hypocalcemia would reduce neurotransmission, due to insufficient calcium diffusing into the presynaptic bulb, you would be absolutely correct. However, that only inhibits synaptic transmission.

We also said earlier that calcium controlled the voltage requirement for the neurotransmission threshold. Is it directly involved in activating the sodium potassium pump? No. But it is important for both the resting membrane potential and the threshold setting for "firing." In severe *hypocalcemia*, the resting membrane potential is elevated and the threshold setting is reduced. That means that the millivolt difference between membrane potential and threshold are closer together. And that means it will take far less stimulus to activate the sodium channels and the sodium-potassium pump. So, the end result of severe *hypocalcemia* could be spontaneous seizure activity. (Hypocalcemia may also cause muscle tremors and twitching. Refer to Chapters 4 and 9 to understand how and why.)

Obviously, sodium is very important for neurotransmission. We said that when the sodium potassium pump is engaged, three Na^+ are pumped into the neuron while two K^+ are pumped out. So, we need sufficient extracellular sodium for neurotransmission, right? That's part of the reason why there are a number of homeostatic mechanisms that force the resorption of sodium by the kidneys. The other important role of sodium, remember, is for maintenance of blood pressure, as we discussed in Chapters 5, 6, and 10. So, how will neurotransmission be affected by ***hyponatremia*** [*hypo-* low + *natr(o)-* sodium + *-emia* blood condition of]? Again, I'm talking about severe *hyponatremia*. If there's insufficient sodium, how can neurons depolarize? It's mighty tough. So, we tend to see extreme weakness, lethargy, and somnolence (sleepiness). There are diseases that can cause hyponatremia, but most of the time severe hyponatremia is ***iatrogenic*** [*iatr(o)-* physician + *gen(o)-* produced + -

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ic pertaining to]. If we ***diurese*** (di-u-rēs' [*diure(o)-* to urinate; i.e., to cause to urinate] patients too much with aggressive ***intravenous*** [*intra-* within + *ven(o)-* vein + *-ous* pertaining to] (IV) fluid therapy, we can cause extreme hyponatremia. I saw this in my mother when she was hospitalized. She became so weak, she could hardly hold her head up or lift her arms. She most certainly couldn't get out of bed. Even her speech was slurred. At first, we thought she may have experienced a stroke. But on close assessment of her electrolytes, we discovered the *hyponatremia*. After changing the sodium concentration of her IV fluids and slowing the delivery rate, her blood sodium levels were restored. Her recovery was quick and dramatic.

Finally, let's think about the other important ion for neurotransmission—potassium. Remember, most of the potassium is within the neuron. Yes, there are a few leaky potassium channels, but comparatively, there is much less outside than there is inside. So, how might ***hyperkalemia*** [*hyper-* excess + *kal(o)-* potassium + *-emia* a blood condition of] affect neurotransmission? Well, think about the requirements for diffusion. For diffusion to take place, the ions must pass through a semipermeable membrane from an area of high concentration to an area of low concentration. Under normal circumstances, it is very easy for potassium to diffuse out of the neuron once potassium channels are opened. But in *hyperkalemia*, there is an abundance of potassium outside the neuron. This will negatively impact depolarization. In fact, do you remember our discussion of hyperkalemia and cardiac function in Chapters 5, 6, and 10? We said that *hyperkalemia* could cause ***bradycardia*** [*brady-* slow + *cardi(o)-* heart + *-ia* condition of] or even stop the heart. Now you know why. The cardiac conduction system is ***neural*** [*neur(o)-* nerve + *-al* pertaining to] tissue. Other neurons are adversely affected too. But the cardiac example is dramatic and important.

What about the flip-side of our potassium coin? How does ***hypokalemia*** [*hypo-* low + *kal(o)-* potassium + *-emia* blood condition of] affect neurotransmission? Because it is one of the key ions involved in depolarization, it stands to reason that deficient potassium will ultimately depress neurotransmission. So, just like *hyponatremia*, weakness and lethargy are likely with hypokalemia too. And because we just focused on the heart with *hyperkalemia*, we

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might as well look at the heart with *hypokalemia*. In hypokalemia, the cardiac muscle (like the skeletal muscle) is weak, due to poor neurotransmission. And weak cardiac muscle is bad news for cardiac output and blood pressure. (Please refer to Chapter 5 if you need to review cardiac output and blood pressure.) And if that's not bad enough, hypokalemia can also cause arrhythmias. Suffice it to say that the heart does not function well with potassium imbalances. You may recall from Chapters 5 and 6 that we talked about furosemide (a loop ***diuretic*** [*diure(o)-* urination + *-tic* pertaining to]) often being used in cardiac patients. This is not a "potassium-sparing" diuretic, so higher doses of it can easily result in hypokalemia.

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Central Nervous System

As we said earlier, the central nervous system (CNS) is made of the brain and the spinal cord. Obviously, these structures are essential for life, especially the brain.

Brain

The brain is the supercomputer for the body. Without it, we cannot perceive, think, or act. Our basal functions controlling digestion, breathing, and the beating of our hearts, all involuntary activities necessary for survival, are controlled by the brain. It is quite an amazing organ, one that ***neurologists*** [*neur(o)-* nerve + *log(o)-* study + *-ist* specialist in] and other scientists are still trying to fully understand. Don't get me wrong, we know a great deal about the brain. Past discoveries helped us to understand and organize the brain into its functional subdivisions. Still, there is much yet to discover and understand.

Cerebrum

The ***cerebrum*** [L. *cerebrum* brain] is the largest part of the brain (Fig. 11.3). This is where conscious thought takes place and memories are stored. It is divided into two, equal ***cerebral*** [*cerebr(o)-* cerebrum + *-al* pertaining to] ***hemispheres*** [*hemi-* half + *sphere* a ball]. As you look at the left cerebral hemisphere, in Fig. 11.3, you'll notice lots of ridges (***gyri***—plural of ***gyrus*** [from Gr. *gyrus* ring, circle]) and folds or indentations (***sulci***—plural of ***sulcus*** [from L. *sulcus* groove, trench]). Why make it so folded?—surface area. You see, all of the cell bodies of neurons needed for data processing are found superficially, in the ***cerebral cortex*** [L. *cortex* bark, rind, shell]. That's what makes up what we call the ***gray matter*** of the brain. We need as much computing-power as possible. And the skull doesn't allow room for expansion. The only way to gain as much surface area for *gray matter* as possible in a confined space is to fold the cerebral tissue. If we were to look at a cross-section of a gyrus, we would see the darker gray matter at the surface and ***white matter*** of the myelinated nerve fibers centrally. And visually there appears to

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be more white matter than gray matter. Don't worry, we haven't sacrificed any of our computing power. Why so much *white matter*? Think about it, if we don't have sufficient wiring for incoming data and information as well as outgoing responses, the brain would be pretty useless. That would be like having a computer with no monitor, keyboard, or mouse. So, the *myelinated tracts* of the *white matter* provide a way for the brain to receive sensory data and send out appropriate motor responses, once the data is processed in the *gray matter*.

And we process certain types of data in specific regions of each *cerebral hemisphere*. We refer to those regions as "lobes," but they are not distinctive lobes like we've seen in the lungs or the liver. Neurologists have mapped them out using specific gyri and sulci. We won't get that specific here. We'll try to be a bit more broad in our mapping, as shown in Fig. 11.4. For instance, most visual data is processed by the ***visual cortex*** in the ***occipital lobes*** of the cerebrum. You may recall from the Chapter 4 that the occipital bone is the caudal bone of the skull. Well, the *occipital lobes* are near that bone, in the caudal portion of each cerebral hemisphere. The occipital lobes not only process incoming visual data but also integrate it with other sensory input.

The ***frontal lobes*** are found near the frontal bone of the skull, forming the rostral portion of each cerebral hemisphere. Here is where much of the complex cognitive thinking takes place. Yes, animals think and problem-solve. That's a necessary ability for survival. Emotional centers are also found in the *frontal lobes*. Yes, here again, animals do have emotions. Did you know that elephants actually cry when they lose a member of their pack? It's true. Dogs often express a wide range of emotions, from joy to sorrow and much more. There are many examples throughout the animal kingdom that demonstrate emotional capacities and profound intelligence of animals. The frontal lobes are also responsible for most movement of skeletal muscles.

The ***parietal lobes*** are found in the middle and dorsal portion of each cerebral hemisphere. The *parietal lobes* are responsible for recognition of numerous sensations, including touch, temperature, taste, pressure, and pain. In terms of cognitive thought, the parietal lobes are important for recognition of shapes and objects. We'll talk

more about the special senses later.

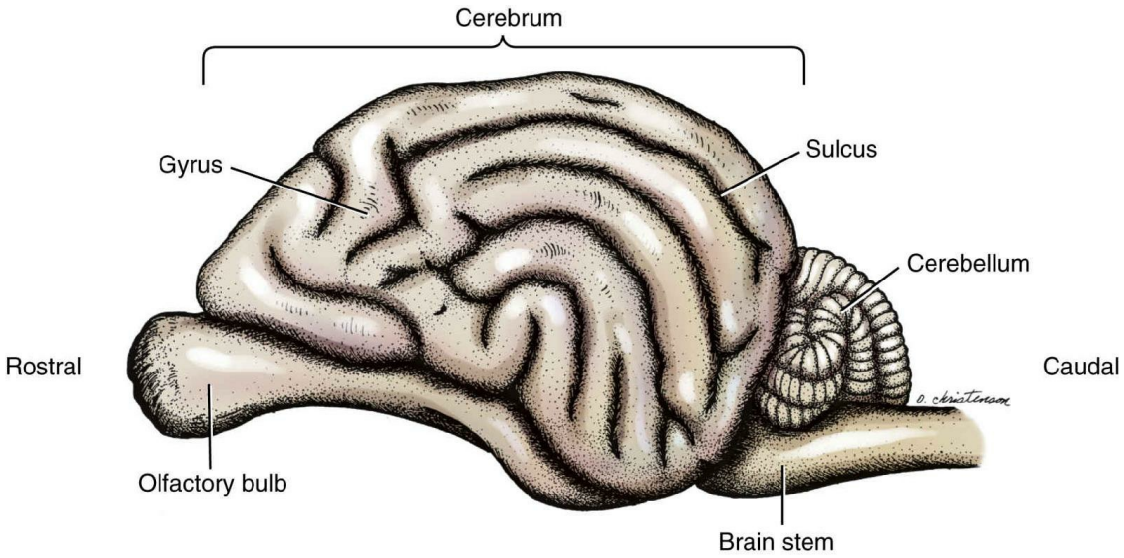

FIG. 11.3 Canine brain (left lateral view).

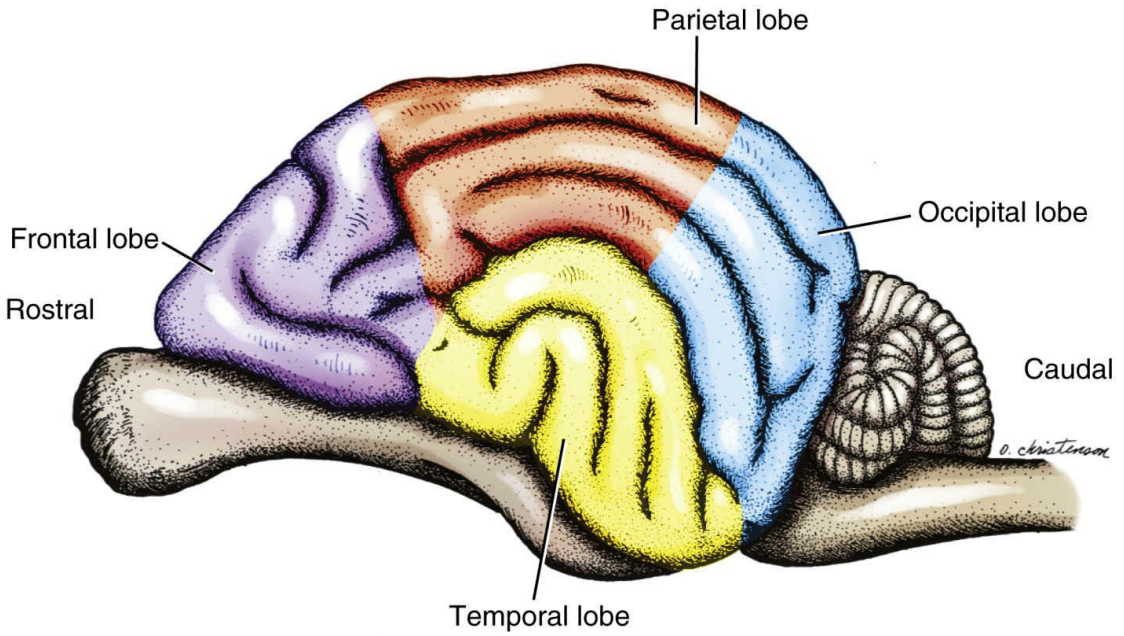

FIG. 11.4 Cerebral lobes.

The ***temporal lobes*** are located in the mid-ventrolateral portion of each cerebral hemisphere. This is where sensory input for smell and hearing are interpreted. Both of these senses are extremely important for survival of animals. Predators need to be able to smell

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and hear their prey. Vision may also be used, but often sound and scent lead a predator to its prey without visual input. A coyote or fox capturing an underground mole would be a good example of this. Likewise, all of the senses are important for prey animals to recognize and avoid predators. Survival in the wild is why both hearing and the sense of smell among animals exponentially exceeds human ability. Are they as necessary among domesticated animals? Not so much. Yet, these innate abilities are retained by domestic animals.

Intersections of the lobes are thought to be important for general interpretation, including recognition of words, for example. And if you don't believe that animals can recognize and understand various words, you've not seen some of the cognitive testing done with dogs. Border Collies are extremely intelligent. I recall seeing a Border Collie that was owned by a human psychiatrist. He began challenging and testing the dog's cognitive ability by teaching the dog names of toys and having the dog select a specific toy by name, from a pile of toys. The dog quickly learned each name and was able to repeatedly select the correct toy, by name, no matter how many different toys were presented. The psychiatrist had to begin writing the name on each toy, because he was having difficulty remembering each one. By the time he finished his "experiment," the dog had more than 1000 toys that he knew and could accurately select by name. I don't plan on challenging my pup to that degree. But she already is retrieving approximately six of her toys by name, and she's only 10 months old. Never underestimate an animal's cognitive ability.

Why bother mapping these various areas of the brain? Will we ever need this knowledge in clinical practice? In most circumstances, no we won't apply this knowledge. However, head injuries are quite common, especially from automotive accidents (e.g., hit by car). Brain tumors are not as common but do occur with some frequency, especially in dogs and cats. Understanding the function of each cerebral lobe may help us to predict deficits and outcomes in head injuries. For instance, a severe blow to the occipital region of the skull could result in cortical blindness (i.e., from trauma to the occipital cortex). It may also help us to understand the clinical manifestations of the deficits we observe in

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head trauma or seizure patients. By the way, I should note here that integration of cerebral input and output is ***contralateral*** [*contra-* opposite + *later(o)-* side + *-al* pertaining to]. For example, conscious motor control of skeletal muscles of the right side of the body takes place in the left frontal lobe. So, mild to moderate injury or disease of the left frontal lobe could cause ***right hemiparesis*** [*hemi-* half + *paresis* weakness; i.e., weakness on the right side of the body]. Significant injury or disease of the left frontal lobe could result in ***right hemiplegia*** [*hemi-* half + *plegia* paralysis; i.e., paralysis on the right side of the body]. This is how the information may apply clinically.

Cerebellum

As you can see in Figs. 11.3 and 11.5, the ***cerebellum*** is caudal to the cerebrum and dorsal to the brain stem. The word *cerebellum* in Latin is actually a diminutive of the word *cerebrum*. So, apparently ancient anatomists thought the *cerebellum* looked like a tiny version of the *cerebrum*. I guess they have a point. After all, it does have lots of tiny sulci and gyri. On the sagittal view in Fig. 11.5, the cerebellum reminds me of cauliflower. Oops. Sorry, I always encouraged my students to never make food analogies in anatomy and medicine. Ah well, sometimes it helps us to make better connections to the information.

Functionally, the *cerebellum* does not engage in any conscious thought. It is strictly involved with involuntary coordination of movement, especially for maintenance of balance and posture. Now, maintenance of balance and equilibrium is complicated, requiring integration of lots of visual, inner ear, and ***proprioceptive*** [*propri(o)-* one's own + *cept(o)-* receptor + *-ive* pertaining to] sensory input. ***Proprioceptors*** [*propri(o)-* one's own + *-ceptor* receptor] are very specialized pressure receptors in the body. They are generally found in key pressure-sensing areas of the body, such as joints and tissues of the feet. And the cerebellum is key in coordinating motor activity in response to that sensory input. We will talk about balance and equilibrium in detail, after we've completed our discussions of the special senses. This will provide better integration of all of the information for you. However, while we are focusing here on the cerebellum, we have a wonderful opportunity

to preview motor coordination for balance by discussing a very specific ***cerebellar*** [*cerebell(o)-* cerebellum + *-ar* pertaining to] problem called ***cerebellar hypoplasia*** [*hypo-* deficient + *plas(o)-* development + *-ia* a condition of].

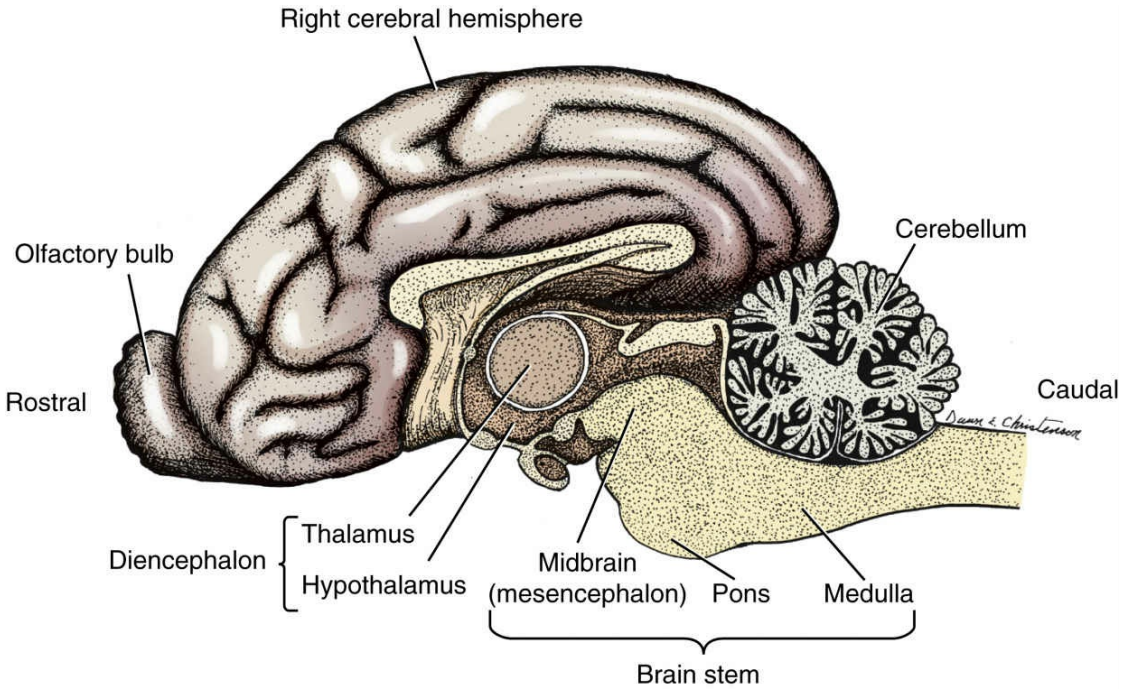

FIG. 11.5 Canine brain (midsagittal view).

Cerebellar Hypoplasia

Cerebellar hypoplasia is truly a physical developmental problem. So, that means that either late fetal development or early ***neonatal*** [*ne(o)-* new + *nat(o)-* born + *-al* pertaining to; i.e., a newborn] development, something minimizes cerebellar growth. This significantly impacts motor coordination, posture, and balance. Any animal can experience this. Through in the course of my career, I have seen it most frequently in kittens. Only on rare occasion have I seen it in other animals.

Among cats, the ***feline*** (cat) ***panleukopenia*** [*pan-* all + *leuk(o)-* white + *-penia* deficiency of; i.e., deficiency of all white blood cells] ***virus*** often targets the cerebellum of the fetus late in pregnancy or of a ***neonate*** [*ne(o)-* new + *nat(o)-* born] in the early days or weeks of life. The cerebellum experiences most of its development late in

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pregnancy and in the early weeks of neonatal life. So, anything like the *feline panleukopenia virus* that injures the cerebellum during these key developmental periods will create impairment. If the queen is exposed to the virus in the environment or through vaccination with a modified-live virus late in pregnancy, some of her kittens may be affected. (If using modified-live vaccines, we should always ask owners of an intact female cat if there is any chance that she could be pregnant.) *Neonatal* kittens exposed to the virus in the environment may also experience *cerebellar hypoplasia*. However, neonatal exposure (depending on age) generally produces the least severe impairment. On ***postmortem*** [*post-* after + *mortem* death] examination, the cerebellums of affected animals are markedly smaller than normal. The severity of symptoms depends on the degree of impairment of cerebellar growth and development.

Most of the kittens I've seen through the years have had mild to moderate cerebellar impairment. Because this made balance and posture challenging, they often had a very base-wide stance for stability. Many of them still lost their balance, especially when walking or turning abruptly. The ***ataxia*** [*a-* without + Gr. *taxis* order; i.e., uncoordinated gait] they experienced was quite variable, from mild stumbling to falling down. However, there is a particular exaggeration in their gait that is very common, and it is pretty characteristic of cerebellar disease. They had a ***hypermetric*** [*hyper-* excess, exaggerated + *metr(o)-* measure, gait + *-ic* pertaining to] gait. As the word implies, the gait when walking is exaggerated. This is very apparent with the front limbs. With each step, these kittens take extremely big, high steps, as though they're stepping over an imaginary tall object obstructing their path. Most of their movements are very jerky, often overcompensated. I've watched affected kittens while eating food from a bowl have difficulty controlling their bobbing heads. And if they bumped their noses on the bottom of the bowl, they often jerked their heads back so abruptly that they tumbled over backwards. As they age, most of these kittens will not improve cerebellar function much, if at all. Can they survive? Yes, provided they are only mildly to moderately affected and have very committed owners. These are special-needs animals that require a great deal of time, care, attention, and patience. They also must be provided a safe environment. Their risk

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of injury from falling down a flight of stairs or off of furniture is pretty high. Life is more of a challenge for the cat and its owner. Still, these cats can lead very happy, fulfilling lives.

Diencephalon

The ***diencephalon*** [*di-* from *dia-* between + *encephal(o)-* brain + *-on* the] includes the ***thalamus*** [from Gr. *thalamos* inner chamber] and ***hypothalamus*** [*hypo-* below + *thalamus*]. Just so you're not confused, the prefix *dia-*, when we've used it before, meant "through." But it also means "between," as it does in the term *diencephalon*. That's why the name of this region is sometimes simplified as ***interbrain*** [*inter-* between + *brain*]. So, just what is the *diencephalon* between? Well, it's between the rest of the brain (cerebrum, cerebellum and brain stem). The *diencephalon* is best shown in Fig. 11.5. The *thalamus* is distinctly visible, because it looks like a bullseye on a target. The thalamus provides major connections for myelinated ***afferent*** [*af-* to, toward + *ferent* to carry, carrying; i.e., incoming sensory] and ***efferent*** [*ef-* out, outward + *ferent* carrying; i.e., outgoing motor] nerve fibers, as well as myelinated fibers of ***interneurons*** [*inter-* between + *neurons* nerves] that connect various parts of the brain.

The *hypothalamus*, the region below the thalamus as its name implies, manages important, core vital functions such as thirst, hunger, and ***thermoregulation*** [*therm(o)-* temperature + *regulat(o)-* regulating + *-ion* act of]. It also provides an important link, integrating ***neurologic*** [*neur(o)-* nerve + *log(o)-* knowledge + *-ic* pertaining to] and endocrine functions of the body. We have touched upon *thermoregulation* in earlier chapters, with our discussion of fever in Chapter 3 and our discussion of heat stroke in Chapter 5. Because the *hypothalamus* is literally central to how the body regulates temperature, this is the perfect place to have a complete ***thermoregulatory*** [*therm(o)-* temperature + *regulat(o)-* regulating + *-ory* pertaining to] discussion. And because by now, you have probably already studied most of the other body systems, we should be able to demonstrate how all of the systems are coordinated for temperature *homeostasis*.

Thermoregulation

Thermoregulation is important for homeostasis in the body. Extremes

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in body temperature can result in cell and tissue death. Organs and tissues do not function optimally at temperature extremes and may even fail. And as we noted in our heat stroke discussion, temperature extremes can be lethal. Consequences of both ***hyperthermia*** [*hyper-* excess + *therm(o)-* temperature + *-ia* condition of] and ***hypothermia*** [*hypo-* low + *therm(o)-* temperature + *-ia* condition of] can be lethal. That is why the body works so hard to maintain body temperature within one degree of the ***hypothalamic*** [*hypothalam(o)-* hypothalamus + *-ic* pertaining to] set point. Yep, the hypothalamus is much like the thermostat on your wall. It has a normal "thermostatic" setting that it tries to maintain by engaging heating and cooling systems. But not everyone's heating and cooling systems function well. Certain species of animals, such as pigs, dogs, and cats, have extremely limited cooling mechanisms, putting them at greater risk of death from heat exposure. In general, ***geriatric*** [*ger(o)-* old age + *-iatric* pertaining to medicine for] patients and *neonates* have poorly functioning thermoregulation, leaving them at increased risk of death when faced with any temperature extremes.

Neonates are especially at risk. From birth until about a week to 2 weeks of age, depending on the species, neonates simply do not have complete thermoregulatory mechanisms. Their primary means of regulating body temperature is behavioral—moving toward or away from heat sources. This is why littermates tend to cluster and pile up together, as well as snuggle close to mom. In the Chapter 9 we briefly mentioned neonatal care of Bulldog puppies, following a C-section. We expressed the need to keep them warm, but we did not address how to safely do that. When caring for a litter of neonates (puppies, kittens, bunnies), especially if they are orphaned, not only do we need to use a safe heat source, but we need to provide space for the youngsters to get away from that source. Warm water circulating blankets provide a very safe heat source. The temperature can be set and maintained at 100°F to 101°F (i.e., close to the normal body temperature of most adult dogs). These blankets can be placed in the box with the neonates but covering only half of the floor of the box. The other half of the box must be a heat-free zone to allow the neonates space to get away from the heat.

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Electric heating pads are **not** recommended due to risk of *hyperthermia*, burns, and electrocution. Electric heating pads cannot be set to a specific temperature. Plus, their thermostatic controls tend to create wide fluctuations in temperature that can far exceed safe levels, especially when placed under neonates. There needs to be significant insulating layers of blankets or towels between the neonates and an electric heating pad, if that is the only heat source available. You need to be careful with warm water bottles too. They can still burn if too hot. A larger concern with warm water bottles is heat loss. They inevitably dissipate heat. And when they fall below an optimal temperature for neonates (90°F to 95°F during the first 3 to 5 days of life), they can actually begin to rob body heat from the youngsters. This could quickly make them *hypothermic*. *Hypothermia* depresses most body functions, including *neurologic*, ***cardiopulmonary*** [*cardi(o)-* heart + *pulmon(o)-* lungs + *-ary* pertaining to], and digestive, to name a few. And it can rapidly lead to death. *Cardiac* ***arrhythmias*** [*a-* without + *rhythm* + *-ia* condition of] develop, even in adult dogs or cats, when body temperature reaches around 93°F. (Remember that with anesthesia/surgical patients.) And if that doesn't kill them, ***hypoglycemia*** [*hypo-* low + *glyc(o)-* glucose + *-emia* a blood condition of] from their inability to nurse or digest a meal due to hypothermia will. Remember, *hypothermia* is precisely what we are trying to avoid with our heat source. Usually, the need for external heat sources are no longer needed when the dam is able to be with and care for her young. Still a reasonable ambient (room) temperature must be provided to prevent significant heat loss from her youngsters. They will not have fully developed thermoregulatory mechanisms until about 4 weeks of age.

So, how does the body of older youngsters and adults respond to fluctuations in ambient temperature? Remember, all of our mechanisms are designed to maintain the *hypothalamic* set point. The average normal body temperature (set point) for most adult domestic animals is around 101.5°F. That's an average. And averages are established by evaluating a large number of subjects and calculating the mean of the majority. There are always those who fall outside the majority. That means that there are always individual variations, even among the vast majority. For example, the average normal human body temperature is 98.6°F. That's

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abnormal for me. My normal body temperature tends to be 97.4°F. So, allowing for the majority individual variations, the *normal core temperature range* for most domestic animals (dogs, cats, cattle, goats, sheep, and pigs) is approximately 100.5°F to 102.5°F, with an average of 101.5°F. The horse tends to run a bit cooler, with a normal range of 99°F to 100.5°F.

Because there is normal variation, as we engage in this discussion, let's consider my 10-month-old pup, with a normal core body temperature of 101°F. Remember, the *hypothalamus* is going to do everything in its power to maintain that temperature within one degree. Let's consider what happens when she's out in cold winter weather. After all, she loves to play in the snow. Her first line of defense against heat loss is her double coat. But even that may get to be a bit chilly after she's out in the snow for a while. And if she gets a little wet, she will begin to lose some body heat. As her core temperature approaches 100°F, the hypothalamus will begin to engage heat conservation measures. So, *arrector pili muscles* (discussed in Chapter 8) in the skin will be stimulated to contract. ***Piloerection*** fluffs her coat to create insulating dead-air space. In addition, ***peripheral vasoconstriction*** [*vas(o)-* vessel + *constrict* + *-ion* act of], especially on those huge Corgi ears, will further minimize loss of body heat by keeping blood at her core and major muscles.

It will help if she has enough fuel on board from her latest meal. The digestive process, especially metabolic processes in the liver, generates a great deal of heat. The liver is a tremendous furnace. In fact, during colder weather, the hypothalamus actually increases appetite to keep that furnace stoked. (That's why those of us living in cold northern climates tend to put on a few pounds during the winter months. Go ahead, blame it on your hypothalamus.) Of course, her physical activity should generate some body heat too. But in really icy conditions well below freezing, she may continue to lose body heat. As her body core temperature drops below 100°F, heat-generating ***shivering*** will be engaged. Between shivering and the rest of her physical activity, hopefully she'll be able to elevate her temperature back over 100°F. It could be a problem if that can't raise her temperature. The shiver reflex often ceases below 97°F. Heart rate also slows as the core temperature plummets, to further minimize heat lost through superficial blood flow. Unfortunately, a

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slower heart rate is a double-edged sword. That will also reduce blood flow to muscles, needed for shivering and other activities. By the time body temperature reaches the low 90s, cardiac arrhythmias may develop. And in the high 80s, the body may have reached the point of no return.

No, I won't permit my pup to stay outside long enough to put her at risk of severe hypothermia like that. But what if something frightened her, making her run off? She could find herself lost out in the cold. There are numerous cases of pets who, for a plethora of reasons and circumstances, find themselves lost in the cold. It happened to friends of mine several years ago. Their two dogs escaped from a boarding facility in an unfamiliar, rural area, miles from home. One of the dogs was older and being managed for heart failure. This happened in February, during a horrible cold snap. Overnight lows were well below zero. The dogs managed to stay together. That was wise, because then they could share bodily warmth. There was evidence from people in the area that they may have sought shelter from the cold, in out-buildings and barns. Somehow, they managed to survive the brutal cold for about 5 days. See? Those thermoregulatory mechanisms can and do work. Unfortunately, on the day they were spotted by rescuers, the dogs had just plunged through thin ice on a pond. Rescuers risked their own safety to pull those dogs from the pond. Hypothermia, with a failing heart, claimed the life of the older dog. Being older put him at risk from the start. His heart failure impaired cardiovascular compensation and limited his physical activity. I tell this story not to make you sad. I tell it to raise your awareness of thermoregulatory impairment in geriatric animals and those with underlying disease. No, most of our patients won't be lost in subzero temperatures. But we will likely subject them to anesthesia and other procedures that put them at even greater risk of hypothermia. And that can cost them their lives. (General anesthesia significantly suppresses the hypothalamus and systemic thermoregulatory responses, resulting in hypothermia. In predisposed pigs, it can cause life-threatening hyperthermia.)

Heat can be equally deadly, as we discussed with heat stroke in Chapter 5. And *hyperthermia* and *heat stroke* can occur, even if an animal is not left in a hot automobile. Let's consider how my pup

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will respond to heat if she's simply out in the yard on a hot summer day. Again, her *hypothalamus* will try to maintain her core temperature within a degree of her normal 101°F set point. As she approaches 102°F (still within a normal range for a dog), hypothalamic mechanisms will engage. First, the mechanisms are subtle. ***Decreased appetite and activity*** will minimize heat production. (She doesn't need to add to her already warm body. And both physical activity and digestion will generate heat.) Behaviorally, she'll probably ***seek a cool place*** to lay down, like under a shady tree. She will also begin to pant. Remember, ***panting*** is a very shallow, nonrespiratory air movement. It cools through convection and evaporation, by moving air over the moist mucous membranes of the mouth. If the ambient temperature is really hot (e.g., upper 90s or into triple digits), panting will not be effective. This is especially true if relative humidity is also high. ***Peripheral vasodilation*** [*vas(o)-* vessel + *dilation*] and ***tachycardia*** [*tachy-* fast + *cardi(o)-* heart + *-ia* condition of] will also occur, in an attempt to dissipate heat from her body. Tachycardia plus peripheral vasodilation quickly circulate warm blood from the core to the surface of the body. Unfortunately, that doesn't dissipate well on a fur-baby. Her coat still has an insulating effect, trapping body heat. If only she were a horse; then she could sweat like mad. But only her little foot pads can perspire a little—certainly not enough to have a cooling effect. The warmer she gets, the more CO_2 will be produced. That will stimulate ***hyperpnea*** [*hyper-* excessive, exaggerated + *pnea* breathing] to blow it off, and unfortunately that muscular activity will only contribute more to her rising body temperature.

Her core temperature can rapidly escalate to 103°F or 104°F, in spite of her physiologic responses. Much warmer and she'll be at risk. Her hypothalamus will be impaired the warmer she becomes. At 106°F to 107°F, she could begin experiencing seizure activity. If she reaches 110°F, she will have significant brain damage and organ failure and most likely die, if she's not dead already. For dogs, cats, and pigs, heat kills more rapidly than cold. Unable to sweat, like horses, they simply cannot effectively cool off. So, protecting them from hot environmental conditions is very important. Providing cooling resources, (water, cooling pads, fans, etc.) is also important,

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especially when air conditioning is not available. We have to compensate for the limitations of their thermoregulatory mechanisms.

Brain Stem

The ***brain stem***, shown in Figs. 11.3, 11.5, and 11.6, includes the ***mesencephalon*** [*mes(o)-* mid, middle + *encephal(o)-* brain + *-on* the; i.e., the midbrain], the ***pons*** (ponz [L. *pons* bridge]), and the ***medulla*** (meh-dul'uh [L. *medulla* inward part] or ***medulla oblongata*** (ob"long-gah'tuh [L. *oblongata* oblong]. Oh my. That's a bunch of pieces and parts with really strange names. We'd better take a look at them one at a time. Collectively, we can simply say that the brain stem is THE main connection of the brain to the rest of the body. And it serves to manage many of the essential involuntary activities of the body, such as *cardiopulmonary* function.

Let's begin with the ***mesencephalon***. It's the ***midbrain*** because it's in the middle, in the deepest part of the brain stem. If the thalamus serves as a central relay station, sort of like Grand Central Station with incoming and outgoing traffic, then the *midbrain* is kind of like a conductor directing that traffic. So, like the thalamus, there are numerous myelinated *afferent* and *efferent* nerve fibers through it. There are also collections of cell bodies. Off to the side of all of the "high-traffic areas," there are portions of the *mesencephalon* that provide activity for some reflexes, such as eye movement for visual tracking when the head is turned.

The ***pons*** is literally a "bridge," as its name indicates. Physically, it bridges the midbrain with the medulla. Functionally, it provides major connections between the cerebrum and medulla, as well as between the cerebrum and cerebellum. It also provides a portion of the ***respiratory centers***, for regulation of rate and depth of breathing. That's pretty important.

The ***medulla oblongata*** is the portion of the brain stem that connects directly to the spinal cord. In fact, structurally, it looks very much like the spinal cord, with predominantly white matter making up its outer portion and a small portion of gray matter centrally. You can probably guess that the white matter of the *medulla* holds major *afferent* and *efferent* myelinated nerve tracts. Those are continuous with the spinal cord. The central gray matter

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is important for reflexes associated with many of the cranial nerves. Plus, it holds a number of extremely important control centers. The ***respiratory centers*** of the medulla team up with the pons to control rate, depth, and rhythm of breathing. ***Cardiac centers*** control heart rate. And ***vasomotor*** [*vas(o)-* vessel + *motor* movement] ***centers*** control the vasculature, to contribute to *blood pressure homeostasis*. So, all of the ***vasoconstriction*** and *vasodilation* that we've mentioned so many times in this chapter and others?—Well, this is where those actions are controlled. But wait, there's more! The medulla also houses nonvital involuntary reflex centers, for things such as *coughing* and *sneezing*. Centers for *swallowing* and *vomiting* are also found in the medulla.

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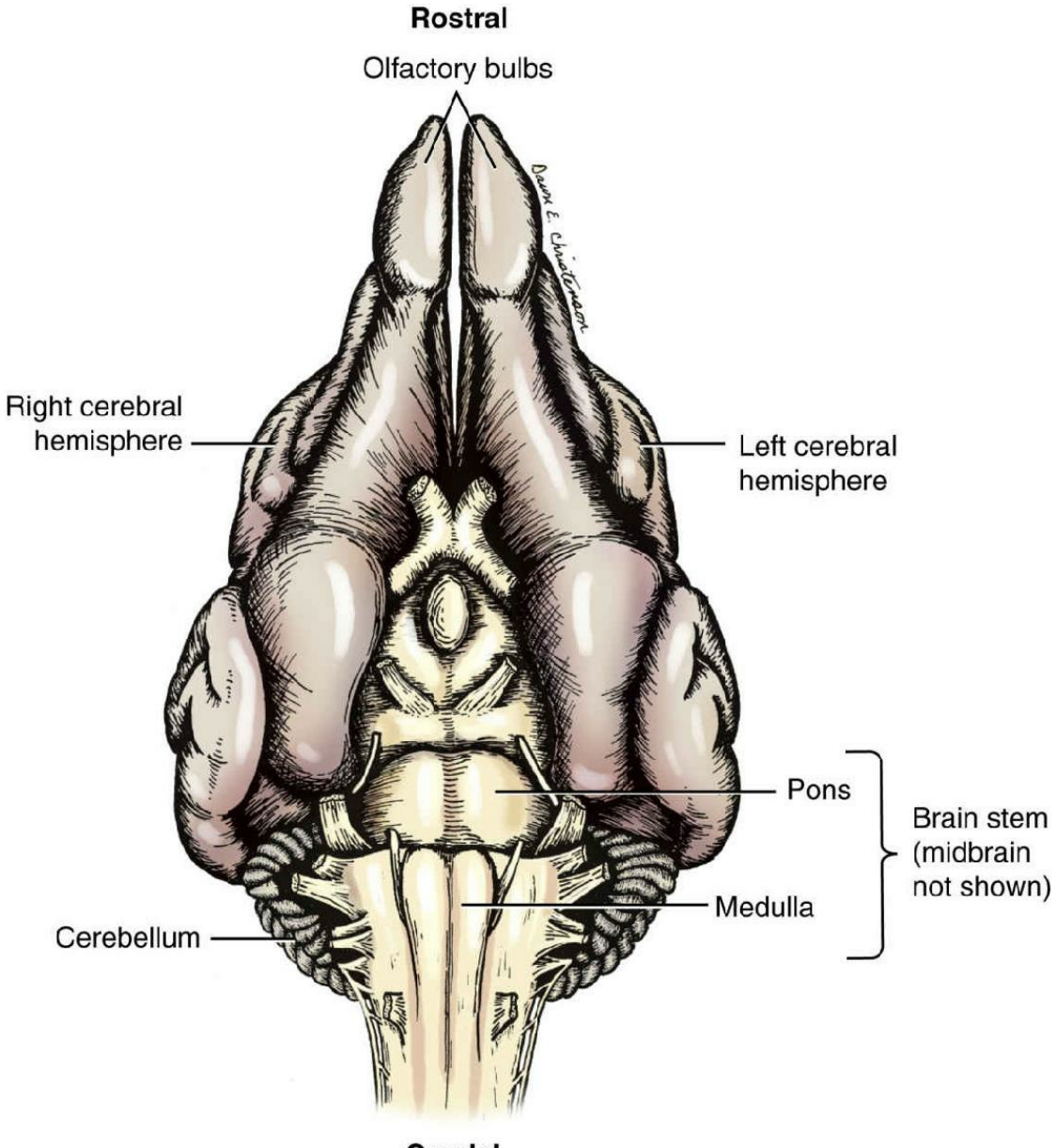

FIG. 11.6 Canine brain (ventral view).

To carry out all of these duties, the brain stem is connected to ***somatic*** [L. *soma* body + *-tic* pertaining to] and ***visceral*** [*viscer(o)-* organ + *-al* pertaining to] structures by way of cranial nerves and the spinal cord. Before we leave the brain and begin our discussion of the spinal cord, let's take a look at some ***encephalopathies*** [*encephal(o)-* brain + *-pathy* disease of].

Seizures

Think about our discussions of *neurotransmission* earlier. Neurotransmission is supposed to be very orderly, right? In

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general, nerve impulses begin in the peripheral nervous system, traveling to and entering the brain with sensory input along *afferent nerve fibers*. Once the input is processed, appropriate motor impulses are sent in response along *efferent nerve fibers* to *somatic* and *visceral* tissues. Obviously, the bulk of the *somatic* tissue receiving those motor impulses is skeletal muscle. Now, imagine complete electrical chaos in the brain, especially the *cerebrum*. That is a ***seizure***—electrical chaos and ***paroxysmal*** [Gr. *paroxysmos*, from *para-* beyond + *oxys(o)-* sharp + *-al* pertaining to; i.e., beyond sharp, over-the-top, violent] mis-firing in the brain.

Minor, very focal areas of chaotic activity, for example associated with muscles of the face and head, will produce minor ***partial seizures***, with facial and head tremors and twitching. I had a Shepherd mix years ago that experienced this. Late in life, she periodically experienced annoying facial twitching, jaw clattering, head-bobbing activity, while remaining completely conscious and aware. Sometimes her partial seizures would last for nearly 5 minutes. In the midst of it, she would often look at me and give a deep sigh, as if to express her discontent with the uncontrolled activity.

Sometimes, mild electrical chaos generalized in the cerebral hemispheres will produce a momentary lapse of awareness, producing what we call a ***petit mal*** (peh-te' mahl [Fr. *petit mal* little illness]) seizure. In animals, *petit mal* seizures are difficult to detect and confirm. In people, the individual may stop in mid-sentence and/or other activity momentarily, expressionless, and seemingly absent. In children, this may be mistaken for daydreaming. Because animals do not talk, it is difficult to know if the momentary pause in activity is intentional to focus on movement or sound in their environment or a petit mal seizure. It helps in recognition if the petit mal is accompanied by minor muscular activity, such as eyelid fluttering or minor jaw clattering. In *partial* and *petit mal* seizures, the person or animal doesn't fully lose consciousness or control. So, they don't fall down.

Depending on the ***etiology*** [*eti(o)-* cause + *-logy* study of] of those minor seizures, they may progress to full-blown ***grand mal*** (grahn mahl [Fr. *grand mal* great sickness]) seizure activity. *Grand mal* seizures may also begin acutely as violent, generalized, *paroxysmal*,

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cerebral chaos. Among domestic animals, grand mal seizures are most common in dogs. So, let's make dogs our focus here, recognizing that any animal can experience them. Most grand mal seizures tend to follow a particular pattern. What I am about to describe as the beginning, middle, and end of a *grand mal* seizure is typical. But not every dog will experience each phase. Even if they experience all three phases, the expression of those phases may be different from one dog to the next. I am merely describing the classic, major symptoms associated with each stage.

During the ***preictal*** [*pre-* before + *ict(o)-* seizure + *-al* pertaining to] phase, the dog may experience something called an ***aura*** [L. "breath"]. During the *preictal aura*, the patient often feels some sort of odd sensation. That's a warning sign of the oncoming seizure. I like to think of this as a tremor that precedes a major earthquake or distant rumble of thunder that precedes a violent storm. For those who have experienced *grand mal* seizure activity, they learn to recognize the aura as a warning sign. Given sufficient warning, the individual may be able to seek a safe location. I had a Poodle in my youth that had ***idiopathic*** [*idi(o)-* one's own + *path(o)-* disease + *-ic* pertaining to; i.e., disease of unknown origin] ***epilepsy*** [Gr. *epilepsia* seizure]. She recognized her *aura*. And when she did, she would either seek one of our family members or hide under a piece of furniture. Service dogs for people with epilepsy are trained to recognize the patient's aura. It's not fully understood how they recognize it. There may be many recognizable features for a dog, from scent to visual cues. Once recognized, the service dog may perform a number of tasks, such activating a life-alert system, seeking someone for help, retrieving a phone, and/or alerting the patient to move to a position or place of safety (e.g., sitting or lying down on the floor).

During the ***ictus*** [L. *ictus* stroke, seizure], the patient loses consciousness and experiences generalized ***tonic*** [*ton(o)-* tension + *-ic* pertaining to; i.e., rigid muscle contraction] and ***clonic*** [Gr. *clonos* turmoil; i.e., rapid, repeated muscular contraction and relaxation] muscle activity. Any and all skeletal muscles may be involved, producing symptoms such as violent shaking and jerking, jaw clenching or chewing, and flailing of the limbs. Paddling motions are sometimes seen. The more violent the muscular activity, the

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more risk for injury for the patient. This can create a significant spike in body temperature, especially the longer *ictus* lasts. Animals should not be restrained. This could result in injury to the restrainer and/or the animal. If the dog is on a hard surface, slipping something like a towel under its head may help to protect the head and face from serious trauma. Contrary to the belief that the tongue can be "swallowed" during a seizure, this is physically not possible. Realistically, the ***frenulum*** [from L. *fraenum* a small bridle] secures the tongue to the floor of the mouth, making it impossible to swallow it. So, please do NOT attempt to grab the tongue. You will be injured, and you may facilitate serious injury to the animal's tongue. I think the misperception of "tongue swallowing" stems from some seizure victims becoming ***apneic*** [*a-* without + *pnea* breathing + *-ic* pertaining to] and ***cyanotic*** [*cyan(o)-* blue + *-tic* pertaining to].

It's really *tonic* spasm of ***laryngeal*** [*laryng(o)-* larynx + *-al* pertaining to] and respiratory muscles that prevent breathing during a *grand mal* seizure. *Apnea* plus *hyperthermia*, (hyperthermia uses more oxygen and produces more CO_2 from increased cellular metabolism), can make prolonged grand mal seizures life threatening. Neither the brain nor the heart tolerates ***hypoxia*** [*hypo-* low + *ox(o)-* oxygen + *-ia* condition of] and ***hypercapnia*** [*hyper-* excess + *capn(o)-* carbon dioxide + *-ia* condition of] well at all. And this is why ***status epilepticus***, a continuous series of grand mal seizure activity, is a life-threatening emergency. Patients in *status epilepticus* rapidly develop severe *hyperthermia*, *hypoxia*, *hypercapnia,* and *hypoglycemia*. The longer the seizure activity and these consequences persist, the less the chances of survival. After 15 to 20 minutes, significant, irreversible brain damage results. So, prompt therapy is needed to stop the seizure as quickly as possible. And because hyperthermia develops in *status epilepticus*, wrapping the dog in a blanket while transporting to a veterinary facility is not a good idea. That will only trap heat, making the hyperthermia worse.

Because *status epilepticus* is so life threatening, it's important for owners of ***chronic*** [*chron(o)-* time + *-ic* pertaining to; i.e., long-term] seizure patients to carefully time each seizure episode. Most grand mal seizures last only a matter of seconds, often 10 to 15 seconds. It

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just feels like an eternity for onlookers. Seizures lasting beyond 60 to 90 seconds can quickly put the dog at risk. It's a difficult call for an owner to make the decision to wait versus when to transport. In my book, it's better to be safe than sorry, especially if it takes a long time to reach the nearest veterinary facility. Sometimes, owners of known seizure patients are told to administer an emergency dose of ***anticonvulsant*** [*anti-* against + *convuls(o)-* seizure + *-ant* being] medication rectally and then transport immediately. Yep, even tablets will be absorbed from the rectal mucosa. Once at the veterinary facility, if the dog is still seizing, we administer an *intravenous* (IV) ***anticonvulsant*** [*-ant* one that is; *anticonvulsant* in the previous sentence was descriptor; but in this context *anticonvulsant* is a thing] to stop the seizure activity. Talk about pressure! Every second counts, and you've got an uncontrollable moving target testing your ***phlebotomy*** [*phleb(o)-* vein + *-tomy* to cut; i.e., puncture of a vein] skills (as if IV injections aren't challenging enough at times). In the emergency room, I became pretty skilled at IV injections in seizure patients. Whatever you do, hold on tight and secure that syringe with your hand holding the leg, as soon as you're in the vessel.

We and the dog can breathe easier, once the animal is in the ***postictal*** [*post-* after + *ict(o)-* seizure + *-al* pertaining to] phase. This stage is incredibly variable. So, let me share some of the most common features. Many times, with full relaxation the dog may urinate and/or defecate. Many dogs gently engage in paddling (i.e., moving the legs as if running, while still unconscious in lateral recumbency). The level of consciousness is altered during the postictal phase, while the brain recovers from the actual seizure. So, the dog may be confused, disoriented, not fully aware, or able to respond to its environment. How long the postictal phase lasts often depends on the length and severity of the seizure. On average, it may last anywhere from 5 to 30 minutes. Don't be surprised if the dog is ***hyperpneic*** [*hyper-* excessive + *pnea* breathing + *-ic* pertaining to; i.e., increased rate and depth of breathing]. Remember, a seizure patient immediately following *ictus* will likely be *hypercapnic* and possibly *hypoxic*, depending on the length of the seizure. So, respiratory centers in the *pons* and *medulla* should stimulate greater rate and depth of breathing to restore blood gas

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homeostasis. Even after the postictal phase is over and the dog is fully conscious and aware, it may sleep for a very long time. Grand mal seizures can be exhausting.

It is very important to diagnose the *etiology* of seizure activity. So many things can cause seizures, including *hypoglycemia*, *hypocalcemia*, other electrolyte disturbances, liver disease, kidney disease, toxins, brain trauma, brain tumors, infectious diseases, and *idiopathic epilepsy*. Much diagnostic testing and history taking are required to make an accurate diagnosis. And accuracy is important. Obviously, simply administering *anticonvulsant* therapy will not help if the patient is hypoglycemic or has ingested a toxin. The underlying problem must be treated. We may start with simple blood work. Progressively, we add more and more diagnostics, ruling out possible etiologies along the way. We may perform CT or MRI scans. We may even perform ***electroencephalography*** [*electr(o)-* electricity + *encephal(o)-* brain + *-graphy* recording] ***(EEG)***, to see if we can determine a particular area or regions of the brain with abnormalities. But sometimes historical information provides the key to our diagnosis.

Do you remember the story I told in Chapter 7 when we discussed nutrition? I told of a *geriatric* dog that was experiencing seizures. We had run multiple diagnostics. I think the only thing left to perform was an *EEG*. As it turned out, we didn't have to do the *EEG*. One simple question led to a diagnosis and treatment that did not require *anticonvulsant* therapy. Do you remember the historical clue that was the key in our diagnosis? It was the dog's diet. The owners were feeding a puppy diet to that old dog. Puppy diets contain large amounts of protein. That dog's aging liver could not manage the ammonia and other by-products of protein digestion, creating the ***hepatic*** [*hepat(o)-* liver + *-ic* pertaining to] ***encephalopathy*** [*encephal(o)-* brain + *-pathy* disease of]. Sure, we could have blindly treated the dog with anticonvulsants, but they would not have been effective. And depending on the anticonvulsant, the dog's hepatic disease could have been made worse.

We need to exhaust every possible etiology of seizure activity before we commit to a diagnosis of idiopathic epilepsy. Patient history is important for diagnosis and treatment. The first time a

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dog has a seizure, we typically do not initiate anticonvulsant therapy. Why not? As we've said, we need to determine the cause. Plus, we need to know the nature of the seizure activity. Often, we don't witness the seizures. Much of the time we rely completely on the owner's assessment and description of the seizure activity. In some cases, that information may be skewed or inaccurate. I remember a lady who was convinced her newly acquired, one-year-old dog was having seizures, two different kinds, in fact. And she wanted treatment for the dog because it was happening so frequently. She said that the dog had what she called "running seizures," where the dog would run full-speed like a maniac through the house. That would often progress into what she called "jumping seizures," where the dog would begin jumping on and off of the sofa. It was challenging to help her understand that this was not seizure activity at all. She finally did understand that it was simply normal, playful behavior of a high-energy, young dog. So, instead of anticonvulsants, we offered tips to help her focus the dog's energy in structured play.

For legitimate seizures, we need to know details about the seizures individually and comparatively over time. Minimally, we want them to keep a log, including date, time of day, length of seizure (especially *ictus*). Yes, time of day for each seizure is important, especially if the dog is experiencing clusters of seizure activity. Ideally, we would like to know length of time (by the clock, not estimated) for each stage (*preictus*, *ictus*, and *postictus*). Descriptions of what they observe in each stage is also very valuable to know. Finally, it would be of value to know events preceding the seizure. For instance, had our geriatric dog owner kept a log, it would have been very telling for us to see that the dog ate a meal approximately an hour or two prior to each seizure. That would have led to our question about diet much earlier (though that should be part of every routine patient history). Still, had the dietary question been asked sooner, we could have saved the dog the stress of some of the diagnostics (such as the CT) and the owner the expense. Typically, we don't initiate *anticonvulsant* therapy until a dog is having seizures more frequently than once every 4 to 6 weeks. However, if the veterinarian observes a pattern where the duration of ictus is increasing significantly, the severity of each

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subsequent seizure is worsening, or the dog begins experiencing clusters of seizures or status epilepticus, therapy may be started sooner. And that's why the log is so important.

Infectious Neurotropic Diseases

There are numerous ***neurotropic*** [*neur(o)-* nerve + *trop(o)-* influencing, loving + *-ic* pertaining to] infectious diseases. Most of these are viral ***pathogens*** [*path(o)-* disease + *gen(o)-* producers]. However, other organisms, such as parasites, bacteria, and fungal agents, can also cause neural disease. For example, the immature larvae of the raccoon roundworm *Baylisascaris procyonis* have an affinity for CNS tissue. That can create life-threatening ***encephalitis*** [*encephal(o)-* brain + *-itis* inflammation of]. By the way, *Baylisascaris* is a ***zoonotic*** [*zo(o)-* animal + *nos(o)-* disease + *-tic* pertaining to; i.e., a disease transmissible between animals and humans] parasite. Bacteria such as *Listeria sp*. (also zoonotic) also cause *encephalitis*. Periodically in the news, we hear reports of various foods being recalled because of possible contamination with bacteria such as *Listeria sp*.

Now, you would think if bacteria are causing *encephalitis*, we simply treat the animal or person with antibiotics, right? Or if it's a fungal *pathogen*, treat with ***antimycotic*** [*anti-* against + *myc(o)-* fungus + *-tic* pertaining to] agents. If only it were that easy. You see, once a *pathogen* sets up housekeeping in the CNS, it's a challenge to get drugs into those tissues to eradicate the organism. Why? There is a protective mechanism called the ***blood-brain barrier (BBB)*** that may limit access to the CNS tissue.

Blood-Brain Barrier

The ***BBB*** is a protective barrier for the brain and spinal cord. It is designed to keep potentially harmful substances out of the CNS. CNS capillaries limit access, just by virtue of capillary wall structure. When we've talked about other capillaries of the body, we've described them as the smallest blood vessels of the body. Remember, capillaries are so small that red blood cells have to pass through them single-file. In other *somatic* tissues and *viscera*, the capillary walls are thin, with only a basement membrane outside and a single layer of *endothelium* inside. We've also described tiny

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gaps between the endothelial cells around teeny-tiny fenestrations (holes) in the basement membrane. That structure, in other tissues, creates ease of simple diffusion, facilitated diffusion, osmosis, and movement of white blood cells from the bloodstream into the tissues. If you'd like to refresh your memory about some of these concepts, you may want to review hematology and lymphatic information in Chapter 3, blood gas diffusion in Chapter 5, renal filtration in Chapter 6, and absorption of nutrients in Chapter 7.

Capillary wall structure in the CNS is a bit different. First, there are no fenestrations. **Strike one** against things getting into CNS tissue. The endothelial cells have no gaps. **Strike two** against things getting into CNS tissue. The capillary walls in the CNS are reinforced by *glial* cells called ***astrocytes*** [*astr(o)-* star + *cyt(o)-* cell]. Have you ever seen a starfish with really long, skinny arms? *Astrocytes* remind me of those. *Astrocytes* create almost a third layer to the capillary wall with their appendages. **Strike three** against things getting into the CNS. Finally, CNS tissue has its own ***phagocytic*** [*phag(o)-* eating + *cyt(o)-* cell + *-ic* pertaining to] cells. It has to because with that capillary wall structure it makes it mighty difficult for white blood cells to exit the bloodstream. The tiny *phagocytic glial* cells of the CNS are called ***microglial*** [*micro-* small + *gli(o)-* glue + *-al* pertaining to] ***cells***. These cells freak out at anything they perceive to be a threat, seek it out, and destroy it. **Strike four** against things getting into the CNS. All of those things contribute to what we call the *BBB*. The *BBB* is well intended. But it is the reason we are limited in drugs that can be used to treat CNS diseases, especially diseases caused by pathogens.

I know what you're probably wondering. If the BBB makes it so difficult for things to get into the CNS, then how on earth do pathogens get in? Well, in cases such as *Baylisascaris* that I mentioned earlier, those larvae simply bore a hole in the capillary wall and slither in. I remember a case of a young girl years ago, who died of a brain abscess (pus accumulation). The bacteria in the abscess was *Escherichia coli*, a pathogen of the digestive tract. If you're wondering how bacteria made their way from the digestive tract to the brain, you're not alone. Numerous doctors, pathologists, and other specialists wondered the same thing. It wasn't until they discovered *Trichinella* sp. larvae in the brain that they could connect

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the dots. Adults of *Trichinella* rapidly reproduce in the small intestine, giving birth to live larvae. Those larvae penetrate the intestinal wall to enter the bloodstream, with the goal of making their way to muscle tissue where they encyst and remain dormant. Well, a couple of those larvae bored through the capillary wall and slithered into the brain, dragging the *E. coli* with them.

There are other organisms, such as viruses, that naturally use cells of the body to reproduce. Viruses don't have all of the *intracellular organelles* they need for reproduction. So, they hijack cells of the body for their own purposes. Compared with many other *pathogens*, viruses have an easier time gaining entry. Let's take a look at a few of the most common and potentially deadly *neurotropic* viruses.

Equine Encephalomyelitis

Equine encephalomyelitis [*encephal(o)-* brain + *myel(o)-* spinal cord + *-itis* inflammation of] is a common viral disease of horses. There are a number of viral subtypes that cause the disease in horses worldwide. The viral agents that cause ***eastern equine encephalomyelitis (EEE)*** and ***western equine encephalomyelitis (WEE)*** are most commonly seen in the United States and Canada. Horse owners routinely vaccinate against these diseases. It is a *zoonotic* disease. And all of the potential signs and symptoms that you might expect with *encephalitis* and *encephalomyelitis* occur in horses and humans alike. In horses, we commonly see fever, reduced mental responsiveness, aimless wandering, circling, head-pressing, ***ataxia*** [*a-* without + *tax(o)-* order + *-ia* condition; i.e., stumbling gait], *paresis*, *paralysis*, and seizures. Many horses die within the first few days. People die too, especially geriatric adults and children.

Don't worry, you can't catch this from a horse. The virus is not transmitted horse to horse or horse to human. It is transmitted by mosquitoes. And the mosquitoes pick up the virus from ***viremic*** [*vir(o)-* virus + *-emic* pertaining to blood; i.e., high concentrations of virus in the blood] birds. Wild birds serve as a reservoir for the virus. In temperate regions, such as Michigan where I live, we tend to see equine cases of *EEE* in late summer and early fall. Human cases often follow horses by about 2 weeks. Warm, wet summers

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promote mosquito reproduction, supporting increased numbers of equine and human cases. Horses can be protected through vaccination. People need to avoid mosquito exposure (evening, nighttime, and dawn) and use mosquito repellents.

West Nile Virus

West Nile virus (WNV) is another *zoonotic, neurotropic pathogen* that's found worldwide. Like *equine encephalomyelitis*, *West Nile* is transmitted by mosquito. And the mosquito population acquires the virus from the wild bird reservoir. In terms of birds, the virus appears to affect blue jays and crows the most. But anyone (bird, mammal, or human) can contract the virus and develop *encephalitis*, with symptoms like that of *EEE*. Birds tend to succumb to the encephalitis the most. So, paying attention to sick birds with neurologic signs, as well as dead birds, is important. The more affected birds we see, the more probable disease in humans and other animals. In Michigan, cases of WNV have been reported every year in animals and people since 2002. August and September are peak months in the state. Numbers of cases depends on the mosquito population. Which is why in Michigan, mosquito pools are tested routinely in every county, multiple times during the year. As of this writing, the State of Michigan reported that, as of November 2018, 154 mosquito pools, 187 birds, 103 humans, 2 horses, 1 alpaca, and 1 deer tested positive for WNV.[a] The Centers for Disease Control and Prevention (CDC) reported 2223 human cases in the United States during the same period; 61% of those people infected developed *encephalitis* and or ***meningitis*** [*mening(o)-* meninges + *-itis* inflammation of].[b] The number of human cases surprised me.

Now, not everyone will develop encephalitis. In fact, many people may have no symptoms to only mild symptoms (like a slight fever and feeling a little "off"). Those with compromised immune systems (animal and human) are the ones who may develop severe or even lethal encephalitis. A vaccine is available for horses, which may be why the number of equine cases reported in Michigan were so low. Most horse owners routinely vaccinate against WNV. To protect ourselves against West Nile, we need to avoid peak mosquito activity and use mosquito repellents.

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Rabies

Rabies is different. Rabies is a *zoonotic, neurotropic* disease of mammals that is directly transmissible between animals and humans. Rabies is probably one of the most lethal mammalian *neurotropic* viruses on the planet. And just to be clear, humans are mammals. And this organism is transmitted directly from one infected individual to the next. We, as veterinary professionals, are probably one of the more at-risk human populations. We handle and treat animals every day. And rabies is one of those diseases that we often push to the bottom of our rule-out list when dealing with neurologic patients. And like I mentioned in Chapter 7, not all cases of rabies present like *Cujo*.[c] Yet, we seem to persist with the notion that rabies is always transmitted by crazed, aggressive, frothing-at-the-mouth animals that bite someone. If that is all we look for, we will likely be wrong—dead wrong.

Now it is true that the rabies virus is transmitted through an infected mammal's saliva. But no bite wound is required. If you've been "in the business" long enough, you know how dry and cracked our poor hands can become from hand washing and cleaning the clinical environment. All we need to do to become infected with the rabies virus is to stick our dry, cracked fingers into an infected animal's mouth. It's more likely, as I pointed out in Chapter 7, to be exposed to a patient in the *"dumb"* (i.e., ***paralytic*** [*paralysis* + *-tic* pertaining to]) form of the disease. We'll be on guard and take precautions if we see the *"furious"* form of the disease.

So, what exactly happens in rabies? Well, this is a neurotropic virus from the get-go. As soon as it enters the body, let's say through our cracked knuckle, it replicates in the tissues there and then rapidly enters motor nerve fibers. It continues to replicate as it moves ***retrograde*** [*retro-* backward + *grade* to step] along the motor nerve fibers. Of course, this causes *neural* injury along the way. So, we may perceive some *paresis* in the affected hand and arm. It will probably produce paralysis of the hand and arm by the time the virus reaches the spinal cord. Once in the spinal cord, the virus uses neurons to travel *retrograde* to the brain. Remember, we were infected through a wound on our hand. There is very little territory to traverse from the infected arm to the brain. Once the virus reaches the brain, it quickly begins replicating in the brain, creating

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encephalitis. (Remember, the *BBB* is of little use against this virus. It has used neurons, not the bloodstream, to reach our brain.)

Before it kills us, the virus is going to follow the *trigeminal* (fifth cranial) nerve to infect our salivary glands. And because of inflammation in the brainstem and other cranial nerves, we'll have ***dysphagia*** [*dys-* difficulty + *phag(o)-* eating + *-ia* condition of; i.e., difficulty swallowing] and ***hypersialosis*** [*hyper-* excess + *sial(o)-* salivation + *-sis* condition of], making us drool and froth-at-the-mouth. Every drop of saliva will be teaming with the virus. Any unsuspecting human medical professionals caring for us, before we die, will also be at risk of becoming infected, especially if rabies is not on the physician's rule-out list. And in the United States, rabies is not a top rule-out among the human population. When we die (not if but **when** at this point in the disease process), it will probably be from paralysis of our respiratory muscles. Until then, the *encephalitis,* ***myelitis*** [*myel(o)-* spinal cord + *-itis* inflammation of], and body-wide peripheral ***neuritis*** [*neur(o)-* nerve + *-itis* inflammation of] will be painful. In fact, we'll probably be ***hyperesthetic*** [*hyper-* excess + *esthes(o)-* sensation + *-tic* pertaining to], making the slightest touch or air movement across our skin a new adventure in pain. It's not a pleasant way to die. Now, here's the thing. The way to confirm a diagnosis of rabies is to microscopically examine the brain for the organism. But unless rabies is suspected, it's not routine to send human brains off to a lab to be examined for the virus. Fortunately, the virus does not live long at all outside the body or in a corpse. So, the mortician is probably safe.

If you're thinking "that sounds like a cheap horror film," you'd be wrong. Much of what I've shared is based on the experience of a young man who died from rabies in the United States. One of his doctors, during the man's hospitalization, had seen someone with the disease years earlier. Had he not had that prior experience, the physician may not have suspected rabies. Because he did, he and the young man were able to document details of the experience. In the end, the young man did die. Numerous people and animals die every year around the globe from rabies. Once infected, the chances of survival in an unvaccinated individual are highly unlikely, even with treatment. If already symptomatic with encephalitis, there is

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probably no chance of survival. Vaccinated individuals stand a much better chance, especially if the wound is cleaned immediately and booster immunizations are administered. But there are no guarantees. Most of us in veterinary medicine are immunized and have our serum titers checked periodically. But no one knows precise values for protective titers. It's not exactly one of those diseases we want to run clinical trials to find out.

This disease should frighten you. It frightens me because I know how deadly it is. And this is one of those diseases where we must work diligently to protect both the animal and human populations. Animals should be immunized. All veterinary professionals should be immunized. And all animals with neurologic signs of encephalitis, especially if unvaccinated, should be treated as if they are rabies positive until proven otherwise. It's the safe play that can save lives. Over the course of my career, I have known of too many cases of human exposure (veterinary professionals and students in our facility and others), who were needlessly exposed because of complacency. And remember this doesn't involve just us. It's the patient's (unvaccinated) human family we need to protect too. And if the animal has rabies, treatment of exposed humans must be initiated as soon as possible to give them the best chance of survival. I hope that I have your attention. This *neurotropic* virus is no laughing matter. It's deadly. Now, let's continue with CNS anatomy.

Spinal Cord

The ***spinal cord***, as we mentioned earlier, is also part of the *CNS*. This is the super-highway for getting sensory input to the brain and motor output to *somatic* and *visceral* tissues. It begins at the foramen magnum and passes through the vertebral foramen (spinal canal), ending in the vicinity of the caudal lumbar vertebrae or ***lumbosacral*** [*lumb(o)-* loin, lumbar vertebrae + *sacr(o)-* sacrum + *-al* pertaining to] joint. Yes, I'm waffling on that because there are subtle species variations for the actual end of the spinal cord. Beyond the end of the spinal cord are numerous nerve fibers that fan out, looking sort of like a horse's tail. Ancient anatomists called that aggregate of nerves the ***cauda equina***. So, in any animal's tail,

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we won't find the spinal cord, only nerve fibers that extend from the *cauda equina*. The schematic in Fig. 11.7 shows the beginning and end of the spinal cord, within the vertebral foramen.

Structure and Function

If you look at Fig. 11.8, you'll see a schematic of a transverse view of the spinal cord, as though you've sliced through the spine and you're looking at the exposed end of the spinal canal. First, look at the cord itself. You'll notice that the *white matter* and *gray matter* are the reverse of what we described for the cerebrum. In the cerebrum we said that cell bodies making up the gray matter were on the surface and myelinated nerve fibers of the white matter were deeper. Of course, the brain does all of the computing, not the spinal cord. We need the spinal cord to make fast-track connections between the brain and peripheral nerves. So, much of the spinal cord is composed of white matter. The *myelinated tracts* in the white matter of the cord provide the fastest transit of nerve impulses **to** *(afferent)* and **from** *(efferent)* the brain. The Evolve animation, Vertebral Column and Spinal Nerves, provides a very good visualization of basic spinal cord structure, including the myelinated tracts.

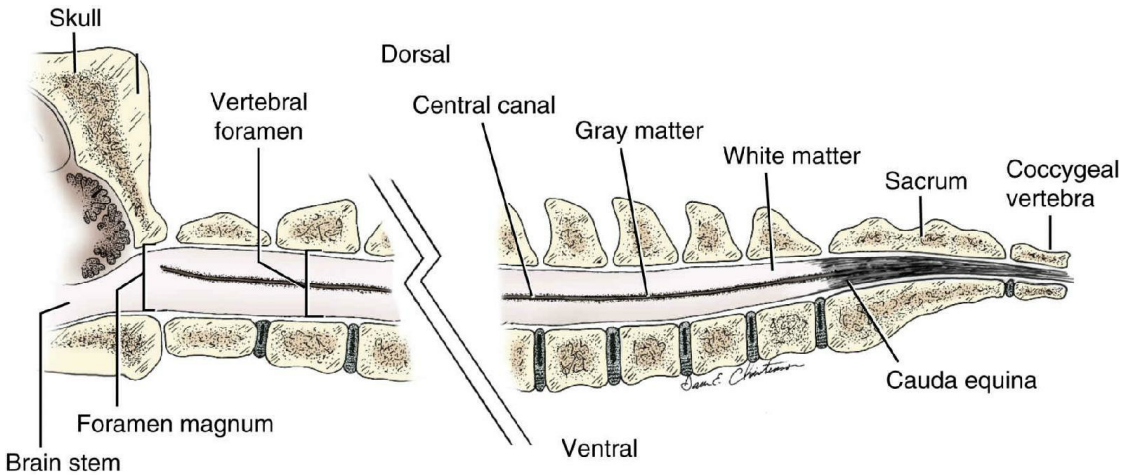

FIG. 11.7 Spinal cord schematic (midsagittal view).

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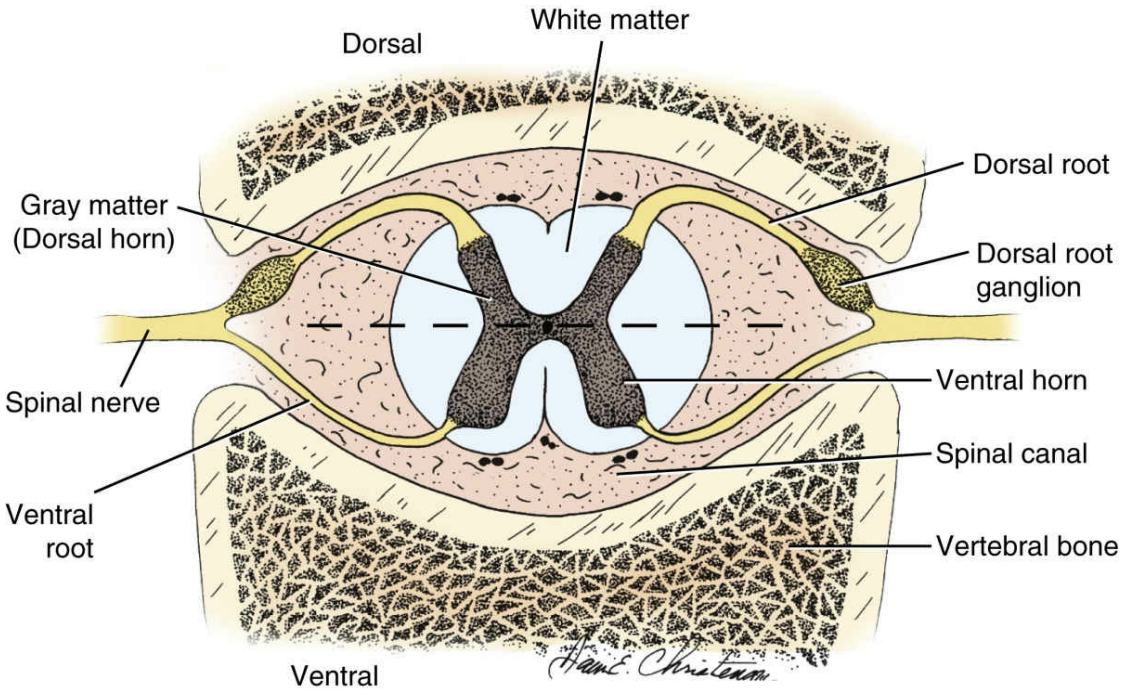

FIG. 11.8 Spinal cord schematic (transverse view).

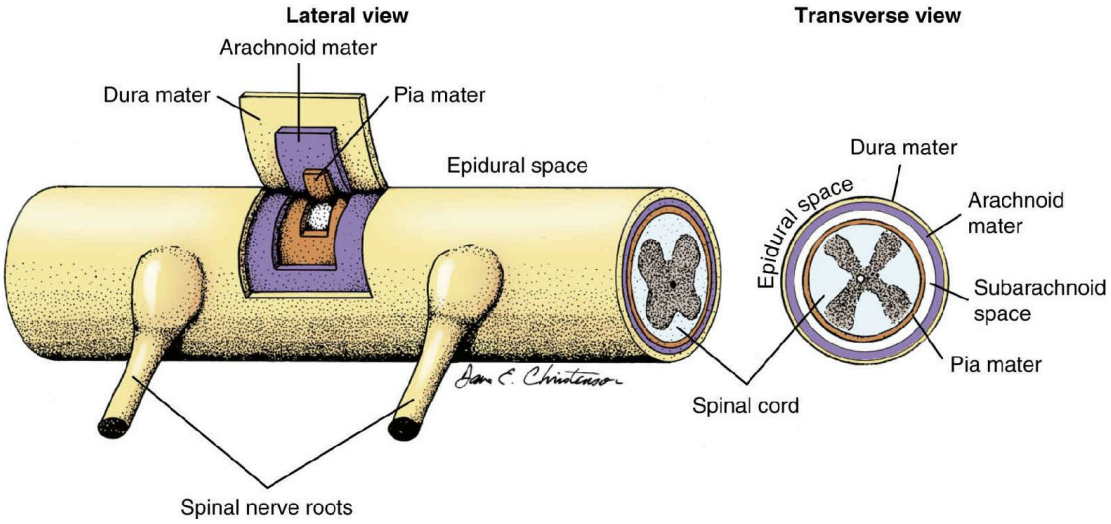

FIG. 11.9 Meningeal schematic.

Why on earth do we need gray matter in the cord? Good question. It's all about making connections. The cell bodies found in the gray matter of the cord are from spinal *interneurons* [*inter-* between] and motor neurons associated with spinal nerves. You may have noticed the horizontal dashed line through the middle of the spinal cord, in Fig. 11.8. This is a gross simplification, but try to think about everything above the line as sensory and everything

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below the line as motor. So, sensory input coming into the cord through the dorsal root of a spinal nerve may need to connect to afferent fibers in that white matter to whisk the sensory information off to the brain. Interneurons make those sorts of connections. And if motor impulses are rapidly coming along efferent fibers from the brain, connections will need to be made to the correct "exit from the freeway," to motor neurons of the spinal nerve. Interneurons make connections like that. When we talk about spinal reflexes later, you'll learn that interneurons may even be used to make connections for some reflex arcs. By the way, most interneurons and motor neurons are multipolar neurons, like the one in Fig. 11.1. You won't find interneurons outside the CNS. In fact, you won't find cell bodies of motor neurons outside the CNS. So, all of the spinal interneuron cell bodies and peripheral motor neuron cell bodies are clustered in the gray matter of the cord, creating that butterfly appearance. To be clear, gray matter in the cord has nothing to do with thinking. It only makes connections and passes on nerve impulses along.

Meninges

The word ***meninges*** (meh-nin'jēz) is the plural of the Greek word *meninx*, meaning membrane. So, *meninges* are membranes. Specifically, the meninges are membranes that surround the brain and spinal cord. There are three meninges, as shown in the crude schematic views of Fig. 11.9. The ***dura mater*** (doo'ruh mah'tur [L. *dura* hard *mater* mother]) is the outer ***meningeal*** [*mening(o)-* meninges + *-al* pertaining to] layer. It is made of tough, dense connective tissue. In the skull, the *dura mater* is physically connected to the bone of the skull. In the spinal column, there is space between the *dura mater* and the vertebral bone. That space is called the ***epidural*** [*epi-* upon + *dura* + *-al* pertaining to] ***space***.

The middle *meningeal* layer is the ***arachnoid*** (uh-rak'noid [*arachn(o)-* spider + *-oid* resembling] or ***arachnoid mater***. It got its name because it looks something like a spider web. And strands of that web-like material connect the *arachnoid* to the innermost meningeal layer. But those connections don't hold those membranes close together. The connections actually keep them

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apart, creating the ***subarachnoid*** [*sub-* beneath + *arachnoid*] ***space***. And filling that space is ***cerebrospinal*** [*cerebr(o)-* cerebrum + *spin(o)-* spine + *-al* pertaining to] ***fluid (CSF)***.

The innermost meningeal layer is the ***pia mater*** (pe'uh mah'tur [L. *pia* tender, soft]). The *pia mater* is the thinnest, most delicate of the meninges. It lies directly on the brain and spinal cord. The vessels woven through the membrane provide important ***vascular*** [*vascul(o)-* vessel + *-ar* pertaining to] supply for the CNS tissue.

Cerebrospinal Fluid

CSF, as we said a moment ago, fills the *subarachnoid space*. That means that this fluid completely surrounds the brain and spinal cord. It is predominantly water. In fact, if you were to look at normal *CSF* grossly, you would think you were looking at water. Just like water, CSF is clear (transparent), colorless, and well—watery. It's pretty much ***acellular*** [*a-* without + *cellul(o)-* cells + *-ar* pertaining to], with fewer than 9 cells/μL.[a] Let me put that number into perspective for you. In ***hematology*** [*hemat(o)-* blood + *-logy* study of], a normal red blood cell count for an adult dog might be more than 7,000,000/μL and a normal white blood cell count might be 12,000/μL. Comparatively, the number of cells in normal *CSF* are hardly worth mentioning. *CSF* is also very, very low in protein. It doesn't even have much in the way of electrolytes. Na^+, Cl^-, and HCO_3^- predominate; and their concentrations are just high enough to draw water into the CSF. Concentrations of Ca^{++} and K^+ are very low. Because CSF contains so little, it's what we call an ultrafiltrate, kind of like filtered water.

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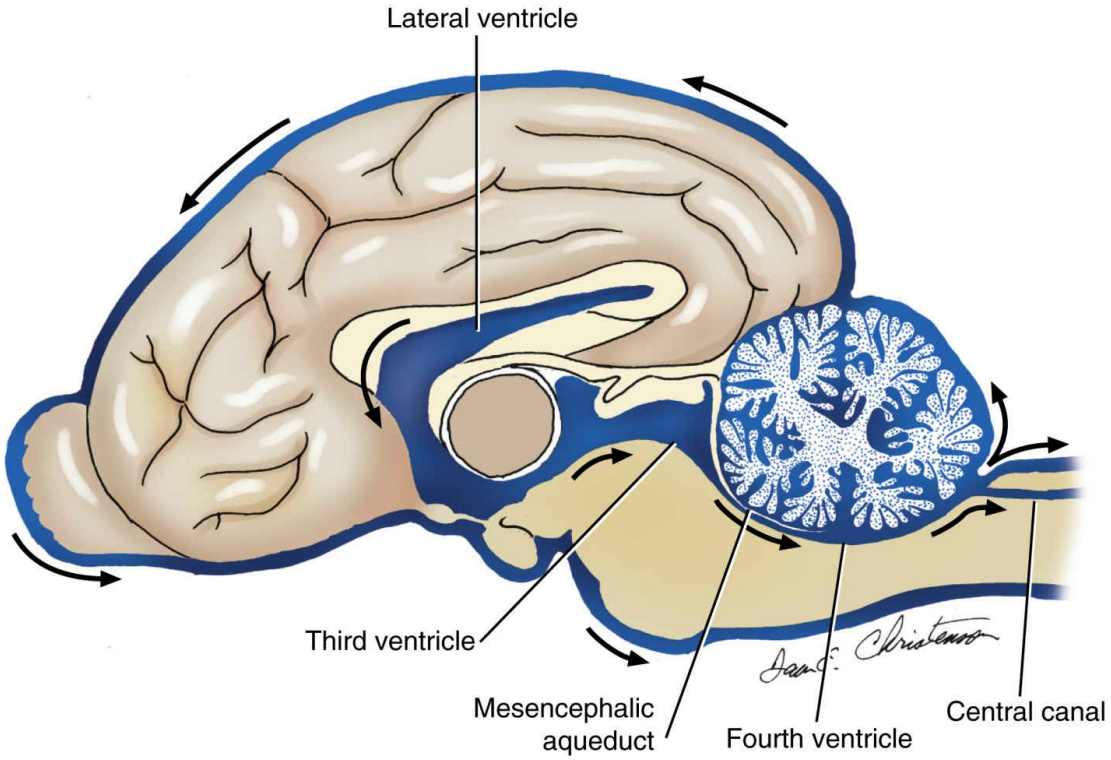

FIG. 11.10 Schematic of cerebrospinal fluid flow.

Where is CSF produced? It's produced by specialized clusters of capillaries in the ventricles of the brain (Fig. 11.10). Most CSF is produced in the lateral ventricles. The third ventricle contributes as well. And it flows one way, from rostral to caudal, through the ventricles: from the lateral ventricles, to the third ventricle, through the ***mesencephalic*** [*mes(o)-* middle + *encephal(o)-* brain + *-ic* pertaining to] ***aqueduct*** [L. "water canal"], and finally through the fourth ventricle. From there, it diverges into the subarachnoid space around the brain and around the spinal cord. And believe it or not, there's a bunch of this produced. CSF is probably replenished about five times a day. So, where does it go? Well, there are bundles of ***dural*** [*dur(o)-* dura mater + *-al* pertaining to] vessels here and there that protrude into the subarachnoid space. So, ultimately the old CSF is eventually reabsorbed into the bloodstream.

Okay, I can just feel your burning question—"So, if CSF is pretty much just water, what the heck does it do?" Excellent and justifiable question! Well, protection is an important role. Remember, the *subarachnoid space* filled with CSF completely surrounds the brain and spinal cord. That fluid-filled space actually provides *shock absorption* for the CNS tissues. This is especially important for the brain. Remember, the dura mater surrounding the

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brain is tight against the skull. So, the only wiggle room the brain has is the CSF-filled subarachnoid space. This cushions the spinal cord too. But in the vertebral canal, there's a lot more wiggle room. In fact, much of the *epidural space* within that canal has adipose (i.e., fat) tissue. So, between the CSF cushion and the adipose, the spinal cord has a little more shock absorption. And believe it or not, by surrounding the brain and spinal cord with fluid like that, it actually makes them somewhat buoyant. That's right, *buoyancy*. The brain and spinal cord float to a degree. Do you want your brain "sitting" on the rock-hard bone of your skull? Over time, that would probably damage neurons. Granted, it's minimal buoyancy. Still, it protects precious neurons.

Homeostasis of CNS tissue is a very important, yet challenging task. Think about the blood-brain barrier for a moment. That makes the CNS like Fort Knox—nearly impenetrable. It would be nice to have some help to maintain homeostasis, at least of the interstitial fluid. Well, CSF helps with that. It is much easier for CNS metabolic wastes, unnecessary ions and molecules, and excess neurotransmitter substances to diffuse into the CSF than the bloodstream in many respects. Think about it. CSF is pretty much water. Diffusion from an area of high concentration to low is pretty easy. Concentrations of things can't get much lower than CSF. And because things such as metabolic wastes need to be removed effectively, it's a very good thing that CSF is replenished as frequently as it is.

With various *encephalopathies*, ***myelopathies*** [*myel(o)-* spinal cord + *-pathy* disease of], and *meningitis*, collection of *CSF* for analysis may be very beneficial in making a diagnosis. Evaluation of CSF usually includes ***cytology*** [*cyt(o)-* cells + *-logy* study of] and chemical (protein and electrolytes) analysis. And ***immunology*** [*immun(o)-* immunity + *-logy* study of] and cultures (bacterial and fungal) may also be used to help diagnose various *pathogens*. The collection site can influence the results of the analysis. For instance, if we're concerned with brain disease, collecting the sample via the atlanto-occipital joint will best reveal conditions of the brain. If we were to collect the sample from the lumbar area, the fluid could be altered as it flows around the spinal cord. So, we wouldn't know if abnormal findings were from the brain or the cord. Of course, if

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we're concerned with spinal disease, then collection from the lumbar area is appropriate. In dogs and cats, we collect between L_5 and L_6. In horses, we use the lumbosacral joint. At all of these collection sites, the subarachnoid space is much larger, making it possible to safely enter the space with a spinal needle, without striking the spinal cord (or cauda equina in lumbar taps, or perhaps brain stem in cervical taps). CSF taps in animals are sterile procedures performed under general anesthesia.

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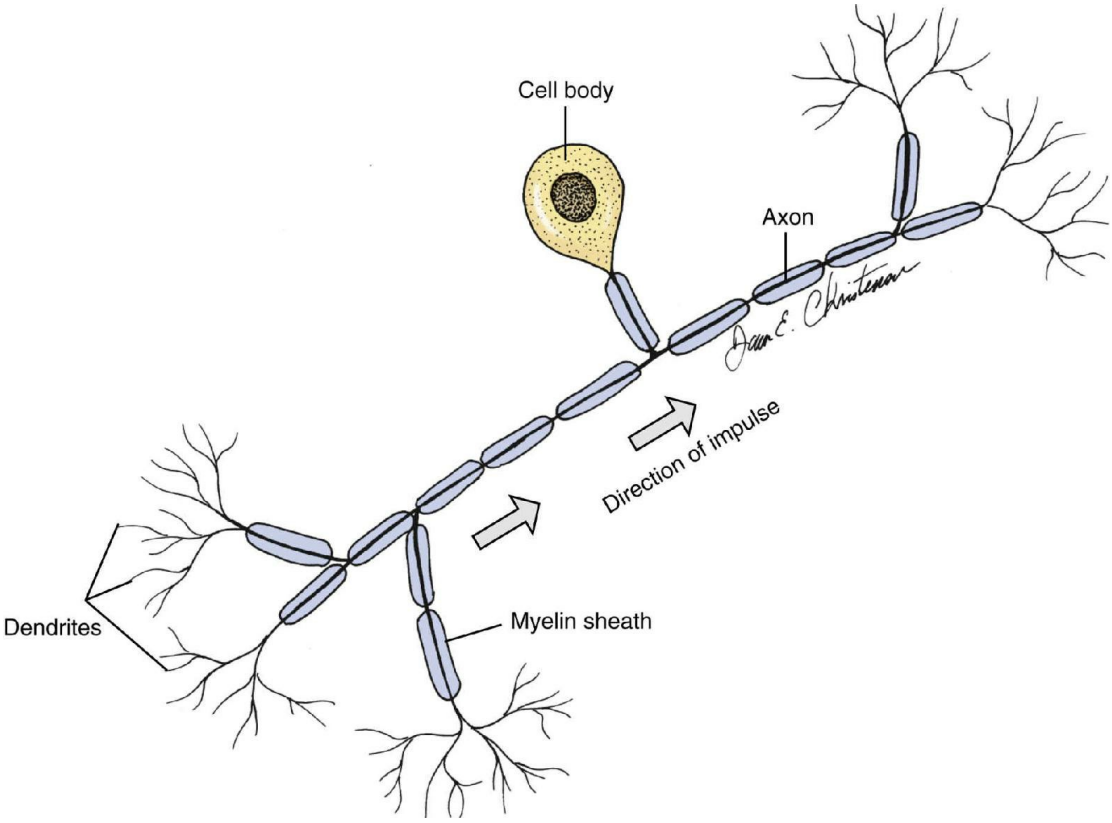

FIG. 11.11 Unipolar neuron.

Hydrocephalus

Hydrocephalus [*hydr(o)-* water + *cephal(o)-* head + *-us* a, the] is a congenital abnormality. Any animal can be born with *hydrocephalus*, but it's most common in toy dog breeds, like the Chihuahua. The lateral ventricles of the brain become extremely dilated with CSF. The animals are usually easy to spot because the dorsal skull is markedly domed. And because hydrocephalus develops in the fetus, as the ventricles dilate so does the skull. You may recall looking at suture lines of the skull in Chapter 4 (Fig. 4.17C). That's where the bones fuse together. In most animals with hydrocephalus, those bones never fuse. That can actually be a very good thing. Because this billowing brain has room to expand, neurologic deficits may be minimal. Yes, some animals are severely affected, having significant difficulty with movement, cognition, and seizure activity. But those with mild impairment can actually lead fulfilling lives. The biggest concern for the owners is head trauma. Remember, the brain is not fully protected by the skull. So, a minor blow to the head could kill the animal.

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Peripheral Nervous System

The ***peripheral nervous system (PNS)*** includes everything outside of the CNS (i.e., outside the brain and spinal cord). This is where we have strategically placed various sensors and specialized receptors to collect and transmit sensory information to the CNS. And on the flip-side of the coin, we need motor nerve fibers for carrying impulses to target tissues and organs, to elicit responses.

Sensory Nerve Fibers

Most sensory nerve fibers in the PNS use a different type of neuron, from the multipolar neuron you've already seen. Sensory fibers are largely composed of ***unipolar*** [*uni-* one + *pol(o)-* pole + *-ar* pertaining to] ***neurons*** (Fig. 11.11). Yeah, it's another weird looking cell. It kind of reminds me of a cyclops, with the cell body set off to the side like that. Beyond that, its functional parts are still the same, with one-way neurotransmission from dendrites to axon.

Now here's a fun fact that I think will blow your mind. We're talking about sensory nerve fibers, right? So, the tiny, dendritic receptors of a unipolar neuron could be in the skin of your big toe. And the rest of the myelinated fiber will stretch all the way up to your spinal cord! Can you believe it?! That's long! Ah, now let's think about that cell body that just seems to be just dangling out there. We're going to cluster cell bodies like that along nerve fibers. Go back and look at Fig. 11.8. Do you see the bulge along one of the dorsal roots, labeled ***dorsal root ganglion*** [Gr. *ganglion* knot]? Well, that's where we clustered all of those cell bodies of all the *unipolar neurons* making up the sensory part of that nerve. Along peripheral nerves, we simply don't have the luxury of space to spread out neuronal cell bodies like they are in the brain and spinal cord. So, whenever you see a *ganglion* along peripheral nerve fibers, you'll know it's a cluster of cell bodies. The actual axonal part of the unipolar neurons of the nerve provide uninterrupted, fast neurotransmission to the spinal cord. That's important, especially if the sensory stimulus is something harmful. In the spinal cord, sensory neurons synapse on other neurons. For simple spinal

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reflexes, they synapse on motor neurons to produce the reflex movement. The patellar (knee-jerk) reflex is a good example of this. More complex reflexes use an interneuron to connect to the motor pathway. (More on reflexes later.) But more powerful stimulation, involving larger numbers of sensory nerve fibers will synapse on sensory nerve tracts to the brain. Interneurons are definitely used to make those connections. That is certainly the case with pain.

Pain

Pain is not pleasant. Under normal circumstances, the initiation for pain begins with a ***noxious*** [L. *noxius* hurtful] ***stimulus***. *Noxious stimuli* cause tissue damage. With greater tissue damage, more sensory neurons are stimulated. And because the stimulation is from trauma of some sort, we need interpretive powers (conscious recognition) to be engaged. We need conscious recognition and decision-making for a response, as fast as possible. That's why such a large bundle of sensory neurons needs to be stimulated at the point of injury. Those sensory nerve fibers, using interneurons, will synapse on large bundles of sensory nerve fibers in the spinal cord. As we mentioned earlier, when we talked about the spinal cord, most myelinated sensory nerve tracts in the cord are in the dorsal cord. Those tracts associated with pain, especially deep pain (i.e., severe, from deep tissue injury), are located deeper in the cord. It is very important to be able to consciously recognize deep pain, as unpleasant as it is. If we do not, further serious injury to the body may occur that leads to death. If we cannot consciously perceive deep, severe pain, there are three primary areas that may be "broken": the peripheral sensory nerve fibers, the spinal cord, or the brain. Often, it's the "middle-man" (spinal cord) that's diseased. We'll discuss this, when we talk about intervertebral disc disease (IVDD) later. Just remember, there should *always* be conscious recognition of pain.

Now, in people, conscious recognition of pain is easy to perceive. We usually verbalize our displeasure with the pain, by saying "Ouch!" or perhaps some other things that cannot be repeated here. Plus, people can be asked to rate their pain on a 10-point scale, with 10 being the worst, excruciating pain anyone could possibly experience. In animals, it's a little more difficult for us to discern if

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they are painful. If they are painful, then we are challenged with discerning the level of that pain. And because each individual (human and animal) has differences in pain tolerance and how they respond to pain, we have an added layer of difficulty ranking pain in our patients. If a patient cries out, we may have a pretty good clue that they may be feeling pain. But some animals, usually pampered little lap dogs, cry out from fear before we even touch them. I've seen other patients that were so stoic that they simply turn their head away and say nothing. And then there are species differences. Dogs react differently to pain than cats. Cats mask pain and disease symptoms very well. Many horses, on the other hand, tend to overreact to pain, with major physiologic responses that in and of themselves can have negative physical consequences. We need to know: can the animal feel pain?—especially when we are doing a neurologic exam for spinal injury. Second, if an animal is in pain, we need to be able to rate the pain to guide our medical management with ***analgesics*** [*an-* without + *alges(o)-* pain + *-ic* pertaining to]. So, to discern pain, actual pain and its level, we need to take a number of things into consideration.

There are numerous pain assessment tools. In human medicine, practitioners promote some sort of medical intervention when pain reaches level 5 (out of 10). Why? If pain reaches a level 8 or greater, many of the ***nociceptors*** [*noci(o)-* injury + *cept(o)-* receptor + *-or* a/an; i.e., a pain receptor] in the brain and spinal cord will be blocked and unable to respond to *analgesics*, even powerful ***opioid*** [*opi(o)-* opium + *-oid* resembling] *analgesics*. It takes a long time to get pain under control when it's escalated to those extreme levels. There are a variety of assessment tools for pain. The human 10-point pain scale has been modified for veterinary use, focusing on behavioral and physiologic changes to guide pain assessment. Other pain assessment tools use a 0 to 4 scale that looks exclusively at behavioral and *psychological* changes for pain assessment. And because there are differences between species and between ***chronic*** (long-term) and ***acute*** (sudden) pain, many of the assessment tools have been modified to address those differences. Regardless of the assessment tool used, it is important for us as veterinary professionals to recognize and minimize pain in our patients. Organizations and links for more detailed pain management

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information are provided at the end of this chapter.

Analgesia and Anesthesia

So, if an animal is in pain, how exactly do various therapeutics reduce or eliminate pain? Well, first we need to remember that pain involves stimulation of *nociceptors*. The stimulus is *noxious* (i.e., from injury, either *acute* injury or *chronic* disease). Tissue injury of any kind causes inflammation.

We talked about ***inflammation*** in detail, in Chapter 3. You may want to go back to that chapter to review inflammation. We said that the cardinal signs of inflammation include ***heat***, ***erythema*** [Gr. *erythema* flush upon the skin, i.e., redness], ***edema*** [Gr. *oidema* swelling], ***pain,*** and ***loss of function***. Well, heat, *erythema*, and *edema* are all *vascular* responses to tissue injury. This is, in part, why your thumb will throb with every heartbeat after you've smashed it with a hammer. (Yeah. Been there, done that.) Can we directly reduce this vascular response as well as nociceptor stimulation directly at the injured site? Yep. That involves cold therapy. In fact, in acute injuries, application of cold can dramatically reduce and control pain. How? First, it causes *vasoconstriction*, just like in our discussion of thermoregulation earlier. Heat and erythema are taken off the table. Edema is prevented by reducing capillary permeability. So, less water seeps into the interstitium. Cellular activity, especially of white blood cells such as neutrophils that tend to increase inflammation, is reduced. And as far as the sensory neurons are concerned, neurotransmission is slowed by cold. In fact, cold therapy not only slows neurotransmission for *analgesia*, but it can even temporarily stop neurotransmission from the injured site to provide ***anesthesia*** [*an-* without + *esthes(o)-* sensation + *-ia* condition/state of; i.e., numbness]. This is why cold therapy is so important during the *acute* stages of injury and the immediate ***postoperative*** [*post-* after + *operat(o)-* surgery + *-ive* pertaining to] period (i.e., first 48 hours).

Analgesic medications work differently depending on the type. In Chapter 3, we talked about ***antiinflammatory medications***. Both steroidal medications, such as prednisolone, and nonsteroidal antiinflammatory drugs (NSAIDs) reduce inflammation. These are given systemically, either orally or by injection. And in various

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ways they break the inflammatory cycle and cascade of events. By reducing inflammation, they effectively help to reduce pain.

Other *analgesic* medications, such as ***opioids***, block *nociceptors* in the brain. That reduces the perception of pain. Each drug is designed to work on specific pain receptors in the brain. Of course, many of these medications also tend to produce sedation and ***somnolence*** [L. *somnolentia* sleepiness], as well as reduce mental acuity and cognition. For most animals, this is not a problem. They don't operate heavy machinery or drive automobiles. And rest while recovering from traumatic injury or surgery is good. But it needs to be balanced. We can't afford to make an orthopedic patient recovering from a total hip replacement so dopey that it falls down, resulting in additional injury to the dog and its surgical site.

Anesthesia does just what the word tells us—it removes all sensation. This is not simply about reducing pain, like *analgesics*, keeping pain minimized and at tolerable levels. Anesthesia is about removing all sensation. How does that work? Well, that depends on the drug and what portion of the nervous system we target with it. Local and regional anesthesia focuses on peripheral sensory nerves.

For ***local anesthesia***, we inject the *anesthetic* agent in a very focal area. Let's say we have a dog with a minor laceration (cut) on the dorsal metacarpus. It's a minor, clean injury, but it still needs to be cleaned appropriately, surgically prepped, and sutured closed. We can inject a short-acting anesthetic agent, such as ***lidocaine***, into the skin surrounding the injury. We encircle the whole injury when we inject the drug. How does lidocaine provide anesthesia so that we can clean, prep, and close the wound without the dog feeling anything? Lidocaine targets the sodium channels of the sensory neurons. It locks them closed. If we cannot engage the sodium channels and the sodium-potassium pump, there can be no depolarization of those nerve fibers. (See how your knowledge of physiology is useful?) But there is a catch. Lidocaine usually lasts for only about 20 to 30 minutes. If it takes us longer to clean, prep, and suture that dog's wound, the dog will be painful before we're finished. We may only need another 15 minutes or so to finish up. So, instead of using plain lidocaine, the veterinarian may decide to use *lidocaine with epinephrine*. How will the epinephrine help? It causes localized vasoconstriction. How are drugs such as lidocaine

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removed from tissues such as the skin? They're absorbed by the bloodstream and removed from the area. By causing vasoconstriction, removal of lidocaine from the tissue is slowed keeping it in the area longer, to provide the needed anesthesia. We can finish our work and have a very happy, painless patient in the process.

For ***regional anesthesia***, we can inject local anesthetic agents around (not in, but *around*) major peripheral nerves or spinal nerve roots. Again, if we use a drug such as lidocaine, it does the very same thing to the nerve or nerve root that we infuse around. It prevents sodium channels from opening. No neurotransmission means no pain. Of course, this time that loss of sensation is over a much larger area—a whole limb, or the whole flank, or the tail. We often use regional blocks for orthopedic surgery. Orthopedic surgery is very traumatic and painful. By removing pain sensation in the surgical limb, less general anesthesia is required to keep the animal safely sleeping during the procedure. And because the drugs used for these limb blocks last for hours, postoperative recovery is much better because the animal is less painful. ***Epidural*** *anesthesia* is frequently used for canine and feline orthopedic surgery in the rear limbs. Most of the nerves supplying the rear limbs, tail, and perineum branch off caudal to the lumbosacral joint. So, we insert our needle through the *lumbosacral* joint into the spinal canal. We do **not** penetrate the meninges. The tip of the needle sits in the *epidural* space. By infusing the drug or combination of drugs in that *epidural* space, we bathe all of the nerves (cauda equina and spinal nerves) in the area. All sensation caudal to the infusion is blocked. Again, this provides anesthesia and analgesia during surgery and postoperatively. There is a catch to regional anesthesia. It's not selective for sensory neurons. It affects sensory and motor neurons. So, if we have blocked sensory neurons to the rear limbs, the dog or cat will not be able to walk for a short time postoperatively. For cows, such as those we discussed in Chapter 7 needing surgery for a *displaced abomasum*, regional anesthesia (e.g., ***paravertebral*** [*para-* near + *vertebr(o)-* vertebrae + *-al* pertaining to] lumbar anesthesia) is great. It lets the cow remain standing and awake during the surgery, without feeling any pain.

General anesthesia focuses on neurons in the brain. In various

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ways, they profoundly suppress neurotransmission. This renders the animal unconscious and unaware. This is good. I appreciated sleeping, totally unaware during my total knee replacement surgery. I did not want to feel what was happening and certainly did not want to hear the surgeon using bone saws and hammers, thank you very much. That's the purpose of general anesthesia. Of course, here again, these agents can't selectively target the cerebrum. The whole brain, including the cerebrum, diencephalon (hypothalamus for thermoregulation), and brain stem (respiratory and cardiac centers) will be suppressed by general anesthetic agents. (Did you catch that? The word *anesthetic* is a descriptor, while *anesthesia* is the actual state of.) So, we need to be very vigilant when we monitor patients under general anesthesia. Monitoring of temperature and cardiovascular and pulmonary function are very, very important.

A few final thoughts on pain management. There is no "silver bullet," "one-size-fits-all" answer to *analgesia*. There are many, many ways to manage pain in veterinary patients. The management protocol is tailored to each patient, guided by the patient's condition, the nature of the patient's injury or disease, *acute* versus *chronic* pain, and so forth. Many times, a ***multimodal*** (i.e., many, varied methods) approach provides optimal results, by "attacking" the pain pathway from multiple angles. And by planning ahead, when we know our medical and surgical procedures will create pain, we can preemptively prevent pain from spiraling out of control. Beyond that, we need to recognize pain in our patients and intervene as appropriate.

Motor Nerve Fibers

Most ***motor neurons***, like the vast majority of neurons in the body, are multipolar neurons. Motor neurons do what their name implies —facilitate movement or other activities (e.g., salivary gland secretion). We actually classify motor neurons into two categories: ***upper motor neurons (UMNs)*** and ***lower motor neurons*** *(LMNs).* The classification is based on location. It's pretty simple, really. As shown in Fig. 11.12, UMNs are located in the *CNS*, and LMNs are located in the *PNS*. So, as the schematic shows, UMNs are in the

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brain and spinal cord. LMNs are found in the PNS, including cranial nerves, the ***vagosympathetic*** [*vag(o)-* vagus nerve + *sympathet(o)-* sympathetic nerves + *-ic* pertaining to] ***trunk***, spinal nerves, and all of their branches.

We classify motor neurons as upper and lower to help us organize this very complex system. When we are dealing with motor deficits, we use our neurologic exam and other diagnostics to isolate the ***lesion*** [L. *laesio* to hurt; i.e., the point of injury/disease]. By isolating the neural lesion, we are better equipped to determine ***prognosis*** [Gr. *prognosis* foreknowledge; i.e., predicted outcome] and possible courses of treatment. Obviously, when dealing with CNS disease and injury, UMN conditions will be more difficult to treat. That said, we can functionally use peripheral nerves (LMNs) to help us isolate CNS lesions. For instance, in head trauma patients, we use the cranial nerves to determine the area(s) and severity of brain injury. But to make those assessments, we need to know what each cranial nerve does and where they integrate in the brain.

Cranial Nerves

All but the first two cranial nerves are directly connected to the brain stem. The ***first cranial nerve*** or the ***olfactory*** [***olfact(o)-*** smell + ***-ory*** pertaining to] ***nerve*** is directly connected to the centers in the cerebrum (temporal lobes) associated with the *sense of smell*. The olfactory bulb (Fig. 11.3) is part of this nerve. Compared with humans, the olfactory nerve is pretty big in most animals. We'll talk about olfactory ability later, when we discuss special senses.

The ***second cranial nerve*** or the ***optic*** [*opt(o)-* vision + *ic* pertaining to] ***nerve*** is also directly connected to the cerebrum, to centers associated with vision. As we mentioned earlier, visual centers are located in the occipital lobes of the cerebrum.

All other cranial nerves arise from the *brain stem*. You will find all cranial nerves and their basic functions listed in Table 11.1. A summary of cranial nerve functions is also discussed and shown in the Evolve animation entitled *Cranial Nerves*. And yes, it is important to know the basic function of each. When we conduct a complete *neurologic* examination, we always evaluate cranial

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nerves. Knowing where they are, what they do, and where they integrate in the brain helps us assess both nerve and brain integrity. By recognizing specific cranial nerve deficits, in addition to other UMN abnormalities (e.g., ↓ mentation and poor motor coordination) we may be able to isolate and determine severity of brain injury. This is very important in head trauma.

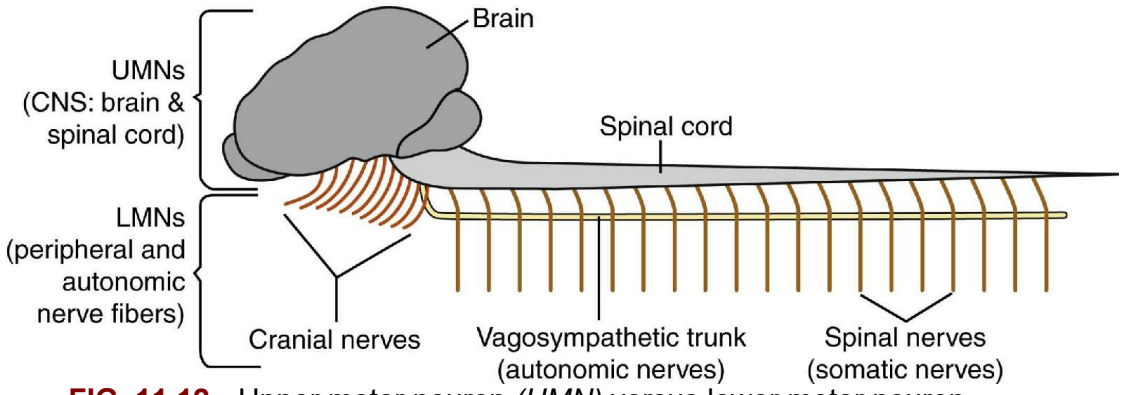

FIG. 11.12 Upper motor neuron *(UMN)* versus lower motor neuron *(LMN)* schematic.

TABLE 11.1

Cranial Nerves

Name	Number	Sensory Function	Motor Function
Olfactory	I	Sense of smell	None
Optic	II	Vision	None
Oculomotor	III	none	Dorsal, dorsolateral, medial, dorsomedial, ventral, and ventromedial eye movements; opening of eyelids; parasympathetic fibers to iris
Trochlear	IV	none	Ventrolateral eye movement
Trigeminal	V	Portions of face, mouth, and head	Salivary glands, lacrimal (tear) glands, muscles for chewing
Abducens	VI	none	Lateral eye movement and retraction of eye into orbit
Facial	VII	Portions of face, including eyelids and nose; taste sensation	Eyelid closure; primary motor input for muscles of face and head for facial expression and lip movement; parasympathetic fibers for salivary glands and lacrimal glands.
Vestibulocochlear	VIII	Hearing and equilibrium	None
Glossopharyngeal	IX	Taste and sensory input from throat	Tongue movement, some pharyngeal muscles for swallowing
Vagus	X	Both visceral and some somatic (e.g., pharyngeal and laryngeal) sensory input	Laryngeal muscles (for arytenoids, epiglottis and vocalization); gag reflex; swallowing; primary motor nerve fibers for parasympathetic branch of autonomics
Accessory	XI	none	Throat, neck, cranial back, and shoulder muscles
Hypoglossal	XII	none	Tongue movement

With traumatic brain injury, edema is problematic. The brain has no room to swell. So, as cerebral edema develops, ***intracranial*** [*intra-* within + *crani(o)-* head + *-al* pertaining to] ***pressure (ICP)***

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increases. Progressively over time (minutes to hours), elevated ICP disrupts cerebral function first. Mentation and cognition (superficial cerebral function) are often affected first, including those areas associated with UMN control of cranial nerve function. Loss of motor coordination points to the cerebellum being affected. As ***ICP*** increases further, the deeper tissues of the brain (*diencephalon* and *brainstem*) that control basal functions, such as *thermoregulation*, heart rate, and breathing, become abnormal. By recognizing early changes in mentation and cranial nerve function, we might be able to intervene and prevent further increases in *ICP*. Doing so prevents serious, life-threatening brain injury. By the way, cerebral edema can develop rapidly following head trauma. So, we evaluate mentation and cranial nerve function, frequently, perhaps as often as every 10 minutes. Many cranial nerve abnormalities observed may indicate an *ipsilateral* lesion. So, a ***subdural hematoma*** over the right cerebrum may create cranial nerve deficits on the right side of the face or head. That's why understanding basic function of each cranial nerve is important. Remember, motor impairment for the rest of the body (e.g., *hemiparesis* [*hemi-* half + *paresis* weakness]) from the same lesion will be exclusively *contralateral*.

Of all of the cranial nerves listed, one of the most important is the ***10th cranial nerve,*** also called the ***vagus nerve***. That nerve is important in so many ways. First, consider its impact on the larynx and pharynx. Functional capacities of swallowing and motor control of the arytenoids and epiglottis are essential for protection of the lower airways. As pointed out in Chapter 5, the vagus nerve is found in the jugular furrow near the jugular vein and carotid artery. If we damage the vagus nerve or some of its branches in the neck, we can impair laryngeal and pharyngeal function. And when it comes to breathing, the ***phrenic*** [*phren(o)-* diaphragm + *-ic* pertaining to] nerve is a branch of the vagus nerve. Plus, as you can see in Table 11.1, the *vagus nerve* is directly involved with the *autonomic nervous system (ANS)*. It is the *vag(o)-* part of the *vagosympathetic trunk*—the *parasympathetic* branch of the *ANS*. You'll learn the functional importance of that a little bit later.

Spinal Nerves

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Spinal nerves are those connected directly to the spinal cord. In Fig. 11.8, you can see two spinal nerve roots, one on either side. You already know that the *dorsal root* is associated with sensory nerve pathways. And because we said earlier that the ventral portions of the spinal cord are predominantly motor neurons (UMNs to be exact), then the ventral spinal nerve roots must be motor neurons. Yep! In Chapter 4, Fig. 4.21 shows a left lateral view of the lumbar vertebrae. The lateral holes formed by the vertebrae are ***intervertebral*** [*inter-* between + *vertebr(o)-* vertebrae + *-al* pertaining to] ***foramina*** [L. *foramen* an opening]. The spinal nerves pass through those *foramina*. Fig. 4.22 shows a bit better how the dorsal and *ventral roots* converge forming a single spinal nerve that passes through the intervertebral foramen and can be seen outside the spine. That means each spinal nerve has both sensory and motor neurons (LMNs to be exact). Outside the spinal column, spinal nerves subdivide into various peripheral nerves. Let's look at a few peripheral nerves of importance.

Peripheral Nerves of Importance

There are numerous peripheral nerves. If we were trying to make you budding neurologists or neurosurgeons, we would talk about each and every one. But that's not our goal. So, let's simply focus on a few that have significant clinical importance. These are the nerves that play a key role in skeletal muscles of the front and rear limbs. Before we talk about them individually, you need to understand that these nerves do not stem from individual spinal nerves. You see, nerves of the limbs are actually formed by a number of spinal nerves. But there's not much room to get a whole bunch of individual nerves into a leg. They need to be gathered into a convenient, concentrated bundle. We refer to a bundle of nerves like that as a ***plexus*** [L. *plexus* braid]. So, the "braid" of nerves (from some cervical and thoracic spinal nerves) that ***innervate*** [*in-* in, into + *nerv(o)-* nerve + *-ate* to be, act of; i.e., to supply with nerves] the forelimb is called the ***brachial*** [*brachi(o)-* arm + *-al* pertaining to] ***plexus***. The brachial plexus enters the leg in the axillary region with the major vessels supplying the limb. The ***lumbosacral plexus*** *innervates* the rear limb. Obviously, from its name, you can probably figure out that lumbar and sacral spinal nerves contribute

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to the *lumbosacral plexus*.

Radial Nerve

The ***radial nerve*** is one of the major nerves that branches off of the *brachial plexus*. The *radial nerve* is of importance because its LMNs innervate the extensor muscles of the elbow, carpus, and digits. It also provides superficial sensory innervation for most of the front limb with the exception of caudal and palmar surfaces. That's an awful lot for one nerve. As the saying goes, "It's not wise to put all of your eggs in one basket." If the radial nerve is damaged, functionally much is lost. And the most vulnerable part of this nerve is where it crosses over the lateral aspect of the mid-shaft humerus. I've seen numerous dogs and cats through the years that have sustained traumatic injuries to the lateral brachium and lost radial nerve function. Whether ***monoparesis*** [*mono-* one + *paresis* weakness of; i.e., of one limb] or ***monoplegia*** [*mono-* one + *plegia* paralysis of; i.e., of one limb] develops all depends on the severity of injury to the nerve. And it's not complete paralysis, because we still have flexor function. So, it's not as bad as a brachial plexus injury. But without extensors, the animal can't bear weight on the limb. They typically drag the leg, scuffing and abrading the skin of the dorsal paw and carpus. And because they've also lost sensation, they can create deep tissue injuries to the digits and carpus.

Many times, limb amputation is the best option. They have no functional use of the leg anyway. But we don't want to rush into that amputation too soon. After all, nerves can repair themselves to a degree. But it's a slow process. So, we need to be patient. While we wait to see if the nerve can recover and regain at least marginal function, we need to protect the paw and carpus. A durable boot needs to be worn whenever the animal is up and active. If radial nerve function is not regained, then amputation may be pursued. And if partial sensation is restored, amputation may be needed sooner than later. Sometimes poorly functioning, damaged sensory neurons can create some strange and intense abnormal sensations. I know this first-hand from the compression of some of my cervical spinal nerves. My tactile sensation was muted, but I had extreme tingling and burning sensations in my left hand and arm that drove me nuts. Animals experiencing those sorts of sensations usually

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begin to bite and chew the limb or foot. (No, I didn't do that.) That often prompts the owners to concede to an amputation. The good news is, because the animal has probably been living with the useless leg for many months, recovery following the amputation is easy. They don't have to learn to walk three-legged. They've already been doing it for a while.

For most amputations, the animal has either been experiencing profound pain in the limb or the sensations I've described with radial nerve injury. Amputating the limb may not stop the pain or sensations. There is a phenomenon called "***ghost pain***" or "***phantom pain,***" where the individual continues to feel those noxious sensations even after the limb is gone. The brain remembers the last sensations felt from the limb. We can prevent phantom sensations by providing regional anesthesia, with a nerve block prior to the amputation. Even early in general anesthesia, although the animal is sleeping, its brain is still able to perceive pain and other sensations. So, if we perform a nerve block, say of the brachial plexus, the last thing the brain feels from that limb is complete loss of sensation—anesthesia. That is precisely what we want.

Sciatic Nerve

The ***sciatic*** [L. *sciaticus*, Gr. *ischiadikos* ischial; pertaining to the ischium] ***nerve*** is the most important nerve of the hind limb. The *sciatic nerve* and its branches (tibial and common peroneal nerves) innervate most of the muscles of the rear limb. The only major muscles of the rear limb NOT innervated by the sciatic and its branches are the adductors, quadriceps, and a portion of the gluteals. So, the semis, gastrocnemius, cranial tibial, and many of the abductor muscles receive motor input from the sciatic nerve and its branches. And sensory nerve fibers provide sensation for all but the medial thigh. You just thought radial nerve injury was bad. This is awful! If we lose complete LMN function of the sciatic nerve, we cannot support weight on that leg. In dogs and cats, the sciatic nerve can be easily damaged by ***intramuscular*** [*intra-* within + *muscul(o)-* muscle + *-ar* pertaining to] (IM) injections into the caudal thigh. It lies just caudal to the femur. We don't even need to hit the nerve directly. If the drug injected is somewhat irritating, it can still

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cause *neuritis* [*neur(o)-* nerve + *-itis* inflammation of] and loss of function. The loss may be temporary or permanent.

I actually know a person who experienced *monoparesis* from an IM injection in his semis as a child. He had just enough nerve function left that he could bear a little weight. But he walked with an extreme limp, dragging the leg and having to shift most of his weight to the opposite leg when walking. He could not run. And he fatigued quickly, especially the affected leg. Decades had passed since the injury. So, his sciatic function would not improve. Fortunately, his condition was not painful. He sustained that *iatrogenic* injury when he was 8 years old. The thigh of an 8-year-old child is larger than many of our feline and smaller canine patients. Yet, his sciatic nerve was still injured. How much more at risk are our patients? So, please, be very, very careful using the semis for intramuscular injections. If other sites are available, use them. Why risk potential disabling injury, like my friend experienced?

Obturator Nerve

The ***obturator*** [L. *obturator* one that closes] ***nerve*** innervates the adductors of the medial thigh. (No, I don't fully understand that Latin meaning. Perhaps it refers to the functional closing of the angle of the rear leg in relation to the rest of the body, when the adductors draw the leg toward the midline? That's the best I can figure out.) Now, functionally the obturator nerve is a minor player, compared with the sciatic and radial nerves. So, why bother mentioning it? Well, it's most commonly damaged by pelvic fractures or ***dystocia*** [*dys-* difficult + *toc(o)-* birth + *-ia* condition of]. Now for dogs or cats with unilateral pelvic fractures that damage the obturator nerve, they can probably adapt to paralysis of their adductors. But for a dairy cow with *dystocia*, whose calf stuck in the birth canal crushes both obturator nerves, she's got a big problem. She's a big girl with a lot of body weight, even if she's in perfect body condition. If she has no functional adductors for her rear legs, she can't stand. Sure, the dairy farmer can try to wait for her to recover, over the next day or so. Waiting much longer than that with a down cow will probably result in pressure ***necrosis*** [*necr(o)-* death + *-sis* condition of] of tissues over bony protuberances. Paralysis of any kind in livestock is almost impossible to manage,

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just because of their size and body weight. So, if she can't recover quickly, euthanasia is probably the kindest solution.

Spinal Reflexes

Part of our neurologic examination always includes ***spinal reflexes***, especially in patients with some sort of *myelopathy* [*myel(o)-* spinal cord + *-pathy* a disease of]. (Note: *myel(o)-* in Chapter 3 meant bone marrow. Context is everything. In neurology, *myel(o)-* means spinal cord.) **This is important:** neural impulses of spinal reflexes go no further than the spinal cord. They do not, I repeat, do ***NOT*** involve the brain at all. That is the nature of most reflexes. What's the purpose of reflexes? Protection. We need fast, reflex actions that do not require contemplation and decision-making. Spinal reflexes provide the fastest responses possible, to prevent serious bodily harm.

The stimulus for any spinal reflex is minor. It could be a light, tickling sensation on a paw. It could be the light touch of a fly landing on the back of a horse. The point being that the stimulus for spinal reflexes is minor. And because it's minor, very few sensory neurons are stimulated. That is why sensory nerve impulses for a reflex synapse only on efferent motor pathways from the spinal cord. Let's consider the "gold standard" for spinal reflexes: the patellar reflex. This provides a perfect example of something we call a ***reflex arc***.

For the ***patellar reflex***, the patellar ligament is gently struck (in the little notch between the patella and tibial crest). This creates a slight tug, ever so slightly stretching the patellar tendon and distal quadriceps muscle. That slight stretching action stimulates sensory stretch receptors in the quadriceps muscle. The afferent nerve impulse rapidly travels along sensory nerve fibers to the spinal cord. In the spinal cord, the sensory neuron synapses directly on a motor neuron. The efferent nerve impulse travels rapidly along the motor nerve fibers to the quadriceps muscle, stimulating contraction of the muscle. The effect is a "knee jerk" or slight extension of the stifle. As explained and shown in the Evolve animation, *Reflex Arc of a Patellar Impulse*, this is the simplest example of a reflex arc. You can see how, especially in the cord, the sensory and motor neurons form an arc. The animation also used

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the term ***monosynaptic*** [*mono-* one + *synap(o)-* synapse + *-tic* pertaining to], in reference to the patellar reflex. There is only one synapse between sensory and motor pathways. You may have also noticed that this reflex arc uses only LMNs. The synapse may be in the cord, but the motor neurons are part of a spinal nerve. That makes them LMNs. By the way, when performing a neurologic exam on a patient, the patellar reflex is the one spinal reflex that should be easily assessed. If there is no trauma to the stifle or quadriceps, absence of a patellar reflex points to a lesion along the spinal nerve, spinal nerve roots, or the spinal cord at that specific lumbar segment (between L_4 and L_6).

There are many other simple reflexes, such as the patellar reflex, that involve simple stretch receptor stimulation and *monosynaptic* reflex arcs. Other monosynaptic spinal reflexes include that of the biceps, triceps, cranial tibial, and gastrocnemius muscles. These are simple reflexes and reflex arcs because the sensory impulse originates from stretch receptors in the muscle mass and the motor impulse returns to the same muscle mass to cause contraction of it. The arc forms a simple loop from start to finish.

More complex, ***polysynaptic*** [*poly-* many + *synap(o)-* synapses + *-tic* pertaining to] spinal reflexes include the ***panniculus*** [from L. *pannus* cloth], ***perineal*** [*perine(o)-* perineum + *-al* pertaining to], or ***anal*** [*an(o)-* anus + *-al* pertaining to], and withdrawal reflexes. With these reflexes, the neural impulse does not begin and end in the same location. The sensory stimulation begins superficially in the skin, and the motor impulse causes contraction elsewhere in a muscle or group of muscles. It's still a reflex arc, but it is more complex. And because of the complexity of connecting a sensory nerve fiber to one or more motor nerve fibers, *interneurons* will be required to make connections. That is why these are *polysynaptic* reflex arcs.

Let's look at the ***withdrawal reflex.*** It takes very little to stimulate superficial sensory receptors in a dog's foot. Just tickling the fur between the toes, touching the pads, or gently grabbing a web of skin between the toes is sufficient. The afferent nerve impulse travels along sensory nerve fibers to the spinal cord. In the cord, the impulse must synapse on numerous motor nerve fibers. Why? Because it will require all of the flexor muscles in the limb to create

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the withdrawal. So, to quickly make those connections, the sensory nerve fibers synapse simultaneously on interneurons that then synapse on all of the related motor nerve fibers of limb flexors. When the efferent motor impulses simultaneously reach the flexors, the limb and foot are withdrawn. What I find amazing with this reflex is that everything I've just described happens in the blink of an eye. No thought. No decision-making. Just pure and simple connections of a reflex arc.

What is even more amazing is the ***panniculus*** [from L. *pannus* cloth] ***reflex***. What the heck does a piece of cloth have to do with this reflex? Well, they probably originally called it the *panniculus reflex* because the muscle involved in the reflex is a broad, thin sheet of muscle (like a sheet of cloth) over the thorax. The actual name of the muscle involved is the ***cutaneous*** [*cutane(o)-* skin + *-ous* pertaining to] ***trunci muscle***. That's a really long a name. Panniculus is shorter and, in my humble opinion, a much better name for the reflex. But if you'd like to refer to the cutaneous trunci reflex, you may. No harm, no foul.

Have you ever seen a fly land on the back of a horse and the horse responded with a big muscular twitch or quiver over its chest? That was a spinal reflex—specifically the *panniculus reflex*. This reflex is complex and is most definitely *polysynaptic*. And the whole pathway for this reflex runs from the tubercoxae in a large animal (iliac crest of a dog or cat) all the way cranial to somewhere in the vicinity of T_1. (Motor fibers that innervate the cutaneous trunci muscle branch off at T_1.) That fly could land anywhere along that horse's back, between the tubercoxae and T_1, and stimulate contraction of that muscle. You see, when the fly landed, it stimulated sensory receptors in the skin. The afferent nerve impulse traveled along sensory nerve fibers to the spinal cord in the vicinity of the fly. In the cord the peripheral sensory nerve fibers synapsed on interneurons to superficial sensory neurons in the spinal cord. The impulse traveled cranially along those sensory nerve fibers and then synapsed on motor neurons around T_1. The efferent nerve impulse then traveled along the motor nerve fibers to the cutaneous trunci muscle, stimulating contraction that we saw as a quiver over the lateral chest. What a complicated pathway!

The beauty of the *panniculus reflex IS* the complexity of the

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pathway. You see, when we are concerned with spinal injury or disease, the panniculus is the one spinal reflex that can actually help us to isolate a spinal lesion. Think about it. The reflex arc of the *panniculus reflex* always has to follow that complicated pathway. And we know its borders. We know in a dog that the cranial border is near the shoulder around T_1 and its caudal border is near the iliac crest. If every step along that pathway is normal, we can elicit the *panniculus reflex* by stimulating the skin anywhere alongside the spine, from the shoulder to the iliac crest. But if there is injury to the cord in between the borders of the reflex arc, we can probably find approximately where that injury is. The pathway will be broken by the spinal lesion. So anywhere caudal to the lesion, we won't be able to stimulate the pathway. That means we won't be able to activate the panniculus reflex caudal to the spinal lesion. For example, let's say that a Dachshund has a spinal cord injury from compression caused by a ruptured intervertebral disc between T_{13} and L_1. We will systematically and sequentially test the panniculus reflex bilaterally, starting at either the shoulder or the iliac crest. Every point that we stimulate and test cranial to T_{13}-L_1 will elicit the panniculus reflex. Every point stimulated and tested caudal to T_{13}-L_1 will not. So, with the *panniculus reflex*, we can isolate the spinal cord compression at T_{13}-L_1. And we can do it with no fancy equipment or diagnostics. How cool is that? And that is the value of spinal reflexes as part of a neurologic exam.

Paresis and Paralysis

Paresis and paralysis can be caused by damage to *upper motor neuron (UMN)* pathways or *lower motor neuron (LMN)* pathways. You already know that paresis is weakness and paralysis is complete loss of motor function. How might UMN pathways be injured to cause either paresis or paralysis? Well, a closed head injury or a brain tumor could damage UMNs. One of my aunts developed ***metastasis*** [*meta-* beyond + *stasis* standing; i.e., spreading beyond] to her brain, from her primary esophageal cancer. She had no idea that the cancer had spread, until she had a *grand mal seizure*. Immediately following the seizure, the right side of her body was paralyzed. The seizure and *right hemiplegia* were

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the direct result of the brain tumor and the surrounding inflammation in her left *cerebral hemisphere*. UMNs in the vicinity of the tumor were damaged, never to be recovered. With those UMN pathways destroyed by the tumor, conscious motor control of the right side of her body was impossible. Remember, neural deficits from UMN damage in the cerebrum will always be *contralateral* (on the opposite side) to the cerebral lesion. If her *neurologist* were to check her spinal reflexes, what do you think would be the outcome of that evaluation? If you said that the spinal reflexes would be intact, you are correct. Because spinal reflexes use a reflex arc from peripheral sensory neurons, to the spinal cord, to peripheral motor neurons (i.e., LMNs).

Where else do we have UMNs? In the spinal cord. So, *myelopathies* can also result in paresis or paralysis. And remember, the UMNs in the spinal cord either originate in the brain and extend through the cord, or they originate along motor tracts in the cord. UMNs are only in the CNS (i.e., the brain and the spinal cord). LMNs merely synapse on other neurons in the ventral horn of the spinal cord. So, is it possible, with myelopathies to damage both UMNs and LMNs? Of course. And depending on severity and location of spinal injury, intervertebral disc disease (IVDD) may impact both UMNs and LMNs.

Intervertebral Disc Disease

IVDD is a degenerative disorder of the discs. The discs deteriorate at an accelerated rate. It typically affects dogs, especially certain breeds of dogs such as Beagles, Dachshunds, Toy Poodles, and Corgis, to name a few. I've seen dogs as young as 3 years of age who have suffered acute, violent intervertebral disc rupture. And the degeneration occurs in all of their discs. So, they may experience violent rupture in cervical, thoracolumbar, and lumbar discs. Because there is a thick, tough ligament along the ventral floor of the vertebral canal in the thoracic region, any discs that rupture between T_1 to about T_{10} or $T_{11,}$ typically do not result in serious damage to the spinal cord. (I am grateful for that anatomic feature, with two ruptured discs in my thoracic spine. The ruptured disc material is pressing the ligament into the canal, but it is "gently" shifting the whole cord there. I only experience pain, from

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the dorsal roots being pressed against the dorsal bone of the intervertebral foramina.) In predisposed large breed dogs, such German Shepherds and Doberman Pinschers, the disc degeneration happens at a slower rate, predominantly affecting middle-aged and older dogs. And the rupturing of discs is slower and more insidious, as are the symptoms. Their diseased discs tend to bulge and ***extrude*** [*ex-* out + L. *trudere* to thrust; i.e., squeeze/press/force out) disc material slowly over time. When the discs rupture, they extrude small amounts of disc material at a time. In either case (*acute*, violent or *chronic*, slow extrusion), neurons in the spinal cord and/or spinal nerves are impacted. The more violent the *extrusion* and the larger the volume of disc material *extruded*, the greater the neurologic impact.

Let's use the previous example of the Dachshund with a ruptured ***thoracolumbar*** [*thorac(o)-* chest + *lumb(o)-* loin, lumbar vertebrae + *-ar* pertaining to] intervertebral disc. Depending on the degree of compression on the spinal cord and/or spinal nerves at T_{13}-L_1, the severity of motor impairment will differ. The least amount of damage will create the least amount of impairment. In this case, minimal impairment would be ***paraparesis*** [*para-* near + *paresis* weakness; i.e., weakness of the hind limbs]. Severe compression would cause ***paraplegia*** [*para-* near + *pleg(o)-* paralysis + *-ia* condition of; i.e., paralysis of the rear limbs]. If a cervical disc had ruptured, symptoms would include ***quadriparesis*** [*quadri-* four + *paresis* weakness; i.e., weakness in all four limbs] or ***tetraplegia*** [*tetra-* four + *-plegia* a paralytic condition of; i.e., paralysis of all four limbs]. (Note that the prefixes *quadri-* and *tetra-* mean "four." So, either one is appropriate to use. It's a matter of preference.)

How and why does a ruptured intervertebral disc cause motor impairment? To answer that question, we need to think about spinal anatomy. Now might be a good time to review the Evolve animation, *Vertebral Column and Spinal Nerves*, as well as the spinal anatomy found in Fig. 4.22. Make note of the structure of the intervertebral disc, the cord as it lies in the spinal canal, and the vertebral structure surrounding the cord. The spinal nerves you see poking out laterally are passing through the intervertebral foramina. With regard to the spinal cord, remember that neurons in the dorsal half of the cord are sensory. Neurons in the ventral half

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of the cord are motor. Some of the neurons in major motor tracts (bundles of motor neurons) in the ventral cord are UMNs. The ventral roots of the spinal nerves are LMNs. Now, look at that intervertebral disc. It lies ventral to the spinal cord. And the dorsal border of the *anulus fibrosus*, closest to the cord, is thin. In IVDD, the anulus fibrosus deteriorates at an accelerated rate, making it fragile. The *nucleus pulposus* also deteriorates, becoming thick and mineralized, no longer able to provide shock absorption between the vertebrae. The disc in our scenario, at T_{13}-L_1, was degenerative. It was stiff, fragile, and inflexible. Any minor movement of normal activity, such as the flexion and extension of the spine (and that's a lot of movement in a long-backed dog like a Dachshund) required to walk up or down a flight of stairs, could have been the final straw, causing the dorsal anulus to break. An IVDD dog jumping off of a piece of furniture has an even higher probability of causing the anulus to finally break. And the impact of landing on the floor when the dog jumps off the couch often results in violent, extreme *extrusion* of disc material.

Now look at Fig. 11.13. When the anulus fibrosus breaks, the contents of that disc will be *extruded* through the broken anulus. That means a portion or all of the nucleus pulposus will extrude dorsally. What's dorsal to the disc? The spinal canal and the spinal cord. Let's see, what types of neurons predominate in the ventral portion of the cord? Motor neurons. What types of neurons are in the ventral spinal nerve roots? Motor neurons. Now do you understand why, with a ruptured intervertebral disc, we get motor impairment? The ventral cord and/or ventral roots are impacted first. If the disc material *extrudes* laterally into the canal, primarily impacting the ventral root on that side, unilateral LMN impairment will develop on that side. And depending on the spinal nerve involved, the motor impairment may only produce monoparesis. Violent, acute (sudden) *extrusion* of the disc in our scenario will result in immediate *paraplegia*. The paraplegia tells us that UMN, efferent pathways from the brain are impaired and interrupted, because she cannot consciously move her legs. What we don't know, from the paralysis alone, is the integrity of LMNs. We evaluate LMN pathways with spinal reflexes.

Are sensory neurons impacted too? They certainly can be. My

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ruptured thoracic discs attest to that. Look again at Fig. 11.13. What has happened to the cord there? It has been crammed against the dorsal bone of the vertebral canal. The dorsal roots are being shoved up against the dorsal bone of the intervertebral foramina. The insult to the spinal nerves is usually painful. And if the dorsal cord has little to no compression, that pain will be felt for as long as those dorsal roots are stretched and pressed against the bone. So, in the vicinity of our Dachshund's ruptured disc, she may have *hyperesthesia*. A gentle touch to the area may cause her to cry out. But compression of the dorsal cord usually results in sensory loss. And here is a very important thing to note about the afferent sensory tracts of the cord related to pain. Those pain tracts for superficial pain (e.g., from skin and other superficial tissue nociceptors) are more superficial in the dorsal cord. So, superficial sensation and superficial pain (e.g., from a skin pinch) are lost first, when the dorsal cord is compressed. Sensory nerve fibers of deep pain tracts (i.e., deep pain such as from bone and joints) are located much deeper, more toward the center of the cord.

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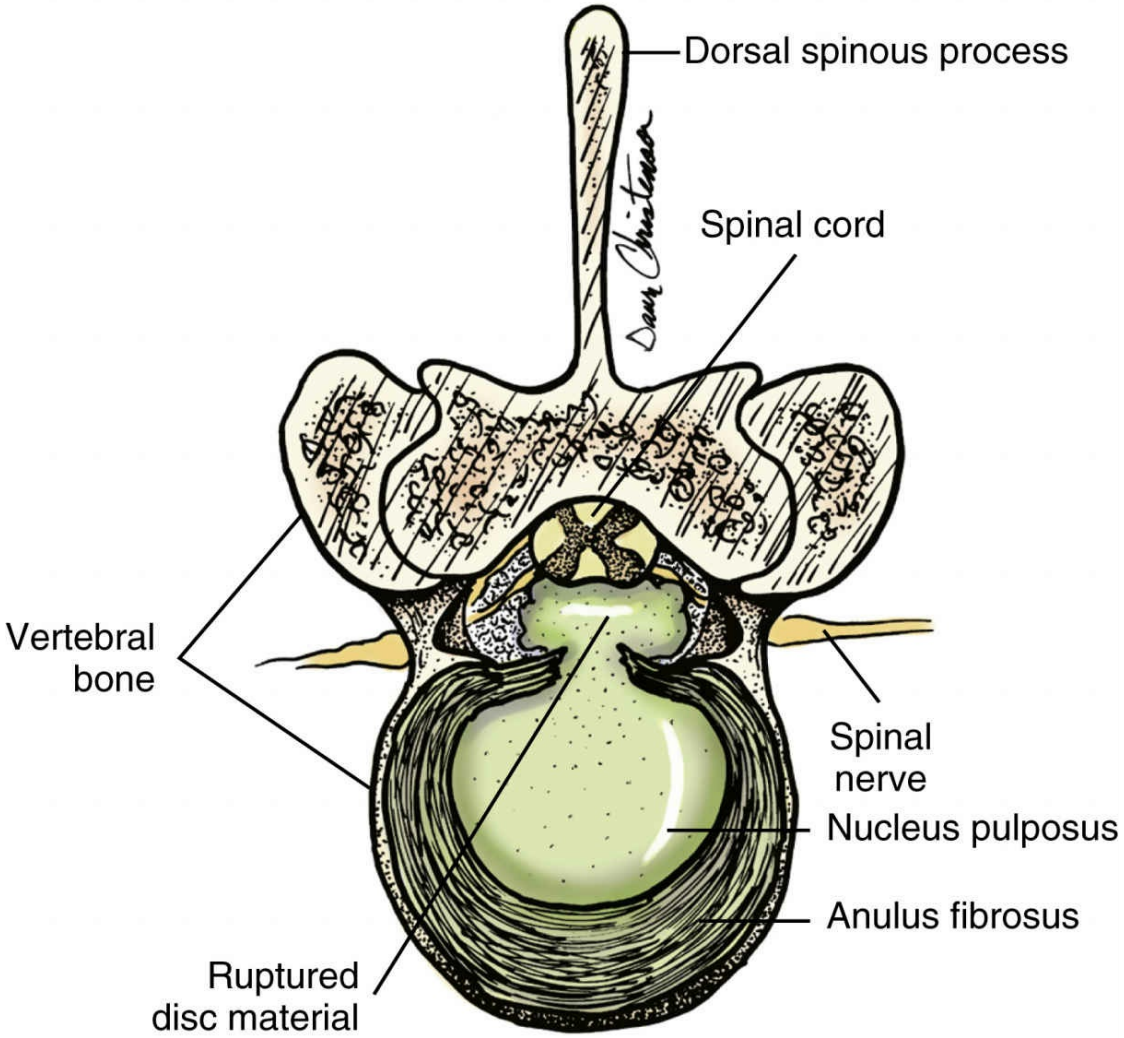

FIG. 11.13 Transverse spinal column showing ruptured intervertebral disc.

Why is it important to know the relative location of superficial and deep pain tracts in the cord? It's important because they provide a significant *prognostic* indicator (i.e., probable outcome). Think about it. If our IVDD Dachshund comes in with motor deficits, even if she's ***paraplegic*** [*para-* near + *pleg(o)-* paralysis + *-ic* pertaining to], if she still has superficial pain sensation (tested by inflicting a minimally painful stimulus, such as a skin pinch), we know that her dorsal cord is intact and unharmed. And that means her deep pain tracts are also intact (so no need to evaluate deep pain). If she's lost superficial sensation in her rear legs, we know that the superficial portions of her dorsal cord are being damaged. But if deep pain is still present, we know that central portions of the cord are still intact. How do we test deep pain? Using our fingers or a hemostat, we apply very firm pressure to a toe, over a phalanx or

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joint. Our goal is to inflict pain. So, we squeeze pretty hard. When we do it, we are looking for conscious acknowledgement of the pain. That acknowledgement may be a whimper, yelp, growl, an attempt to bite us, turning and looking at us and/or the toe, a change in facial expression—anything that tells us the dog felt the pain. We will test it multiple times, to make sure that we've gotten a consistent, accurate result. If we cannot elicit a conscious reaction to deep pain, we know that there is severe cord damage. And chances for neurologic recovery are grim.

If the dog withdraws the foot during our superficial or deep pain test, why is the withdrawal not an indicator of pain? Withdrawal is a spinal reflex. Withdrawal of the hind limb evaluates sensory and lower motor nerve fibers from L_6 to S_2. As explained earlier, this is a *polysynaptic reflex arc* along those nerve fibers. And that reflex arc evaluates only LMNs. Superficial and deep pain evaluates UMNs in the brain and spinal cord to create the conscious response of something such as turning, growling, or biting. We know that the UMN fibers (ventral cord) for conscious control of the rear legs in our Dachshund are damaged, because she cannot consciously move her rear legs. She's currently paraplegic. But if she can feel pain, we know that sensory pain tracts (dorsal cord) are still intact. I hope this helps you to see the difference between UMNs, LMNs, spinal reflexes, and pain assessment.

Can anything be done for our Dachshund? Yes. Although there are no guarantees in neurology. In general, if deep pain is intact and the duration of the paralysis is not too long, surgical ***decompression*** [*de-* reduced + *compression* squeezing/pressing together] of the spinal cord and spinal nerve(s) offers the best chance of restoring her mobility. And the sooner we do that, especially following acute paralysis, the better. But first, we need diagnostic imaging to show precisely where and how the disc material has extruded. CT scans and MRIs provide excellent visualization. Yes, we may already know that the disc is extruded in the vicinity of T_{13}-L_1. But is the disc material simply ventral to the cord? Does some of it also extend lateral to the cord—unilaterally or bilaterally? What is the condition of adjoining discs? This information will guide the surgeon's approach.

If the disc material is beneath the cord and to one side, the

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surgeon may perform a ***hemilaminectomy*** [*hemi-* half + *lamin(o)-* lamina, layer + *-ectomy* cutting out]. If the disc material is disbursed equally under and around the cord, the surgeon will likely perform a ***dorsal laminectomy*** [*lamin(o)-* lamina + *-ectomy* cutting out]. Okay, that word "laminectomy" is challenging. Just what is the lamina or layer begin cut out? Look at Fig. 11.13 again. Do you see that dorsal arch of vertebral bone that the cord is compressed against? That's what is being removed in a *dorsal laminectomy*. In a *hemilaminectomy* the vertebral bone from approximately midline extending laterally to the intervertebral foramen is removed. Once the bone is removed, the neural tissue is decompressed (i.e., no longer pressed up against the rock-hard bone). The surgeon will carefully try to remove as much of the extruded disc material as possible. We can never get it all, because we can't lift the cord out of the way to safely get at it. The most important part of the surgery is decompression, giving the cord and spinal nerves room to "breathe." Don't worry, the cord is not left completely unprotected. The "window" made in the bone is not that big. And soft tissues, such as fat and the epaxial muscles, provide soft, flexible protection.

Postoperatively, we may see immediate improvement. (As soon as I was awake, after my latest cervical decompression, all of my neurologic symptoms of numbness, *monoparesis*, tingling, and burning in my left hand and arm were completely gone. Well, the arm was still weak, but that was because of muscle ***atrophy*** [*a-* without + *troph(o)-* development + *-y* state of].) It's very gratifying to see immediate restoration of sensation and conscious motor control (changing from *paraplegia* to *paraparesis*). But sometimes *laminectomy* patients may become temporarily worse. This is from surgical trauma and inflammation. So, for a dog who was *paraparetic* with superficial sensation, he/she may temporarily lose superficial sensation and become *paraplegic*. When the surgical inflammation subsides, the patient's condition should improve. Remember, this was a neural injury. Recovery from neural injuries takes time, usually measured in weeks and months, sometimes over a year. So, patience is important, especially on the part of the owner.

No, not all IVDD cases have good outcomes. This is especially true for patients who have lost deep pain. Can a *paraplegic* dog, without deep pain, lead an enjoyable life? Yes, provided the dog

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has willing owners AND provided it still has UMN control of urination and defecation. I know of numerous dogs who are very active, tooling around in their specially made two-wheel carts. But if the dog cannot control urination and defecation, especially if urine is unable to be voided from the bladder even by manual expression, euthanasia is probably the only option. For cervical IVDD patients with caudal cervical lesions, the consequences can be far worse than simply the *quadriplegia*. And that is because the neural injury may impact ***autonomic*** function as well. You see, spinal nerves from about C_8-T_1 to $L_{4\text{-}5}$ contribute to nerve fibers of the *vagosympathetic trunk* of the autonomic nervous system.

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Autonomic Nervous System

We've already mentioned bits and pieces of the ***autonomic*** [*auto-* self + *nom(o)-* control + *-ic* pertaining to] ***nervous system (ANS)***. Let's see if we can give you more thorough understanding of this branch of the PNS. (Yes, though the ANS seems independent it is part of the peripheral nervous system, because it is made up of peripheral nerves.) In cooperation with the diencephalon and brainstem, the ANS helps to control normal, involuntary bodily functions, such as heart rate and digestive activity. Motor nerve fibers (LMN—because this is part of the PNS) are found in the *vagosympathetic trunk*. Based on what we've covered so far, you know that the vagosympathetic trunk is formed by the *vagus nerve* and *spinal nerves* between the last cervical vertebra and mid-lumbar vertebrae. Synapses between CNS and ANS nerve fibers, as well as synapses between ANS neurons and their cell bodies, are found in the *ganglia* of the vagosympathetic trunk. It may help you to understand the ganglia of this system better if you view the Evolve animation: *Impulse Conduction in the Autonomic Nervous System*.

Within the ANS, there are two branches that are in a constant "tug-of-war" for control. Their competitive nature is a good thing because they tend to keep each other in check, until needs of the body warrant one of the branches to have the upper hand. Each branch has important duties.

Sympathetic Branch

The ***sympathetic branch*** of autonomics is responsible for the body's basal survival response of ***fight or flight***. Sympathetic nerve fibers arise from the *spinal nerves* in the vagosympathetic trunk. When the body goes into survival mode, it's the sympathetic branch that kicks into high gear. We've talked about many of those sympathetic responses, in this chapter and in other chapters, such as Chapter 5. You'll find key bodily responses to sympathetic stimulation in Table 11.2. Please note that *only* sympathetic nerve fibers innervate the *adrenal glands*.

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TABLE 11.2

Primary Responses to Sympathetic and Parasympathetic Stimulation

Body Structure/system	Sympathetic Stimulation	Parasympathetic Stimulation
Pupil	Mydriasis (dilation)	Miosis (constriction)
Salivary glands	Decreased secretion	Increased secretion
Heart	Tachycardia	Bradycardia
Airways	Bronchodilation	Bronchoconstriction
Adrenal glands	Increased secretion of epinephrine	None
Digestive tract	Decreased activity and secretions	Increased activity and secretions
Lacrimal glands	Decreased secretion of tears	Increased secretion of tears
Blood vessels	Vasodilation to heart, brain, and major muscles; peripheral and digestive vasoconstriction	None

If you watched the animation *Impulse Conduction in the Autonomic Nervous System*, they spoke of two different neurotransmitters that are used along the sympathetic branch. *Acetylcholine* is used at ***preganglionic*** [*pre-* before + Gr. *ganglion* knot + *-ic* pertaining to] synapses. Remember, *acetylcholinesterase* is the enzyme used to remove *acetylcholine* from the synaptic cleft. And at ***postganglionic*** [*post-* after + *ganglion* + *-ic* pertaining to] synapses, *norepinephrine* is used. Remember, *monoamine oxidase* is the enzyme that removes norepinephrine from the synaptic cleft.

Parasympathetic Branch

The ***parasympathetic branch*** of autonomics is responsible for the body's less-urgent needs, such as digestion. Parasympathetic nerve fibers arise from the *vagus nerve* in the vagosympathetic trunk. In general, after a meal, we tend to sit back and relax, right. That's why the parasympathetic branch of autonomics is often said to be responsible for ***rest and repose*** or ***rest and digest***. You'll find key bodily responses to parasympathetic stimulation in Table 11.2. Remember, acetylcholine is the exclusive neurotransmitter used along the entire parasympathetic branch, at *preganglionic* and *postganglionic* synapses.

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Autonomic Give and Take

Now for the million-dollar question: how do each of these organs know which branch of the ANS it should "listen" to? Excellent question. This is explained and shown very well in the Evolve animation: *Autonomic Neurotransmitters*. Remember, it's the brain that ultimately stimulates the LMNs of the ANS. The brain is in the driver's seat, stepping on the throttle or the brake, as needed. And because the terminal, *postganglionic synapse* of each branch uses different *neurotransmitters*, each organ knows exactly "who" is giving the orders and how to respond. Remember, only acetylcholine is used along the parasympathetic branch. Along the sympathetic branch, only norepinephrine is used at the terminal synapses (i.e., at the organs).

Let's play with these concepts briefly. Consider the heart. The sympathetic branch is the throttle for the ***myocardium***, [*my(o)-* muscle + *cardi(o)-* heart + *-um* the] and the parasympathetic branch is the brake. So, if the myocardium is stimulated by acetylcholine, it will slow down. And ***vagal*** [*vag(o)-* vagus nerve + *-al* pertaining to] influence over heart rate can be profound. Stimulation of vagus nerve (i.e., parasympathetic) fibers, from things such as pressure or trauma to the vagus in the neck, pressure exerted on the eyeballs, or stimulation of the abdominal viscera during surgical procedures, can all produce significant *bradycardia* [*brady-* slow]. That's pretty powerful *vagal influence*. On the flip-side, if the myocardium is stimulated by norepinephrine, it will speed up, resulting in *tachycardia* [*tachy-* rapid]. Now think about the digestive tract, from salivary glands to the colon. Here the parasympathetic branch is the throttle and the sympathetic branch is the brake. So, when the digestive tract is stimulated by acetylcholine, all of the digestive tract activities, such as secretion and motility, increase. If on the other hand the digestive tract is stimulated by norepinephrine, it hits the brakes and slows down or stops digestive activity.

And remember, hormones can mimic or block the effects of those neurotransmitters. We've mentioned the hormone ***epinephrine*** in this chapter and in others, such as Chapters 5 and 10. In fight or flight, because the adrenal glands are only innervated by sympathetic nerve fibers, epinephrine is secreted in abundance. Epinephrine is ***sympathomimetic*** [*sympathy(o)-* the sympathetic

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branch + *-mimetic* pertaining to mimicking] because it *mimics* the effects of the sympathetic branch. Epinephrine is very similar to norepinephrine. That is why sympathetic nerve fibers are also said to be ***adrenergic*** [*adren(o)-* adrenal gland + *erg(o)-* work, working + *-ic* pertaining to]. Frankly, organs like the heart don't know the difference between the two. Although, functionally epinephrine targets *β-adrenergic* and *α-adrenergic* receptors on organs. We talked about these receptors quite a bit in Chapters 5 and 10. As you may recall from those discussions, each type of adrenergic receptor is found in abundance in key areas. That's how epinephrine can cause *vasodilation* in some areas and *vasoconstriction* in other areas at the same time. In emergency situations, we often administer ***exogenous*** [*ex-* out, outside + *gen(o)-* produced + *-ous* pertaining to; i.e., produced outside the body; synthetic] epinephrine IV, for its *sympathomimetic* benefits. Of course, if we have *sympathomimetic* medications, then we must also have ***sympatholytic*** [*sympathy(o)-* the sympathetic branch + *-lytic* pertaining to breaking] agents too. And that is very true. There are a number of sympatholytic medications used in cardiovascular diseases, such as "beta-blockers," that reduce the overall influence of the sympathetic effects by blocking β-adrenergic receptors.

When we talked about neurotransmission at the beginning of this chapter, we said that nerve fibers using *acetylcholine* in their synapses are called *cholinergic* nerve fibers. Obviously, based on what you know now, all of the nerve fibers along the *parasympathetic branch* are *cholinergic*. And the pharmaceutical industry has done a fine job of producing various medications that either mimic or block the effects of acetylcholine. If you've spent much time in veterinary practice, you are probably very familiar with a ***parasympatholytic*** [*parasympath(o)-* the parasympathetic branch + *-lytic* pertaining to breaking] or ***anticholinergic*** [*anti-* against + *cholin(o)-* acetylcholine + *erg(o)-* work + *-ic* pertaining to] drug called ***atropine***. There are many applications for the use of an *anticholinergic* drug such as *atropine*. Let's say that our IVDD Dachshund during the *laminectomy* developed severe *bradycardia* [*brady-* slow + *-cardia* heart condition]. According to Table 11.2, the parasympathetic branch slows the heart. If we administer IV atropine, we block the *cholinergic* receptors on the myocardium and

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the heart rate speeds up. It's a fast solution to a potentially life-threatening problem. What does the parasympathetic branch do to the pupils of the eyes? It makes them constrict. And in painful eye conditions that we'll discuss later in this chapter, extreme pupillary constriction can snowball and add to the pain. So, to break the pain cycle in the affected eye, we can apply an *anticholinergic* such as *atropine* topically. By blocking the *cholinergic* receptors on the muscles of the iris that cause pupil constriction, we in effect paralyze the muscles. That breaks the pain cycle and results in pupillary dilation. Since we're talking about the use of ***ophthalmic*** [*ophthalm(o)-* eye + *-ic* pertaining to] drugs to manage pupil size, we might as well talk about the flip-side of the coin. Yes, we sometimes use ***parasympathomimetic*** [*parasympath(o)-* the parasympathetic branch + *-mimetic* pertaining to mimicking] agents to cause pupillary constriction. We'll talk about this in the management of ***ocular*** [*ocul(o)-* eye + *-ar* pertaining to] diseases such as glaucoma in a little while. And that sounds like a great segue into special senses!

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Special Senses

Special senses are just that—special. They provide highly specialized sensory information that helps to orient us to the world around us, perceive threats, find food, find companions and mates, and contribute to the overall enjoyment of life. We will touch on each of the senses. But when it comes to hearing and vision, we will go into much greater depth.

Taste

The ***sense of taste*** or ***gustation*** [L. *gustatio*, from *gustare* to taste] is probably best discerned by the human palate. I am convinced that any animal, like a dog, that will gleefully consume its own vomit or the feces of another animal has no accounting for taste. Yet, all animals have some sense of taste. (Obviously, some more than others.) All animals have tongues, and those tongues have specialized ***gustatory*** [L. *gustatio* + *-ory* pertaining to] ***receptors*** that we commonly call ***taste buds***. Most of those receptors are concentrated on the tongue. But in some animals (even humans), sensory receptors may also be found in cheeks and throat. Sensory input from the *gustatory* receptors travels along afferent fibers of the ***facial***, ***glossopharyngeal*** [*gloss(o)-* tongue + *pharyng(o)-* throat + *-al* pertaining to], and *vagus* nerves to the *parietal lobes* in the cerebrum.

Now you might think that ***omnivorous*** [*omnivor(o)-* all eating + *-ous* pertaining to] and ***carnivorous*** [*carnivor(o)-* flesh eating + *-ous* pertaining to] animals would have the largest numbers of taste buds. (Humans, dogs, and pigs are *omnivores,* and cats are the only true *carnivores*.) Following that line of thinking, that would leave ***herbivores*** [L. *herba* herb + *vorare* to eat; i.e., plant eaters], already being at the bottom of the food chain, with the least amount of taste buds. Newsflash: that way of thinking is incredibly flawed. As it turns out, most herbivores have the largest numbers of taste buds. Horses and cattle top the list with approximately 25,000 taste buds. Rabbits are close behind with 17,000. I guess that helps all of them to select the best blades of grass and sweetest clover to eat. Among omnivores, pigs have the most, with 15,000. That's more than

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humans, who average about 9000. Dogs on the other hand have only about 1700. No wonder they can eat nasty stuff and enjoy it! That's still enough gustatory receptors to enable dogs to taste sweet, salty, bitter, and sour things. (So, that nasty, bitter oral medication?—dogs can taste it and that's why they'll struggle with you and try to spit it out.) Oh, and then there is the poor cat … top predator in the food chain, that has less than 500. Ah, but that explains why being able to smell food is so important to cats! The tactile "feel" in a cat's mouth is also important, and for some more important than taste.

Touch

The ***sense of touch*** is also very important. I learned to appreciate this as I slowly lost ***tactile*** [L. *tactilis*, from *tangere* to touch] sensation in my left hand from my cervical issues. Before my surgery, I had difficulty grasping and picking things up because I could not feel them. I had to look at what I was grabbing and rely on deeper pressure sensors. Even then, because superficial tactile sensation was all but lost, I had difficulty and often dropped things. Thank goodness it was restored with surgery to decompress the cord and spinal nerves innervating my hand and arm.

Now, most animals, especially domestic animals, do not have opposable thumbs. So, they would not be troubled by an experience like mine. Still, tactile sensation is important for them. Think about it. Horses rely on *tactile* sensation of their lips. That's what gives them the fantastic ***prehensile*** [L. *prehensio* to grasp + *-ile* pertaining to] ability that we talked about in Chapter 7. In Chapter 8, we talked about whiskers, saying that they are ***tactile hairs***. *Tactile hairs* are longer and much more stiff than other hairs, with a complex network of capillaries and sensory neurons surrounding the base of the hair. Fluids transfer energy, even sound waves, pretty well. And waves created in the capillary network of these tactile hairs magnify sensations, for stimulation of the sensory receptors. That's why the slightest touch of something brushing by or air currents gently blowing by these tactile hairs are so readily felt by the animal. Tactile sensory receptors are found in abundance throughout the skin, enabling us and animals to feel so many

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things, even things slight and small such as the fly landing on the horse's back to activate the *panniculus reflex*.

When you combine the collective sensory input from tactile receptors, temperature receptors, and pressure receptors, we and our animal kin are empowered with tremendous functional and protective capacities. They enable all of us to feel where and on the type of surface we place our feet when walking. (Just try walking if you can't feel your feet. I can almost guarantee you'll stumble and probably fall.) Tactile sensation allows us to feel texture. This is how cats develop substrate and surface preferences for places of elimination. (Hopefully, they prefer the box and type of litter we've selected.) It helps animals to select appropriate materials for bedding. All of this sensory input enables us to feel potential hazards that might injure us, such as sharp objects or surfaces that are too hot or too cold. The collective input from tactile, temperature, and pressure receptors, as subtle as those sensations may be, permits us to withdraw a hand, paw, or foot by reflex action (a spinal reflex, remember) to protect us from harm. We often take the sense of touch for granted. But it is actually an important key to our survival.

Smell

We have already mentioned the ***olfactory*** [*olfact(o)-* smell + *-ory* pertaining to] ***nerve*** (first cranial nerve). ***Olfaction*** is the ***sense of smell***. In the Evolve resources, you'll find a brief overview of olfaction in the animation called *Smell*. *Olfaction* is far more important for survival of many mammals other than humans. In fact, even in domestication, *olfactory* ability is important. That is why all domestic animals have much larger *olfactory* nerves with far more sensory nerve endings in the nasal passages than humans. *Olfaction* is so important for cats that they will not eat if they cannot smell their food. (Remember, cats can't taste much.) Many cloven hooved animals, such as sheep and goats, use their superior sense of smell to pick up on the *pheromone* trail left behind as they and the herd walked. You may remember from Chapter 8, we talked about the ***interdigital*** [*inter-* between + *digit* toe + *-al* pertaining to] glands of these animals that secrete ***pheromones***—chemical ***scent-markers***

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—that permit them to find their way or find each other if they become separated. Believe it or not, even certain insects have a powerful *sense of smell*. Termites and ants follow pheromone trails too, to guide them to and from the colony and food resources. And blow flies have an amazing sense of smell! Blow flies are those shiny metallic flies that you see around garbage cans, manure, and dead animals. Okay, those things stink, so what's so amazing about a blow fly being able to find them? This is how well they can smell —if an animal drops dead right now, within minutes a blow fly can smell it from well over a mile away and be there to lay her eggs. Is that amazing or what?!

Among domestic animals, when it comes to olfactory ability, dogs are among some of the best. But believe it or not, cows are actually superior to dogs. Still, I would love to be gifted with a dog's sense of smell for just a few hours, just to "see" the world through their noses. It would be a fascinating experience to be sure. I love watching our Corgi pup excitedly sniffing all over our property. We live on 10 acres in the country, with fields and woods. We have wildlife galore from chipmunks, squirrels, song birds, turkeys, deer, moles, and opossums to coyotes. We mow walking paths through the back field. When we take our Corgi pup on a walk, she races around checking "messages" from all sorts of animals. (We check email, dogs check scent-markers.) Oh my gosh, she has so much fun every time!

Have you ever watched a dog sniff? No, I mean *really* watched a dog sniff. If you haven't, do it sometime. You'll see the dog sniff-sniff-sniff-sniff-sniff-sniff. Then periodically the dog blows out somewhat forcefully from its nose. Functionally, what is going on? You see, everything has an ***odor***. That *odor* is actually the ***volatilization*** [L. *volatilis,* from *volare* to fly + -i*zation* act, process of] of various chemical compounds. So, odors are molecules. Professionals who either study *olfaction* or train dogs in nose work and tracking refer to odor molecules as ***odorants***. And those *odorants* surround the point of origin in what's called a ***scent cone***. We know when our pup has just picked up on the fringe of a *scent cone* as she races through the field. Her head whips around, and she quickly doubles-back in search of that fascinating scent cone. When she finds it, she intently sniffs. As she sniffs, she is actually drawing

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those odor molecules into her nasal passages. They become trapped and concentrated in the *nasal turbinates*. And in her turbinates, as we mentioned in Chapter 5, she has literally millions of ***olfactory receptors***. (It is estimated that the average dog has approximately 300 million scent receptors. Humans might have only about 6 million.) As those *olfactory receptors* are stimulated, the *afferent* nerve impulse follows sensory nerve fibers through the ***cribriform*** [L. *cribrum* sieve + *-form* shaped] ***plate*** to the ***olfactory bulb*** (shown in Figs. 11.3, 11.4, 11.5 and 11.6). (Yes, the bone between the nasal passages and cranial vault is full of holes, like a sieve. How else would we get all of those sensory nerve endings of the olfactory nerve to the brain?) From the olfactory bulb the impulses are fast-tracked to the *temporal lobes* of the cerebrum for interpretation and conscious recognition. There is a large portion of the cerebrum devoted to the sense of smell in dogs, compared with humans—approximately 40% more, in fact. So, even if we stuck our noses where the pup is sniffing, there's probably no way we will ever smell what she smells. Perhaps that's just as well.

Okay, that explains how dogs actually smell various odors. What about that "blowing-the-nose" thing? It's kind of like hitting the "refresh" button on your computer. By forcing air out of the nose, those molecules sniffed in are blown out. It clears the plate, so to speak, so that new odorants can be sniffed in and evaluated. And because of dogs' olfactory abilities and their intelligence, it is very easy to train dogs to recognize the respond to specific odors. This type of training is used by law enforcement for drug and explosive detection, as well as for tracking criminals. Search and rescue agencies train dogs to find missing persons. Think about that one for a moment. When a child has been lost, a dog will be given one of the child's toys or an article of clothing to sniff. And that dog can track for miles on that scent to find the missing child. On the medical side, dogs are trained as service animals for people with diseases such as epilepsy and diabetes. Yes, these dogs may be picking up on subtle behavioral clues, warning of an impending crisis. But they are also picking up on odorants that precede seizures and diabetic crises. The dog can then call for help (another person or first responders) and warn the victim. There have even been dogs known to point to the location of tumors in people.

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Suffice it to say that the sense of smell in dogs is amazing. So, why some dogs try to find the smelliest, rotten stuff to roll in is beyond me.

Hearing

Hearing is another special sense that, in animals, is far superior to humans. Scientists have actually "mapped" the range of hearing frequencies of people and numerous animals. The frequencies of sound waves are measured in ***hertz*** (pronounced hurts; i.e., cycles per second). The measurement value (hertz, ***Hz***) was named after the 19th century German physicist Heinrich Hertz. For those of you familiar with music, it may help you to correlate hertz to pitch. The higher the hertz measurement, the higher the pitch. The lower the hertz measurement, the lower the pitch.

So, as we examine the ***auditory*** [*audi(o)-* from L. *audire* to hear + *-tory* pertaining to] abilities of humans and animals, we know that the average human ***acoustic*** [*acoust(o)-* sound + *-ic* pertaining to] range is approximately 65 to 20,000 Hz. And while that may be limiting, most everyday noises that we hear are between 250 and 6000 Hz. So, our *acoustic* range is quite functional for our needs. But for animals such as dolphins and bats, who need to echo-locate, their acoustic range may be as high as 100,000 Hz. Ever wonder why dogs can hear that dog whistle and you can't? Well comparatively, dogs have an average *acoustic* range of about 40 to 60,000 Hz. Remember this when training your dog in obedience. With a normal dog, you can whisper the commands and the dog will hear you just fine. There is no need to raise your voice if the dog doesn't respond. Either the dog didn't *understand* the command or it is *choosing* not to respond. A loud command doesn't teach the command itself. Yelling your commands teaches the dog to respond to you *only* when your raise your voice. That's neither optimal nor necessary. Cats have a similar range to dogs, with an average acoustic range of approximately 45 to 65,000 Hz. There are actually some animals whose bottom end of their acoustic range may be as low as 0.5 Hz. It's animals' lower acoustic range that warns them well in advance of oncoming storms, earthquakes, and volcanic eruptions. That's how they manage to vacate the area long before

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we're even aware of the oncoming threat.

But just how do animals and people hear? Well, it's a matter of transforming sound waves into mechanical energy in order to stimulate specialized sensory receptors for neurotransmission. Wow, doesn't that sound complex? Let's take a look at the anatomy that makes up the *auditory pathway*, step by step, shall we?

Otic Anatomy

External Ear

The most prominant part of ***otic*** [*ot(o)-* ear + *-ic* pertaining to] anatomy is part of the ***external ear*** and that is the ***pinna*** [L. *pinna* wing; plural pinnae] (Fig. 11.14). I guess some ear *pinnae* do resemble wings, especially when I picture my Basset Hound with her ear pinnae flapping in the wind. Sometimes she looked like she could fly. But flying is not the purpose of ear pinnae. Their purpose is to capture and funnel sound waves into the ***ear canal*** or ***external acoustic meatus*** (me-a'tus [L. *meatus* a way or path; i.e., an opening or passageway]). And depending on the structure and mobility of an ear pinna, some animals funnel sound waves better than others. Functionally, pinnae of animals such as the Basset on the right of Fig. 11.14 probably don't hear quite as well as animals with erect pinnae. But Bassets are scent hounds, not guard dogs. Look at the large, erect pinna on the left of Fig. 11.14. This would be typical for a number of dog breeds, such as most shepherds. Size and proportion make a difference too. Proportionally, my Corgi has huge ear pinnae. They're like satellite dishes. And she hears and alerts us to *every* little thing. The erect pinnae of horses, mules, and most rabbits are not only large, but the animals can rotate them around in every conceivable direction. Think of how important that is for survival, as prey animals. Predators, on the other hand, tend to have forward-facing pinnae, to hone in on their prey.

Okay, so ear pinnae of all shapes and sizes funnel sound waves into the external acoustic meatus. Look at the shape of the ear canal of a dog, shown in Fig. 11.15. This is typical of most domestic animal ear canals. It's not a straight passage, like a human ear. It's almost "L" shaped. This can be beneficial and problematic. It's beneficial because that shape prevents damage of the ear drum

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from foreign objects. It can be problematic because things such as water, ***cerumen*** (suh-roo'men [from L. *cera* wax; i.e., ear wax]), and pus are difficult to clean out of that horizontal part. And certainly, anything accumulating in the canal will interfere with sound waves reaching the ***tympanic*** [*tympan(o)-* drum + *-ic* pertaining to] ***membrane*** (aka. ***ear drum***). We'll talk more about problems like that when we discuss ***otitis*** [*ot(o)-* ear + *-itis* inflammation of] later. The *tympanic membrane* marks the separation between the external ear and the middle ear. When sound waves strike it, the *tympanic membrane* moves. Have you ever seen those big kettle-like tympani drums in an orchestra? The percussionist strikes the tight "skin" of that drum and it moves, right? Well, when sound waves strike the ear drum, it moves too.

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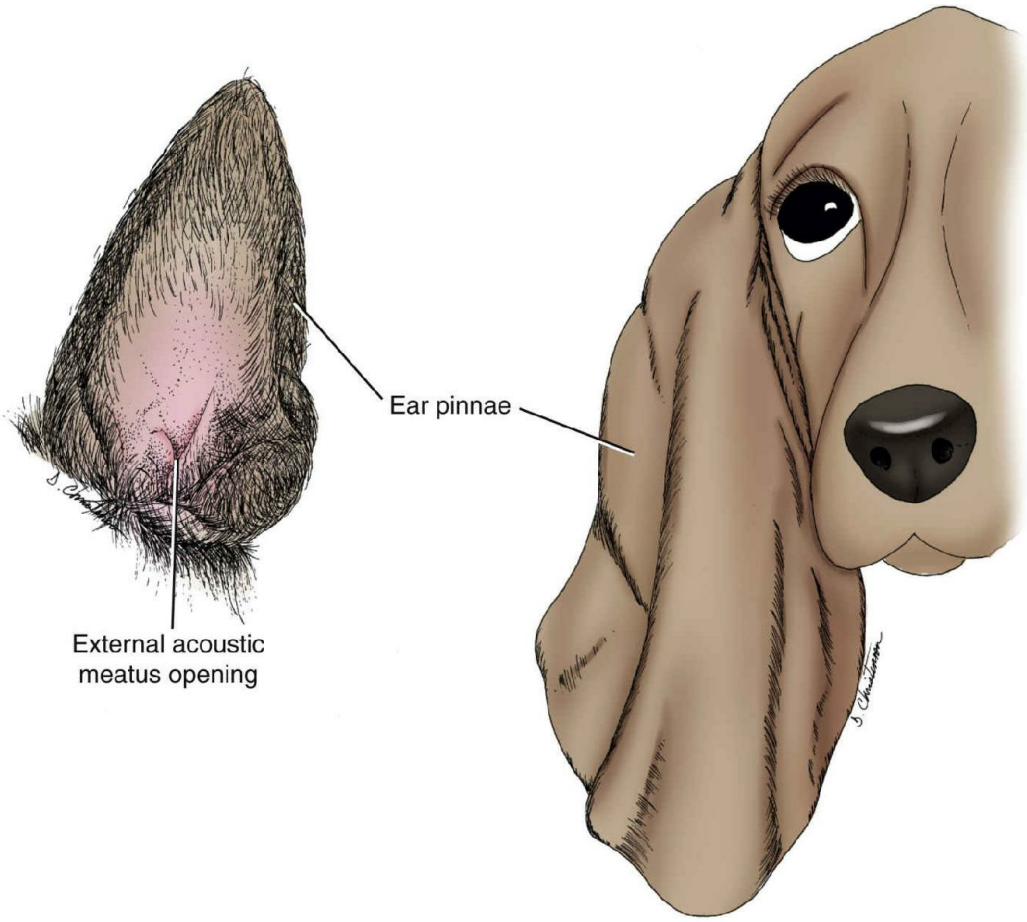

FIG. 11.14 Ear pinnae.

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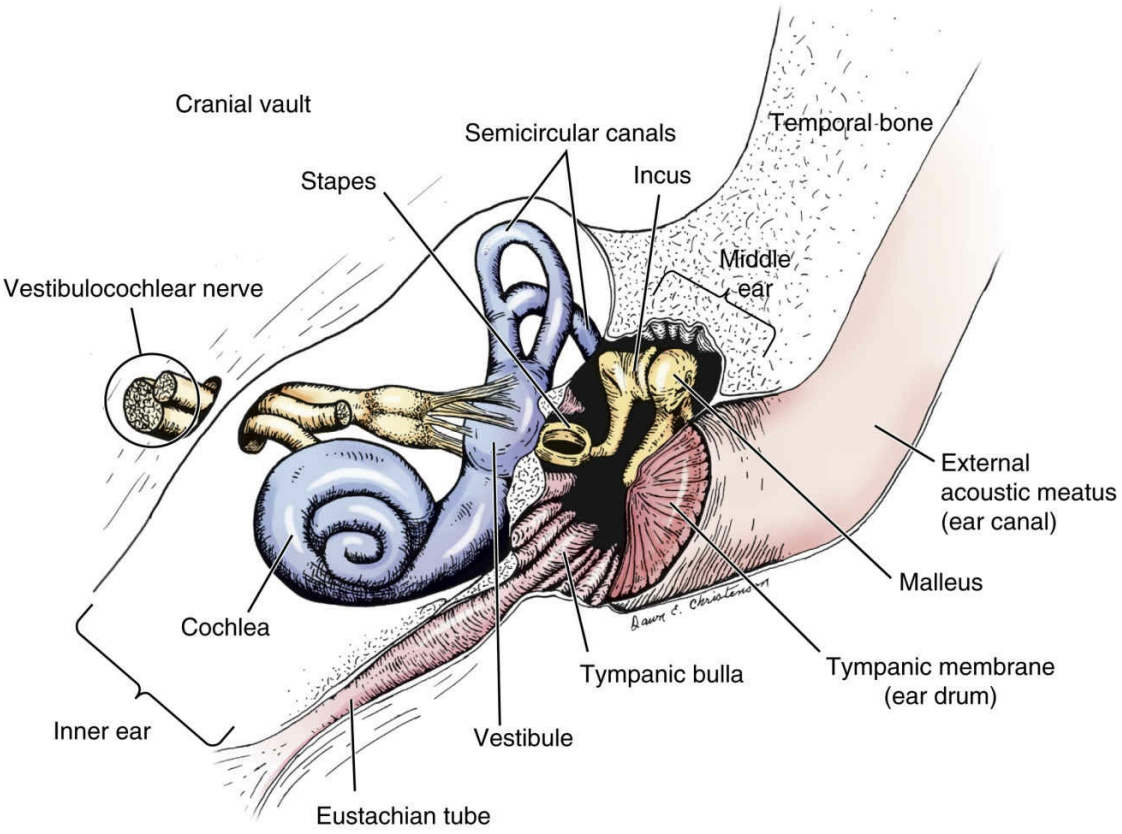

FIG. 11.15 Schematic cross-section of a canine ear.

Middle Ear

On the other side of the ear drum is the ***middle ear***. The middle ear is housed in the ***tympanic bulla*** [L. *bulla* blister]. A blister is a rounded structure, right? Well, the *tympanic bulla* is a hollow, rounded structure formed by bone of the skull. You can see this in Fig. 11.16. And in Fig. 11.17, you can see the working parts of the middle ear. In here are three of the smallest bones in the body. And here is where we really transform sound waves into mechanical energy. You see, those tiny bones move when the ear drum moves. They have no choice, because they are physically attached to the ear drum by the first bone in the series. The bones themselves are tied together by itsy-bitsy ligaments, just like all other joints in the body.

Ancient anatomists named each of those ***otic ossicles*** [L. *ossiculum* a small bone] by comparing them to the tools and a product of blacksmiths. There are two essential tools that every blacksmith needs to forge things from steel. They need a hammer and an anvil to shape and form the hot steel, to create something, such as a stirrup for a saddle. So, the first *otic ossicle* that is attached

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to the ear drum is the ***malleus*** [L. *malleus* hammer]. Hammers need something to strike against. So, the second *otic ossicle* is the ***incus*** (ing'kus [L. *incus* anvil]). And the last *otic ossicle* represents what is forged, the ***stapes*** (sta'pez [L. *stapes* stirrup]). So, between the ear drum and the ossicles, the ear transforms sound waves into physical, mechanical movement. This is shown in the Evolve animation, *Hearing*.

Now, you may have also noticed, in Fig. 11.17, that there are also two tiny muscles in the middle ear. We can't afford too much movement of the ear drum or those ossicles. That might create damage. And in case you're wondering, yes, we can develop ***arthritic*** [*arthr(o)-* joint + *-itic* pertaining to inflammation] changes at those tiny joints. More importantly, too much movement of the ossicles might damage components of the inner ear. So, when subjected to acute, high decibel (i.e., really loud) sounds, reflex arcs will result in contraction of the muscles. Notice I said "arcs," plural. To protect sensory neurons in the inner ear, we have built in redundancies. Each of those muscles is innervated by branches of different cranial nerves. The ***tensor tympani muscle*** (attached to the *malleus*) is innervated by a branch of the *trigeminal nerve* (V). And the ***stapedius*** [L. *stapes*, from L. *stares* to stand + *pes* foot + *-us* the] ***muscle*** is innervated by a branch of the *facial nerve* (VII). See? We're not even using nerves directly involved with hearing. Why? Because the ***vestibulocochlear*** [*vestibul(o)-* vestibule, chamber + *cochle(o)-* cochlea, snail + *-ar* pertaining to] ***nerve*** or ***auditory nerve*** (VIII) is strictly sensory. By using different cranial nerves, we don't put all of our eggs in one basket, so to speak. By activating those reflex arcs, we minimize or stop movement of the ossicles momentarily. That's why, if you've ever been exposed to a sudden, loud noise, like a gun being fired or an explosion of some kind, momentarily you can't hear or your hearing is extremely muted. As soon as the muscles relax, hearing is restored.

One final feature of the middle ear is the ***eustachian tube***. (The *eustachian tube* was named after an Italian anatomist, Bartolommeo Eustachio.) That tube connects the environment of the middle ear in the *tympanic bulla* to the *pharynx*. We need a flexible ear drum to permit freedom of movement for hearing, right? But changes in air pressure may alter that flexibility. And we experience changes in air

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pressure all the time, with weather changes and as we move from one altitude to the next. Higher altitudes have lower air pressure. Let's see how that affects the ear drum.

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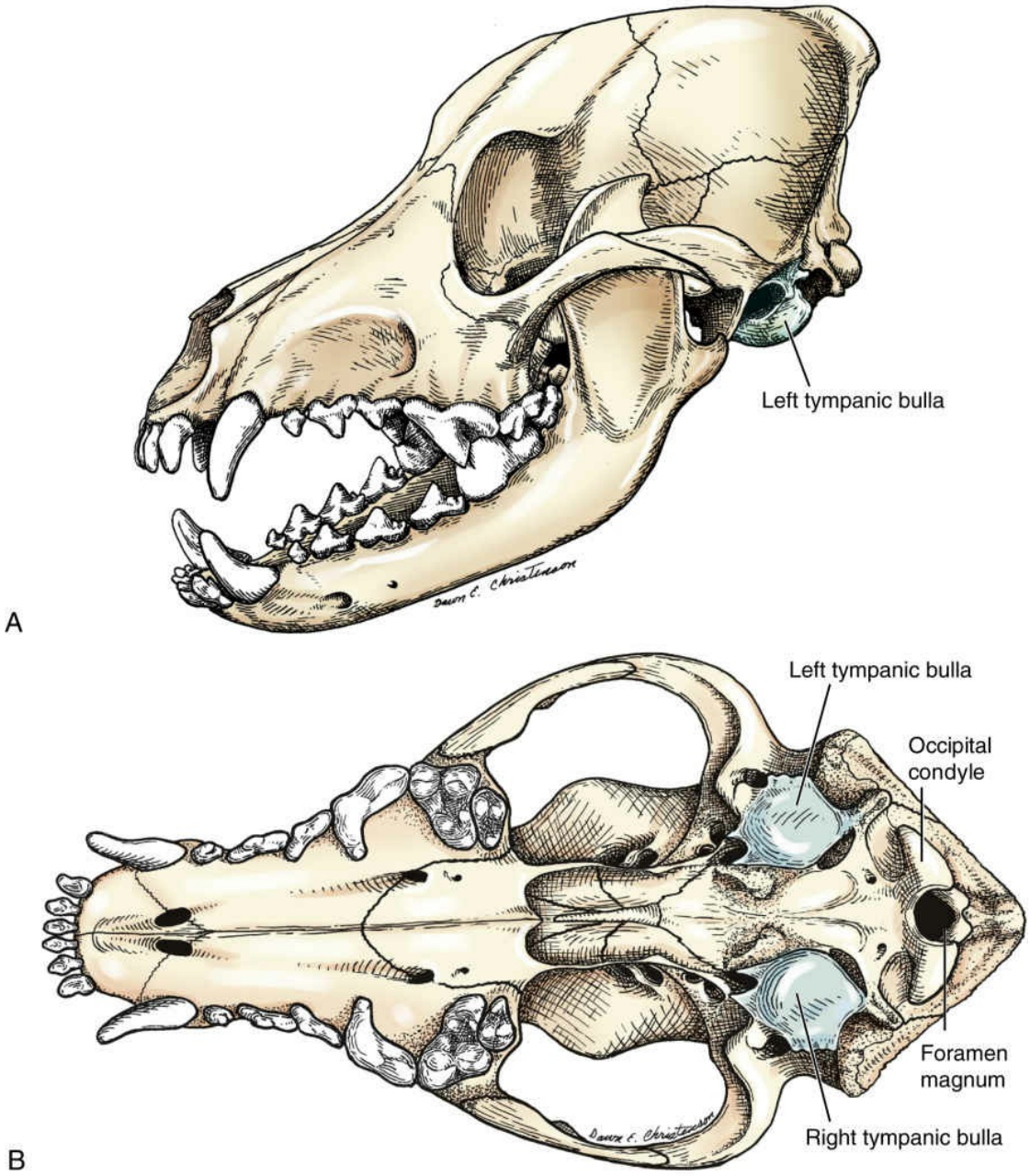

FIG. 11.16 Tympanic bullae of canine skull; (A) left lateral view; (B) ventral view.

Hypothetically, let's begin a journey at sea level. Atmospheric pressure at sea level, as we said in Chapter 5, is approximately 760 mm Hg. Let's travel from Galveston, Texas, (sea level) to Denver, Colorado (mile-high city). When we begin our journey, air pressure around us and in the middle ear is 760 mm Hg. When we end our journey in Denver, air pressure around us is approximately 620 mm Hg, while within the middle ear it is still 760 mm Hg. That interior pressure will make the ear drum bulge out into the ear canal. And with that much tension, the ear drum and ossicles won't be able to

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move very well at all. Our hearing is horribly diminished. We need a way to equalize the pressure, to match that of the surrounding air. That is precisely what the eustachian tube is for. And because it opens into the pharynx, movements of pharyngeal muscles, (e.g., swallowing) alters the patency (openness) of the eustachian tube. Because it's a soft, flaccid tube, much of the time it's rather flat and closed. Each time we swallow or chew on our chewing gum, the eustachian tube opens up briefly. And because air moves from high pressure to low pressure, the pressurized air in the tympanic bulla rushes out through the eustachian tube into the pharynx, reducing pressure in the middle ear. Once pressure is equalized inside and out, the ear drum moves freely again and our hearing is restored. If we could not equalize pressure in the middle ear, marked pressure differences inside and out could rupture the ear drum.

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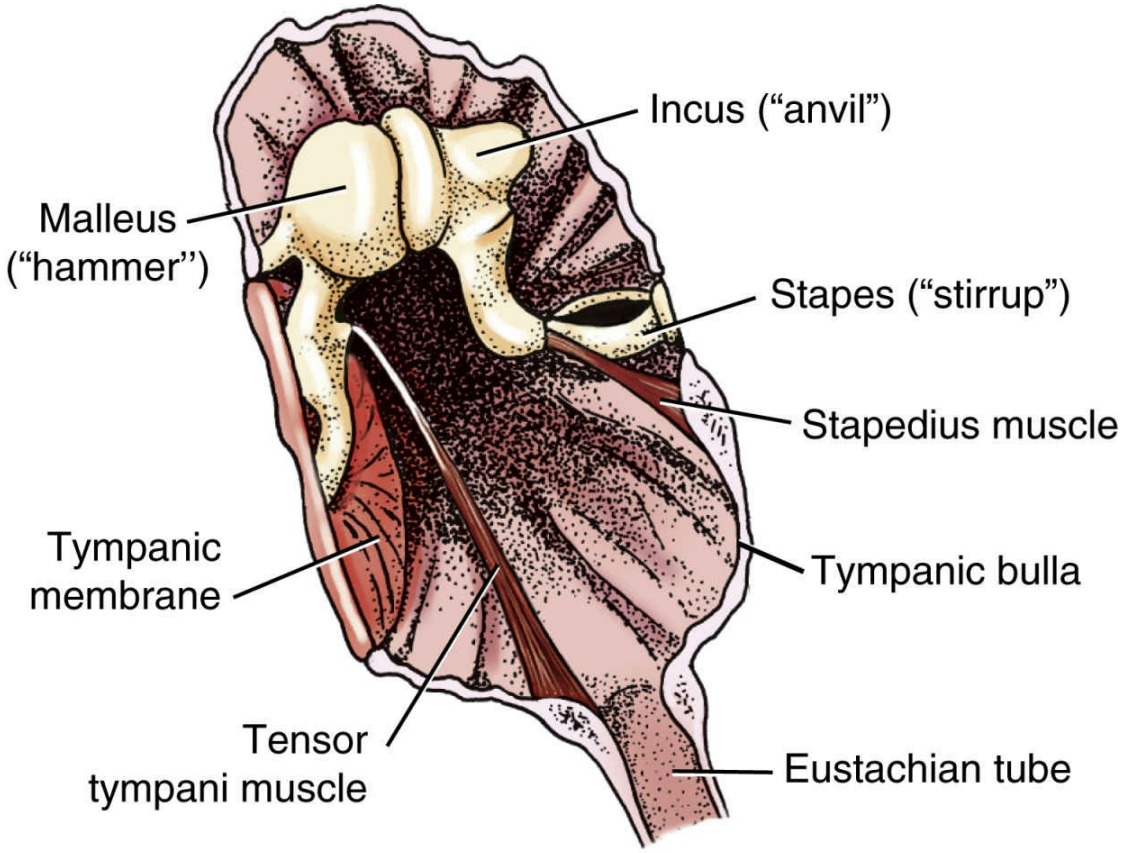

FIG. 11.17 Middle ear.

Of course, the eustachian tubes can lead to problems. In animals and people who have ***pharyngitis*** [*pharyng(o)-* throat + *-itis* inflammation of] from infectious upper respiratory diseases, *pathogens* can move *retrograde* (backward) through the eustachian tube(s) and cause middle ear infections (***otitis media*** [L. *media* middle]). Hopefully, the eustachian tube(s) remain somewhat patent (open), to drain any fluids produced by the inflammation/infection.

Of any domestic animal, horses can have the greatest problems with their eustachian tubes. That’s because horses have large pouches along their eustachian tubes. These are the ***guttural*** [from L. *guttur* throat + *-al* pertaining to] ***pouches*** in a horse (Fig. 11.18). Most of the time, you wouldn’t even know horses have *guttural pouches*. But if air becomes trapped in them or they become inflamed or infected, they can become very painful. And because they are associated with the eustachian tubes, they can negatively impact pressure equalization of the middle ear.

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Inner Ear

Okay, so far we've talked about sound waves being transformed into mechanical movement of the ear drum and *otic ossicles*. But how does that result in stimulation of sensory neurons for hearing? Well, we need to transfer that mechanical energy from the middle ear to the inner ear. We do that at the ***oval window***. The *oval window* is really not a window per se, certainly not an open window. The *oval window* is a flexible, connective tissue membrane. The stapes is attached to that membrane. When the stapes moves, the oval window flexes and moves with it.

On the opposite side of the oval window is the ***cochlea*** [L. *cochlea* snail shell]. The *cochlea* is that portion of the ***inner ear*** that is responsible for hearing. It along with the other portions of the inner ear are completely surrounded by the temporal bone of the skull. The cochlea is literally shaped like a snail shell; and within it is a twisted labyrinth of spiraled, fluid-filled ducts or canals. As discussed in the Evolve animation called *Hearing*, movement of the oval window causes the fluid (***endolymph*** [*endo-* inside + *lymph* water] in the middle, cochlear duct and ***perilymph*** [*peri-* around + *lymph* water] in the outer ducts) to move. The fluid waves created actually correspond to the frequency of sound waves that originally struck the ear drum. So, low-frequency sound waves create broad, rolling fluid waves. High-frequency sound waves create narrow, rapid fluid waves. And as the animation said, that fluid movement (fluid waves) makes the *basilar membrane* of the ***organ of Corti*** (named after a 19th century Italian, Dr. Alfonso Corti) move. This makes the sensory receptors or ***hair cells*** on top of the basilar membrane to rub against the ***tectorial*** [from L. *tectum* roof + *-al* pertaining to] ***membrane***. And it's that contact that stimulates the sensory receptors, resulting in neurotransmission via the ***cochlear*** [*cochle(o)-* cochlea + *-ar* pertaining to] branch of the ***vestibulocochlear nerve*** to the *auditory* centers in the *temporal lobes* of the cerebrum. The one thing that the animation does not discuss is the dissipation of the fluid waves. At the very end of the labyrinth of ducts is the ***round window***. This is a flabby, flexible, connective tissue membrane that acts like a shock absorber. When the fluid waves hit the round window, because it's rather loose and flabby, the momentum is rapidly dissipated. Without that "shock

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absorber," those fluid waves within the cochlea would probably reverberate (rebound) and create all sorts of weird, extraneous stimulation of the hair cells.

Auditory Pathway Summary

Let's pause to put this whole hearing process together. You may want to watch the *Hearing* animation again, especially for the inner ear events. Sound waves are funneled into the ear canal by the pinna. When the sound waves strike the ear drum, it moves. Because the malleus is attached to the ear drum, when the ear drum moves the malleus along with the incus and stapes also move. Movement of the stapes in the oval window creates fluid waves in the ducts of the cochlea. Those fluid waves cause the basilar membrane of the organ of Corti to move, rubbing the sensory receptors against the tectorial membrane. This stimulates the sensory receptors, generating a neural impulse that follows the cochlear branch of the vestibulocochlear nerve into the brain, where it is interpreted in the temporal lobe.

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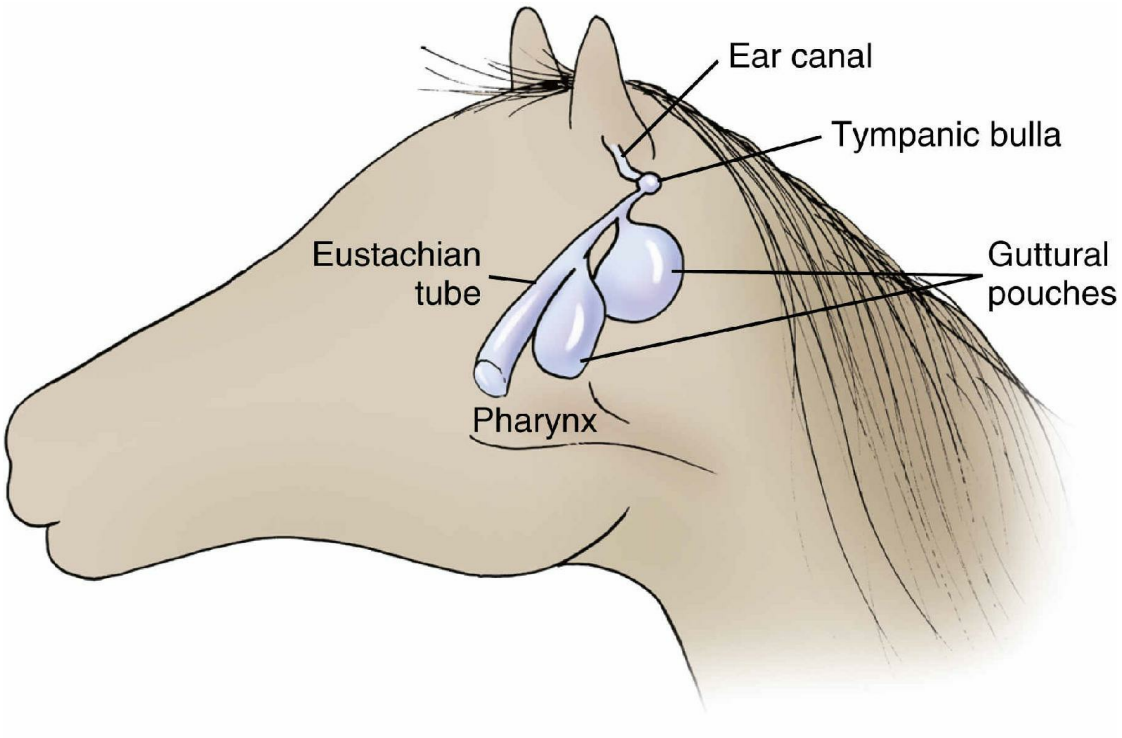

FIG. 11.18 Equine guttural pouch schematic.

Now, if you really want to blow your mind, think about music. It doesn't matter the type of musical genre. They all have one thing in common: series of notes, various frequencies of sound, and different tonal qualities. But let's think about listening to an opera, so that we have a variety of vocalists and a complete orchestra accompanying them. Among the vocalists we have sopranos, mezzo-sopranos, altos, tenors, baritones, and basses. And while two tenors may sing the very same note eloquently, the vocal characteristic of each voice is different. Within the orchestra, we have numerous and varied instruments. The percussion section has tympani, snare drums, cymbals, chimes, and a xylophone. The woodwind section has flutes, oboes, clarinets, piccolos, saxophones, and bassoons. The brass section includes trumpets, French horns, trombones, and tubas. And in the string section are violins, violas, cellos, and basses. Now here is the mind-blowing part. When we listen to the performance, with the entire vocal cast and the entire orchestra singing and playing simultaneously, our remarkable auditory pathway and cerebral integration permit us to hear and distinguish each and every sound all at once. Is that amazing or what?!

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Hearing Loss

Of course, there are those who have reduced hearing ability or have lost all hearing ability and are deaf. Both humans and animals experience hearing loss. Let's see if we can apply what we know about the auditory pathway to figure out how hearing deficits and deafness might occur. Let's work backward from the brain.

Well, brain damage to the *temporal lobes* could certainly result in deafness. If we have no functional neurons to receive and interpret auditory input, we cannot hear. This is a good example in which integration of the sensory input is ipsilateral from the source. So, head trauma or disease in the left temporal lobe will create hearing loss for the left ear. Of course, the other CNS components that could be damaged are the thalamus and brains stem. But if we have problems with those portions of the brain, hearing loss is the least of our worries. The next point in the pathway that could be disrupted is the cochlear branch of the vestibulocochlear nerve (eighth cranial nerve). Probably the whole eighth cranial nerve would be diseased or damaged. And that could also create problems with balance and equilibrium. We'll talk about that later.

What about the cochlea? This is certainly a key component of the auditory pathway. Congenital (i.e., born with) deafness is not uncommon among animals. This is often a problem in albino animals. White cats, not necessarily albino, often experience congenital deafness. There are a number of dog breeds that may have congenital deafness. I've seen quite a number of deaf dogs in my career, but Dalmatians seemed to top the list of those with congenital hearing loss. A former colleague of mine did extensive research in congenital deafness in animals. She shared with me that some animals who are deaf from birth have structural defects in the cochlea, especially the organ of Corti. That makes sense. However, she also shared that many animals are born with normal cochlear structure and function, only to develop hearing loss very early in life. And for many, the hearing loss developed from progressive loss of the endolymph bathing the organ of Corti. Once the organ of Corti shriveled up, the cochlear nerve atrophied and the animal was deaf.

In people, hearing loss often develops from chronic exposure to high-decibel noise. In terms of the cochlea, these types of hearing

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deficits develop because of hair cell damage, from being literally "beaten up" against the tectorial membrane. The reflexes involving the tensor tympani and stapedius muscles work only to protect against acute high-decibel sounds. They cannot protect against chronic high-decibel noise. My father developed hearing deficits from his repeated exposure to the firing of big guns aboard ship when he served in the Navy during World War II. Hearing protection was not a top priority when called to battle stations during a naval engagement. And some of those naval barrages could go on for minutes or hours. It takes its toll. And that is something to bear in mind with canine military veterans. If those dogs have been exposed to similar situations, they will likely experience hearing loss too. That's not something we often think about, but we should. And because a dog's acoustic range is much greater than that of humans, our canine veterans have far more to lose. ***Ototoxic*** [*ot(o)-* ear + *tox(o)-* poison + *-ic* pertaining to] drugs also damage and destroy hair cells. ***Gentamycin***, a powerful antibiotic, is a good example of an *ototoxic* drug. And whether administered in an otic preparation or given systemically, the ototoxic effects are the same.

In the middle ear, *arthritis* will make the joints between the *ossicles* stiff. Diminished movement of the ossicles ultimately results in reduced stimulation of hair cells in the cochlea. And while *arthritic* changes may very well develop in animals, especially in those who have experienced middle ear infections, it's probably more likely that reduced movement of the ear drum is the culprit. Remember, there shouldn't be anything in the middle ear except the ossicles, muscles, and air. If fluid, such pus from a middle ear infection, is accumulating in the tympanic bulla, neither the ossicles nor the tympanic membrane will move freely. That creates hearing deficits. Obviously, inflammation of the ear drum itself will reduce its flexibility. Inflamed tissues become edematous, and a swollen tympanic membrane is much less flexible.

If all of the functional components of the middle and internal ear are intact and normal, the only way hearing deficits can be created is by blocking the sound waves. That means something must be obstructing the ear canal. Certainly, excess cerumen (ear wax), hair, and other materials (like pus) can obstruct the canal. In fact, those

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things frequently do. But polyps and tumors within the ear canal or at the orifice (opening) can also partially or completely obstruct the canal. And for those animals who have experienced repeated external ear inflammation from allergies and/or infections, ***hypertrophic*** [*hyper-* excess + *troph(o)-* development + *-ic* pertaining to] changes to the walls of the canal frequently cause hearing loss due to obstruction. We'll talk more about this when we discuss otitis externa in a moment.

Regardless of the *etiology* (cause) of the hearing loss, deaf animals require special handling and care. And this is especially true for us in our veterinary facilities. Remember, when animals are in our facilities, they are usually frightened (whether they can hear or not). And for a deaf patient, it can be even more frightening. Think about it. They can't hear us coming. Our words and tone of voice do nothing to get their attention or soothe their high anxiety. And if we're not careful, startling a deaf animal can result in serious harm to us. I'd bite too, if I were a dog or cat being suddenly awakened in a strange place by a complete stranger. So, what can we do, to avoid this? Well, we need to tap into their other senses. The aroma of a warm plate of canned food would provide a very pleasant awakening. Of course, that's not always practical, especially when we're extremely busy and/or the patient is being fasted for diagnostic testing or surgery. Whenever I approached the cage of a deaf animal in my care, I always tried to get their attention visually, before opening the cage door. If the patient was sleeping, I tried to activate tactile and pressure receptors, by gently bumping the cage door or cage floor. Many of them would be awakened by the vibrations. Then once I had their visual attention, I would slowly enter. For those that I had cared for a long time and knew their behavioral responses (typically very docile, well-adjusted animals), I might carefully awaken them by gently stroking and petting them. The key is to avoid startling the patient. That only serves to put them in fight or flight mode, and that's when they are more likely to bite first and ask questions later. So, go slow, be patient, and use a large measure of understanding and compassion for an animal that has lost a vital sensory ability, leaving them very vulnerable and afraid.

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Otitis

We actually classify ***otitis*** based on the segment of the ear involved. So, *otitis interna* involves the inner ear. *Otitis media* involves the middle ear. And *otitis externa* involves the external ear, the ear canal in particular. Most of the time, veterinary patients develop otitis externa. I'd like a nickel for every time my Basset Hound developed otitis externa. I've mentioned in other chapters some of her chronic disease problems. In Chapters 3, and 8, I shared that she had allergies—environmental allergies and food allergies. And that was the root of her chronic, recurrent otitis. She was a "poster-child" for the link between allergies and otitis. On average, more than 85% of all dogs with chronic, recurrent otitis have an underlying allergic condition. A simple "dietary indiscretion," like eating a small piece of a corn chip (Sadie was allergic to corn), would initiate the inflammatory process. Within minutes her left ear would flare and become red, hot, and ***pruritic*** [*prurit(o)-* itch, itchy + *-ic* pertaining to]. Within the hour, her right ear would flare. After that, in a matter of few hours, her whole body was *pruritic*.

Inflammation like that usually stimulates excessive ***ceruminal*** [*cerumin(o)-* cerumen + *-al* pertaining to] production. *Otitis* also provides a wonderful environment to support microbes (usually yeast and/or bacteria). And overgrowth of microbes resulting in infection doesn't take long at all. Often within a couple of days after her allergic flareup, Sadie would develop an infection in one or both ears. Of course, those big, droopy, heavy pinnae didn't help. They kept those ear canals dark and moist, just what microbes love. And believe it or not, the microbes in each ear could be completely different. I know because I did the ***otic cytology*** [*cyt(o)-* cells + *-logy* study of]. One ear could be loaded with yeast and the other ear could be loaded with just bacteria or combination of two different types of bacteria, plus yeast. Oh well, there was a bright side to her numerous otitis episodes. Sadie's ears contributed to numerous teaching slide sets for *otic cytology*.

I think the most difficult part about treating inflamed ears like that is cleaning them out before putting the medication in. There is no point in putting otic antibiotics in there when the canal is loaded with cerumen and pus. It will never reach the deepest part of the canal or the tissues. And that's precisely where we need it go. So,

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we need to clean them, gently of course. But, and this is a very big but, if there is any chance at all that the tympanic membrane may not be intact, do NOT put a cleaning solution in the ear! If intact, cleaning solutions can be used. Suffice to say that most otitis patients will object to this. It helps to warm the cleaning solution. (Test the temperature on your wrist first, to make sure that it's not too warm!) Squirting in a liberal amount of cleaning solution in the ear canal and squishing it around by massaging the canal is very important. (You should hear squishy noises or you're not doing it right.) Gently sop up and wipe out the dirty liquid with cotton balls and repeat if needed. Now, the dog or cat will, I repeat WILL, shake its head. Keep your mouth closed, and wear eye protection. You know what, it is actually very beneficial during the cleaning process for the animal to shake its head. Centrifugal force makes all of that gunk way down in the ear canal, even from the horizonal canal, fly out. That's a very good thing. Once the canal is clean and relatively dry, the actual medication can be applied. For most medications, it's usually only a few drops. Working that down into the canal by massaging the canal and base of the ear is also important. I mentioned gentamicin earlier. With certain bacterial pathogens, gentamicin is the antibiotic of choice. We need to be careful that it is applied in the right volume over the correct period of time. If we don't, we may cause *ototoxicity* and irreparable damage.

Over the years, I have seen numerous animals, most of them dogs, with extreme *chronic*, recurrent, severe *otitis externa*. In many of them, the inflammation and infection often progressed to *otitis media* too. *Hypertrophic* changes to the *external acoustic meatus* often made it impossible to treat the *otitis*. These animals endured much misery, even when their owners were completely compliant. Sometimes disease like that (especially in an animal whose body over reacts, producing profound *hypertrophic* changes in the canal) is beyond medical management. And in cases like that, the best option was surgical. Sometimes we simply removed the lateral wall of the ear canal so that it could be kept clean and dry and easily treated if otitis developed. Those patients didn't have middle ear involvement. But for the most severe cases involving the middle ear too, we opted for a ***total ear canal ablation*** [L. *ablatus* carried away, removed] and ***bulla osteotomy*** [*oste(o)-* bone + *-tomy* to cut; i.e,.

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cutting into] *(TECABO).* Yep, you read that right. We remove the entire ear canal, and cut into the tympanic bulla to remove the ossicles and the ear drum. The skin over the surgical site is closed, so to look at the dog after it heals, the average observer would never even know what was missing. Doesn't a ***TECABO*** make the animal totally and permanently deaf? Yes, especially if done bilaterally. But with the extreme disease leading up to this moment in time, the patient was deaf already. Once it recovers from the surgical trauma (which is *very* painful), the patient will lead a much more comfortable life, free from *chronic* ***aural*** [*aur(o)-* ear + *-al* pertaining to] disease and pain.

Ear Mites

We cannot talk about otitis externa without talking about ***ear mites***. In dogs and cats, this condition is caused by ***Otodectes cynotis*** (o"to-dek'tēz sin-o'tis). The genus name *Otodectes* literally means "ear biter" [*ot(o)-* ear + *dectes* biter]. And in spite of the fact that its species name is *cynotis* [*cyn(o)-* from Gr. *kynos* dog], this little mite is most often seen infecting cats. Kittens are most severely affected, probably due to their poor immunity. But even adult cats infected for the first time can have pretty significant otitis. As if otitis in and of itself isn't bad enough, otitis caused by *Otodectes* is often worse for the animal. Remember, that cat (or dog) has perhaps hundreds of tiny mites running around in the ear canal, scurrying, scratching, and biting. And they are so easy to see during an ***otoscopic*** [*ot(o)-* ear + *scop(o)-* to view + *-ic* pertaining to] ***exam***. Under the warm glow of the light source of the ***otoscope*** [*ot(o)-* ear + *scope* a viewer; i.e., an instrument used to examine the ears], those little buggers run around like crazy. And because there is usually lots of black, coffee-ground-looking debris (cerumen plus mite excrement) as a backdrop, it's really easy to see the little white mites.

I will never forget the story of a veterinarian who infected his own ear with *Otodectes*. I always shared the story in detail with my students when I taught ***parasitology*** [*parasit(o)-* parasites + *-logy* study of]. Wait, what?! The guy infected his own ear?! Yep. And he did it intentionally, not once, not twice, but three times. The first time, it was mostly curiosity. He wanted to know what his patients were experiencing. What he discovered is that the mites were most

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active at night. In the evening, he could feel and hear their activity increase. Scratching noises would get louder and louder as the night progressed. By the time midnight rolled around and into the wee hours of the night, the scratching noises became almost deafening and the ***pruritus*** [*prurit(o)-* itch + *-us* an] from their biting made it impossible for the man to sleep. And they didn't simply stay in his ear. He could feel them running across his face overnight! As the morning approached, the mite activity would settle down. During the daytime hours, the infestation was pretty tolerable. But as evening approached, the aggravating activity cycle would start all over again. As the days progressed, his ear canal began to fill with debris. After a week or two, his ear canal was completely plugged and he couldn't hear a thing (except the mites of course). By about the third week, the activity seemed to reduce. When all activity seemed to end, he flushed out his ear. There were no residual ill effects.

Wanting to know if his result could be repeated, he infected his ear again. (I know, right?!) The cycle of activity was just as before, with one major exception. It didn't last as long. This time after only a couple of weeks, activity seemed to cease. And the amount of debris was not as great. So, his hearing impairment was not as severe. Okay, he felt the need to repeat his self-sacrificing experiment one more time. Again, the cycle of ***nocturnal*** [L. *nocturnus* night] mite activity was repeated. This time the infestation lasted only about a week, with very little debris. The conclusion that he drew from this was that he developed local immunity to the mites, reducing the severity and longevity of each subsequent infestation. And he extrapolated from that conclusion that the reason we see more severe infestations in kittens and not in adult cats is all due to development of local immunity. Of course, we have to take this with a grain of salt because humans are not dogs and cats. We are certainly not intended hosts for those tiny creatures. And our ear anatomy does not provide an equally suitable environment as the ear canal of a dog or cat. That said, I think he was onto something.

So, does this mean we shouldn't worry about treating dogs and cats for ear mite infestations? Absolutely not! First and foremost, to paraphrase the veterinary technician oath,[a] we are to serve animals

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to the best of our abilities, aiding animals by providing excellent care and alleviating suffering. Veterinarians have similar ethical standards. Second, if we do not treat the animal, the infestation could lead to secondary microbial infection that complicates and worsens the otitis. Third, if we do not eradicate the infestation, this animal serves as a reservoir to infect other animals. And the pet owners could serve as fomites (i.e., transport vehicles) between animals. So, yes, we should treat otitis due to ear mites. Clear? Good.

I know that there is more to the ear than what we have discussed thus far. We still need to discuss the rest of the inner ear and how it contributes to balance and equilibrium. However, because balance and equilibrium require integration of other sensory input, such as vision and proprioception, I think it will be best to tell the complete story after we look at the eye. Get it?—We're going to *look* at the eyes? Okay, never mind. The pun was funny to me.

Vision

Vision is another very important special sense. Of course, there are nonvisual (blind) animals and people who can navigate life quite well, despite their disability. But loss of sight imposes limitations and challenges. Think about it. Vision permits us to see where we are, to orient ourselves in the surrounding environment. Vision enables us to see obstacles and threats in our path. And many animals have outstanding visual aptitude, enabling them to see those threats and obstacles, even in extremely low-light conditions. People need ***optical*** [*opt(o)-* vision + *-al* pertaining to] aids, such as night-vision goggles, to see in extremely low-light conditions. I'll explain why later on. Vision enables us and our animal kin to more easily find a mate, shelter, water, and food. Even honey bees and butterflies can see colorful flowers in the world, guiding them to the nectar they need for food. And differences in ***ocular*** [*ocul(o)-* eye + *-ar* pertaining to] structure and placement, across the spectrum of animal species, create differences in visual aptitude and acuity.

For instance, ***visual fields*** (i.e., spatially, how much can be seen) vary tremendously among animals. Placement of the eyes in the skull helps to determine an animal's visual field and visual acuity,

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especially with regard to ***depth perception***. Animals with forward-facing eyes (that includes humans) have ***binocular*** [*bi-* two + *ocul(o)-* eye + *-ar* pertaining to] ***vision***. With *binocular vision*, an animal visually triangulates distance between itself and the subject it's looking at. That provides *depth perception*. Predators need good depth perception to be able to capture prey. Without *binocular vision*, it would be much more difficult to determine distance to its prey. Without it, when lunging to make its capture, the predator might miss. Of course, the negative feature of forward-facing eyes is the way it limits the *visual field*.

If you look straight ahead at the page you're reading, you are probably using your narrow range of *binocular vision* to read. But you are probably also visually aware of other things beside you because of your ***peripheral vision***. And it doesn't matter that much of your peripheral vision is ***monocular*** [*mono-* one + *ocul(o)-* eye + *-ar* pertaining to]. What's important is that you have a broader *visual field* through your peripheral vision. And the broad visual field provided by your peripheral vision enables you to quickly become aware of someone walking up alongside you. The average visual field (horizontal) of humans is about 200 degrees. Perhaps 60 degrees of that field (centrally) is binocular and the rest is monocular. And because your visual field stops at about your shoulders, you can't see anyone approaching directly from behind. Compare that with a prey animal, like a rabbit. A rabbit needs to be able to see predators, to avoid becoming dinner. The laterally placed, bulging eyes of the rabbit provide a very broad visual field of nearly 360 degrees. And that's just thinking about the horizontal plane. That bunny has pretty good vertical vision too. And that's important to see a hawk or an owl flying above. Most of that rabbit's vision is monocular. But it probably doesn't need much binocular vision to feed on clover. Horses' eyes are somewhat laterally placed too, although not as much as a rabbit. Plus, a horse's eyes don't bulge out like a rabbit's either. Because of those differences, a horse's visual field is not as broad as a rabbit. In fact, directly in front of a horse's nose is a blind spot. Standing there, a horse may not see you. But if you stand caudal to the head, at the level of the shoulder, the lateral eye placement permits the horse to see you. In part, that's why you should lead a horse at its shoulder.

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Directly behind that horse, you won't be seen either, unless the horse turns its head. Let's just say that you don't want to startle a horse from behind. You could be seriously injured. And that is a valuable example of how visual field, dictated by an animal's anatomy, can guide our interactions with the animal. Now, let's take a closer look at *ocular* and ***periocular*** [*peri-* around + *ocul(o)-* eye + *-ar* pertaining to] anatomy.

Ocular and Periocular Anatomy

The ***eyeball*** or ***globe*** sits in the ***orbit*** [from L. *orbita* mark of a wheel circuit] of the skull. There are differences in the bony structure of the orbit, among various species and breeds of animals. Most animals, including humans, have a complete bony orbit that surrounds the full circumference of the globe. But the orbit of dogs and cats is incomplete, as shown in Fig. 11.19. Notice how the lateral border of the orbit is open. The wider that lateral opening, the easier it is for the globe to ***proptose*** [Gr. *proptosis*, from Gr. *pro* before, forward + *ptosis* to fall]. In ***proptosis***, the globe is displaced from the orbit, literally "falling forward." In animals (and humans) with complete orbits, the bony orbit needs to be fractured for *proptosis* to occur. The orbit not only protects much of the globe from injury, but with complete orbits, it also serves as a restraint device for the eye, like a seatbelt. With a lateral gap, it's as if the seatbelt is unbuckled. And in some ***brachycephalic*** [*brachy-* short + *cephal(o)-* head + *-ic* pertaining to] breeds that lateral gap in the orbit is exaggerated. In fact, in some *brachycephalic* dog breeds, such as Pugs, Pekingese, and Cavalier King Charles Spaniels, the orbit is also shallower than other breeds, creating ***exophthalmos*** [*ex-* out + *ophthalmos* eye; i.e., eyes bulging out]. This makes proptosis even more likely. So, be careful when you restrain these bug-eyed beasts. You don't want to cause *proptosis*, by squeezing the beast a little too much with your restraint.

Of course, there is more to protecting and securing the globe than just the orbit. The *ocular adnexa* [L. ***adnexa*** appendage] helps too. Appendages? What? No, the eye doesn't have arms and legs, but it does have ***eyelids*** or ***palpebrae*** [L. *palpebra* eyelid; palpebrae - plural]. The eyelids (Fig. 11.20) provide very important protection for the globe. Obviously, most animals have dorsal and ventral

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eyelids. Certainly, all mammals do. When closed, the globe is covered and protected. Blinking helps to disburse the tear film, to keep the anterior, exposed surfaces of the eye moist. When performing *neurologic* and *ophthalmic* exams, it's important to evaluate eyelid function.

Time out. This is a good place to pause to point out the spelling of words like ***ophthalmic*** and ***ophthalmology*** [*ophthalm(o)-* eye + *-logy* study of]. The *ophthalm(o)-* root has a "ph" followed by a "th." In pronouncing words with this root, the "ph" creates an "f" sound. Please make note of this, so that you spell and correctly pronounce words using this root. ***Ophthalmologists*** [*ophthalm(o)-* eye + *log(o)-* study + *-ist* specialist in] often become quite annoyed with budding veterinary professionals who fail to do so. Now, back to our story.

Neurologically, we want to know if sensory and motor function of the adnexa is intact. Testing the ***palpebral*** [*palpebr(o)-* eyelid + *-al* pertaining to] ***reflex*** helps us to evaluate that. By gently touching *periocular* skin at multiple points, we evaluate multiple cranial nerves that provide superficial sensation and motor activity for both closing and opening the eyelids. This simple test actually evaluates three cranial nerves: the ***oculomotor*** [*ocul(o)-* eye + *motor* movement] (third cranial) ***nerve***, the ***trigeminal*** [from *tri-* three + L. *geminus* twin + *-al* pertaining to] (fifth cranial) ***nerve***, and the ***facial*** (seventh cranial) ***nerve***. The *oculomotor nerve* helps to retract (open) the eyelids. The *facial nerve* helps to close the eyelids. And the *trigeminal nerve* provides sensation. ***Lagophthalmos*** [Gr. *lagos* hare + *ophthalmos* eye; i.e., the eyelids don't fully close] may indicate damage to the *facial nerve*. (No, I don't know how that's related to a hare. I simply like to think of the "lag" part of that word as "lagging behind.") ***Ptosis*** [Gr. *ptosis* to fall; i.e., inability to open the eyelids fully, the upper lid "falls" or droops; by the way, the "p" is silent], on the other hand, would indicate damage to the *oculomotor nerve*. If the animal can open and close its eyelids but does not respond to your touching skin on and near the eyelids, then there may be damage to the trigeminal nerve. Isn't it amazing how much a simple evaluation like the *palpebral reflex* can tell us?

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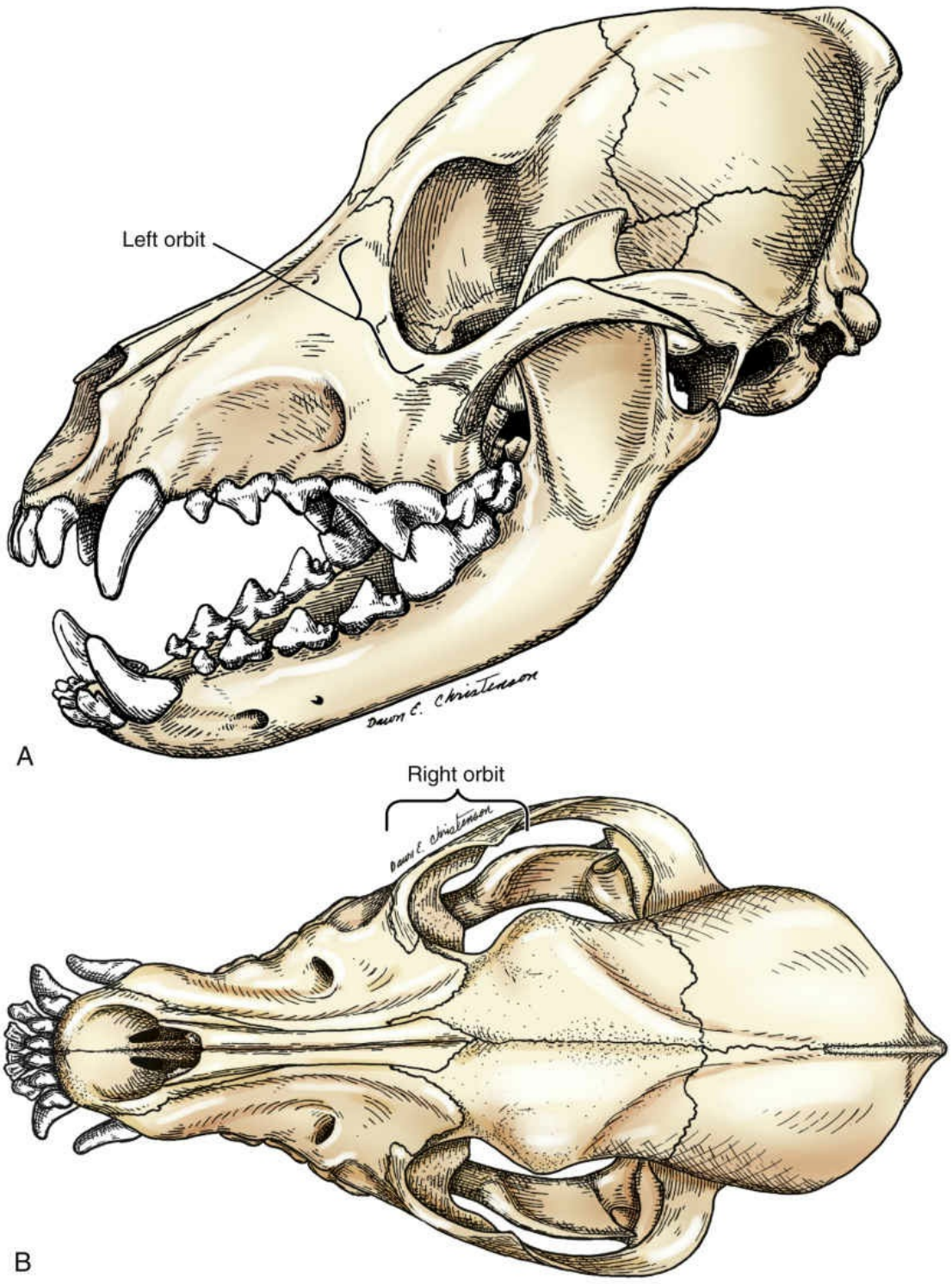

FIG. 11.19 Canine orbit; (A) left lateral view; (B) dorsal view.

Of course, we also need to structurally look at those lids. We look at the lids and each ***canthus*** [Gr. *kanthos* angle]. And if we find abnormalities at these "angles" formed by the eyelids, we need to be very specific when we record the information in the medical record. We should correlate the abnormality to either the ***medial***

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canthus or the ***lateral canthus***, as well as which eye (right or left). By the way, if you look closely at the *medial canthus*, you'll be able to see the ***puncta*** [L. *punctum* a tiny spot; *puncta* plural]. The *puncta* are the openings into the ***nasolacrimal*** [*nas(o)-* nose + *lacrim(o)-* tears + *-al* pertaining to] ***ducts*** that drain tears into the ***nasal*** [*nas(o)-* nose + *-al* pertaining to] ***passages***. Yes, the ***nasolacrimal apparatus*** (Fig. 11.21) is the reason your nose runs when you cry. You can't see it when you look at an animal, but the ***lacrimal*** [*lacrim(o)-* tear + *-al* pertaining to] *gland* is located near the lateral aspect of the dorsal eyelid. In dogs and cats that also have a ***third eyelid*** or ***nictitating*** [from L. *nictitare* to wink] ***membrane***, the ***gland nictitans*** associated with the third eyelid also contributes a little bit to tear production.

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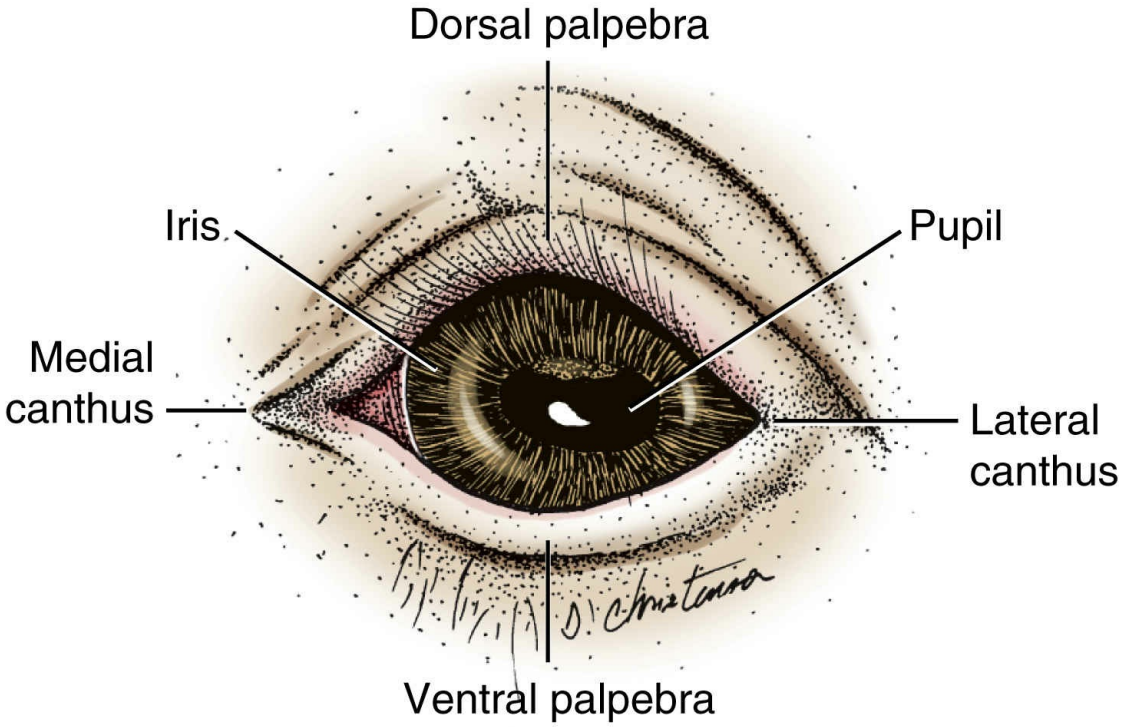

FIG. 11.20 Anterior view of an equine eye and adnexa.

You can see the ***nictitating membrane*** at the *medial canthus* of the eye. Most of the time, we can see only a very small border of the third eyelid. Relaxation of the muscles that retract the third eyelid, when the animal is sleepy or ill, allows it to elevate and cover much of the eye. But in a normal, wide-awake dog or cat, if you want to see more of the *nictitating membrane*, you'll need to apply gentle pressure to the globe, by pressing on the dorsolateral upper eyelid. It will make it look like the animal is winking (and that's probably why anatomists called it the nictitating—"winking" membrane). One final thought about the *nictitating membrane*—sometimes inflammation will cause the *gland nictitans* to swell and ***prolapse*** [from L. *prolapsus* to fall before; i.e., protrusion]. This is commonly called "***Cherry eye***" because it looks like a pinkish-red, round blob (like a cherry) at the medial canthus. Because the gland nictitans contributes to the tear film, it is important to try to preserve it if it *prolapses*. That involves returning and securing it in its normal position but also reducing inflammation.

It's not just the vessels within the gland nictitans that make it look so red in cherry eye. The vessels of the ***conjunctiva*** [from L. *conjungere* join together] contribute to it too. The conjunctiva is a thin, delicate, vascular mucous membrane that is "joined together"

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with the eyelids and globe. The ***conjunctival*** [*conjunctiv(o)-* conjunctiva + *-al* pertaining to] ***membrane*** is a continuous sheet of tissue that lines all surfaces of the third eyelid, the "underside" of the upper and lower eyelids, and the exposed, anterior surface of the globe up to the ***limbus*** [L. *limbus* hem, fringe]. The *limbus* is the border between the transparent cornea and the ***sclera*** [L. *sclera* hard; i.e., the "white" of the eye]. Vasodilation of conjunctival vessels makes the structures it covers appear more pink or even red. Ever had or seen someone with "bloodshot" eyes? You were looking at conjunctival vessels. In general, you can barely see the conjunctiva. In fact, because it is so thin, when we look at the sclera through the conjunctiva, all we see is the white sclera. The conjunctiva on the underside of the eyelids (i.e., ***palpebral conjunctiva***) provides a really smooth surface for gliding over the globe, especially important for the cornea. (By the way, the conjunctival tissue on the eyeball itself is referred to as the ***bulbar*** [L. *bulbar* "bulb"] ***conjunctiva***.)

Because the conjunctiva is a continuous sheet of tissue that runs from the margins of each eyelid to the limbus, it forms a little pouch-like area under each lid. Those are the ***conjunctival sacs***. Why are they important? Well, foreign material can become lodged in them. That's especially true for the ***ventral conjunctival sac***. It's always a good idea to take a peek in there when examining the eyes. The ***dorsal conjunctival sac*** is frequently used for application of *ophthalmic* medications (Fig. 11.22). Application of ophthalmic ointments, as shown, reduces risk of injury to the eye, namely the cornea. (The applicator tip of ophthalmic ointments is designed for application as shown. It can touch the conjunctiva, but ONLY for single patient use. For multipatient use, such as the sterile ophthalmic ointment used to lubricate the eyes of anesthetized patients, the applicator tip cannot touch the animal. Why not? Contamination from one patient to the next.) Even for application of ophthalmic solutions, the conjunctival tissue is much less sensitive than the cornea. So, the patient won't object as much if you drop it into the dorsal conjunctival sac. Could we use the ventral conjunctival sac? Yes. However, the medication is distributed over the eye much better if it is applied in the dorsal conjunctival sac, due to gravity. NOTE: ophthalmic solutions

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should *always* precede ophthalmic ointments when multiple medications are being applied; and at least 5 minutes should elapse between medications. We'll talk more about eyelids later, when we discuss palpebral disorders.

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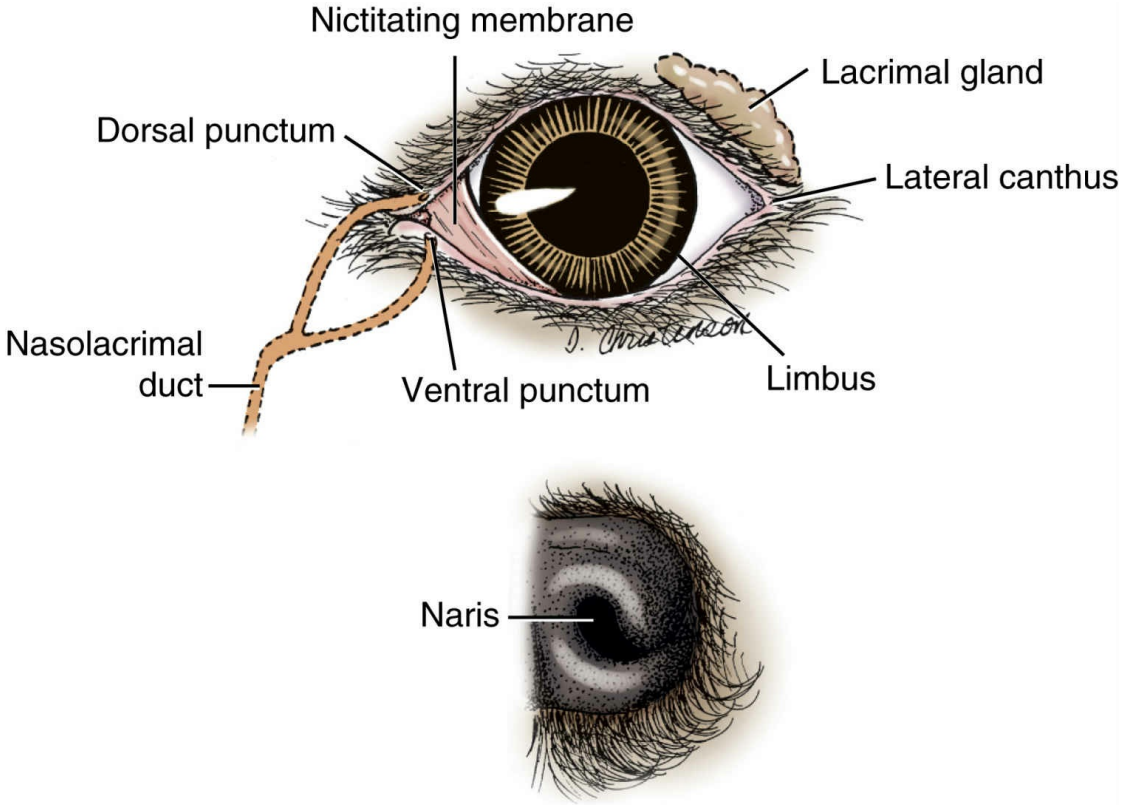

FIG. 11.21 Nasolacrimal apparatus.

Because we've mentioned tears a couple of times, let's take a little detour and talk about tears and tear production, before we move on to look at minute detail of ocular anatomy.

Tear Production

Tears are important to lubricate the superficial parts of the eye. The majority of the watery portion of tears is produced by the ***lacrimal gland***, located in the dorsolateral upper eyelid. The *gland nictitans*, as I mentioned, may also contribute to aqueous (watery) tear production. And it's the watery part that most of us think of when we think of tears. But tears are more than just water alone. There are actually three parts to the tear film: a ***mucus*** (mucin) layer, an ***aqueous*** (watery) layer, and an oil or ***lipid*** layer. Because oil and water don't mix and oil floats on water, the outer part of the tear film is the lipid layer. This is a good place for it. It helps to reduce evaporation of the watery component (the middle layer of the tear film). Finally, right on the eye itself is the mucin (mucus) layer of the tear film. A balance of all three layers keeps the exposed surfaces of the eye moist and lubricated.

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To produce such different components of the tear film, they all can't possibly come from the same place, right? Right. You already know that the ***lacrimal gland*** produces the *aqueous* part of the tear film. Mucus is usually produced by mucous membranes. Guess what, the ***conjunctiva*** is a *mucous membrane*. We certainly have lots of that right where we need it. In Chapter 8, what type of glands produced oily substances? That's right, ***sebaceous*** glands [from L. *sebaceus*, pertaining to *sebum* suet]. And along the margins of the eyelids we have sebaceous glands that are called ***meibomian glands***, named after the German anatomist, Heinrich Meibom. Ideally, all three components of the tear film are produced in a balanced way, to moisten and lubricate the anterior external surfaces of the eye. If we don't have sufficient lubrication, it's kind of like rubbing sand paper over the surface of the eye every time we blink. That's what it can be like with dry eye.

Dry eye

Dry eye is just what you'd think—a dry eye from insufficient tears. And usually this involves the aqueous part of the tear film. How and why does this develop? Well, there may be a number of factors. Certain drugs can reduce lacrimal secretions. And if you think back to our discussion of autonomics, we said that the sympathetic branch decreased tear production and the parasympathetic branch increased tear production. So, in terms of drugs that might adversely affect aqueous tear production, both *sympathomimetic* and *parasympatholytic* agents could do that.

But most of the time, it's an inflammatory problem. Chronic or recurrent inflammation of the lacrimal gland(s), from trauma or an autoimmune disorder, over time chips away at the functional capacity of the lacrimal gland(s). (I've put the "s" in parentheses because trauma is often a unilateral problem.) Controlling the inflammation is important. And if we can reduce or eliminate the inflammation before all of the functional lacrimal tissue is lost, we may be able to stimulate better production of the aqueous part tears with medication. If not, we'll have an ongoing, uphill battle trying to keep those eyes moist and lubricated.

Now, the *meibomian glands* and *mucous membranes* will try to pick up the slack. And it helps a little. But really overproduction of

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mucus and sebaceous secretions just seems to create gross, goopy, crusty, irritated eyes. But it's not the reddened conjunctiva we need to be concerned with, it's the cornea. And really dry eyes can lead to ***keratoconjunctivitis*** [*kerat(o)-* cornea + *conjunctiv(o)-* conjunctiva + *-itis* inflammation of] ***sicca*** [L. *siccus* dry]. This is a very painful and serious condition. It can lead to erosion and ulceration of the cornea. We'll talk more about corneal ulceration later. Management of *keratoconjunctivitis sicca* requires tremendous owner compliance because they need to put apply artificial lubricants to the eyes many times each day. With enough *exogenous* lubrication, hopefully, the animal can be kept comfortable and secondary disease and complications prevented. If not, the animal may ultimately lose one or both eyes.

Don't cry, but that concludes our lacrimal detour. Now, let's take a closer look at the eye itself.

Ocular Anatomy Continued

In Fig. 11.23, you see a schematic of the eye as it sits in the orbit. Notice that it has a number of ***extraocular*** [*extra-* outside + *ocul(o)-* eye + *-ar* pertaining to] ***muscles***. We need numerous muscles to move the eyeball horizontally, vertically, and diagonally and to retract it deeper into the orbit. And if you go back to Table 11.2, you'll see that three of the 12 cranial nerves innervate the various *extraocular muscles*. Those nerves are the *oculomotor nerve* (third), ***trochlear*** [from L. *trochlea* a pulley + *-ar* pertaining to] ***nerve*** (fourth) and the ***abducens*** (ab-doo'sunz [L. *abducens* drawing away]) ***nerve*** (sixth). Again, in a neurologic exam, familiarity with the functional input of these nerves can help us to isolate the neural injury. For instance, medial ***strabismus*** [Gr. *strabismos* a squinting; i.e., deviation of the eyeball that cannot be overcome; medial *strabismus* makes the animal look "cross-eyed"] might point to damage to the *abducens nerve*, paralyzing the muscles that produce lateral eye movement.

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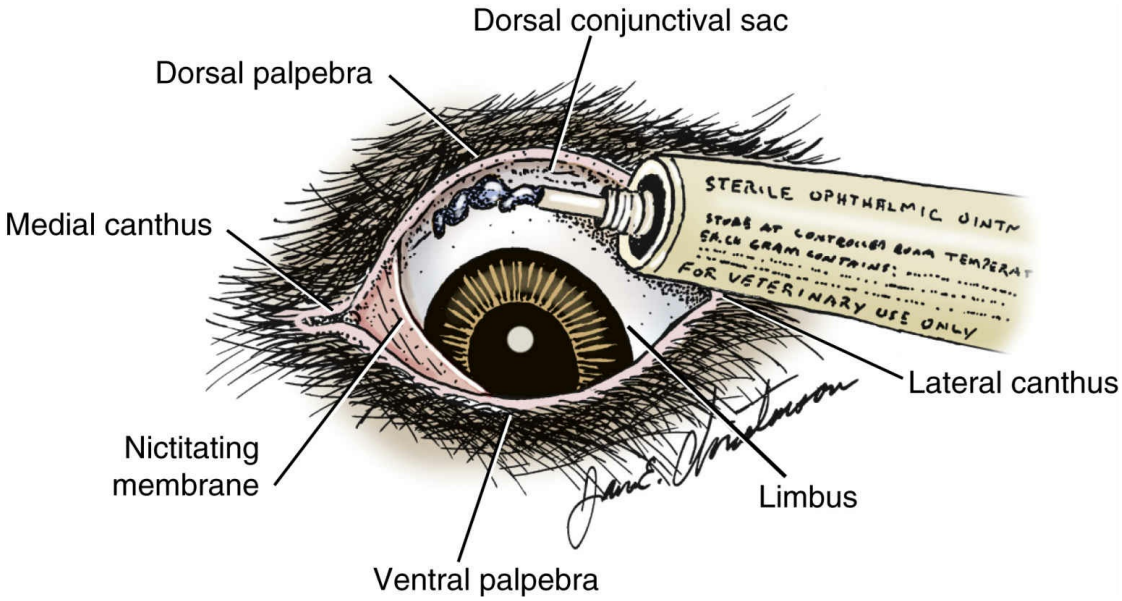

FIG. 11.22 Application of ophthalmic ointment.

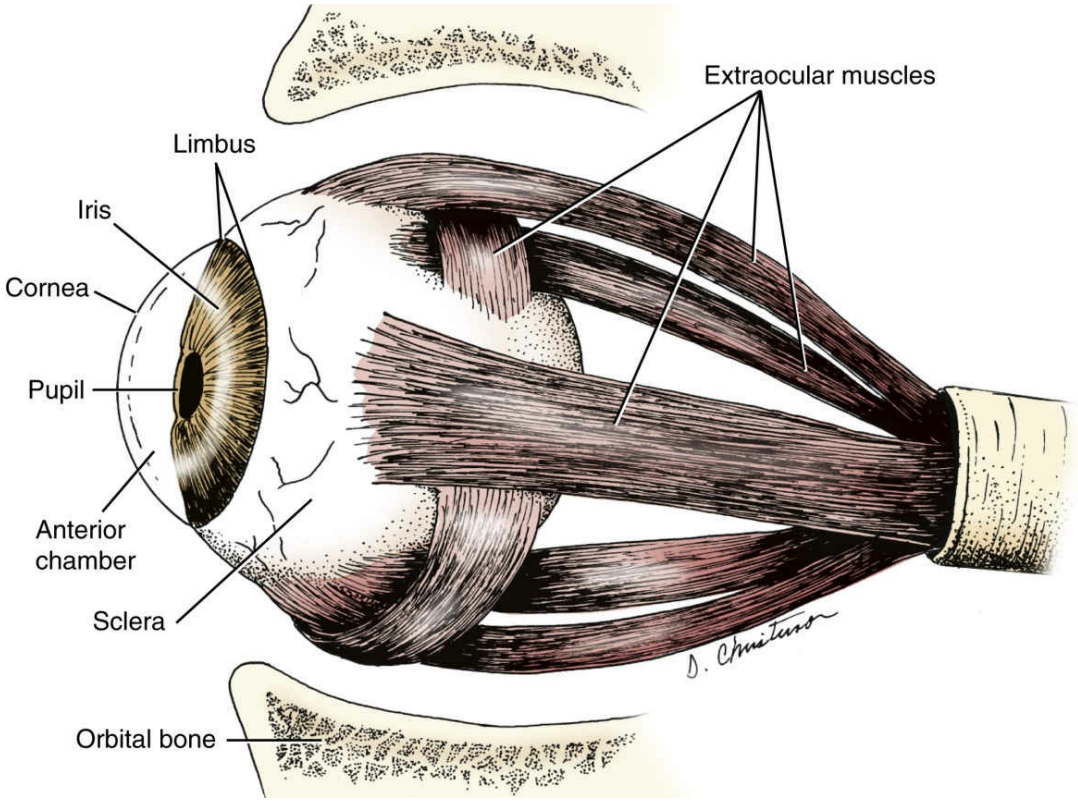

FIG. 11.23 Gross eye schematic.

Notice that the extraocular muscles attach directly to the globe, specifically the ***scleral*** [*scler(o)-* hard + *-al* pertaining to] layer of the eye. This should help you to understand why they named the *sclera*

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the way they did. It is kind of "hard" tissue. No, it's not rock-hard like bone, but it is pretty firm. To form and maintain that ball-like shape of the eye, the *sclera* needs to be ultra-firm, especially when all of those extraocular muscles start tugging on it. This image also shows the *limbus* a little bit better (i.e., that junction between the sclera and cornea). That's because you can actually see the transparent, domed structure of the cornea. (Just remember, the limbus is not a single point. It is a circular border between those two structures.) The fluid-filled space directly behind the cornea is the ***anterior chamber***. And because the cornea and the fluid in the *anterior chamber* are completely transparent, when we look at someone's eyes, we tend to ignore the transparent stuff and notice the colored ***iris*** instead.

The ***iris*** is actually an ***intraocular*** [*intra-* inside + *ocul(o)-* eye + *-ar* pertaining to] ***muscle***. The central hole formed by the iris is what we refer to as the ***pupil***. There are actually two sets of muscle fibers in the iris. One set of fibers radiates out from the pupil, like the spokes of a wheel. These are innervated by *sympathetic nerve fibers*. When stimulated by the sympathetic nerves, they contract resulting in ***mydriasis*** [Gr. *mydriasis* pupil dilation]. The other muscle fibers of the iris are oriented in a circular pattern. These circular muscle fibers are innervated by *parasympathetic nerve fibers*. ***Miosis*** [Gr. *meiosis* making smaller; i.e., pupil constriction] results from parasympathetic stimulation of the circular muscle fibers. We expect pupil size to be bilaterally symmetrical. However, there are times that we may see ***anisocoria*** [*anis(o)-* unequal + *cor(o)-* from Gr. *kore* pupil + *-ia* condition of]. We need to carefully evaluate a patient with *anisocoria* to determine if it is a PNS problem or a CNS problem. The latter is obviously far more serious. But frequently, *anisocoria* develops from disruption of the sympathetic innervation to one eye. And if that's the case, we will see more than just the miotic pupil of the affected eye. Portions of the facial nerve contribute to the sympathetic innervation. So, in addition to the *unilateral miosis*, we may also see *ptosis* and elevation of the nictitating membrane, as well as other facial nerve deficits on the affected side. Head trauma could result in damage to those peripheral nerve fibers. And since we also talked about *otitis media*, you should know that a portion of the path of those nerve fibers is

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near the *tympanic bulla*. So, severe otitis, especially *otitis media*, may damage the peripheral sympathetic nerve fibers. *Anisocoria*, with the collective neurologic symptoms mentioned, is often referred to as ***Horner's syndrome***.

If the pupils are normal, there are times we may need to alter pupil size, for ***ophthalmoscopy*** [*ophthalm(o)-* eye + *-scopy* viewing of; i.e., viewing the eye using an ***ophthalmoscope*** and/or special lens] for instance. Of course, we can artificially create changes in ***pupillary*** [*pupill(o)-* pupil + *-ary* pertaining to] size with ophthalmic medications that disrupt the neural control. For example, applying a *parasympatholytic* or ***mydriatic*** [*mydriat(o)-* mydriasis + *-ic* pertaining to; i.e., an agent that causes *mydriasis*] ophthalmic medication, we can create *mydriasis*. By applying a *sympatholytic* or ***miotic*** [*miot(o)-* miosis + *-ic* pertaining to; i.e., an agent that causes *miosis*] agent, we can create *miosis*. But the iris is not the only *intraocular muscle*. There are also ***ciliary*** [from L. *cilium* hair + *-ary* pertaining to] ***muscles*** behind the iris that are attached to the lens of the eye, as you can see in Fig. 11.24. These muscles are also disabled with mydriatic medications. We'll talk about these more when we discuss the visual pathway later. In brief, contraction and relaxation of the *ciliary muscles* alters the shape of the lens, altering visual focus.

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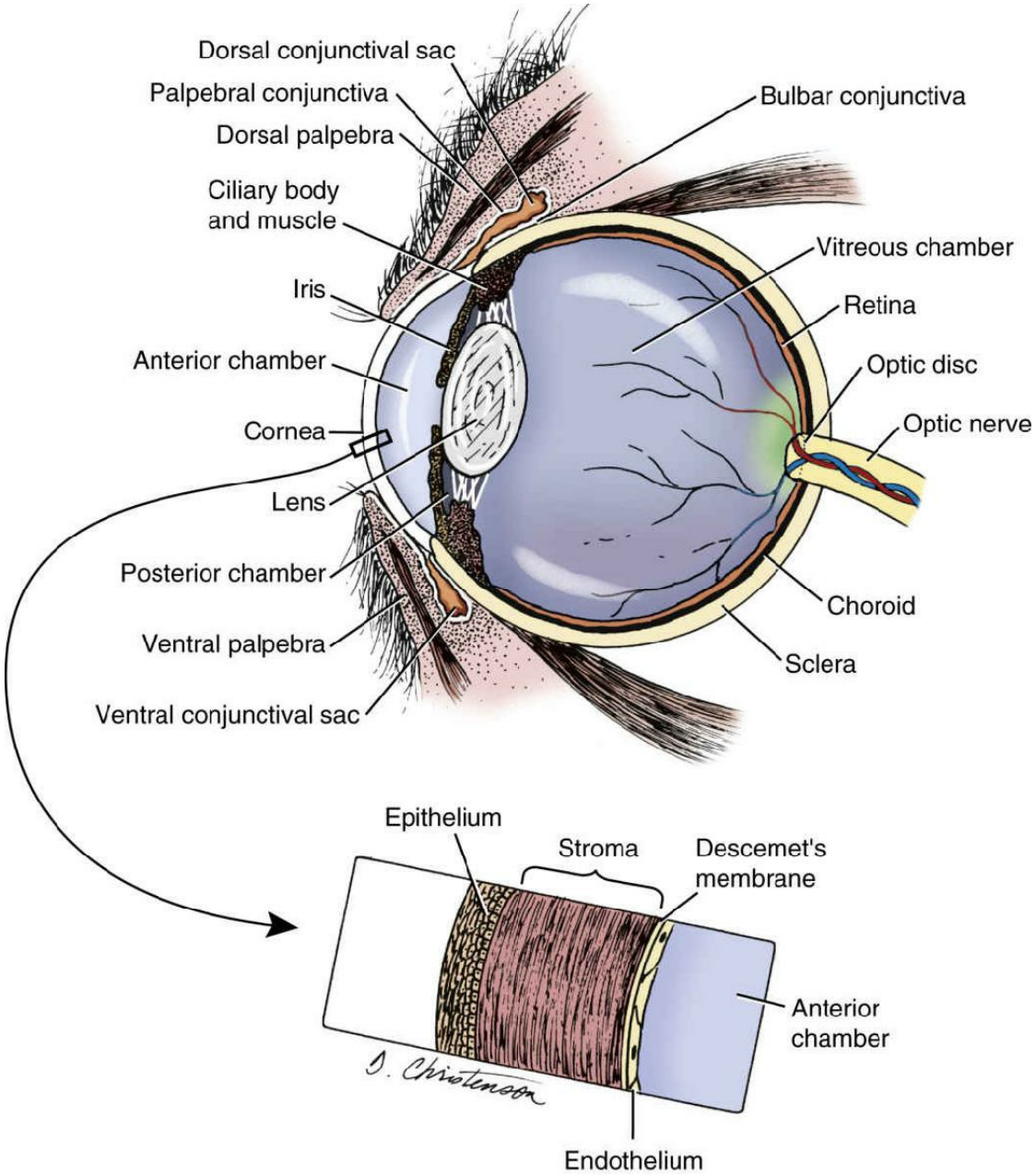

FIG. 11.24 Sagittal schematic of the eye.

Earlier we said that the *anterior chamber* was a fluid-filled chamber between the cornea and the iris. There are two other "chambers" within the eye. The ***posterior chamber*** is a tiny fluid-filled chamber (more like a tiny closet) directly behind the iris. It's so small, the liquid there is just a puddle, compared with what's in the *anterior chamber*. We'll talk more about the "puddle" found in the tiny *posterior chamber*, when we discuss ***aqueous humor*** [L. *humor* a liquid] production and flow. The huge chamber that you see behind the lens, in Fig. 11.24, is the ***vitreous*** [from L. *vitreus* glassy + *-ous* pertaining to] ***chamber***. The *vitreous chamber* is filled by

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a firm, transparent mass of gelatinous material called ***vitreous humor*** [L. *humor* a liquid]. Because it is dense, gelatinous material, it's sometimes referred to as the ***vitreous body***. Okay, so is this stuff liquid or gelatinous? It's gelatinous. But when ancient anatomists originally saw *vitreous humor*, they probably had a difficult time classifying it. After all, gelatinous material has properties of both a liquid and a solid. And if the language of the time provided only those two binaries, I probably would have defaulted to "liquid" too. The one thing they nailed was the "*vitreous*" part of the name. Because it is crystal clear and has mass, it really does look "glassy." Now for the next big question: what does *vitreous humor* do? Nothing. It just sits there. Well, that's not entirely true. It plays a supportive role. Because of its density and mass, it contributes to maintaining the spherical shape of the globe. So, between the *sclera* and the *vitreous humor*, we can maintain the shape of the eye-*ball*. (Get it? …eye-*ball*?) The other really important thing that vitreous humor does, despite the fact that it simply sits there like a couch-potato, is help to hold the ***retina*** in place. Beyond that, it just sits there, occupying space. It's a pretty easy job.

While we're back here in the vitreous chamber, we might as well talk about the *fundus* [L. *fundus* bottom or base]. Okay, here again the Latin is not all that helpful. So, let's see if clinical application will help you to understand what the fundus is. When we use ***ophthalmoscopy*** [*ophthalm(o)-* eye + *-scopy* viewing of] to look at the interior, back wall of the eye's vitreous chamber, what we can actually see is the *fundus*. So, if you look at Fig. 11.24, what we can visualize (depending on how well our *mydriatic* agent dilates the pupil) is maybe the back half of the wall that forms the *vitreous chamber*. Skilled *ophthalmologists* can visualize more. A typical ***fundoscopic*** [*fund(o)-* fundus + *scop(o)-* view + *-ic* pertaining to] view is shown in Fig. 11.25. But wait, in Fig. 11.25 the retina is not labeled, but it is in Fig. 11.24. That's no mistake. When we do our *fundoscopic* exam, we can't actually see the retina because it's transparent. So, we actually look through the retina to see the vessels and the ***tapetal*** [L. *tapetum* a rug or tapestry + *-al* pertaining to] tissues.

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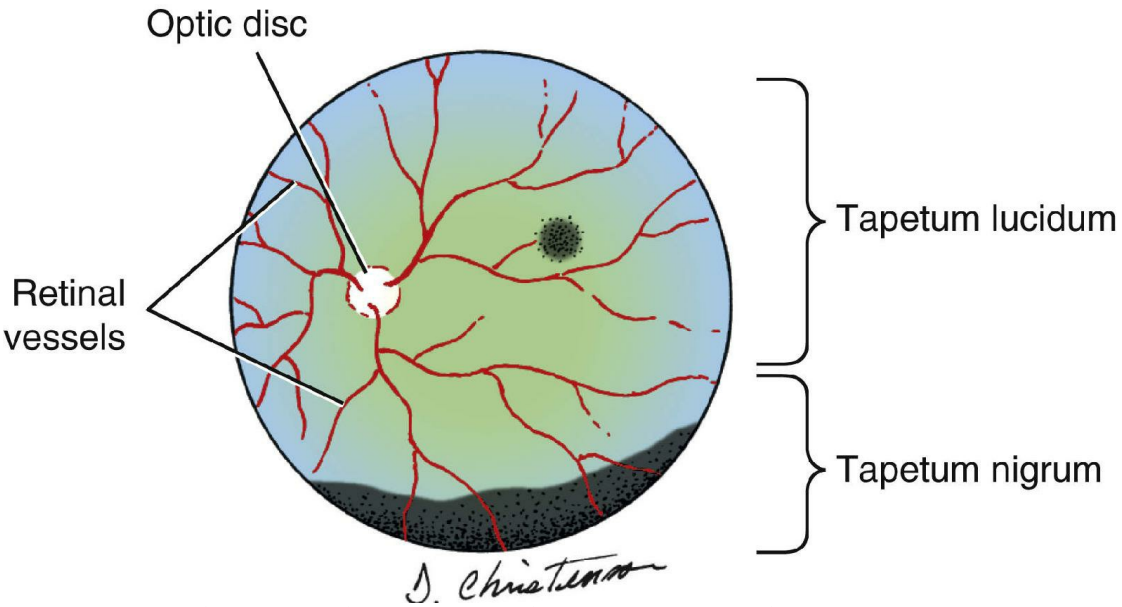

FIG. 11.25 Central view of a normal canine fundus.

Are you overwhelmed by all of the weird ophthalmic terms yet? If you are, you're not alone. There are lots of tiny but important bits and pieces to the eye. And each one of them has a special name. In time, you'll get a handle on them. Be patient with yourself and remember to breathe. You can do this. Now, let's deal with that "rug" that was just mentioned. A rug covers the floor and most tapestries cover the wall, right? So, if we have *tapetal tissue* in an animal's eye, it must cover some sort of floor or wall. And in the case of many animal eyes, they have some beautiful "rugs" and "tapestries." One of the most beautiful rugs we can see in the *fundoscopic* view of Fig. 11.25 is the *tapetum* [L. "rug"] ***lucidum*** (loo'si-dum [L. *lucidum* bright, shiny]. And in many cases the *tapetum lucidum* is an iridescent greenish-blue. Have you ever been driving at night and had your headlights reflect off of the eyes of a cat, dog, raccoon, or deer? It was probably a bright greenish reflection, wasn't it? Your headlights were actually reflecting off of the animal's *tapetum lucidum*. You've also probably seen photographs of friends or family members with "red-eye." You see, the human fundus is actually pretty boring. (Not my words. Talk to my ***optometrist*** [*opt(o)-* vision + *metr(o)-* measure + *-ist* specialist in].) Humans don't have colorful tapetal tissue like animals do. So, the ***fundic*** [*fund(o)-* fundus + *-ic* pertaining to] reflection is red from the blood in the vasculature.

What is the purpose of the *tapetum lucidum*? Because it is so

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brightly colored, in low-light situations it actually enhances the dim light. We don't want light bouncing off like it did with your headlights. We just want some enhancement because the ***retina*** (the actual neural tissue of the eye responsible for vision) is ***photosensitive*** [*phot(o)-* light + *sensitive*]. I like to compare the enhancement to the clothing someone might wear while walking after dark. Which will enhance the ability of others to see that person?—light-colored clothing or dark-colored clothing? It's the light-colored clothing. The *tapetum lucidum* does a similar thing. And because of the light enhancement, it will be much easier for the light-sensitive specialized receptor cells to be stimulated. And we strategically locate the colorful *tapetum lucidum* on the central part of the *fundus*, where optimal vision is generated. The rest of the fundic "carpet" is ***tapetum nigrum*** [from L. *nigra* black]. The portions of the *retina* over the *tapetum nigrum* are probably used for daylight and peripheral vision. In the dark, it's probably more important to be able to see well directly in front of the animal to avoid obstacles. Peripheral vision serves a better purpose in daylight. We'll touch on the retina and tapetal tissue more when we talk about the visual pathway. What lies underneath the tapetal tissue? The ***choroid*** [Gr. *chorion* membrane + *-oid* resembling] is the middle layer of the eye. And the tapetal tissue is the superficial pigmented surface of the *choroid*. So, from inside out we have the retina, the choroid, and the sclera.

The ***choroid*** is part of what is referred to as the *"vascular tunic"* of the eye, also known as the ***uvea*** [from L. *uva* grape]. Okay, let's see if we can connect the dots on this vascular tunic—uvea business. First, if the tissue is vascular, it is full of blood. Crushing vascular tissue squeezes out its "juices," namely blood. When we crush grapes, we squeeze out colorful juices too. So, maybe ancient anatomists thought all of the uveal tissues were, for lack of a better word, "juicy." Sorry. It's a stretch, but it's the best I can do. Those old, crusty, ancient anatomists are no longer around to ask questions. If you remember anything at all, remember that the ***uvea*** is the highly vascular *intraocular* tissue that includes the ***choroid***, the ***iris***, and the ***ciliary tissues***. ***Uveitis*** [*uve(o)-* uvea + *-itis* inflammation of] is inflammation of some or all of those tissues. Because the *iris* and *ciliary tissues* are in the anterior part of the eye,

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if the inflammation involves only those two parts of the uvea, it is referred to as ***anterior uveitis***. And *anterior uveitis* can have a tremendous impact on production and flow of the *aqueous humor*.

Aqueous Humor Production and Flow

Aqueous humor is the watery, ***intraocular liquid*** that fills the anterior and posterior chambers of the eye. (Remember, the *posterior chamber* is really just a small "water closet" behind the iris.) Of course, by filling the *anterior chamber* with this liquid, it helps the cornea to maintain a nice domed shape. But it is also important to maintain the actual tissue of the cornea too. If you look again at Fig. 11.24, especially the magnified part of the cornea, you'll notice that there are no blood vessels. Most other tissues of the body have direct blood supply for delivery of nutrients and removal of wastes. But we can't afford to have vessels running through the cornea. It needs to be a perfectly, crystal-clear dome, so that we can see. Somehow, we have to provide nutrients to all of those tissue layers you see in that diagram. And that's where aqueous humor comes in. But remember, it's not only delivery of nutrients that we need; it's removal of wastes too. Cellular activity, especially by the epithelial and endothelial cells, produces wastes just like other cells of the body. So, we need a constant refreshing flow of aqueous humor all the time.

In Fig. 11.26, you see a close-up schematic of the anterior globe. Remember, this is a two-dimensional diagram providing a sagittal view of the anatomy. Bear in mind that anatomic features, such as the ***iridocorneal*** [*irid(o)-* iris + *corne(o)-* cornea + *-al* pertaining to] ***angle***, are found around the full circumference where the iris and cornea meet. The other important point to make is that this is a *closed system*. The fluid is always recycled within the body.

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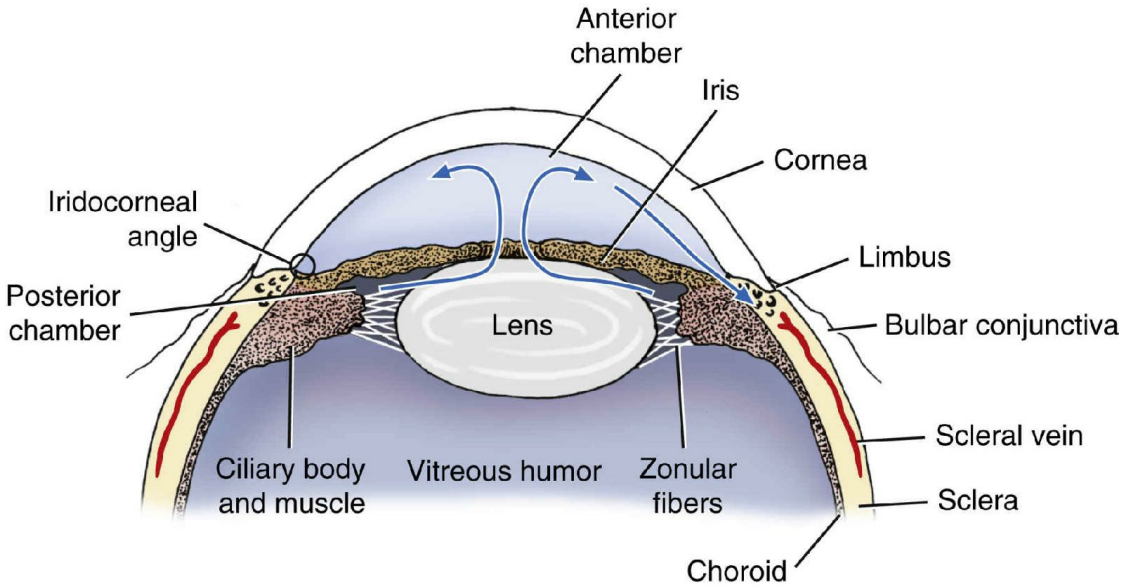

FIG. 11.26 Schematic of aqueous humor production and flow.

So, we need a faucet, a little sink that overflows into a big fountain, and a drain that recycles the water back to the faucet. We said earlier in one of our anatomic discussions that the ciliary muscles are attached to the lens to provide focusing ability for vision. In Fig. 11.26, you'll see a label for the "ciliary body and muscle." Those are two very distinct tissues. Unfortunately, the ***ciliary body*** is simply too difficult to actually show here. But because it's secretory tissue, it stands to reason that we need the ciliary body right next to the *posterior chamber* (our tiny water closet). That secretory tissue is our "faucet." And the ciliary body is constantly producing aqueous humor and pouring it into the posterior chamber. If it's constantly produced, that fluid has to go somewhere. And our goal is to get it into the anterior chamber. So, the path of least resistance is to flow from the posterior chamber, over the surface of the lens, and through the *pupil* into the *anterior chamber*. That flow of fluid through the pupil creates our *intraocular* "fountain." So, as this fresh aqueous humor is gushing into the anterior chamber, it washes over the interior surface of the cornea, providing a new supply of needed nutrients. (No, it doesn't blast against the cornea. But that fountain description gives you a good mental image of the aqueous flow.) The nutrients diffuse into and through the corneal endothelium and the other corneal layers, while waste products are removed and carried away to the drain. Aqueous humor drains at the *iridocorneal angle*. There are a bunch of

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tiny, little drain holes all around the iridocorneal angle that lead off to scleral vessels. Ultimately, *scleral veins* carry the fluid off to general circulation. And as we learned from our cardiovascular discussions in Chapter 5, eventually the arterial blood will circle back around to the uvea (including the ciliary body), to support further production of fresh aqueous humor.

This is a fantastic water, waste-management, recycling system. And it works very well until something goes wrong that results in excess fluid accumulations in the anterior globe. Remember, the eye is a closed structure. There are no pressure-relief valves. And if *intraocular pressures* continue to rise and persist for too long, structures within the eye will be damaged. But how might excess aqueous humor develop? Well, we have one way in and one way out. The ciliary body pumps it in, and the iridocorneal angle drains it away. So, we have two possibilities: a) the ciliary body is producing too much fluid, or b) the drain at the iridocorneal angle is blocked.

Overproduction of aqueous humor is a possibility. For instance, a tumor in the anterior uveal tissues might cause the ciliary body to go overboard with production. But that would be a rare circumstance. So, that leaves us one possibility—a blocked drain. That is the most frequent cause of excess aqueous humor and increased intraocular pressures. We mentioned *uveitis* earlier, specifically anterior uveitis. In *anterior uveitis*, the structures directly involved in the inflammation are the iris, *ciliary muscles,* and the *ciliary body*. What happens to most tissues when they are inflamed? They become *edematous* (swollen). If the iris becomes edematous, what happens to the iridocorneal angle? The angle is reduced. Hypothetically, if a normal iridocorneal angle is 30 degrees, with iris edema that angle might be reduced to 10 degrees or less. Wow, that would severely reduce access and flow to the actual drainage holes. And if the ciliary body continues production, fluid and pressure build. But wait, it can get worse.

One of the cardinal signs of inflammation is pain, right? Do you suppose that anterior uveitis is painful? You bet it is. Do you suppose that increased intraocular pressure is painful? You bet it is. How do muscles typically respond to pain? They often contract and go into spasm. The iris and ciliary muscles are muscles, just like

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other muscles of the body. They'll probably contract and go into spasms too. And that creates a catch-22 of inflammation and pain—muscle spasm—lactic acid—increased inflammation and pain—more and continued muscle spasm—more lactic acid … you get the idea. And motor input feeding this cycle is *parasympathetic*. So, spasm of the iris causes *miosis*—marked miosis, giving us a pinpoint pupil. Now we have all but stopped flow of fluid. Very little will get through that tiny pupil. *Stasis* [Gr. *stasis* "a standing still"] of aqueous flow and the close proximity of that swollen, inflamed iris just sitting on the lens capsule is a recipe for disaster.

Proteins are bound to ooze out of the iris capillaries. You might recall some important plasma proteins from Chapter 3, such as *fibrinogen*. That fibrinogen is going to turn into fibrin, and for all practical purposes glue that iris to the lens. There's a term for that. It's called ***synechia*** (sin-e'ke-uh [Gr. *synecheia* continuity; i.e., adhesion]). We can't see that right now, as we examine our patient. All we can see is a *miotic* response to the intraocular pain. The animal probably has ***blepharospasm*** [*blephar(o)-* eyelid + *spasm* acute, violent muscular contraction] too. (Blepharospasm makes it look like the animal is squinting.) The best way to relieve intraocular pain and muscular spasm is to apply a *parasympatholytic*, a *mydriatic*, also sometimes called ***cycloplegic*** [*cycl(o)-* circle + *pleg(o)-* paralysis + *-ic* pertaining to] agent. The circular muscle fibers of the iris, innervated by the parasympathetic nerve fibers, create the miosis. They are the ones in spasm. By paralyzing them, *mydriasis* will result, ending the painful, spasmodic catch-22 cycle. On the bright side, this provides analgesia. On the downside, where the *fibrin* glued the *iris* to the *lens capsule*, the iris will probably become torn as the pupil dilates. Now, we have intraocular bleeding.

That blood will seep into the anterior chamber. We call this ***hyphema*** [from Gr. *hypaimos* suffused with blood; i.e., blood in the anterior chamber]. Oh my, blood outside the vasculature clots, right? And now we have blood pooling in the *anterior chamber*. Our drainage system for aqueous humor is designed to drain watery fluid. Now we have cells and clots that will compound our drainage problems. And *mydriasis* of our swollen iris probably reduces our drainage angle even more as the iris bunches up at the

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margins. We are caught between a proverbial "rock and a hard place." With time and the right drug combinations, we may very well be able to get the inflammation under control and ultimately resolve the anterior uveitis. In the process, we need to keep a close watch on intraocular pressures with ***tonometry*** [*ton(o)-* pressure + *-metry* measuring]. Adjusting and adding medications as needed to reduce fluid abundance and pressures. It is not an easy task, but it can be done. I've seen it done many, many times.

The funny thing about *uveitis* is that once an animal develops uveitis, even if it was from a single traumatic event, that patient is likely to experience uveitis again. And with multiple episodes of uveitis, with all of the pressure consequences and physical alterations (such as scarring) made to the eye, especially at the iridocorneal angle, chronic recurrent disease perpetuates increased intraocular pressure.

Glaucoma

Glaucoma is a *chronic* condition of the eye. Most often it develops from abnormalities affecting the iridocorneal angle. Those abnormalities may be from repeated inflammatory insults, like what was just described. Or it may be from inherent structural abnormalities, like my father with closed-angle glaucoma. The common thread lies in the perpetual increased intraocular pressures. Even if those pressures are not extreme, causing the globe to stretch and bulge, over time the increased pressures cause intraocular damage. The lens may be affected by influx of fluid into its matrix. And if fluid gets into the tissue of the lens, it becomes somewhat opaque (cloudy). With extreme pressure increases, the cornea may become opaque as well. And the word *glaucoma* [G. *glaukos*, *glaukoma* "bluish-gray"] actually refers to that grayish opacity.

But probably the most crucial functional structures of the eye are the *retina* and the *optic nerve* (cranial nerve II). Slowly, progressively neurons of the retina are destroyed from the pressure. Those most anterior in the globe tend to be lost first. So peripheral vision is the first to be lost. Neurons continue to be lost, as long as pressures remain high. Even if we get pressures under control, whatever neurons were lost will never be regained. And the ***optic disc***

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experiences change too. It often appears flattened and cupped from the pressure. The *optic disc* is the connection of retinal neurons and the optic nerve. Destroy the optic disc, which is the point of origin of the optic nerve, and we lose vision totally.

You see, *glaucoma* is not just increased intraocular pressure. It is a chronic disease with all of the intraocular damage that results from chronic increased intraocular pressure. It's an insidious disease, especially in animals. They can't verbalize the early symptoms that they may see and feel. So, often, by the time we evaluate them and diagnose the problem it's too late to save any vision. Our goal at that point becomes saving the globe itself. And sometimes we can. But many times, in the best interest of the patient (especially if pain is a factor, as it so often is), ***enucleation*** [*e-* out + L. *nucleus* kernel + *-ation* act or process of; i.e., surgical removal of the eye] is the best course of action. It's an esthetic loss, not a functional one. The animal was blind in that eye already. Don't worry. We don't leave an open, empty orbit. A prosthetic support device may be placed in or over the orbit and then the eyelids are sutured closed. The prosthetic device prevents the lids from sinking into the orbit. Then it simply looks like the animal is holding its lids closed.

Common Eyelid Disorders

Two of the most common eyelid disorders are ***ectropion*** [*ec-* out + *trop(o)-* turning + *-on* an] and ***entropion*** [*en-* in + *trop(o)-* turning + *-on* an]. Ectropion usually affects the lower eyelid. Instead of the *palpebra* nestling up against the globe, it turns out, away from the globe. When it's severe, the eyelids can't close completely because the lower lid is hanging out, flapping in the breeze. But that may not be as problematic as foreign material falling into the exposed *ventral conjunctival sac*. The conjunctiva and eyelid are more likely to be injured. So, ***blepharoplasty*** [*blephar(o)-* eyelid + *-plasty* reconstructive surgery] is often performed to alter the palpebral structure, so that the margin of the eyelid nestles against the globe as it should.

Entropion is more problematic. That's because in this condition the eyelid has rolled in, so that the furry surface is now rubbing on the cornea. Ouch! Look at Fig. 11.24 again. Imagine rolling the furry surface of one or both lids onto that cornea. Now look at the

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magnified section of cornea. Imagine each time the animal blinks and moves its globe. That epithelium will be scraped and abraded. It may abrade through that epithelium, layer by layer through those stratified cells. Did I mention that the corneal has an abundance of sensory nerve endings including *nociceptors*? This is an extremely painful situation. And it's one that frequently leads to corneal ulceration.

The thing we need to figure out, with an *entropion* patient, is whether this is a structural problem or a ***neuromuscular*** [*neur(o)-* nerve + *mucul(o)-* muscle + *-ar* pertaining to] problem. You see, with *ocular* pain from something like *uveitis* or corneal ulceration, the animal generally has *blepharospasm*. Many times, that just makes the animal look like it's squinting. But with really severe blepharospasm, they can close those lids so tightly that they begin to roll in. And if the animal actually has a corneal ulcer, then we need to figure out—which came first, the chicken or the egg (i.e., the ulcer or the entropion)? A drop of topical anesthetic might just give us the answer. By relieving the corneal pain, the entropion (if caused by blepharospasm) will likely resolve because the lids are no longer in spasm. If that doesn't happen, we need to treat the corneal ulcer and correct the eyelid conformation. Yep, that involves *blepharoplasty* again.

Corneal Ulceration

Because we've mentioned ***corneal ulceration*** multiple times, we might as well discuss it in a little more detail. Corneal ulceration can result from many, many things, such as *keratoconjunctivitis sicca*, *entropion*, trauma, and infection. That makes sense based on what's been discussed already. But believe it or not, one of the most common events to cause corneal ulceration is bathing. Shampoos tend to be very alkaline, and the detergent acts as a ***surfactant***. We talked about surfactant in Chapter 5, when we discussed surface tension in the alveoli. As you may recall, *surfactant* stands for "surface acting agent." Let's see, what is our tear film composed of? —mucus, water, and oil. So, what will detergents do to the tear film? (1) Detergents emulsify oils and fats, breaking them into smaller bits. (2) Detergents with their surfactant properties reduce the surface tension of the watery part of the tears. And (3)

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detergents are pretty effective at stripping organic material such as *proteinaceous* [*protein* + *-ceous* pertaining to] mucus. That shampoo can thoroughly destroy the tear film. Then we have the cornea. What do detergents do to the skin on our hands? It makes them dry and flakey. Let's see, we destroyed the tear film. The cornea is dry because of it. The corneal epithelium is now dry and flakey from that and the direct impact of the detergent. And every time the animal blinks, its dry, sticky eyelids are abrading the epithelium. What great recipe for creating a corneal ulcer! We don't need any major systemic or *ophthalmic* disease. All we need is some shampoo. This is why a sterile, over-the-counter ophthalmic ointment should always be applied to the eyes before bathing.

Corneal ulcers need to be diagnosed and treated early. They can become very serious, very fast, especially when bacteria jump on the bandwagon. Look again at the magnified corneal cross-section in Fig. 11.24. A *superficial corneal ulcer* involves the corneal ***epithelial layer***. Deeper ulcers get down into the ***stroma*** [Gr. *stroma* anything laid out for lying on]. *Stroma* is a transparent connective tissue of sorts. It's one thing to repair epithelium. We've got cells right there to quickly repair the area. But repair of connective tissue?—that's a tall order. We spoke about the difficulty in repairing connective tissues in Chapters 4 and 8. And the cornea is ***avascular*** [*a-* without + *vascul(o)-* vessel + *-ar* pertaining to]. So, we have no direct delivery route for cells and other supplies. We'll need vascular supply and cells migrating to the defect. It takes time to grow new, temporary vessels into that cornea. Suffice it to say that healing of a deep corneal ulcer will NOT be a rapid process. And if that deep ulcer gets all the way down to the ***Descemet membrane*** (named after the French anatomist, Jean Descemet), we have one ultrathin *basement membrane* and a single layer of *endothelium* keeping the aqueous humor inside that eyeball. And at the middle of that deep, deep ulcer, the *Descemet membrane* bulges up like a blister. We call that a ***descemetocele*** [*Descemet membrane* + *-cele* herniation]. And it won't take much to pop it, like a water balloon. Most of the time, if a *descemetocele* ruptures, the iris herniates through the hole. If that happens—game over. The chances of saving this eye are slim to none. And this is precisely why corneal ulcers need to be taken seriously and treated promptly.

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For deep ulcers and *descemetoceles* (that have NOT ruptured), we often use a natural "Band-aid" over the ulcer. For an *intermediate stromal ulcer*, we may simply perform ***blepharorrhaphy*** [*blephar(o)-* eyelid + *-rrhaphy* suturing of; i.e., suturing the eyelids closed]. This is a temporary situation. We suture the lids closed, leaving enough space near one *canthus* to administer the needed ophthalmic solutions to treat the ulcer. The sutures are removed when the ulcer is sufficiently healed. For *descemetoceles* we often do a ***conjunctival graft***, where the conjunctiva (still attached to the eyelid) is sutured directly over the ulcer. The value of this is providing a whole team of cells, in that conjunctival tissue, directly in the wound to heal the ulcer.

By the way, remember corneal ulcers are painful. Ocular pain usually snowballs, by causing spasm of the *intraocular* muscles. That can lead to *uveitis*. With uveitis the animals are often ***photophobic*** [*phot(o)-* light + *phob(o)-* aversion, fear + *-ic* pertaining to]. Why would they have *photophobia*? Well, the iris constricts when the eye is exposed to light. And that's just going to compound the intraocular muscle spasm and pain. That, in part, contributes to the *blepharospasm* too. By holding the lids tightly closed, the eye isn't exposed to light. All of these symptoms may develop with corneal ulceration and/or uveitis. And because of all intraocular muscle activity, corneal ulceration can easily lead to uveitis.

Okay, we have skirted around the whole purpose of the eyes—vision. And since we just mentioned *photophobia*, it seems to be a good time to finally take a look at the visual pathway, including how it may become impaired.

Visual Pathway and Visual Impairment

Many of the things we have discussed so far can disrupt the ***visual pathway***. Just look at Fig. 11.27 and think about all of the structures in the path of that light source before it shines on the *fundus*. And that's the goal, right? The goal is for light to shine on the fundus to stimulate ***photoreceptors*** [*phot(o)-* light + *receptor* one that receives] in the *retina*. Once a sensory impulse is generated, it is whisked away via the *optic nerve* to the visual centers in the *occipital lobes*. It seems simple enough–well, except for that refraction part that

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actually flips the image upside-down as it shines on the fundus. Thank goodness the occipital lobes can right the image. If they didn't, our world would literally appear to be turned upside-down!

Honestly, the overall design of the eyes is amazing, but there are so many pieces and parts that can be "broken," impairing our vision. Of course, even the *photoreceptors* ***(rods and cones)*** themselves in normal individuals can create visual impairment. For example, humans have abundant cones in the retina. ***Cones*** are the type of sensory receptor that permits us to see **color**. We have rods too but not as many. ***Rods*** can be stimulated in **low-light** situations but do not perceive color. So, with lots of cones that require bright light, too few rods, and no *tapetum lucidum* to enhance low light entering our eyes, we have impaired night vision. Most domestic animals have an abundance of rods, fewer cones, and some degree of tapetum lucidum. So, they can see very well in low light, but during the daytime, they probably couldn't tell a purple petunia from a bright yellow daisy. They have color impairment with their vision. Of course, they never had much color vision, so they don't know what they're missing. And that's just thinking about how normal anatomy can result in visual impairment. In case you're wondering, animals can also experience focus issues, such as near-sightedness and far-sightedness. No, we don't make optical corrections with glasses. We just avoid things like agility or equestrian competitions when they have optical abnormalities like these. They might miss a jump and be injured. Now that's just thinking about some things outside the realm of disease. With disease processes, the possibilities seem endless for creation of visual impairment.

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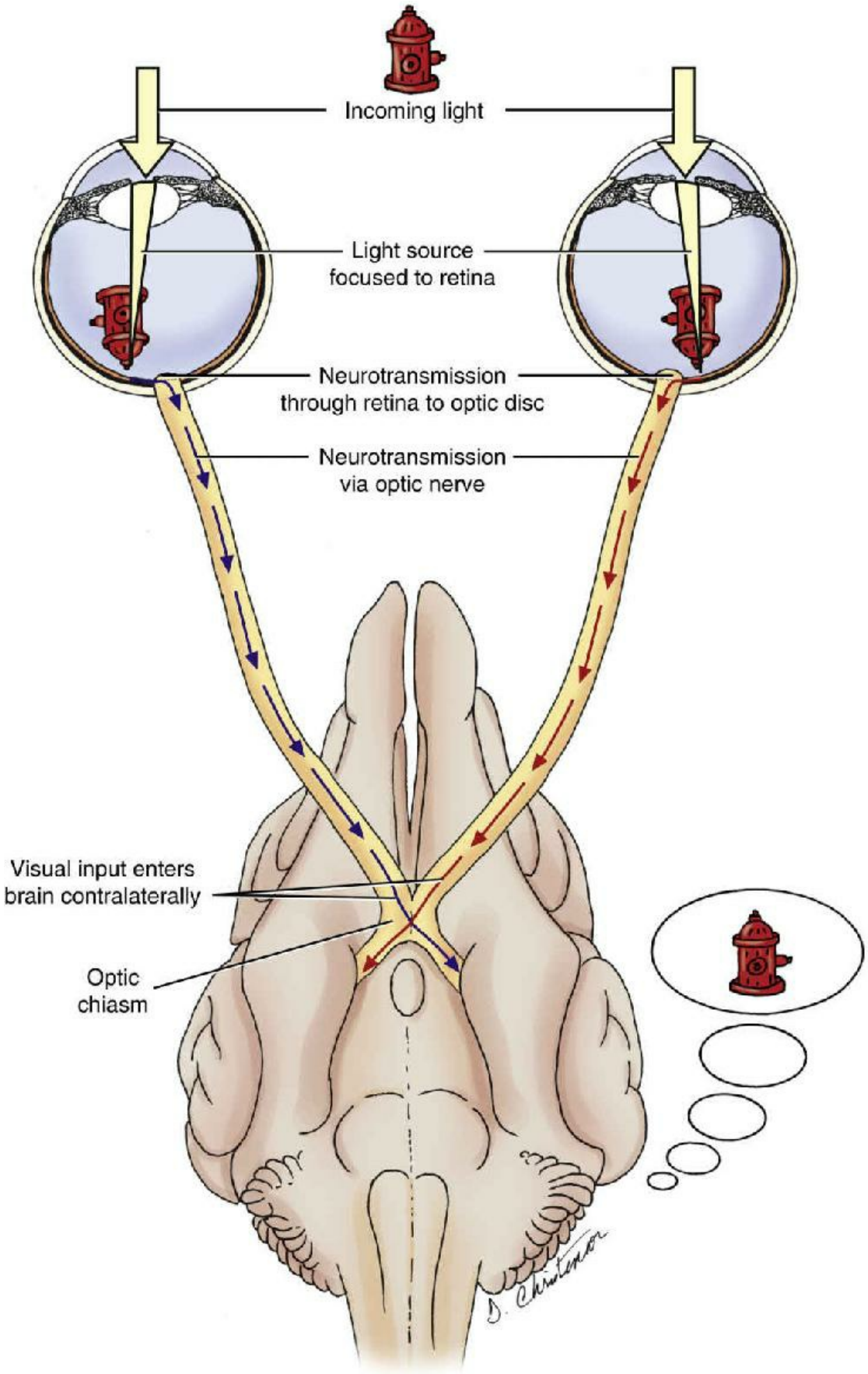

FIG. 11.27 Visual pathway schematic.

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Think about it. The *corneal ulceration* that we just talked about can impair vision. Like any injury, there will be inflammation with a corneal ulcer. But when the cornea becomes inflamed, edema results in opacity. *Corneal edema* makes it look bluish-gray, and it may make it slightly cloudy to completely opaque. Little light may get through. And even if it does, it will not be refracted as it should be. It could be like putting a sheet over your head—you can perceive some light but can't see anything but the sheet.

The whole purpose of the *cornea*, the *aqueous humor* in the anterior chamber, and the *lens* is to ***refract*** (bend) light. They need to be crystal-clear to do that. The cornea and aqueous humor refract the light source, concentrating it toward the pupil. And the lens provides the ultimate in focus. In a young, health animal, even though the crystalline lens material is rather firm, it is still flexible. It's just flexible enough, so that the ciliary muscles can alter the shape of the lens. How is that possible? Well, if you look at Fig. 11.26 again, you'll see tiny, thin tendons of sorts, called ***zonular*** [Gr. *zonula*, from *zona* a girdle] ***fibers.*** They connect the ***ciliary muscles*** to the ***lens capsule***. When the muscles contract and relax changing tension on the *zonular fibers*, the shape of the lens changes. That shape is constantly changing from plump and rounded to more ovoid and flattened. It really is as if the lens has a "girdle" helping it to "tuck its tummy" or to "let it all hang out." Each shape-shift alters how the incoming light source is critically focused on the ***fundus***. This is what provides ***visual acuity*** to see minute detail. If the lens or the lens capsule become opaque, as happens with ***cataracts*** [Gr. *katarraktes* waterfall; i.e., opaque, white, like the turbulent white water and froth seen at the base of a waterfall], light will not pass through appropriately, if at all. As the lens hardens ***(nuclear sclerosis)*** with aging, its shape cannot be altered enough for visual acuity. Fortunately, most animals don't need to read fine print (or boring text books like this). So, visual impairment from *nuclear sclerosis* isn't as limiting as what we experience. (Without my glasses, trifocals no less, I have trouble recognizing faces, let alone reading books or road signs!)

And then there is the neural tissue itself. ***Retinopathies*** [*retin(o)-* retina + *path(o)-* disease] directly and negatively impact vision. It's one thing to have poor focusing ability. But without the ***retina***,

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there is no vision. Yet we have to wonder, in the protected environment of the eye, how the retina could be damaged. Let's look at a few examples. We talked about chronic increased intraocular pressure crushing and destroying ***retinal*** [*retin(o)-* retina + *-al* pertaining to] neurons when we discussed *glaucoma*. If *neurotransmission* cannot be generated by the retina, we cannot see. ***Retinal detachment*** from ocular trauma or intraocular inflammation can occur. If those *photoreceptors* are not imbedded in the *choroid*, they lose their vascular support and ultimately can't be stimulated. And there are genetic conditions, such as ***progressive retinal atrophy*** [*a-* without + *troph(o)-* development, from Gr. *atrophia* wasting away] ***(PRA).*** An affected dog with *PRA* will progressively go blind. There is no stopping the disease. That's what makes testing for diseases like PRA so important, to ensure that they are not passed on to future generations. And reputable dog breeders work hard to maintain and improve the integrity of the breeds by not breeding affected animals, as well as by not breeding carriers to other carriers of diseases such as PRA.

Even if all of the ocular anatomy is healthy and intact, blindness can still develop. For example, we mentioned ***cortical blindness***. The ***visual cortex*** in the *occipital lobes* of the cerebrum is absolutely essential for vision. If those neurons are damaged, say by head trauma, blindness may result. (This is a good example of contralateral sensory input and interpretation. So, if trauma damages the right occipital lobe, the left eye will experience the blindness.) So, you see (pun intended), for any animal brought to us for visual impairment, we need to think about the plethora of *etiologies* that might result in vision loss. Then we must consider, is there anything we can do to "fix" the part of the visual pathway that's broken to restore sight? If *cataracts* are the problem, we can fix that by removing the opaque lens and putting in a new one. Cataract surgery is very common in people and animals. If vision can't be restored, blindness may not be the only issue for the animal. Vision needs to be coordinated with inner ear input, and proprioceptors to maintain balance and equilibrium.

Balance and Equilibrium

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When ***balance and equilibrium*** work well, life is good. When balance and equilibrium don't work, we have neurologic chaos. You know this if you suffer from motion sickness. In that simple example, there is a discontinuity of all of the sensory input—vision, proprioception, and function of the inner ear. But to understand that or even normal balance, you need to be familiar with the ***vestibular*** [*vestibul(o)-* vestibule, small chamber + *-ar* pertaining to] ***apparatus*** in the inner ear.

Vestibular Apparatus

There are actually two distinct, functional components of the ***vestibular apparatus***: the *vestibule* and the *semicircular canals*. The ultimate purpose of each is to provide sensory input that helps us to maintain balance. However, the type of sensory input is quite specialized for each. Let's look at the vestibule first (Fig. 11.28). In the left portion of the diagram, you can see that there are actually two tiny chambers in the ***vestibule***. The alignment of each is a bit different, but their fluid-filled interiors are quite the same (shown on the right side of the diagram). So, the inner walls of each chamber are lined with ***specialized receptor (hair) cells***. They are specialized to be stimulated by weight (i.e., the pressure exerted by weight). Over the surface of the sensory receptors is a sticky, gelatinous, mucoid sort of substance. Sitting on top and loosely stuck to the mucoid material are ***otoliths*** [*ot(o)-* ear + *lith(o)-* stone]. Yep, you read that right—stones (really a crystalline structure). Nevertheless, the term *otolith* means "ear stone." And that means we all have rocks in our heads.

Obviously, ***gravity*** will be the principal force influencing where the *otoliths* rest, right? And wherever they rest, their weight will exert pressure, stimulating the sensory receptors. So, if you're sitting up right now reading your book and your head is held vertically, the otoliths will slide down to the ventral part of the chambers. The receptor cells there are stimulated. That sensory information, carried off via the ***vestibulocochlear nerve*** to be interpreted by the *temporal lobes*, will indicate that you are upright. And you have input from other sensors in your body to corroborate that input (i.e., from your eyes and from proprioceptors in your buttocks). If you tilt your head to read because you placed the book

in your lap, the *otoliths* will slide where gravity dictates and stimulate receptors there. Again, *vision* and *proprioception* support the evidence that you are still upright, just tilted a bit. And based on interpretation of your body position, the *cerebellum* will coordinate whatever muscular activity is needed to keep you upright, so that you don't fall out of your chair. All of this is part of what we refer to as ***"static equilibrium."*** It's static because we're not moving.

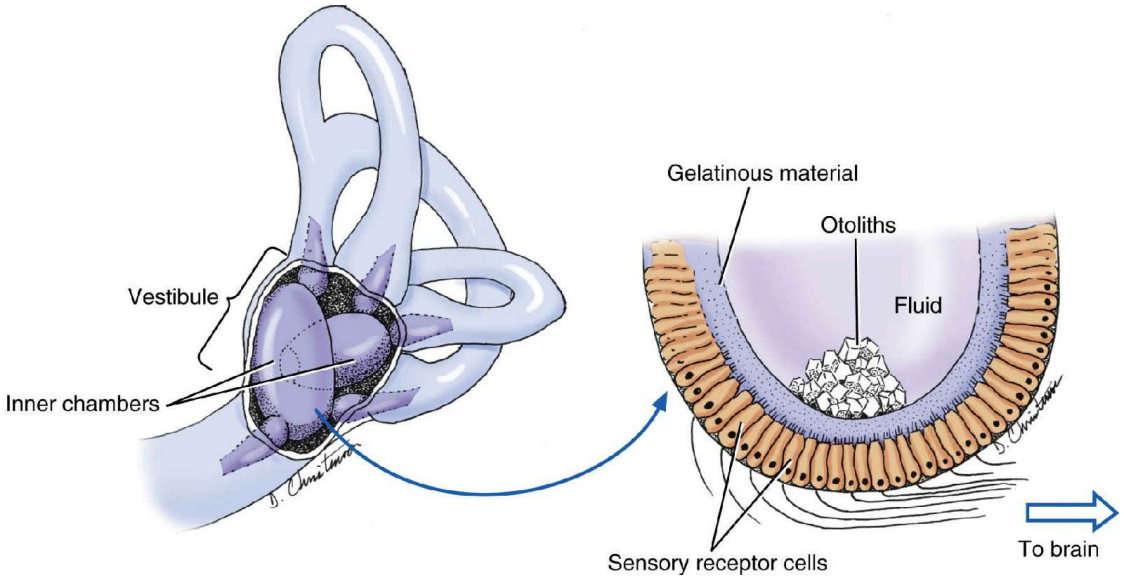

FIG. 11.28 Vestibule.

If you climb into your car to go to work or class, now you have different gravitational forces applied. How? When you accelerate, G forces (i.e., gravitational forces) of the acceleration will overcome standard gravity. The *acceleration* actually makes the otoliths slide from the bottom of the chambers (there when you weren't moving) to the posterior part of the chambers. The faster you accelerate the greater the impact on the otolith position and weight (pressure) exerted on the sensory cells. When you *decelerate* (slow down) as you approach a stoplight, the otoliths slide to the bottom of the chambers again, perhaps a bit anterior if you stop hard and fast. Again, coordinating this input with visual and proprioceptive input, your temporal lobes perceive that you are upright and moving. The sensory input is synchronous. Now, technically YOU are still not moving yourself. You're still just sitting there. So, this is still part of *static equilibrium*. It's technically the automobile that's

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moving.

Now, imagine a dog or cat in a crate in the back of an automobile, unable to see out of the windows. What does the *visual* and *proprioceptive* input indicate to that animal? They indicate that the animal is sitting still. As you drive down the road, what does the *vestibule* indicate? It indicates that the animal is moving, stimulated by the acceleration and deceleration forces. That discontinuity of sensory input creates chaos in the temporal lobes. And for those ultrasensitive to that discontinuity, they become nauseated. And if it's bad enough, they'll even vomit. It happens to me if I am a passenger in the back seat. If I can't see out, especially if I can't see straight ahead down the road, I will be extremely nauseated. That's one manifestation of ***motion sickness***. But that's not the only contributor. The other parts of the vestibular apparatus that can contribute to this are the semicircular canals.

If you look at Fig. 11.29, you can see the ***semicircular*** [*semi-* partial + *circul(o)-* circle + *-ar* pertaining to] ***canals***. Notice that there are three of them. Remember all of the body planes that we talked about, way back in Chapter 1? Well, each of the ring-like *semicircular canals* is oriented with the major body planes. If you're at all familiar with flying an airplane, the semicircular canals are the parts of our "instrumentation" (like a gyroscope [Gr. guros "a ring") that gives us information about pitch and roll movements. Notice that I said *movement*. That's what this part of the *vestibular apparatus* is designed to do—provide sensory input related to body *motion*. Motion is dynamic, and that's why the *semicircular canals* provide sensory input for ***dynamic equilibrium***. Warning: this concept is difficult to grasp for some folks. So, if you struggle with it, you're not alone. Let's take a closer look at the interior structure of the semicircular canals, to help us understand how the sensory input from motion is created.

You'll notice that each semicircular canal has kind of a fat area, where it connects to the wall of the vestibule. That "fat" area is called the ***ampulla*** [L. *ampulla* a jug]. The *ampulla* is where we find the *sensory receptor cell*. These receptor cells are stimulated by bending. How do we bend them? We do it by dragging them through the fluid that fills the semicircular canals. If you have ever gone swimming or dragged your hand in the water when sailing

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along in a boat or a canoe, you know that your hand was bent in the process. That's the functional principle used in the semicircular canals. Remember, the whole vestibular apparatus is completely encased in temporal bone. They can't move. They are simply along for the ride, wherever the skull moves. That means that when you turn your head, you are literally turning the structural canals around the fluid within them. I know, that's difficult to imagine. To prove how a structure can move around a fluid, try this experiment. Fill a bowl with water. Place an ice cube in the bowl. Now turn the bowl. The ice cube didn't move did it?—at least not at first. Now, repeat the experiment. This time, before you fill the bowl with water, find some of those little plastic 3M self-stick colored place-holders/tags. Stick a few to the inside wall of the bowl, so that the nonstick colored part of the tag sticks out away from the wall. You'll probably have to put a crease in it to accomplish this. Now fill the bowl with water. What happens to those self-stick tags when you turn the bowl full of water? They began to bend, didn't they? That is precisely how those special receptor cells in each *ampulla* are bent and stimulated. And it's a *reciprocal* bending and sensory input, from the right and left inner ears. The ***reciprocity*** of bending from the movement is how the cerebrum is able to interpret which way the head is turned.

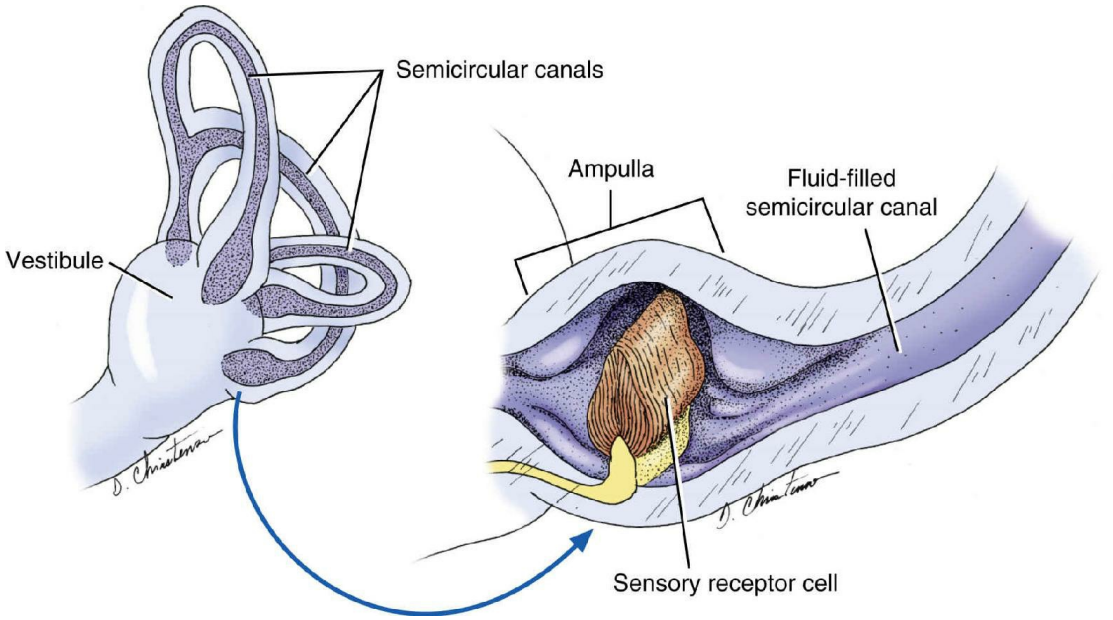

FIG. 11.29 Semicircular canals.

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Now, spin the bowl a few times in one direction. What happens when the bowl movement stops? Ah, the water continues to move, right? That's because of ***inertia***. And now what happens to those little tags? Oh, now they bend in the opposite direction, don't they? This would be very much like what happens if we close our eyes momentarily and spin ourselves quickly in one direction. When we stop spinning, we tend to lose our balance and fall down, right? But why? Let's see if we can figure that out. But we need to consider all of the sensory input for this. Let's see, by spinning, eventually the *fluid* in the *semicircular canals* began to move because of *inertia*. When we stopped spinning, the inertia of fluid movement bent the receptor cells in the opposite direction. The sensory input from that indicated to the brain that we were now spinning in the opposite direction. But, the *proprioceptors* in our feet and major joints of our legs indicated that we were standing still. Visually, our brain was told a similar story of standing still. Even the chambers in the *vestibule* indicated that we were standing still. That disconnect of sensory input creates chaos. *Cerebellar* coordination of motor responses to the disjointed sensory input will probably be overdone, causing us to look rather silly as we stumble around.

We can create other sensory disparities that lead to similar results. For instance, driving in a snow storm at night can create such a disparity. The snowflakes coming straight at us create overstimulation of the *visual* centers. *Proprioceptive* and *inner ear* inputs, on the other hand, probably indicate that we're standing still. In that kind of a snow storm, we're probably driving at a snail's pace. That too creates a disparity in sensory input (i.e., no acceleration or deceleration forces). Combine all of that and that's what people experience as ***vertigo*** [L. *vertigo* illusion of revolving]. It literally is as if you or your surroundings are spinning out of control. And that is very much what it is like for patients with vestibular disease.

Vestibular Disease

Vestibular disease is a relatively common disorder. I've known numerous people who have experienced it. People usually refer to it as "vertigo." And hearing my friends' experiences of the disease has been enlightening for me in understanding what my patients

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with vestibular disease are experiencing. In both people and animals, it is an *acute* (sudden) disorder, and I mean *acute*. People have described their experience as an abrupt change, literally turning their world upside down. "I was fine one minute, and then the whole room began to spin." Imagine how disorienting that would be. How does the body respond to disparities in sensory input? Well, it tends to react, overreact, in an attempt to maintain balance. Feet are set wide to keep from falling down. Jerky movements of the body occur, in an attempt to maintain balance, in response to the "moving world." Of course, the reality is that the world is not moving. So, those jerky attempts to "stay upright" often result in stumbling and falling down. The *ataxia* can be profound.

The eyes try to "spot," like ice skaters, to coordinate visual and inner ear input. And in vestibular disease, we see this as ***nystagmus*** [Gr. *nystagmos* drowsiness, nodding]. You know how it is when you're sitting up, really sleepy and begin to nod off, right? As you begin to dose off, you awaken and jerk your head up, right? That's how they came up with the word *nystagmus*. They eyes are literally "nodding," with a rhythmic jerking motion. Most often it's a side-to-side "bouncing" or jerking of the eyes *(horizontal nystagmus).* That is frequently associated with peripheral vestibular disease, involving the vestibular apparatus itself. *Vertical nystagmus* (bouncing up and down) tends to be associated with central (i.e., brain) ***pathology*** [*path(o)-* disease + *-logy* study, knowledge of]. Sometimes even ***rotary nystagmus*** is seen—the eyes are literally rotating around, as if the animal is watching a spinning object in front of its face. Of course, the *nystagmus* may not always be present. Sometimes a simple positional change, such as when we place an animal in lateral recumbency, will elicit the nystagmus. For that, we'll make note of ***positional nystagmus***. But if it's there from the get-go, we note ***spontaneous nystagmus***. And we also note whether it is horizontal, vertical, or rotary.

Now, think back to our discussion of *motion sickness*. But this time, the patient is not moving in the world. It simply perceives that it is moving in the world with a huge disparity in sensory input. These poor animals and people are usually extremely nauseated. We've all had the stomach flu and know how miserable it is to be

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profoundly nauseated to the point of vomiting. Imagine feeling like that and trying to navigate your way to the restroom, as well as trying to keep yourself upright over the toilet while your world is spinning. And the physical movement of trying to get there makes the "spinning" even worse. There is a high probability that you'll miss the target and fall into your own vomit. Animals don't necessarily try to hit a target, like we do. But they sure do often fail to remain upright and fall into their own vomit. It's a miserable feeling, to say the least. Friends who have experienced vestibular disease have described the slightest movements (e.g., lifting their head from the pillow or turning over in bed) as enough provocation to make the sensations of spinning profoundly worse. And that usually resulted in a powerful "wave" of ***nausea*** that often led to ***vomiting***. That knowledge has been very guiding for me, in caring for patients with vestibular disease. I am now very slow, methodical, and deliberate in my handling of these patients. And I've found that my slow, deliberate actions do help minimize exacerbation of those symptoms for them.

Okay, so that's what animals with vestibular disease experience. But how or why does all of it develop? Well, somewhere in the system, something is likely creating *inflammation*. If the inflammation is a central (brain) problem due to an *encephalopathy*, interpretation of and response to sensory input are disrupted. If the inflammation is peripheral in the *inner ear*, the sensory input itself is abnormal. The brain can only respond to the sensory input it gets. And whether the input or the perception of it is abnormal, the response is pretty much the same: *nausea*, *vomiting*, *ataxia*, and *nystagmus*. But wait, there's more. We may also see a ***head tilt***. Why? Think of the *vestibular apparatus* alone. If the chambers within the vestibule are inflamed, it may be perceived that the animal is standing on unlevel ground. So, to keep themselves "level" (or so their brain's think), they tilt their head and often lean. Head tilts are very common in vestibular disease—central and peripheral disease. Obviously, most central causes of vestibular disease will have a poorer *prognosis* than peripheral disease. So, we need to know the source. That means beyond our history, physical examination, and laboratory data, diagnostic imaging may be needed to conclusively rule out central *etiologies*, such as brain tumors. CT scans or MRIs

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are used for this. That imaging may or may not reveal evidence of peripheral disease.

Peripheral vestibular disease is the most common cause of all of the symptoms we've discussed. But "peripheral vestibular disease" is not a conclusive diagnosis. It's merely labeling the general place of origin of the symptoms. If we know the animal has been receiving potentially *ototoxic* medication, we may be able to link it to the medication. Diagnostic imaging won't show us anything in this case. But if stopping the medication resolves the symptoms, we'll have our answer. Imaging may help us in the case of *otitis interna* that develops from *otitis media*. In a case like that, we'll likely see abnormalities in the *tympanic bulla*. Honestly, we may never know the actual *etiology* of peripheral vestibular disease. In fact, that's often the case. That's why we often call it *idiopathic peripheral vestibular disease*. This is common in cats and in *geriatric* dogs. The first few days for them are the worst. After that most animals progressively improve. Vomiting, nausea, and anorexia (loss of appetite) diminish over the course of about a week. After a couple of weeks, many have returned to normal. Some animals may take a little longer, perhaps 3 weeks. And while most patients fully recover, I've seen a few dogs that have a persistent slight head tilt. But that's the exception rather than the rule. I've also seen some patients that experience persistent or periodic "motion sickness"-like symptoms. Those patients may be treated, as needed, with medications to relieve the nausea and vomiting.

Case Study

Frankie is a 13-year-old, spayed female, mixed breed dog. Her owners were quite frantic when they brought her in to be evaluated. Frankie fell down when she tried to get up from her bed. The owners reported that she thrashed around on the hardwood floor, unable to get back up. Frankie had been napping just prior to this event. We were uncertain, based on the limited history from her emotional owners, if Frankie experienced a seizure, acute quadriparesis, or an orthopedic injury. We approached our physical examination cautiously.

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We began our assessment with a neurologic examination. We would not want to create further trauma, for instance if there was a ruptured intervertebral disc. She had no spinal pain. All spinal reflexes were intact. We had difficulty placing Frankie in lateral recumbency to check her spinal reflexes because she thrashed wildly, especially when we attempted to turn her over. Her thrashing and resistance to our restraint immediately ruled out any possibility of myelopathy. Probably the thrashing the owners witnessed was not seizure activity as we suspected. But we needed to evaluate her further to rule out an encephalopathy. As we began to evaluate her cranial nerves, we noted that Frankie had hypersialosis and horizontal nystagmus. Pupillary light reflexes were normal, and there was no evidence of anisocoria. Facial sensation and motor function were intact. Visual, auditory, and olfactory abilities appeared to be intact. When we slowly helped Frankie into sternal recumbency, we noticed a significant head tilt to the left. We carefully lowered Frankie to the floor and supported her in a standing position on a rubber mat. Her stance was base-wide. As we attempted to encourage Frankie to walk, her severe ataxia caused her to fall. We caught her, preventing injury. Immediately after falling Frankie vomited, just missing the clinician's shoe. The owners reported that she did this in the car on the way to our facility too. The remainder of her physical examination was within normal limits.

All of Frankie's blood work was within normal limits. A CT scan ruled out obvious cerebral, cerebellar, diencephalon, mesencephalon, pons, and medulla pathology. Frankie has no prior history nor current physical evidence of otitis. So, the veterinarian has concluded that Frankie has geriatric onset idiopathic peripheral vestibular disease. Frankie was hospitalized for 3 days, receiving IV fluid therapy and medications to control her symptoms of nausea and vomiting. After 48 hours, her spontaneous nystagmus was reduced to positional. After 72 hours, she began eating and her ataxia seemed somewhat improved. She was sent home 4 days following her admittance, with a head tilt and mild ataxia. She was evaluated at weekly recheck appointments, showing improvement at each reevaluation. Three weeks later, Frankie was completely back to normal.

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Case Study Questions

1. Based on her initial history, there was a concern that because Frankie could not get up, she may have _____, or weakness in all four limbs.
2. Because spinal reflexes and Frankie's ability to use all four limbs to struggle against us, our rule-out of _____, or a disease of the spinal cord, was eliminated as a possibility.
3. We were less suspicious of seizure activity. However, we needed more clinical information to rule out a disease of the brain, medically termed _____.
4. Frankie did not have _____, or unequal pupils.
5. Frankie did have _____ (i.e., rhythmic jerking of her eyes).
6. Frankie's _____ ability, or sense of smell, was intact.
7. Frankie's _____ ability, or hearing, was intact.
8. Frankie had a lot of difficulty walking, due to her _____, or uncoordinated, stumbling gait.
9. A CT scan was done to rule out any pathology of the _____, or mid-brain, as well as other portions of the brain.
10. There was no evidence of _____ (inflammation of the ear) in Frankie's clinical history or on physical examination.
11. Frankie's disease was called _____ because the cause was unknown.
12. Because Frankie's problems stemmed from the inner

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ear apparatus that is composed of the semicircular canals and vestibule, the final diagnosis was that of geriatric-onset peripheral _____ disease.

The Answer Key to these case study questions may be found in Appendix B.

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Pain Management Information

American College of Veterinary Anesthesia and Analgesia (ACVAA; www.acvaa.org)
American Veterinary Medical Association (AVMA; www.avma.org)
American Animal Hospital Association (AAHA; www.aaha.org)
https://www.aaha.org/globalassets/02-guidelines/pain-management/2015_aaha_aafp_pain_management_guidelines_for_dogs_and_cat
University of Glasgow School of Veterinary Medicine (www.gla.ac.uk/schools/vet)
https://www.gla.ac.uk/schools/vet/sah/services/anaesthesiaandp

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[a] *Medusa* was a monster from Greek mythology. She was a winged human-like creature, with venomous snakes for hair. Anyone who looked at her face would turn to stone.

[a] www.michigan.gov, West Nile virus (WNV) in Michigan Emerging Disease Issues: Diseases that may affect humans or animals, Weekly Summary, Michigan 2018, November 5, 2018.

[b] www.cdc.gov, West Nile virus, Statistics and Maps, Preliminary Maps & Data for 2018, November 13, 2018.

[a] *Cujo*, a novel by Stephen King that became the basis for the movie *Cujo*, Paramount Pictures, 1983.

[a] Latimer KS. *Duncan and Prasse's Veterinary Laboratory Medicine Clinical Pathology*. 5th ed. Chichester: Wiley-Blackwell; 2001:355.

[a] The veterinary technician oath was first created and adopted by the National Association of Veterinary Technicians in America (NAVTA) in the 1980s; The entire oath reads: "*I solemnly dedicate myself to aiding animals and society by providing excellent care and services for animals, by alleviating animal suffering, and promoting public*

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health. I accept my obligations to practice my profession conscientiously and with sensitivity, adhering to the profession's Code of Ethics, and furthering my knowledge and competence through a commitment to lifelong learning."

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12

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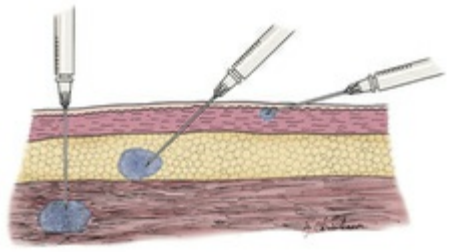

Applied Terminology for Pharmacology

Applied Terminology for Weights and Measures,
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- Weight,
- Volume,
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  - Ratio Solutions,
- Conversion Between Metric, Apothecary, and Household Measures,

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- 5 Rights of Medication Administration,
  - Right Patient?,
  - Right Drug?,
  - Right Dosage?,
  - Right Route?,
  - Right Time?,

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Applied Terminology for Pharmacodynamics and Pharmacokinetics,
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Types of Pharmaceuticals,
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  - Antibiotics,
  - Antimycotics,
  - Antiseptics and Disinfectants,
- Biologicals,
- Analgesics, Anesthetics, and Sedatives,
  - Agonists Versus Antagonists,
- Anti-Inflammatories,
  - Steroidal Anti-Inflammatory,
  - Non-Steroidal Anti-Inflammatory,
- Pesticides,
  - Insecticides and Acaricides,
  - Anthelmintics,
- Symptomatic Therapeutics,
  - Antipyretics,
  - Antitussives,
  - Antiemetics and Antidiarrheals,

Prescription Writing and Transcribing,
- Rx Practice Exercise #1,
- Rx Practice Exercise #2,
- Rx Practice Exercise #3,

Case Study,

GOALS AND OBJECTIVES

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By the conclusion of this chapter, the student will be able to:

1. Recognize common root words, prefixes, and suffixes related to pharmacology.
2. Divide simple and compound medical terms into their respective parts.
3. Recognize, correctly pronounce, and appropriately use common medical terms related to pharmacology.
4. Demonstrate an applied understanding of the metric system with regard to length, weight, volume, and conversions to apothecary and household measures.
5. Demonstrate an applied understanding of medication administration with regard to the “five rights.”
6. Demonstrate an applied understanding of medication administration with regard to relative rates of drug uptake per the various routes.
7. Demonstrate a basic understanding of pharmacokinetics and pharmacodynamics.
8. Demonstrate a basic understanding of common types of pharmaceuticals.
9. Demonstrate an applied understanding of prescription writing and transcription.

Important Note: *The purpose of this chapter is to familiarize you with the common terminology and abbreviations used in pharmacology. Dosage calculations, drug classifications, and specifics with regard to pharmacokinetics are not covered because these topics stray from the intent of this text. It is my hope that the tables and descriptions will provide valuable resources for you elsewhere in your studies. I strongly recommend that you complete all other chapters before this one. If you do this, you will find the background knowledge and redundancies of terms and word parts extremely helpful.*

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Applied Terminology for Weights and Measures

There are a number of ways to measure things. In the kitchen, we often use household measures of teaspoons, tablespoons, cups, and pints. We even use ***apothecary*** (uh-poth'ĕ-ka"re [Gr. *apotheke* "storehouse"; pharmacy] measurements in the kitchen, like pounds and ounces. While we may convert measurements of medications to household and apothecary units, for the sake of owner convenience and compliance, the preferred system of measurement in ***pharmacology*** [*pharmac(o)-* drug, pharmacy + *-logy* study of] is ***metric***.

Why? The metric system provides a clean decimal system of measurement. Within this system, everything is multiplied or divided by factors of 10. The prefixes used to indicate the decimal equivalents are standardized across the metric system. It doesn't matter if you're measuring length, weight, or volume, the same prefixes are used. This provides ease of use—that is, once you're familiar with the prefixes (Table 12.1). Commit them to memory once and you've got the whole metric system covered! I believe the metric system also provides precision, unlike any other unit of measure. And when we're dealing with medications, precision is very, very important.

Length

In metrics, the basic unit for measuring length is the ***meter*** (roughly 41 inches, if you want to visualize that). We don't generally apply measurements of length to medications. Occasionally, a ribbon of ointment may be applied using a length measurement. But this is the exception rather than the rule. Generally, measurements of ***millimeters*** [*milli-* one thousandth of a *meter*] and ***centimeters*** [*centi-* one hundredth of a *meter*] are used for measuring wounds, tumors, or other types of lesions. If you remember our brief discussion of diagnostic ultrasound in Chapter 1, we talked primarily about how structures appeared (i.e., light vs. dark, etc.). We often measure

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structures in sonographic images, including the various layers of some of those structures, in millimeters and centimeters. In the laboratory, we often measure things under the microscope in ***micrometers*** [*micro-* on millionth + *meter*]. A *micrometer* is also called a *micron* (μ). So, you see, metric measurements of length are used frequently in veterinary medicine. We simply don't use this in pharmacology much, if at all.

Weight

In metrics, the basic unit for measuring weight is the ***gram***. In pharmacology, most drug dosages are given as so many grams, ***milligrams*** [*milli-* on thousandth + *gram*] or ***micrograms*** [*micro-* one millionth + *gram*] of drug per ***kilogram*** [*kilo-* one thousand + *gram*] of body weight. Wait, you say that your scale only weighs in pounds? No problem. You can easily convert pounds to kilograms by dividing the weight in pounds by 2.2.

It goes without saying that there is a huge difference between a *milligram* (mg) and a *microgram* (mcg). Most drugs tend to be dosed in mg/kg. But there are some drugs, like dexmedetomidine—a powerful sedative—that are dosed in mcg/kg. Yet, the concentration on the bottle is in milligrams. In order to calculate the final volume of the drug, we need both the dose and concentration expressed in the same unit of measure. So, how on earth do we get from point A to point B? We can't afford to make a mistake with this drug, because we could easily kill the animal. The relationship between each unit of weight is the key. And how we figure out the relationship is by looking at the terminology. If a milligram is one thousandth of a gram, then there are 1000 mg in every gram. If a microgram is one millionth of a gram, then there are 1,000,000 mcg in every gram. So, micrograms are smaller than milligrams by a factor of one thousand, right? Right. That means there are 1000 mcg in every mg. Now we have something we can work with. Hypothetically, let's say that you calculate the dosage of dexmedetomidine for a patient to be 50 mcg. Unfortunately, you find that the concentration of the drug is 0.5 mg/mL. How can we use our relational knowledge of these units to change the 0.5 mg/mL to micrograms per mL? If there are 1000 mcg for every 1

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mg, then what if we simply multiply 0.5 mg by 1000? That reveals 500 mcg/mL. With both the concentration and the dosage expressed in micrograms, we can finish calculating the volume of drug to be administered. Why go through all of this trouble? Because we can't afford to make a mistake. It is essential to maintain the same unit of measurement for both the dosage calculated and the concentration, to avoid deadly errors. To do that, you need to remember the relational difference between the two units of measure. To help you with the typical units of metric weight used in pharmacology, take a look at Table 12.2.

TABLE 12.1

Metric Prefixes

Prefix	Phonetics	Abbreviation	Decimal Value	Scientific Notation	Meaning
tera	ter'ah	T	1,000,000,000,000	10^{12}	Trillion
giga	jĭ'gah, gig'ah	G	1,000,000,000	10^{9}	Billion
mega	meg'ah	M	1,000,000	10^{6}	Million
kilo	**kil'o, ke'lo**	**k**	**1,000**	**10^{3}**	**Thousand**
hecto	hek'to	h	100	10^{2}	**Hundred**
deka	**dek'ah**	**dk**	**10**	**10^{1}**	**Ten**
deci	**des'ĭ**	**d**	**0.1**	**10^{-1}**	**One tenth**
centi	**sen'tĭ**	**c**	**0.01**	**10^{-2}**	**One hundredth**
milli	**mil'ĭ**	**m**	**0.001**	**10^{-3}**	**One thousandth**
micro	**mi'kro**	**µ or mc**	**0.000 001**	**10^{-6}**	**One millionth**
nano	nan'o	n	0.000 000 001	10^{-9}	One billionth
pico	**pi'ko**	**p**	**0.000 000 000 001**	**10^{-12}**	**One trillionth**
femto	**fem'to**	**f**	**0.000 000 000 000 001**	**10^{-15}**	**One quadrillionth**
atto	at'to	a	0.000 000 000 000 000 001	10^{-18}	One quintillionth

Do we ever use smaller units of weight than micrograms? Fortunately, in pharmacology, we don't. But we do use smaller units of measurement in the laboratory. For example, the mean corpuscular hemoglobin (MCH) is reported in ***picograms*** [*pico-* one trillionth + *gram*]. You thought micrograms were small. *Picograms* (pg) are puny! Aren't you glad that you don't need to calculate drug dosages with these?

Volume

In metrics, the basic unit for measuring volume is the ***liter*** (L). You're somewhat familiar with this, with the two-liter bottles of

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soda in the grocery store. Generally, we don't deal with such large volumes for medications. Now, ***intravenous*** [*intra-* within + *ven(o)-* vein + *-ous* pertaining to] (IV) fluids come in liter bags, and even 3 L and 5 L bags for fluid therapy. But most medications are not delivered in such large volumes. Most of the time, we deliver liquid medications in ***milliliters*** [*milli-* one thousandth + *liter*] (mL). Don't let the one thousandth confuse you. If it helps, just remember that there are 1000 mL in every liter. Table 12.3 has the metric volume equivalencies for those units typically used in pharmacology. By the way, 1 mL is the same as 1 cc.

Do we ever deal in smaller volumes? You bet! But we don't tend to use volumes like ***microliters*** [*micro-* one millionth + *liter*] in pharmacology. But we do use *microliter* (μL) measurements in the laboratory. It's not unusual at all to measure blood, plasma, or serum in μL for various diagnostic tests. Yes, we have special pipettes to measure such small volumes. Come to think of it, the laboratory is the one place that uses quite a few small volumes for reporting purposes. In hematology [hemat(o)- blood + -logy study of], the mean corpuscular volume (MCV) is reported in ***femtoliters*** [*femto-* one quadrillionth + *liter*]. That's small! Still other laboratory values, like the hemoglobin (Hgb), are reported in grams (g) per ***deciliter*** [*deci-* one tenth + *liter*] (dL).

TABLE 12.2

Metric Weights

Weight	Symbol	Equivalency
1 kilogram	1 kg	1,000 grams
1 gram	1 g	1,000 mg; (1/1,000 kg)
1 milligram	1 mg	0.001 gram; 1/1000 gram; (1,000 mcg)
1 microgram	1 mcg	0.000,001 gram; 1/1,000,000 gram; (1/1,000 mg)
1 picogram	1pg	0.000,000,000,001 gram; 1/1,000,000,000,000 gram

TABLE 12.3

Metric Volumes

Volume	Symbol	Equivalency
	1 L	1,000 mL

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1 liter		
1 deciliter	1 dL	0.1 liter; 1/10 liter; 100 mL
1 milliliter	1 mL	0.001 liter; 1/1,000 liter
1 cubic centimeter	1 cc	0.001 liter; 1/1,000 liter
1 microliter	1 μL	0.000,001 liter; 1/1,000,000 liter
1 femtoliter	1 fL	0.000,000,000,000,001 liter; 1/1,000,000,000,000,000 liter

Whether in the laboratory or the pharmacy, metric measurements are the same. And understanding one volumetric measurement compared to another is a matter of understanding the meaning of the prefixes used and their relationship to one another. Let's take that last volume mentioned in the previous paragraph: *deciliter*. Relationally, we know based on the prefix that a deciliter is one tenth of a liter. But how might we express a deciliter in milliliters? There are a number of ways we could try to figure this out. Perhaps the easiest way would be to use what we already know. We know that there are 1000 mL in every liter. If 1 dL is one tenth of a liter, then we could simply multiply 1000 mL by 1/10. That gives us a fraction of 1000 mL over 10. Cross out a zero from each of those numbers and we are left with 100 mL. So, 100 mL is equivalent to 1 dL. How easy was that?

Concentration

How the concentration of a medication is expressed depends on the form of the medication. For instance, the concentration of tablets and capsules are generally expressed as milligrams per tablet or capsule. If you're looking at a bottle of Ampicillin, a common ***antibiotic*** [*anti-* against + *bi(o)-* life + *-tic* pertaining to], you may see the following: Ampicillin 250 mg capsules. Other manufacturers may label a drug as 10 mg/tablet or 10 mg tablets.

Solutions are a bit different. Here the concentration is expressed as weight over volume. A word of caution: not all manufacturers make this information easy to find or easy to understand. Most often a medication solution will be expressed as a number of milligrams per milliliter (mg/mL). For example, Acepromazine, a common tranquilizer, is produced in a concentration of 10 mg/mL. Sometimes a manufacturer will express a particular drug's

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concentration as 100 mg/10 mL. If we misread the volume portion of that concentration as one mL, our dosing of the drug will be wrong. So, you need to pay close attention when reading the concentration on the bottle. Remember our dexmedetomidine example earlier? We don't want to make deadly errors in dosing. So, be vigilant.

Percent Solutions

Percent [*per-* for each + *cent* hundred] solutions are merely a variation on a theme. This is still a matter of weight per volume. You simply need to know how to interpret it because it looks a little different. As an example, furosemide is a drug used to stimulate the kidneys to produce urine. Most often, furosemide is found in a 5% solution. How do we interpret 5% as weight versus volume? Well, we need to remember that the word percent means "for each hundred." Generally, the number given indicates grams. The volume is always based on 100 mL ("for each hundred," remember?). So, our 5% solution is interpreted as 5 grams per 100 mL.

But if you need to perform a dosage calculation using milligrams per milliliter, how do you get there? It's easier than you might think. Look at Table 12.1. The prefix *milli-* means one thousandth, right? Looking at it another way, we can say that there are 1000 milligrams per gram. So, with our 5% solution, what if we simply change the 5 grams to 5000 milligrams? Now we have the same prefix for weight and volume (milli-). If we set this up as a fraction, with weight on top and volume on the bottom, we can simply cross off zeros until we are left with a single mL on the bottom. It might look something like this to start: $\frac{5000\text{mg}}{100\text{mL}}$. Then, we cross off some zeros: $\frac{50\cancel{00}\text{mg}}{1\cancel{00}\text{mL}}$. What we are left with, after crossing off those zeros, is 50 mg/mL. And that is precisely the concentration of a 5% solution. See? How easy was that?!

Ratio Solutions

There are some drugs, like the emergency drug epinephrine, that are expressed as ratios. In fact, a typical concentration of

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epinephrine is 1:1000 (read that as one to one thousand). Here again, if we are going to calculate the volume of this emergency drug to be delivered, we need to express the dosage and the concentration in the same unit of measurement. So, how do we interpret that ratio of 1:1000? The first number represents grams. The second number of the ratio represents milliliters. So, a 1:1000 concentration is literally interpreted as 1 gram per 1000 milliliters. If you look up epinephrine in a ***pharmaceutical*** [*pharmaceutic(o)*- drug + *-al* pertaining to] reference, you will find that it is dosed in mg/kg. So, to complete our dosage calculation, we need to express the concentration of the drug in mg/mL. How do we do that? Again, it's about relationships. A milligram is one thousandth of a gram, meaning that there are 1000 mg in every gram. So, let's take the 1:1000 concentration and express it first as 1 g/1000 mL. Then let's multiply the 1 g by 1000 to give us milligrams. Then we find that we have 1000 mg/1000 mL. With this, all we need to do is cross off zeros. There's no rocket-science here to figure out that there is 1 mg/mL in that bottle of epinephrine. So, why on earth do they label the drug with a ratio? Why not simply put the mg/mL big and bold on the label, instead? I wish I had the answer for you. I doubt we'll facilitate a change in the manufacturer's labeling. So, that puts the burden on us to know how to interpret the meaning of the ratio (grams per volume) on the label *and* know the relationships between the various units of measure.

Conversion Between Metric, Apothecary, and Household Measures

The metric system is understood and used throughout the world. In fact, in most other countries[a] around the world, outside the United States, the metric system is the exclusive system of measurement used. It is the universal system of measurement in medicine. Unfortunately, not everyone is familiar with it. Many of our clients are more familiar with apothecary and household forms of measurement. So, for the sake of owner convenience and compliance, we may need to convert the dosing of medications to more familiar forms of measurement for them.

Think about it. How many people know their own weight or the

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weight of their pets in kilograms? Probably very few. This may be, in part, due to the fact that most U.S. scales provide weight in pounds not kilograms. And since most medications are dosed in milligrams per kilogram, we need to be able to convert pounds to kilograms. Let's say, hypothetically, that we have calculated the final volume of an oral liquid medication in milliliters. You say: "no problem. We'll simply send a syringe home with the owner." Not so fast. What if the owner is elderly, with such arthritic hands that she can't draw back on the syringe plunger without great difficulty? What if her vision is poor, such that she can't read the tiny markings on the barrel of the syringe? Do you know what's going to happen? That owner probably either won't give the needed medication or won't give the correct volume of it. And that is not in our patient's best interest. An owner like this will likely be more compliant and accurate if he or she can use a more familiar measuring device. So, there are times when converting from metric to more familiar forms of measure will be best for everyone. Let's say the volume of drug to be given is 5 mL. We could easily convert that to 1 tsp. If that makes life easier for the owner and helps ensure that the animal gets the medication, why not do it?

TABLE 12.4

Metric, Apothecary, and Household Equivalencies

Metric	Apothecary	Household
Weight		
1 kg	2.2 lb	2.2 lb
453.6 g	16 oz	1 lb
~30 g (31.1 g)	1 oz	
1 g	~15 gr (15.4 gr)	
~65 mg	1 gr	
~16 mg	1/4 gr	
~0.5 mg	1/120 gr	
Volume		
3.8 L	128 fl oz	1 gal (4 qt)
946.3 mL	32 fl oz	1 qt (2 pt)
473.2 mL	16 fl oz	1 pt
~240 mL (236.6 mL)	8 fl oz	1 c
~30 mL	1 fl oz	2 tbsp
~15 mL	0.5 fl oz (4 fl dr)	1 tbsp (~3 tsp)
~5 mL	1 fl dr	1 tsp

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Note that approximate (~) weights and volumes in the table above are commonly used for calculation purposes. Actual equivalencies follow some approximations in parentheses.

Can we always easily convert to household or apothecary measure? No. Sometimes the amount of medication to be given is way too small. My cat, for instance, was receiving 0.4 mL of methimazole orally for her ***hyperthyroidism*** [*hyper-* excess + *thyroid(o)-* thyroid + *-ism* condition of; i.e., an overproductive thyroid gland]. There is no way for us to convert that small volume to household measure and deliver it accurately. So, we stick with the metric volume. I won't belabor this. Table 12.4 has been provided to give you a quick reference for these conversions. You never know when you might need it.

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Applied Terminology Administration of Medications

This section is designed to provide you with generalized knowledge about medication administration. For learning how to safely administer medications, via the various routes, you will need to rely on other resources and courses in your veterinary education. But this will give you a good foundation to build on.

5. Rights of Medication Administration

Before we give any medication to a patient, there are five questions we should always, and I mean *always*, ask ourselves. Do I have the right patient? Is this the right drug? Is this the right dosage? Am I giving it via the right route? And is this the right time for it to be given? It is our professional responsibility to protect the welfare of each and every patient in our care. By asking these questions, we provide a series of checks to prevent mistakes. Mistakes in medicine are costly … they cost lives at times. We cannot afford to put the lives of our patients at risk. That is why we follow the five rights of medication administration. Even after all these years of being in veterinary medicine, I still follow the "five rights." Nobody's dying on my watch, not if I can prevent it.

Right Patient?

This seems so logical, doesn't it? Yet, in human and veterinary medicine alike, medications are given to and surgical procedures are performed on the wrong patients all the time. In veterinary medicine, we have significant species variations and even breed variations that put patients at risk from drug intolerances. When I think of sight hounds, like Greyhounds, Whippets, and Borzois, with their lean body type, they cannot tolerate certain types of ***anesthesia*** [*an-* without, no + *esthes(o)-* sensation + *-ia* condition of]. Barbiturate ***anesthetic*** [*an-* without, no + *esthet(o)-* sensation + *-ic* pertaining to] agents can be lethal for sight hounds. Many of the topical ***insecticidal*** [*insect* + *cid(o)-* kill, killing + *-al* pertaining to]

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agents labelled for use in dogs can be lethal for cats. As a breed, Collies cannot receive ***anthelmintic*** [*ant-, anti-* against + *helmin(o)-* worm + *-tic* pertaining to; i.e., dewormer] agents containing ivermectin as the active ingredient. That ingredient can be deadly for them.

What if an animal is given or inadvertently consumes a drug that could be potentially ***toxic*** (poisonous)? Depending on the drug that is consumed, we might be able to give an ***emetic*** [*eme(o)-* vomiting + *-tic* pertaining to] agent to induce ***emesis*** [*eme(o)-* vomiting + *-sis* process of]. This needs to be done quickly, before the drug is absorbed. We might be able to wash off a topical agent to minimize absorption. But injectable medications cannot be taken back. This can have very serious consequences for patients receiving the wrong medication. What if we have two domestic shorthair cats in the hospital? One is being treated for a minor injury and scheduled to go home later that day. The other is in end-stage ***renal*** [*ren(o)-* kidney + *-al* pertaining to] failure and is to be ***euthanized*** [*eu-* good + *than(o)-* death + *-ize* process of; i.e., humanely killed] that day. If we do not ensure that we have the right patient, we may euthanize the wrong cat. It happens. That is a catastrophic ***iatrogenic*** [*iatr(o)-* physician + *gen(o)-* producing + *-ic* pertaining to] consequence. We can't take it back. We can't fix it. It is an error that should not be made. Do I make my point clear? Good.

Right Drug?

There are a number of factors that go into ensuring that we have the right drug for our patient. First, we need to verify the veterinarian's drug order. We should compare the drug order to the drug label. We should evaluate that label for the drug name, concentration, and the expiration date. We should evaluate the contents of the container for contamination or spoilage. And we should check to see that we have the right form of the drug. Some drugs, like ***antibiotics*** [*anti-* against + *bi(o)-* life + *-tic* pertaining to; i.e., drugs used to inhibit or kill bacteria], come in oral and injectable forms. Table 12.5 provides common prescription abbreviations for the various forms of medications. Which form of the medication did the veterinarian order? Is that what you have in your hand?

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TABLE 12.5

Medication Form Abbreviations

R_x Abbreviation	Meaning
aq	Water [L. *aqua*]
cap	Capsule
elix	Elixir
emuls	Emulsion
ext	Extract
mixt	Mixture
supp	Suppository
susp	Suspension
syr	Syrup
tab	Tablet
tinct	Tincture [L. *tinctura*]
ung	Ointment [L. *unguentum*]

Are you unfamiliar with the medication? Then look it up in a ***pharmaceutical*** reference or the manufacturer's insert. This is important for veterinarians and veterinary technicians alike. No, credentialled technicians cannot prescribe medications. Still, we need to be familiar with the medications we administer, to know how they work as well as their side effects. Who will be responsible for monitoring for the side effects? Most likely the veterinary technician who administered the medication. That was my duty when I worked in Emergency and Critical Care Medicine. Believe me, I received all kinds of drug orders, from multiple doctors, interns, and residents, for a large number of patients in the ICU. I had to make sure that I was not only giving the right drug to the right patient, but that I knew what to look for in terms of appropriate and adverse effects in my patients.

We used the example of dexmedetomidine earlier. As I said, this is a commonly used sedative. It comes in two different concentrations: 0.5 mg/mL and 0.1 mg/mL. Remember, I said that this drug is usually dosed in micrograms per kilogram. The volumes given are usually tiny. It doesn't take much of this drug to heavily sedate a patient. Did I mention that this drug significantly slows the heart rate? What if I were to calculate a final dose of 10

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mcg for a patient? What if I calculated the volume to be given based on the lesser concentration of the drug (0.1 mg/mL)? And what if I grabbed the wrong bottle, drawing up that volume from the high concentration (0.5 mg/mL)? I could stop that patient's heart. And that death would be my fault entirely. This could be avoided, if I were to simply pay attention and make sure that I have the right drug in my hand when I draw it up.

There were times when we were slammed with emergency and critical cases. The chaos exponentially increased the possibility of medical errors. That's why my fellow technicians and I had to be vigilant in our patient care and medication administration. During those crazy times, when our doctors were frantically trying to juggle orders for diagnostics and therapeutics, they occasionally made errors. Chaos does that. Those errors could have been made because the doctors were thinking about the wrong patient when giving the drug order. The errors could have been because the doctor was not aware of changes in the patient's condition. Both of these examples could result in ***contraindications*** [*contra-* opposed, against + *indicate* advise + *-tion* to make; i.e. to make inadvisable] for the drug ordered. I had to be vigilant and question any drug orders that I perceived to be *contraindicated* for my patients. This is not disrespectful. This is about teamwork. I am my patient's advocate. And I am the final responsible person of our veterinary medical team to ensure that the right drug is given to the right patient. If I fail in my duty here, my patients may die. Not on my watch.

TABLE 12.6

Medication Unit Abbreviations

Abbreviation	Meaning	Abbreviation	Meaning	Abbreviation	Meaning
kg	kilogram	gal	gallon	gt	drop [L. *gutta*]
g, gm	gram	qt	quart	gtt	drops [L. *guttae*]
mg	milligram	pt	pint	U	unit
mcg, µg	microgram	fl oz	fluid ounce	lb, #	pound
L	liter				
cc	cubic centimeter	fl dr	fluid dram	oz	ounce
ml, mL	milliliter	TBL, tbsp	tablespoon	gr	grain
mEq	milliequivalent	tsp	teaspoon	dr	dram

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Right Dosage?

Okay, I am going to sound like a broken record. To ensure that I am giving the right dosage of a medication, I need to verify the veterinarian's drug order. Was the drug ordered in milligrams per pound or milligrams per kilogram? What is the recommended dosing for this medication by the manufacturer? Are there species variations in dosing? Sometimes the same drug may be dosed higher in cats and lower in dogs or vice versa. Sometimes, as I pointed out in the previous section, drugs come in various concentrations. Am I giving the correct dosage based on the concentration in my hand? There is a huge difference between a 100 mcg/mL solution of dexmedetomidine versus a 500 mcg/mL solution. (See? It looks like a more dramatic difference when expressed in micrograms, compared to 0.1 mg/mL and 0.5 mg/mL, doesn't it?) Did I calculate using the correct units? Table 12.6 provides common units used in medication dosing. Remember, you may need to convert from one unit to another, as in our dexmedetomidine example. Double check to ensure that your conversion is correct. Acepromazine is another widely used sedative in dogs in cats. It too may be found in different concentrations. Believe me, there is a huge difference between giving 1 mL of a 1% solution of acepromazine versus 1 mL of a 2 mg/mL solution. (Did you figure that out? A 1% solution contains 10 mg/mL of the drug.)

Once you've drawn up an injectable medication, look at it. Double check your calculations. Ask yourself: "Does this volume make sense?" Seriously, if you're about to give 10 mL of a medication to a 3-pound kitten, all sorts of red flags, bells, and whistles should go off in your head. Ask a colleague to double check it for you. I did that, even after nearly 40 years in the profession. One calculation error, moving the decimal point the wrong way, can kill a patient. That patient is way more important than your pride. And I would be way more concerned about my professional reputation and liability in a malpractice suit if the patient dies due to my error, if I were you. Mistakes happen. I still recall a case where a student nearly gave 100-times the actual dose of a drug to a cat, all because of a calculation error. Instead of drawing up the correct 0.12 mL of the drug, the student incorrectly

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drew up 12 mL. That could have easily killed the cat. Fortunately, a colleague caught the error before it was too late. How did such a huge calculation error happen? The student was unfamiliar with percent solutions and did not know how to interpret the 5% concentration on the drug's label. If you're ever uncertain, unfamiliar, or confused, ask an experienced professional or look it up. Don't put a patient in jeopardy.

What if an oral drug has been ordered but the calculated dosage doesn't match the size of the tablets or capsules available? Some tablets can be split to accommodate dosing issues like this. But ***enteric*** [*enter(o)-* intestine + *-ic* pertaining to] coated tablets should not be split. The *enteric* coating is designed to protect the stomach and control the absorption rate. If we split one, we can dramatically speed up the absorption rate. We may also, depending on the drug, irritate the stomach lining. Capsules absolutely cannot be split. If we give the tablet or capsule concentration below the calculated dose, we could significantly underdose the patient, rendering our treatment useless and jeopardizing the patient's health. If we give the tablet or capsule concentration above our calculated dose, we could significantly overdose the patient jeopardizing its health. So, what do we do if our calculated dosage doesn't match the available drug? Well, consult with the ordering veterinarian. He or she may be able to alter the dosage a little, to accommodate the available drug concentration. Or the doctor may simply choose a different drug. Ask. Question. This is all about your patient, and your patient should always come first.

Right Route?

This is redundant, but always verify the veterinarian's drug order. Which route was ordered? (Table 12.7 shows you the common routes of medication administration, with their abbreviations and meanings.) Is the route ordered appropriate for the patient? Obviously, a vomiting patient shouldn't receive a drug per os (PO, "by mouth"). Does the doctor know that the patient is vomiting now? If not, inform them. Is the route ordered appropriate for the medication? Some drugs should never, ever be given IV. Others should never be given IM (*intramuscular* [*intra-* within + *muscul(o)-* muscle + *-ar* pertaining to]). Sometimes there are volume limitations

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for the prescribed route in a given patient. It's one thing to give 10 mL of drug IM to a horse, but we sure can't give that volume IM to a Chihuahua! Sometimes, when the doctor orders a particular drug to be given IM, it turns out that the calculated volume to be given is way too much for the patient. When that happens, I consult with the ordering veterinarian. If possible, they may choose a different route for the medication. If that's not possible, they may have to select another drug.

TABLE 12.7

Route of Medication Administration Abbreviations

R_x Abbreviation	Meaning
AD	Right ear [L. *auris dextra*]
AS	Left ear [L. *auris sinistra*]
AU	Both ears [L. *auris uterque*]
ID	Intradermal
IM	Intramuscular
IO	Intraosseous
IP	Intraperitoneal
IT	Intratracheal
IV	Intravenous
OD	Right eye [L. *oculus dexter*]
OS	Left eye [L. *oculus sinister*]
OU	Both eyes [L. *oculus uterque*]
PO	By mouth [L. *per os*]
PR	By rectum [L. *per rectum*]
SQ, SC	Subcutaneous

There have been many times that I have had difficulty reading the drug orders. Penmanship is pretty poor when I can't clearly discern "IM" from "IV." I'm telling you, I have seen written orders where it was impossible to tell the difference between IM and IV, let alone be able to make out the drug name itself, or discern a 6 from an 8 or 0. What did I do? I questioned the ordering doctor. I needed clarification in order to ensure that I was giving the right drug to my patient, at the right dosage by the correct route. If I can't be certain of all those things, I jolly-well shouldn't be giving the

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medication.

Routes of administration

Let's take a brief look at some of the most common routes of medication administration. Again, I won't be telling you how to administer medications via each of these routes. I will simply be sharing some insights into the value of each, including ***indications*** [*indicate* advise, advisable + *-tion* to make; i.e., advisable circumstances] and *contraindications* (inadvisable circumstances), as appropriate.

Enteral

The ***enteral*** [*enter(o)-* intestines + *-al* pertaining to] routes of medication administration are probably the most widely used, especially for drugs given by owners at home. There's a saying: "if the gut works, use it!" That's because it works so well for nutritional needs and medication delivery. Of course, generally owners give enteral medications ***PO*** [L. *per os* by mouth). But there are circumstances when they may need give meds ***PR*** [L. *per rectum* by rectum]. Seriously?! Seriously. There are many owners of seizure patients who are instructed to give a "rescue" dose of an ***anticonvulsant*** [*anti-* against + *convulse* seizure + *-ant* one that], like diazepam or phenobarbital, to their pet PR in the event of a grand mal seizure. Believe it or not, the absorption rate from the rectum is actually very good. This emergency dose of PR medication at home may actually stop the seizure activity and make it possible for the owner to safely transport the pet to their veterinarian.

Obviously, PO is the most common route to administer *enteral* medications. The challenge for owners and veterinary professionals alike is getting the animal to take it and swallow it, without spitting the drug back out. And if the drug tastes awful, good luck with that! Fortunately, many manufacturers and compounding pharmacies produce oral solutions and suspensions that are very palatable. The compounding pharmacy that made my cat's methimazole suspension made it taste great. She licked it up, like a kid licking an ice cream cone. She probably wouldn't have gotten the drug if it tasted bad. She was one cat I did not want to tangle with. In fact, I told my spouse many times that if Bobbie (the cat)

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ever needed to be hospitalized in ICU, I did *not* want to be on duty. Bobbie had a very short fuse and could turn from purring to full-on vicious-attack-cat in a heartbeat.

This sort of problem is something many owners face with PO medications. It's the biggest factor influencing noncompliance with medication administration. Sometimes it's the offensive taste of the medication that makes the animal fight the owner. (You think a dog or cat is difficult? Imagine a horse refusing its PO meds.) Sometimes it's the form of the medication that the animal finds objectionable. I've known any number of dogs and cats who hate to be pilled. They're also the ones who will find the pill in whatever it's hidden, eat the treat, and spit out the pill. They're masters at it. Yet, many of these same patients will gladly take an oral liquid. If there are options for PO liquid versus tablets or capsules, it always pays to find out from the owner which form of the drug will be easiest for them to administer.

Compared to most injectable routes of administration, PO has one of the slowest absorption rates. But if the speed of absorption is not a concern, it is an excellent choice. Owners don't need any special skills to be able to give PO meds to tolerant patients. Of course, we do need to be sure to inform owners of any special requirements surrounding the oral medication administration. For example, some medications should be given with a meal. Others should be given an hour or two before (ac) or after a meal (pc). Still others should not be given with dairy products, like tetracyclines. Whatever those requirements are, we need to clearly label the medication bottle. *Pharmaceutical* suppliers have ready-made stick-on labels, with clear instructions like: "give with food" or "refrigerate after opening." This makes our job much easier.

Parenteral

There are many ***parenteral*** [*par-, para-* around + *enter(o)-* intestines + *-al* pertaining to; i.e., not via the intestines] routes of medication administration. The following are some of the most common.

Intradermal

We use the ***intradermal*** [*intra-* within + *derm(o)-* skin, dermis + *-al* pertaining to] route of administration for diagnostic testing. We're

looking for a localized reaction to the agent at the injection site. Have you ever had a TB (tuberculosis) test? That was an i*ntradermal* (ID) injection (Fig. 12.1). We use ID injections for TB testing in cattle and primates. We also use intradermal injections for allergy testing. Obviously, the ***vascular*** [*vascul(o)-* vessel + *-ar* pertaining to] layer of the skin (dermis) is not very thick. So, we can only inject very small amounts, usually 0.2 mL or less. And because the vessels in the dermis are so tiny, we can safely inject without first aspirating (drawing back on the plunger).

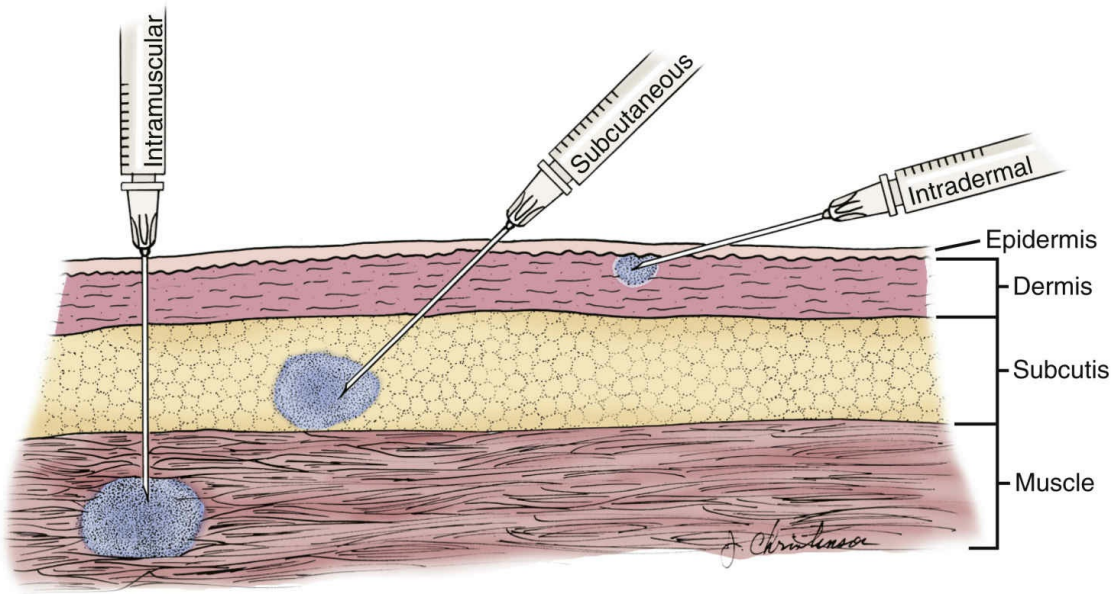

FIG. 12.1 Common parenteral routes of administration.

Transdermal

The use of transdermal [*trans-* across + *derm(o)-* skin + *-al* pertaining to] (ID) medications, in the form of creams, ointments, patches, and so forth has grown in recent years. They have been used in human medicine much longer. Why the slow rise in popularity in veterinary medicine compared to human medicine? Well, let's just say that most people are not as furry as veterinary patients. We need the medication to come into direct contact with the skin where it will be absorbed. Fur gets in the way. However, there are areas of exposed skin on most veterinary patients—like the ear pinna (flap). That works well for the application of creams and ointments. A

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good friend of mine has a cat that needs daily medication. The cat is impossible to give PO medications to. So, her veterinarian has the drug compounded in a cream. She applies it to the cat's ear pinna, alternating the ears each day.

For a fentanyl transdermal patch, we will need to *carefully* shave an area of the body (inaccessible to the patient) to apply the patch. Why do we need to be so careful with our shaving? If we create small scrapes on the skin, those areas expose the drug directly to the bloodstream. Plus, like any abrasion, those areas will become inflamed, increasing blood flow to the area. Bottom line: the drug will be absorbed more rapidly than it should be. This could have adverse, potentially lethal, consequences for the patient. Transdermal patches are designed to deliver the drug at a slow, even rate over time (3 days with fentanyl patches). This is another reason why you should never, ever cut a fentanyl patch. That could result in a lethal bolus of fentanyl to the patient.

By the way, any medication that is designed to be given transdermally can be absorbed by us as well as our patients. Whenever applying transdermal medications, always wear gloves. The same is true for the owner who applies these medications at home.

Subcutaneous

Subcutaneous [*sub-* below + *cutane(o)-* skin + *-ous* pertaining to] (SQ) injections can be given anywhere where there is enough loose skin on the animal (Fig. 12.1). The *subcutaneous* route is frequently used for immunizations, as well as a variety of medications. The rate of absorption from a ***SQ*** injection will be relatively slow compared to IM or IV injections. This is because the subcutaneous tissue is not as vascular as muscle. However, because the ***subcutis*** [*sub-* below + *cutis* skin] is a very loose matrix of connective tissue and fat deposits, we can inject much larger volumes of medication than we can into muscle. Most volumes of medication given in SQ injections will be less than 5 mL. If necessary, depending on the animal and the location of the SQ injection, we may be able to inject 50 to 100 mL of fluids in one site. For example, for noncritical canine or feline patients who require minor rehydration, we often give SQ fluids.

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Intramuscular

Intramuscular [*intra-* within + *muscul(o)-* muscle + *-ar* pertaining to] (IM) injections are administered to large muscles masses. Common ***IM*** injection sites are provided in Chapter 4, by species. The rate of absorption of a medication from an intramuscular injection will be reasonably fast, especially compared to SQ injections or PO administration. For instance, if we are trying to sedate an animal for a minor procedure with an IM injection, we may actually achieve sedation in roughly 15 minutes. The same sedative given SQ may take 30 minutes or more before adequate sedation is realized. Why is it faster with the IM injection? This is because we have greater blood supply to the muscle tissue for rapid absorption and distribution of the drug. Of course, we are more limited in the volumes of drug that can be delivered IM. Depending on the size of the dog or cat and the muscle mass being injected, we might be able to comfortably deliver between 1 and 6 mL of medication max. In cattle and horses, we might be able to inject up to 10 or 20 mL. In smaller ruminants (goats and llamas), we'll probably find volumes limited between that of dogs and cattle (i.e., the lower limit).

Intravenous

Intravenous [*intra-* within + *ven(o)-* vein + *-ous* pertaining to] (IV) injections and infusions provide the most rapid delivery of medications. The actual effect of the drug given is almost instantaneous—basically as long as it takes for the bloodstream to carry the drug from the injection site to the heart. From there it's just a heartbeat away from the brain or whatever organ is targeted by the drug. Common intravenous sites are detailed in the cardiovascular section of Chapter 5. Aside from the speed of drug effects, why give medications ***IV***? Well, the intravenous route allows us to give much larger volumes. This is why the IV route is the preferred route for fluid therapy. We can provide daily maintenance volumes of fluids, as well as large replacement volumes in emergent and critical patients. Secondly, there are times when medications are contraindicated to be given by any other route. Often medications that have a high potential for tissue irritation are labelled for IV use only. Some of these IV medications may require administration into a very large vein to minimize risk

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of creating ***phlebitis*** [*phleb(o)-* vein + *-itis* inflammation of]. Some of those agents should only be given through a central line that places the agent into the vena cava or heart, where it will be rapidly diluted by the blood.

Obviously, any injection should be given in an ***aseptic*** [*a-* without, no + *sep(o)-* infection + *-tic* pertaining to] manner. Because IV administration goes directly into the bloodstream, ***asepsis*** [*a-* no, without + *sep(o)-* infection + *-sis* process, state of] is crucial. Additionally, we cannot afford to infuse suspended particles *intravenously*. Generally, this means that IV fluids and medications should be transparent. One of the rare exceptions to this rule is the *anesthetic* agent propofol—a white, opaque suspension. Finally, there are some IV medications that have a tendency to form crystals or other precipitates. Mannitol frequently does this. Drugs like this should only be administered through an IV filter.

Intranasal

Obviously, the ***intranasal*** [*intra-* within + *nas(o)-* nose + *-al* pertaining to] route of administration is limited in its use. The extreme sensitivity of the nose makes it very difficult to infuse even small volumes of medications there. We usually wind up "wearing" much of what we infuse when the animal sneezes. So, we tend use this route almost exclusively for the administration of certain immunizations, usually infectious respiratory diseases. That said, I frequently administered a topical *anesthetic* agent, like lidocaine, *intranasally*. This was to make patients more comfortable during the placement of a ***nasogastric*** [*nas(o)-* nose + *gastr(o)-* stomach + *-ic* pertaining to; a tube that extends from the nose to the stomach] tube (NG tube). Most animals do not appreciate it when we try to pass a tube through their nose. But it often becomes necessary for the nutritional support of patients who refuse to eat.

Intratracheal

The ***intratracheal*** [*intra-* within + *trache(o)-* trachea + *-al* pertaining to] (IT) route of medication administration is generally reserved for use in emergency situations. For example, if an emergency patient developed ***cardiac*** [*cardi(o)-* heart + *-ac* pertaining to] ***arrest*** before we were able to place an IV catheter, we delivered emergency

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drugs through the ***endotracheal*** [*endo-* within + *trache(o)-* trachea + *-al* pertaining to] ***tube***. We need to add a little fluid volume (usually with sterile saline) to the drugs to carry them down into the lungs. The beauty of this route is that the lungs are very *vascular* and the blood leaving the lungs goes directly to the heart. In cardiac arrest, that is precisely where we want those drugs to go. The rate of absorption through the ***IT*** route is comparable to an IV injection.

Intraosseous

Like the intratracheal route, the ***intraosseous*** [*intra-* within + *osse(o)-* bone + *-ous* pertaining to] (IO) route is typically reserved as a last resort in emergency situations. In my experience in emergency and critical care medicine, most often we administered *intraosseous* fluid therapy to extremely small puppies or kittens. When the emergent fluid replacement and blood pressure needs of those patients outweighed the time it would take to do a vascular cut-down for placement of an IV catheter, we opted for IO administration. Sometimes the IV catheter placed was so very small that we could not deliver IV fluids fast enough. IO administration in those cases was much faster. As we discussed in Chapter 4, IO administration places the fluids and/or drugs into the bone marrow. Because the bone marrow has a rich vascular supply, it is an effective and efficient emergency site for fluid therapy. Generally, we will place the largest needle we can (20-18 gauge) into the proximal end of a long bone, like the femur or humerus. Is this painful? Yes. But when given the choice of trying to save the life of the animal versus letting it die, we choose life. Often the patient is unconscious and unaware of the trauma we inflict at that time.

Intraperitoneal

Intraperitoneal [*intra-* within + *periton(o)-* abdomen + *-al* pertaining to] (IP) injections and infusions are very limited. When I think of pocket pets and laboratory animals, like gerbils, hamsters, and mice, *intraperitoneal* injections are commonly used. Think about it: these creatures have tiny muscles and veins, making it nearly impossible to safely administer IM and IV medications. The restraint factor adds another layer of difficulty for IV and IM administration. We might attempt a SQ injection. However, it is

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often easier to safely and comfortably restrain these tiny creatures in dorsal recumbency in one hand, while injecting *intraperitoneally* with the other hand. Most of the time, they seem to mind the restraint more than the injection itself.

In the ICU, we sometimes used ***intraperitoneal dialysis*** for ***renal*** [*ren(o)-* kidney + *-al* pertaining to] ***failure*** patients. The fluid (dialysate) was infused into the peritoneal (abdominal) cavity, picking up the toxic wastes, and then flowed back out into a collection system to be measured and ultimately discarded. This same route may also be used for the safe and efficient rewarming of severely ***hypothermic*** [*hypo-* below, insufficient + *therm(o)-* temperature + *-ic* pertaining to] patients. The warmed IP fluids warm the core organs first, providing adequate blood flow to those organs while minimizing the risk of developing severe ***hypotension*** [*hypo-* low + *tens(o)-* pressure + *-ion* state of; i.e., low blood pressure].

Right Time?

Let's see, so far, we've addressed four of the five rights for medication administration: right patient, right drug, right dosage, and right route. As with the other "rights," we should begin thinking about the "right time," by verifying the veterinarian's drug order. There is much to be considered with regard to timing (see Table 12.8 for prescription notations regarding time/frequency). We need to know how frequently the drug is to be given. But timing goes beyond mere frequency. For instance, to ensure adequate absorption of a PO medication, should it be given before meals? Should it be given after a meal, to avoid stomach upset? Is timing critically important to maintain steady drug levels in the patient's system, like with phenobarbital administration in a seizure patient? (To maintain steady levels of that drug for seizure control, it must be given on time, every 12 hours [***q12h***]. It cannot be given simply twice a day [***BID***] at the convenience of the owner's schedule.) Give a drug too soon and we could risk ***toxicity*** [*toxic(o)-* poison + *-ity* state of] or overdose complications. In whatever manner, the frequency is prescribed, and we should adhere to those orders. Yet, there are times when the prescribed frequency falls into question. If you are unfamiliar with a particular drug, you should look it up to ensure that the frequency prescribed is appropriate for the drug

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and the patient. I have cared for many patients in the ICU who were receiving ***analgesics*** [*an-* without + *alges(o)-* pain + *-ic* pertaining to; i.e., pain relievers]. Often, there is a frequency range for these drugs. In a patient with "every 8 hour" (q8h) analgesic orders, who is in pain 6 hours after receiving the drug, I would consult a drug reference. Why? Because administration every 8 hours appeared to be inappropriate for that patient. So, if the manufacturer gave a dosing range of q6-8h, I would ask the clinician to adjust the order. If the frequency could not be adjusted, the doctor may have to consider other medications or methods for pain control.

TABLE 12.8

Time/Frequency of Medication Administration Abbreviations

R_x Abbreviation	Meaning
a.c., ac	Before meals [L. *ante cibum*]
ad lib.	As desired [L. *ad libitum*]
b.i.d., BID	Twice daily [L. *bis in die*]
c̄	With
h	Hour [L. *hora*]
hs	At bedtime [L. *hora somni*]
noct.	At night [L. *nocte*]
NPO	Nothing by mouth [L. *nil per os*]
p.c., pc	After meals [L. *post cibum*]
per	By
PRN	As needed [L. *pro re nata*]
q	Every [L. *quaqua*]
qAM	Every morning
qd	Every day [L. *quaqua die*], cf. s.i.d.
qh	Every hour [L. *quaqua hora*]
q2h	Every 2 hours
q4h, qqh	Every 4 hours [L. *quaqua quarta hora*]
q6h	Every 6 hours
q8h	Every 8 hours
q12h	Every 12 hours
q.i.d., QID	4 times daily [L. *quarta in die*]

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q.o.d., QOD	Every other day
s.i.d., SID	Once daily [L. *semel in die*] , cf. qd
s⁻	Without
Sig., SIG:	Give [L. *signa*]
STAT	Immediately [L. *statim*]
t.i.d., TID	3 times daily [L. *ter in die*]

Another factor that I will include under "timing" is the rate of delivery of a drug. What is the right rate of infusion? If an IV drug can be given as a single bolus injection, how rapidly can it be injected? Sometimes, we need to give the agent slowly over 5 to 10 minutes to avoid complications like *phlebitis* or ***acute*** (sudden) changes in blood pressure. I have delivered many drugs via ***constant rate infusions*** (***CRI***). It is very important to set up the infusion pump at the appropriate rate. There is a big difference between a rate of mcg/kg/min versus mcg/kg/h. Selecting the wrong rate of delivery on the infusion pump could quickly kill a patient.

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Applied Terminology for Pharmacodynamics and Pharmacokinetics

Wow, those are two really big words. Let's see if we can figure out what they mean. First, they both begin with *pharmac(o)-*. So, they obviously have something to do with drugs. What's going on with the drugs is determined by the other root in each word.

Pharmacokinetics

Pharmacokinetics [*pharmac(o)-* drugs + *kinet(o)-*, *kines(o)-* to move, movement + *-ic* pertaining to] is that part of pharmacology that looks at how drugs *move* into, through, and out of the body. Obviously, the route of administration influences this. We addressed this a little bit when we talked about the rate of absorption via various routes. We said that blood flow to the area was a factor influencing absorption and redistribution. The greater the blood flow, the faster the absorption. But that's not all. The rate of absorption is also influenced by the composition of the medication.

Consider two different drugs being given *subcutaneously*. We know that, based on the limited vascularity in subcutis, absorption of any drug will be slower when given SQ than say IM or IV injections. But a ***hydrophilic*** [*hydr(o)-* water + *phil(o)-* loving + *-ic* pertaining to] drug will be absorbed faster from a SQ injection than a ***lipophilic*** [*lip(o)-* fat + *phil(o)-* loving + *-ic* pertaining to] drug. Why? Consider the nature of the ***interstitial*** [*inter-* between + *stiti(o)-* tissue + *-al* pertaining to] fluid. It is primarily water. So, a *hydrophilic* drug will likely diffuse a bit more rapidly through the SQ interstitial fluid. Because oil and water don't mix, it may take a *lipophilic* drug a bit longer to make its way to vessels in the area. The opposite might be true when comparing the absorption of PO medications. Because cellular membranes along the digestive tract have a ***lipoprotein*** [*lip(o)-* fat + *protein*] structure, a lipophilic drug may have an easier time diffusing across. Sure, active transport may

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help the hydrophilic drug across the cellular membrane. So, we may be splitting hairs when it comes to the actual difference in speed with these examples. This is merely to help you understand that the composition of the drug in a given environment may either enhance or hinder the rate of distribution and absorption.

Where do drugs go, once they've been absorbed or injected into the bloodstream? Well, that depends on the route of administration. Obviously, something given IV will go to the heart first and then get pumped throughout the body. At some point in its journey, the drug will eventually be distributed to the target tissue or organ. For something given PO, it's a little different story. Everything absorbed from the digestive tract into the bloodstream will go to the liver first. "So what?" you say. Hypothetically, let's say that we are giving a drug that is rapidly metabolized by the liver. Perhaps the ***biotransformation*** [*bi(o)-* life + *transform(o)-* change + *-tion* process of] by the liver is so rapid that it is difficult to achieve therapeutic levels, where it is needed in the body. No matter which route of administration we choose, eventually the drug will wind up in the liver. But if we would like it to be distributed to other parts of the body before reaching the liver, it wouldn't make any sense to give the drug PO. We know it will go directly to the liver from the digestive tract. So, we may choose a parenteral route for that particular drug to keep the liver out of the loop for a little while. Bear in mind that nearly all drugs are eventually *biotransformed* by the liver.

The final movement of a drug will involve its ***excretion*** [*ex-* out + *cret(o)-* sift, separate + *-tion* process, act of; i.e., elimination, removal from the body]. Both the liver and the kidneys are the principal sites where drugs and their by-products "disembark" from their journey through the body. Exit stage left! Seriously, these two organs are very effective at removing things, like drugs, from the body. The drugs and their by-products actually pass in either the urine, feces, or both. This is something to keep in mind if you're caring for a cancer patient receiving ***chemotherapy*** [*chem(o)-* chemical + *therapy* treatment]. You could be exposed to the *chemotherapeutic* agent or its by-products in the urine and feces, endangering your health. Know the excretion mechanism as well as the rate and duration of excretion for these agents. Protect yourself

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with knowledge and the appropriate gear (like gloves).

Of course, because the liver and kidneys are so important for *biotransformation* (primarily liver) and *excretion*, they may be damaged by medications, especially with prolonged administration. We have already mentioned the anticonvulsant phenobarbital. In dogs with epilepsy, phenobarbital may be given for years, perhaps for most of the dog's life. It is a very effective anticonvulsant. Unfortunately, the drug may take its toll on the liver. Phenobarbital-induced ***hepatotoxicity*** [*hepat(o)-* liver + *toxic(o)-* poisoning + *-ity* state of] can develop. This is one of the reasons why these patients should have routine blood work at appropriate intervals to evaluate liver function. There are many other drugs that have a high potential for *hepatotoxicity*, like some powerful drugs used to treat deep fungal infections. Likewise, because the kidneys are a principle point of drug excretion, they may experience significant damage from ***nephrotoxic*** [*nephr(o)-* kidney + *tox(o)-* poison + *-ic* pertaining to] agents. This is why we need to know and understand *pharmacokinetics*. We need to know if an agent is potentially ***hepatotoxic*** [*hepat(o)-* liver + *tox(o)-* poison + *-ic* pertaining to] or *nephrotoxic*, even with appropriate dosing. By knowing this, we and the owners can be vigilant for any early symptoms of toxicity. We may need to check ***hepatic*** [*hepat(o)-* liver + *-ic* pertaining to] and *renal* blood values regularly to catch early changes. Once these organs fail, there's no turning back.

Pharmacodynamics

Let's see if we can decipher the term ***pharmacodynamics*** [*pharmac(o)-* drugs + *dynam(o)-* power + *-ic* pertaining to]. I've known students who have had difficulty keeping the terms *pharmacokinetics* and *pharmacodynamics* straight. So, maybe this will help. Think about dynamite. It's a powerful explosive, right? So, pharmacodynamics is that facet of pharmacology that looks at the powerful actions that drugs have on the body.

How a drug exerts its "power" over specific tissues or organs all depends on the drug's compatibility with receptors on those tissues and organs. You might think of receptors as on and off switches, in key locations, for very specific effects. Pharmacodynamic effects

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result from a drug binding at specific receptors and flipping the particular switch on or off. The pharmaceutical industry has done fabulous research to identify specific receptors and their responses to various drugs. Only certain drugs can bind at specific receptors. It has to be a perfect fit, sort of like Cinderella[b] and the glass slipper. She was the only one who could wear that slipper, resulting in the happy story-book ending.

As I mentioned earlier, I have cared for numerous patients in the ICU who needed analgesics. For extreme pain, we often administered constant rate infusions of the ***opioid*** [*opi(o)-* opium + *-oid* resembling; i.e., a synthetic narcotic with similar actions to that of opium] fentanyl. Fentanyl binds with mu receptors in the brain to effectively block the pain pathways in the brain (pain switch—off). Unfortunately, fentanyl's activity in the brain may also significantly depress breathing, heart rate, and blood pressure. The good news is, if a patient's vital ***cardiopulmonary*** [*cardi(o)-* heart + *pulmon(o)-* lung + *-ary* pertaining to] functions become too depressed, we can stop the infusion and give a reversal agent, like naloxone. Naloxone will take over the mu receptors, blocking the cardiopulmonary depressant effects. Of course, we also lose the analgesic effects of the fentanyl (pain switch—on). Yes, we will address the pain control needs of the patient. But the first order of business is making sure that the patient lives. And that's where the reversal drug comes into play. We'll talk more about this kind of drug "competition" for the same receptor in a little bit. For now, let's look at some commonly used types of pharmaceuticals. Some of these discussions may help your understanding of pharmacodynamics.

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Types of Pharmaceuticals

Antimicrobials

Antimicrobial [*anti-* against + *microb(o)-* microbes + *-al* pertaining to] agents are used against all sorts of ***pathogenic*** [*path(o)-* disease + *gen(o)-* producing + *-ic* pertaining to] organisms, including bacteria, viruses, fungi, and protozoa. So, these types of agents target the organism to either kill it [*cid(o)-* to kill] or stop [*-static* pertaining to inhibition] its activities, like reproduction. There are a plethora of antimicrobials for use on or in animals as well as for decontaminating the environment. Regardless of which agent is used, we need to ensure that it is used properly, in the right concentration, at the right frequency and for the appropriate duration. Failure to do this can place the patient we are treating at risk. It may also jeopardize the health of other animals and people, by exposing them to ***nosocomial*** [*nos(o)-* disease + Gr. *komeion* to take care of + *-al* pertaining to; i.e., hospital-acquired disease] infections or ***zoonotic*** [*zo(o)-* animal + from *nos(o)-* disease + *-tic* pertaining to; disease in humans acquired from animals] disease. Leptospirosis is a *zoonotic* disease caused by the bacterium, *Leptospira sp*. It can be lethal for animals and humans alike. We also live in a time when we are seeing an upsurge in resistant organisms (e.g., MRSA, Methicillin-resistant *Staphylococcus aureus*). Failure to use antimicrobials properly can increase the likelihood that an organism develops resistance to the agents.

Antibiotics

Most of us have taken ***antibiotics*** [*anti-* against + *bi(o)-* life + *-tic* pertaining to] at some point in our lives. It was probably to treat some sort of bacterial infection. Did you know that not all antibiotics work the same way? They don't. Some are ***bactericidal*** [*bacteri(o)-* bacteria + *cid(o)-* killing + *-al* pertaining to]. Our intent is to kill the *pathogenic* bacteria, with bactericidal agents. Unfortunately, sometimes we kill good ***commensal*** [*com-* together, with + L. *mensa* table + *-al* pertaining to; i.e., bacteria living in harmony with the animal; cf. symbiosis] bacteria along with the

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bad. This could result in side effects, like antibiotic-induced diarrhea. Other agents don't actually kill the bacteria. ***Bacteriostatic*** [*bacteri(o)-* bacteria + *-static* pertaining to inhibition] agents inhibit important life-sustaining activities of the organism, like reproduction. What's the point? Well, if we can stop the bacteria from colonizing (reproducing), we give the body a fighting chance to destroy the organism itself. Without the inhibition, the bacteria may overwhelm the body's immune system. *Antibiotics* are manufactured in both *enteral* and *parenteral* preparations.

Antimycotics

Antimycotic [*anti-* against + *myc(o)-* fungus + *-tic* pertaining to; i.e., antifungal] agents are produced in a number of forms, too. But here's the thing—fungal infections are often much harder to eradicate than bacterial infections. Fungi (plural of fungus) transform from stages that are susceptible to antimycotic agents to protective spores. For "deep" infections (e.g., lungs or other organs), it takes powerful antimycotics, like ketoconazole (ke"to-kon'uh-zol) to treat the patient. Unfortunately, given *parenterally*, drugs like ketoconazole have the potential to be *hepatotoxic*.

Antiseptics and Disinfectants

Antiseptic [*anti-* against + *sep(o)-* infection + *-tic* pertaining to] and ***disinfectant*** [*dis-* to reverse + *infect(o)-* infection + *-ant* one that] agents are those used superficially to either treat or prevent the growth and spread of *pathogenic* microbes. What's the difference? *Antiseptics* are used on the body, like surgical scrub for preparing a surgical site or alcohol to prepare a parenteral injection site. *Disinfectants*, on the other hand, are used on inanimate objects and surfaces. Did you notice something there? By adding an "s" to the suffix of each of these terms, I have changed them from modifiers (adjectives) to things (nouns). It seems like such a subtle change. Yet, it makes a big difference in how the words are used.

It is important to understand which form and concentration of *antiseptic* agent is appropriate for which conditions and circumstances. As we said, we use surgical scrubs, like those containing chlorhexidine or an iodophor (i-o-do-for'; i.e., iodine-based), to prepare surgical sites. These products not only contain

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the *antimicrobial* component, but also some sort of detergent (soap). The concentration of the antimicrobial is often relatively high (4% chlorhexidine scrub for example). These two factors mean that we should only use these products superficially, on intact skin. They should not be used in open wounds on the exposed tissue because both the detergent and the high concentration of the antimicrobial are ***cytotoxic*** [*cyt(o)-* cell + *tox(o)-* poison + *-ic* pertaining to]. (Notice that I keep referring to these products as antimicrobials. That's because they are effective against both bacteria and fungi.). Both the scrubbing action for the removal of contaminants and contact time are important. For example, chlorhexidine needs 2 minutes of contact time to allow it to bind to the skin. This provides roughly 12 hours of antimicrobial activity. This is why we don't want to wipe off the excess scrub until it has been allowed to sit on the skin for at least 2 minutes.

So, if we want to cleanse an open wound, what should we use? Well, simple mechanical cleansing can be done by flushing with sterile saline. If we want antimicrobial activity, we'll use an antimicrobial solution. That solution, whether it contains chlorhexidine gluconate or an iodophor, must be diluted to avoid ***cytotoxicity*** [*cyt(o)-* cell + *toxic(o)-* poisoning + *-ity* state of]. And in the case of iodophors, they must be diluted in order to release the iodine from the solution's stabilizing agents. It's only free iodine that provides antimicrobial activity. So, if a noncytotoxic concentration is 0.01% and the stock solution has a concentration of 10%, we have some major diluting to do. It looks like weak tea compared to the molasses-like solution from the manufacturer. But it's at this dilution that it is safe and effective. Chlorhexidine solutions need to be diluted too, but only to make them safe for the tissues. Again, we could be talking about taking a 2 to 4% stock solution down to a 0.05 to 0.1% solution. Why choose one over the other? Both can be quite effective. However, if we want to reduce the frequency of wound treatment and bandage changes, we may want to go with a chlorhexidine solution. Chlorhexidine gluconate, once it has been in contact with the tissues for 2 minutes, may provide antimicrobial activity for over 12 hours. Comparatively, iodophor solutions do not bind to the tissues and provide antimicrobial activity for only 4 to 6 hours. It all depends on what's

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best for the patient and its particular circumstance.

Disinfectants come as solutions and detergents. Many of them are *bactericidal*, ***fungicidal*** [*fung(o)-* fungus + *cid(o)-* kill, killing + *-al* pertaining to], and ***virucidal*** [*vir(o)-* virus + *cid(o)-* kill, killing + *-al* pertaining to]. Their killing abilities rely, in part, on the chemicals, appropriate dilutions of those chemicals, and contact time with the surface. That means actual contact with the surface. If urine, feces, vomit, or other potentially infective material are present, the bulk of that organic material must be removed to allow the disinfectant to actually make contact with the surface of the contaminated item. The dilution of the agent may be different, depending the type of organism to be targeted. And not all disinfectants, regardless of dilution, are effective against bacteria, fungi, and viruses. In fact, whether a virus is enveloped (i.e., with a protective layer) or nonenveloped may dictate the efficacy (effectiveness) of a given disinfectant. Is there really that much to be considered? Yes. So, you need to be sure that you understand the product you are using (read the label!) and the known or suspect microbe you're targeting. When we began this *antimicrobial* section, we touched on the idea of *nosocomial* infection and ***zoonosis*** [*zo(o)-* animal + *nos(o)-* disease + *-sis* condition of; i.e., disease transmitted between animals and people]. Disinfectants play a huge role in nosocomial infection control, as well as prevention of zoonoses (zo-o-no'sēz; plural of zoonosis). It is our professional duty to serve our patients well *and* protect the public welfare.

Biologicals

Generally, when we think of ***biologicals*** [*bi(o)-* life + *log(o)-* knowledge, study + *-cal* pertaining to], we tend to think about vaccines. Vaccines certainly make up the lion's share of the *biological* agents we use. Immunizations were discussed in Chapter 3. Generally speaking, vaccines contain either killed or attenuated (modified) forms of the organism. We routinely vaccinate against a number of pathogenic bacteria and viruses.

Do you recall in the previous section when we were discussing *antibiotics*, that we said antibiotics may kill ***pathogens*** [*path(o)-* disease + *gen(o)-* producer] as well as good bacteria? We said that

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this may cause antibiotic-induced diarrhea. Well, a growing segment of biologicals now includes ***probiotics*** [*pro-* for + *bi(o)-* life + *-tic* pertaining to]. *Probiotic* products come in a variety of forms, from dairy products with live cultures of good, nonpathogenic bacteria to tablets and capsules. Replenishing the good bacteria along the digestive tract has been proven to be very beneficial for animals and humans alike.

Analgesics, Anesthetics, and Sedatives

Okay, let me begin this section by saying that there may be overlap between the drugs included in these categories. The effect and whether we call a given agent a sedative or analgesic may be dose dependent. Many of these drugs are nothing alike. So, why create such a huge category as this? Because there is enough common ground to lump them together.

Agonists Versus Antagonists

This is one of those areas of common ground. There are some analgesics and some sedatives that have competition for receptors. We touched on this briefly in the pharmacodynamics section when we talked about the fentanyl. We said that fentanyl was an *opioid analgesic* agent that bound to the mu receptors in the brain. This is an example of an ***agonist*** [*agon(o)-* to compete + *-ist* one that]. What is an agonist competing with? Well, those chemicals in the body that stimulate the pain pathways. In fact, that competition can be pretty fierce. You see, if pain is allowed to spiral out of control, those chemicals stimulating the pain pathways will be so tightly bound to the receptors that even powerful analgesics like fentanyl cannot work. This is why we never want our patients to reach a level 8 out of 10 on the pain scale or higher. It will take high doses of analgesics way too long to achieve pain relief.

When talking about some of the adverse side effects of fentanyl, we also said that naloxone would reverse the effects (good and bad) of the fentanyl. So, naloxone is an example of an ***antagonist*** [*ant, anti-* against + *agon(o)-* to compete + *-ist* one that; i.e., a reversal agent]. Most *opioid agonists* compete for the same receptors. So, naloxone can be used as the *antagonist* for most opioids.

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Comparatively, butorphanol is a commonly used mild analgesic and sedative that has agonist and antagonist properties. If administered following an opioid, butorphanol may antagonize (reverse) the opioid, while acting as an agonist to provide some analgesia. It is an agonist-antagonist. How does it do both? It targets different receptors. So, it acts as an antagonist at mu receptors while acting as an agonist at k receptors. Freaky, huh?

Of course, there are a number of agonists for analgesia or sedation. You can bet your boots that if they have figured out agonist receptors, and if the agonist can have serious adverse effects in the body, then pharmaceutical researchers have probably created an antagonist for that agent. We mentioned the sedative dexmedetomidine in earlier sections. It is an agonist. This sedative can markedly depress cardiopulmonary function. So, being able to reverse its effects may be necessary. Atipamezole is its antagonist. The other value in being able to antagonize dexmedetomidine is quickly reviving an outpatient following a minor procedure, reducing wait-time for the owner. We can get things done and quickly send them on their way.

Okay, so we've talked about sedatives and analgesics a little bit. What exactly is the difference between an *analgesic* agent and an ***anesthetic*** [*an-* without + *esthe(o)-* sensation + *-tic* pertaining to] agent? Don't they both remove sensations? Well, I guess you might look at it that way. But analgesics reduce or remove pain sensation only. Anesthetic agents remove all sensation. Let me see if I can clarify that further.

Let's say we're going to remove a small wart from a patient. It's a pretty minor procedure. There is certainly no need to make the animal unconscious for this. So, we might give an intradermal injection of a local anesthetic agent, like lidocaine, around the wart. This will temporarily remove all sensation surrounding the wart. The animal won't feel it at all when we cut into the skin. By the way, whenever you see the suffix *-caine*, you are probably looking at a local anesthetic agent. Lidocaine, bupivacaine and proparacaine are all examples of local anesthetic agents. If we inject them around major nerves or nerve roots, we can create ***anesthesia*** [*an-* without + *esthes(o)-* sensation + *-ia* condition of] in the region that the nerves supply. We use regional anesthesia like this for some abdominal

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surgeries in cattle. The animal is wide awake, maybe even chewing her cud, while we're busily puddling around in her abdomen. She doesn't feel a thing. Of course, the ultimate in removing all sensation involves general anesthesia. Under general anesthesia, the animal is completely unconscious. No sensations (touch, pain, sound, etc.) are (consciously) perceived by the patient. I hope this helps you understand the difference between analgesics (pain relievers) and anesthetic agents.

Anti-Inflammatories

It may be of value to you to review the inflammation and hemostasis sections ofChapter 3 for this discussion. The latter will be important when we talk about NSAIDs. It may also be helpful to review Chapter 10 for our steroid discussion. That said, why would we want to reduce or prevent inflammation? Well, some of the characteristic signs of inflammation, like edema (swelling) and pain, can significantly slow or complicate a patient's recovery. This is especially true following surgery. Yet, not all ***anti-inflammatory*** [*anti-* against + *inflamm(o)-* fire, inflammation + *-tory* pertaining to] agents drugs are the same. In fact, some would be contraindicated immediately preceding or following surgery.

Steroidal Anti-Inflammatory

Corticosteroids [*cortic(o)-* cortex + steroid] are naturally produced in the cortex of the adrenal gland. You can review this in Chapter 10. The pharmaceutical industry has produced numerous synthetic corticosteroids that closely resemble the composition and activity of those secreted by the adrenal glands. In fact, these drugs are so much like the real thing that, depending on the dosing and duration of systemic therapy, can actually shut down the adrenal cortex. If the drug is abruptly stopped, these patients can develop severe complications. Immune-suppression from corticosteroids can also place patients at risk of infection. In surgical patients, the powerful ***anti-inflammatory*** effects of corticosteroids (from drugs or the adrenal glands—as seen in ***chronic*** [*chron(o)-* time + *-ic* pertaining to; i.e., long-term] disease) can significantly slow the healing process. Yet, there are times when PO or parenteral steroids are

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very beneficial.

Corticosteroids are prescribed when anti-inflammatory and/or ***immunosuppressive*** [*immun(o)-* protection + *suppress* inhibit + *-ive* pertaining to] effects are absolutely necessary. For example, in Chapter 3, we discussed ***immune-mediated hemolytic*** [*hem(o)-* blood + *-lytic* pertaining to breaking, destroying] ***anemia*** [*an-* without, deficient + *em(o)-* blood + *-ia* condition] (IMHA). As you'll recall, in IMHA for whatever reason the body's immune system has targeted and is destroying the red blood cells. If we do not stop the targeting and destruction, the patient will die. So, we administer *immunosuppressive* doses of corticosteroids. The same is true for patients with immune-mediated ***myasthenia*** [*my(o)-* muscle + *asthen(o)-* weakness + *-ia* condition of] ***gravis*** [L. *gravis* heavy], as we spoke about in Chapter 4. Even some profound ***allergic*** [*allerg(o)-* allergy + *-ic* pertaining to] reactions may require temporary treatment with corticosteroids. These patients may also experience side effects, like excess thirst and urination—leaving them at risk of dehydration, if adequate drinking water is not available. Owners may need to limit the animal's exposure to infectious diseases while receiving steroidal therapy. However, the benefits when dealing with profound disease typically far outweigh the adverse effects. To avoid a health crisis, when the steroidal therapy is stopped, we gradually reduce the dose over time. By tapering the dose, we permit the adrenal glands to slowly "reawaken" and begin secreting their own ***cortical*** [*cortic(o)-* cortex + *-al* pertaining to] hormones. It's a necessary, delicate dance, and it works. Dexamethasone (parenteral) and prednisone (PO) are commonly prescribed corticosteroids.

By the way, whether given parenterally or PO, corticosteroids can significantly impact the digestive tract. So, while receiving steroidal therapy, ***gastrointestinal*** [*gastr(o)-* stomach + *intestin(o)-* intestines + *-al* pertaining to] (GI) protectants are often administered. GI protectants in addition to the administration of PO corticosteroids with food often minimize or prevent *gastrointestinal* upset.

Non-Steroidal Anti-Inflammatory

When we want anti-inflammatory effects without the side effects of immunosuppression, delayed healing, and so forth, we turn to ***non-***

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steroidal anti-inflammatory drugs (NSAIDs). However, these too can have profound GI side-effects. In fact, they can lead to ulceration along the GI tract so severe that perforation may occur. They should *never* be given with corticosteroids. The combined GI consequences can be devastating. Another factor to consider is *renal* integrity. NSAIDs are *contraindicated* in the presence of renal disease because they adversely affect blood flow to the kidneys.

When are NSAIDs indicated? We often administer them following surgery because they reduce inflammation without delaying healing of the surgical site. We frequently administer them for *chronic* conditions, like ***arthritis*** [*arthr(o)-* joint + *-itis* inflammation of]. No, we won't alter the deterioration in the joints, but by reducing the inflammation we can provide some level of *analgesia*. But what if we have a German Shepherd with hip dysplasia (discussed in Chapter 4) who is scheduled for a total hip replacement? The NSAID given ***preoperatively*** [*pre-* before + *operat(o)-* surgery + *-tive* pertaining to] for the chronic pain may need to be stopped long before the surgery. Why?—Because some NSAIDs can put the patient at risk of significant blood-loss during the procedure. That impact is determined by what the drug principally targets in the inflammatory pathway—that is, either cyclooxygenase 1 (sik"lo-ok'si-jen-ās; COX1) or cyclooxygenase 2 (COX2).

COX1 and COX2

Cyclooxygenase is one of many pieces of the inflammatory puzzle. We won't discuss the fine details of inflammation here. Suffice it to say that by inhibiting cyclooxygenase, we reduce inflammation. That's the desired outcome, when administering COX1 and COX2 NSAIDs. But there are also functional consequences. In the body, cyclooxygenase 1 is important for platelet function and protection of the GI mucosa. By giving a COX1 inhibiter, like aspirin, platelets cannot stick together. In fact, this is why people at risk of heart attacks may take aspirin on a regular basis. Unfortunately, giving aspirin preoperatively (before surgery) to our total hip patient would put the dog at risk of profound ***hemorrhage*** [*hem(o)-* blood + *-rrhage* to escape; i.e., bleeding] at the surgical site. So, aspirin must be stopped. With aspirin therapy, we also lose the GI protection of

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COX1. This is why ***gastric*** [*gastr(o)-* stomach + *-ic* pertaining to] upset and ulceration may occur with aspirin administration. *Enteric* coated aspirin tablets help reduce risk of *gastric* consequences, by delaying exposure to the drug until it reaches the intestines (hence the name: enteric coated). Buffered aspirin also helps minimize gastric consequences.

By the way, the ***half-life*** of aspirin in dogs and cats is way longer than it is in people. The half-life of a drug is the amount of time it takes for the concentration of the drug in the body to be reduced by half. This is how pharmaceutical companies determine the duration of action of a given drug. Once reduced by half, the drug may still be present but not in sufficient concentration to be therapeutic. The half-life of aspirin in humans is less than 4 hours. In dogs, the half-life of aspirin is nearly 9 hours. And in cats, the half-life of aspirin may be 1 to 2 days or more, depending on the dose. Why tell you this? Because pet owners must be made to understand that they cannot give aspirin to their dogs or cats at the same dosing and frequency that they would take it themselves. In fact, cats have a very difficult time with the *biotransformation* and *excretion* of the drug. So, they are highly susceptible to toxicity.

Are there any NSAIDs that our hip dysplasia dog could safely take preoperatively? Yes. Those that principally target cyclooxygenase 2 do not interfere with platelet function as much or not all compared to COX1 inhibitors. Carprofen is probably the most widely used COX2 inhibitor in dogs. Because carprofen, in normal therapeutic dosages, does not interfere with platelet function, it can be given up to the day of surgery. This is important for preoperative analgesia. Now, COX2 inhibitors also interfere with GI protection and reduce renal blood flow. And because a COX2 inhibitor, like carprofen, is biotransformed and partially recycled in the liver, chronic use can be damaging to the liver. By the way, the half-life of carprofen in dogs may be up to 12 hours. So, it should not be given any more frequently than twice a day.

Pesticides

There are many ***pesticides*** [*pest(o)-* pest + *cid(o)-* to kill; i.e., pest killers] used in veterinary medicine. They come in a variety of

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forms, including parenteral, topical, and oral preparations for animal use. Still others are produced to treat the environment (the typical context for use of the term "pesticide"). Of course, the pests I refer to here are parasites. So, really the term ***parasiticide*** [*parasit(o)-* parasite + *cid(o)-* to kill] is probably more appropriate when thinking of administering a product to an animal. You see, parasites are an inherent part of veterinary medicine. Many parasites can cause significant disease in animals. Some are zoonotic. Some are deadly. In order to eliminate, prevent, or minimize disease in animals and people, we need to control parasites. In our discussions here, we will barely scratch the surface of parasites and their control. You'll need a ***parasitology*** [*parasit(o)-* parasite + *-logy* study of] course to fully understand the nature of parasites and controlling or eradicating them. In brief, some of the best control measures use our knowledge of the parasite's life cycle to find points of vulnerability. Those are the points we target to either kill the organism or to keep it from reproducing.

Insecticides and Acaricides

Insecticides [*insect* + *cid(o)-* to kill] are typically used to control lice and flea infestations. ***Acaricides*** [*acar(o)-* mite + *cid(o)-* to kill] are used to control mites and ticks. Mites and ticks are ***arachnids*** [*arachn(o)-* from Gr. *arachne* spider], like spiders. That's why we use a different term for products used to control these parasitic arachnids. Some products, used in dogs and cats, are effective at controlling both fleas and ticks. The forms of some of these products are topical preparations and others are PO. Our five rights of medication administration still apply to these agents, in spite of the fact that many pet owners do not view them (especially topical forms) as "medications." If you recall from our discussion of the ***neuromuscular*** [*neur(o)-* nerve + *muscul(o)-* muscle + *-ar* pertaining to] ***junction*** inChapters 4 and 11, even pests like insects and arachnids have ***cholinergic*** [*cholin(o)-* acetylcholine + *erg(o)-* working + *-ic* pertaining to] receptors. So, if the insecticide or acaricide targets those receptors to kill the parasite, in sufficient dosages it may kill the host animal in the same way. That's why we need to be sure that the right agent is given to the right animal, at the right dosage, by the right route, in the right frequency. Failure

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to do this may result in toxicity and even death. Please note that some products labelled for use in dogs are toxic for cats, even in small dosages. For environmental *pesticides* used to control insects, mites, and ticks in and around the home, care must be taken to protect humans and animals living there. Care must also be taken to protect nontarget organisms, like honeybees and wildlife.

Anthelmintics

Anthelmintics [*ant-, anti-* against + *helmin(o)-* worm + *-tic* pertaining to] or de-wormers are used to control worms. (Go figure). There are many different kinds, each targeting a particular type of worm or a particular life-stage of the parasite. For instance, heartworm preventatives are designed to kill migrating larvae (baby worms). When given, up to a month after a mosquito infects the dog with heartworm larvae, many of these agents will kill the larvae. The goal is to prevent the organism from reaching the dog's heart. Once they arrive there, it's too late. Yes, we can treat adult heartworm infections, but not without great risk to the patient. And the damage done to the heart and lungs from heartworm disease may be irreversible. If you've read the label or advertising for heartworm preventatives, you may have noticed that they also control other common intestinal parasites, like roundworms and hookworms. Here again, the product is designed to target migrating larvae of these worms. Once we have adults in the intestines, we'll need other anthelmintics to kill off the adult population. Because a number of intestinal parasites are zoonotic, we would prefer to prevent adult infestations. By preventing adult infestations, we prevent environmental contamination—an infectious source for humans and animals.

But there are some worms, like certain tapeworms, that require a multifaceted approach to control. We use special types of anthelmintics to kill adult tapeworms. But the immature life-stages of tapeworms are not found in our dog, cat, or horse. They are found in what's called an *intermediate host*—where the necessary development of the immature parasite occurs, until it is finally able to infect the final host animal. For example, *Dipylidium* (di"puh-lid'e-um) is a common tapeworm of dogs and cats. The intermediate host for *Dipylidium* is the flea. So, we can kill the adult

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tapeworms in our dog or cat with an anthelmintic. But if we don't also eradicate fleas from the animal and the environment, the pet will likely consume another infected flea and become re-infested with the tapeworm. Of course, that's easy with flea products on the market today. Comparatively, trying to keep a horse from eating infected forage mites, the intermediate host of *Anoplocephala* (an"o-plo-sef'uh-luh), the horse tapeworm, is nearly impossible. Forage mites may be anywhere in the grains or hay that horses eat. So, to minimize disease in the horse, we simply treat periodically with anthelmintics, to keep numbers of *Anoplocephala* to a minimum. Knowledge is a wonderful thing. I could go on and on about parasites (it was my favorite subject to teach). But let's move into some symptomatic pharmaceuticals.

Symptomatic Therapeutics

Symptomatic pharmaceuticals are, as the name implies, used to control the symptoms of disease. They probably will not get to the root of the disease problem. But sometimes symptom control is an important part of supportive care, not only for patient comfort but also to minimize or prevent serious complications.

Antipyretics

NSAIDs can be used as ***antipyretic*** [*anti-* against + *pyr(o)-* fire, fever + *-tic* pertaining to] agents. Both COX1 and COX2 NSAIDs have *antipyretic* properties. However, we typically don't use antipyretics unless absolutely necessary (i.e., if the fever is approaching dangerous levels). Why wait to treat a fever? Well, let's consider a patient who has a fever from a bacterial infection. We'll be treating the infection with antibiotics, right? We'll know that our antibiotic therapy is working when the fever goes down. If we give an antipyretic with the antibiotic, how will we know which drug is actually reducing the fever? We won't. We could actually mask a serious problem with regard to the infection. Plus, as we discussed in Chapter 3, fever is beneficial for immunity. It also slows us down so that we get enough rest for recovery. And unlike us, feeling the need take antipyretics so that we can function at work or school, most animals can take the time to rest and allow their immune

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systems to do what they were designed to do. Truth be told, we should wait at least 24 to 48 hours after a fever breaks naturally before going back to work or school. Masking our fever might make us feel a little better, but it also exposes a lot of other people to us when we're infectious. Just sayin'.

Antitussives

Have you ever had a respiratory infection that made you cough so much and so hard that you couldn't rest and which made you so unbelievably sore that it hurt to breathe? And it seems like the more we cough, the more our airways are irritated, making us cough even more. It can become a vicious catch-22. I actually had a friend who coughed hard enough to crack ribs. That's an out-of-control cough that should be treated with an ***antitussive*** [*anti-* against + *tuss(o)-* cough + *-ive* pertaining to; i.e., cough suppressant] agent.

There are a number of conditions that can stimulate coughing. Many upper and lower airway diseases that do this are discussed in the respiratory section of Chapter 5. In brief, some of those diseases may include pneumonia, asthma, collapsing trachea, heart disease, and infectious diseases like kennel cough in dogs and upper respiratory infections in cats. Now, with a productive cough from a microbial infection, we need to be judicious in our use of antitussives. We don't want to suppress the cough so much that the animal can't physically move all of the mucus and phlegm up and out of their airways. We may also add an ***expectorant*** [*ex-* out + *pector(o)-* chest + -ant one that; i.e., to get out of the chest] to the antitussive. This makes the productivity (removal of airway secretions) more effective when the animal does cough.

But if the cough is nonproductive, a powerful antitussive may be in order. This is frequently the case in dogs with ***kennel cough*** (***infectious tracheobronchitis*** [*trache(o)-* trachea + *bronch(o)-* bronchus + *-itis* inflammation of]). These dogs have loud, honking coughs that keep them and their owners (maybe even the neighbors) awake at night. So, while the dog fights the infection (sometimes viral), we may suppress the cough with an antitussive agent like PO butorphanol. They may need it for only a week or so. Dogs with collapsing tracheas often require chronic therapy with antitussives. Coughing for these animals can actually lead to airway

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obstruction and death. So, controlling the cough is extremely important. Due to that importance, we often use more powerful opioid antitussives, like hydrocodone. And because many of these patients are really small (e.g., Toy Poodles or Yorkshire Terriers), the hydrocodone is usually in a typical "cough syrup" liquid form. This makes accurate dosing possible, usually in volumes less than 1 mL.

Antiemetics and Antidiarrheals

Vomiting and diarrhea are not pleasant. And in young and/or smaller animals, vomiting and diarrhea can rapidly lead to dehydration, low blood pressure, and death. So, we may need to employ ***antiemetics*** [*anti-* against + *eme(o)-* vomit + *-tic* pertaining to] and ***antidiarrheals*** [*anti-* against + *diarrhe(o)-* diarrhea + *-al* pertaining to] to prevent dangerous complications. Of course, every case of vomiting and/or diarrhea does not necessarily warrant symptomatic treatment. We really need to figure out the cause. Numerous GI disorders associated with vomiting and diarrhea are discussed in Chapter 7.

Like I said, we need to know the cause of the vomiting. If chemotherapy in our cancer patient is creating nausea and vomiting, then giving a parenteral antiemetic, like ondansetron, makes sense. That patient is fighting a big enough battle without having to feel nauseated and become dehydrated from vomiting. But a puppy or kitten could be vomiting from an infectious disease (perhaps warranting an antiemetic) or GI obstruction from a foreign body. Puppies and kittens explore the world with their mouths. They may ingest all sorts of strange objects that may obstruct and even perforate the GI tract. So, in the case of an undisclosed foreign body, masking the symptom may mask a very serious problem that requires immediate surgery. Delay of surgery could allow the foreign body to perforate the bowel. And by then, it may be too late to save the patient. Obviously, most of the time with vomiting patients, we will give centrally acting (i.e., in the brain) parenteral antiemetics. There are situations when vomiting, from motion sickness for example, can be prevented with PO medication before "hitting-the-road."

Diarrhea may have a number of causes too. Certain parasites

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cause diarrhea, along with infectious diseases, dietary intolerance, disorders affecting digestion and absorption of nutrients, stress, and immune-mediated disorders, like inflammatory bowel disease. We really need to try to figure out the cause before simply giving an antidiarrheal. For example, we've already talked about antibiotic-induced diarrhea. An antidiarrheal is not going to fix the problem. We need to reestablish the population of commensal bacteria. Sure, an antidiarrheal might slow or stop the diarrhea. But when we stop the antidiarrheal, the diarrhea will return. As with vomiting, if we mask the symptom, we may also mask a much more serious underlying disease process. So, it behooves us to figure out the cause. Unlike many of the other drugs we've talked about, there are only a few antidiarrheals available for use, mostly for dogs, including bismuth subsalicylate, kaolin/pectin, and loperamide. All are PO medications.

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Prescription Writing and Transcribing

Okay, we've waded through an awful lot of information surrounding drugs and their administration. All of this and even more that you'll learn in your pharmacology courses will be extremely important to you and the patients you serve. But I think the writing and transcribing (translating) of prescriptions is really where the "rubber meets the road." I'm a Licensed Veterinary Technician. The scope of my professional license does not permit me to actually prescribe medications. Aha! But I still need to know how to write, interpret, and transcribe what the veterinarian has prescribed. Why? Because I may receive a verbal order (VO) from one of my doctors that I in turn need to record in the patient's medical record. (By the way, I need to precede the actual prescription that I record with the VO and the doctor's name.) If handed a prescription, I need to be able to interpret it so that I can follow through with administration of the medication. And I need to be able to transcribe the prescription in a standardized way on the medication label for the client. The fact that I cannot actually prescribe in no way removes my professional responsibilities for the rest. So, whatever role you play on the veterinary healthcare team, this information is important for you to know.

To help you develop your skills in writing and transcribing, I will discuss some of the requirements of written prescriptions and medication labels, providing an example. Then, I will provide you with practice exercises. You'll need to refer to many of those tables, in earlier sections, to complete those exercises. Let's begin by looking at the standardized structure of a written prescription (Fig. 12.2). Wait. Do we actually give written prescriptions to animal owners? Yes. In fact, this is becoming more common-place today, especially when it comes to narcotics. So, if this prescription is to be filled by the local pharmacy and not our veterinary hospital, it had better have all of the essential information on it in a logical order.

First, the name, address, and phone number for our facility must be printed on the prescription. The name of the prescribing veterinarian must also be printed on it. The signature of the prescribing veterinarian is required, along with their DEA# (Drug

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Enforcement Administration number) for controlled substances. Obviously, the date that the prescription is issued and signed must also be present. Prescriptions will not be honored by the pharmacy indefinitely.

Second, we must supply the patient's information. Because this will be filled by an outside (human) pharmacy, they may require more than just the patient's name. They may also need a medical record (MR) number and the patient's signalment (species, breed, gender, date of birth/age, color/markings). The name, address, and phone number of the owner may also be required, especially if narcotics or other controlled substances are prescribed.

That's a lot of necessary information, and we haven't even talked about the actual drug order yet. There are three key components related to the drug order itself. First, there is the superscription or heading of the prescription that is usually marked with the notation "***R***$_{x}$." That is a very old abbreviation derived from the Latin word *recipere*, meaning "recipe." Here is where we find the name of the drug and its concentration. Often the total volume or number of tablets will be included here as well. Second, the actual orders for administration are preceded by the notation "***Sig***" [from L. *signa* "mark"]. We interpret the notation Sig (or SIG) as meaning "give" (for human patients, "take"). In the case of *transdermal*, topical, ***ophthalmic*** [*ophthalm(o)-* eye + *-ic* pertaining to] or ***otic*** [*ot(o)-* ear + *-ic* pertaining to] medications, we would interpret it to mean "apply". Finally, the ***pharmacist*** [*pharmac(o)-* drug, pharmacy + *-ist* one who specializes in] will need to know if the prescription can be refilled. If so, how many times may it be refilled? There is often a separate space below the administrative orders where the number of refills may be clearly noted.

So, as you can see on our sample prescription (Fig. 12.2), we have all of the necessary information for the doctor and hospital. There are three veterinarians in this facility, all may be listed on our prescription form. We may simply circle the name of the prescribing doctor, to ensure that the correct doctor's name appears and is spelled correctly on the medication label. Because Dr. Smith has prescribed a controlled substance, her DEA number is documented. If this was a prescription for anything other than a controlled substance, this area would be left blank. We have

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included all of the patient and owner information, including the dog's medical record (MR) number and signalment. The drug (codeine), tablet concentration (30 mg/tab), and total number of tablets (30) to be issued are clearly written. The instructions for administration (Sig) indicate a range for dosage (30 to 60 mg), as well as a range for frequency (q6–q8h). The notation "prn for pain" means that the drug can be given less frequently than every 6 to 8 hours, depending on the patient's pain. The medication may be refilled once.

So, how should this prescription appear on the medication label? Well, the information will be simplified. Because the pharmacy, not veterinary practice, is filling the prescription, the pharmacy's name and contact information must be on the label, as well as the pharmacist's initials. The pharmacy must also include a unique prescription number. The doctor's name must be included, but not her DEA#. The date on the label is the actual date filled, not the date on which the prescription was written. The patient information will probably be consolidated to include her name and perhaps her species. The owner's name and address will be included as well. The instructions for administration will precede the actual drug name and concentration and be printed in large, bold text. The number of refills must be included, as well as by what date those refills may be requested (often within one year of the initial prescription, sometimes less). An expiration date for the drug may also be included on the label if it has a short shelf-life. Generally, it is assumed that the drug will expire and should be discarded one year after it is filled. Considering all of that, the medication label for the previous prescription may appear something like the one shown in Fig. 12.3.

Notice how the patient's name, prescription number, and instructions for administration are highlighted with bold text. This provides good visual cues to the owner and helps to ensure that the right animal receives the drug at the right dosage and frequency. The same is true for the pharmacy name, phone, and prescription numbers. This makes it easy for the owner to call with questions or to request refills. Notice that the dosing is listed in number of tablets, not milligrams. Owners cannot be expected to perform dosage calculations. The pharmacist must write the prescribed

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dosing in a clear, easily understood way. Notice too that the frequency on the label is listed precisely as Dr. Smith wrote it in her prescription. If she wanted to give the owner flexibility in frequency, she could have written TID-QID (3 to 4 times daily). She was precise in prescribing q6-q8h. Therefore "every 6 to 8 hours" must appear on the medication label. If there was a question regarding this, the pharmacist would need to call the doctor to verify or request a change. Even if I was filling this prescription in our veterinary facility, I would not be able to alter the veterinarian's prescription without her authorization. Okay, you have all of the basic information you need to record and transcribe prescriptions. Let's put that to use.

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Veterinary Medical Center, P.C.
2500 S. Fork Road, Anytown, MI 01234
(012) 345-6789 Fax (012) 345-6790
J.A. Smith, DVM T.L. Johnson, DVM P.M Jones, DVM

Patient: Serena
MR# 642351 Canine, Gordon Set., FS, 13 yrs
Owner: Dawn Christenson
1234 N. Pine St., Anytown, MI 01234
(012) 345-1212

R_x: Codeine, 30mg tabs, #30

Sig: 30-60mg, PO, q6-8h, prn for pain

Refills: 1

Signature: *Julie A. Smith,* DVM DEA#: 001234501

Date: 01-23-19

FIG. 12.2 Prescription example.

Brown´s Pharmacy, 1565 E. Main St., Anytown, MI 01234
(012) 345-0070

R_x # 555005 Smith, Julie A. DVM 01-24-19 (JMT)

Serena Canine
Dawn Christenson, 1234N. Pine St., Anytown, MI 01234

Give 1 to 2 tablets, by mouth, every 6 to 8 hours, as needed for pain

Codeine 30mg tab Qty 30 tab

Refills – 1 until 01-24-20

FIG. 12.3 Medication label example.

R_x Practice Exercise #1

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You are working the overnight shift caring for a patient (Fuzzball, canine, MN, mix, 3 years old, 30 pounds) in your hospital. He jumped from his owner's moving car today, fracturing his left tibia and sustaining numerous bruises and abrasions. It's 02:00 a.m. and Fuzzball appears to be in a great deal of pain (7/10 on the pain scale) in spite of receiving PO tramadol. When you call Dr. Jones about this, she instructs you to discontinue the tramadol and give the opioid methadone at zero-point-five milligrams per kilogram every four hours intravenously. Our stock solution of methadone is ten millgrams per milliliter. How should you record this verbal order for the new medication in the patient's medical record? (write on a separate piece of paper)

R_x Practice Exercise #2

After administering the methadone to Fuzzball, he appears a little more comfortable. You perceive that his pain level has been reduced to 5/10 on the pain scale. Two hours after administration, his pain seems to be increasing. By 3 hours after administration, his pain level appears to be back up to 7/10. Again, you call Dr. Jones, at 05:00 a.m. She tells you to discontinue the methadone and start an intravenous constant rate infusion of fentanyl at a range of two to five micrograms per kilogram per hour. Our stock solution of fentanyl is fifty micrograms per milliliter. How should you record this verbal order for the new medication in the patient's medical record? (write on a separate piece of paper)

R_x Practice Exercise #3

After surgical repair of Fuzzball's fractured tibia and 3 days of hospitalization, he is ready to be discharged. Dr. Jones is sending Fuzzball home on the following medications: Ampicillin 250 mg cap, Sig: 250 mg PO QID X 10d (#40); Carprofen 25 mg tab, Sig: 25 mg PO q12h pc X 14d (#30); and Tramadol 50 mg tab, Sig: 50 mg PO BID-TID, prn for pain (#60). You need to fill these prescriptions to send home with Fuzzball. How will you complete the administration instructions for each medication on their respective labels?

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Veterinary Medical Center, 2500 S. Fork Rd., Anytown, MI 01234
(012) 345-6789

R_x # 120052 Jones, Pat M. DVM 06-25-19 (DEC)

Fuzzball Canine
Ally Pruitt, 95 W. Putnam St., Anytown, MI 01234

Ampicillin 250mg cap Qty 40 cap

Refills – 0

Veterinary Medical Center, 2500 S. Fork Rd., Anytown, MI 01234
(012) 345-6789

R_x # 120053 Jones, Pat M. DVM 06-25-19 (DEC)

Fuzzball Canine
Ally Pruitt, 95 W. Putnam St., Anytown, MI 01234

Carprofen 25mg tab Qty 30 tab

Refills – 0

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Veterinary Medical Center, 2500 S. Fork Rd., Anytown, MI 01234
(012) 345-6789

R_x # 120054 Jones, Pat M. DVM 06-25-19 (DEC)

Fuzzball Canine
Ally Pruitt, 95 W. Putnam St., Anytown, MI 01234

Tramadol 50 mg tab Qty 60 tab

Refills – 0

Case Study

Bridgēt is a 15-year old, female-spayed, dilute calico, domestic shorthaired cat. The owner brought her in with complaints of chronic vomiting and weight loss. When she is not nauseated and vomiting, Bridgēt eats very well. In fact, the owner said that she is eating more food now than ever before. Per the owner, she seems ravenous, frequently annoying the owner and begging for food. The owner attributed this behavior to Bridgēt's nausea and vomiting. "If Bridgēt isn't vomiting all the time, maybe she'll actually gain some weight," she said. So, the owner requested an antiemetic for Bridgēt.

On physical examination, Bridgēt does appear quite thin. Her body condition score is 2/5, and her body weight is 3.6 kg. She has lost roughly half of her body weight compared to 1 year ago. Other than this, the remainder of her physical examination appears normal.

To investigate her vomiting and weight loss further, the veterinarian ordered a diagnostic ultrasound of Bridgēt's abdomen, with a note to the sonographer to focus on the gastrointestinal system. Bridgēt was quite uncooperative for the abdominal ultrasound. Sedation was needed. So, the veterinarian gave verbal sedation orders for low dose dexmedetomidine (i.e., 0.1 milligrams

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per milliliter). The order is for four micrograms per kilogram intravenously. The intravenous administration of 0.14 mL went smoothly. Fortunately, we only needed to give such a small amount, since Bridgēt struggled and pulled her leg away immediately after the injection was complete. Very soon after, Bridgēt was resting comfortably, sleeping through most of the abdominal ultrasound. The abdominal ultrasound appeared to be within normal limits. After drawing blood for laboratory analysis, 0.14 mL of the antagonist (atipamezole) was administered to Bridgēt. Her recovery from sedation was quick, permitting the owner to take her home within 15 minutes after the ultrasound was complete. Most of Bridgēt's laboratory data appeared quite normal, except thyroid values, which were elevated. Here are the reported findings:

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Test	Patient	Reference range
Hematology		
PCV	36%	34%–51%
TS	7.0 g/dL	6.5–8.5 g/dL
Hgb	12.1 g/dL	11.9–17.5 g/dL
RBC	7.9×10^6	$7.9–11.6 \times 10^6$
MCV	46 fL	38–52 fL
MCH	16 pg	13–17 pg
MCHC	33 g/dL	33–37 g/dL
Platelets	$265 \times 10^3/\mu L$	$179–569 \times 10^3/\mu L$
WBC	$12.7 \times 10^3/\mu L$	$4.4–15.6 \times 10^3/\mu L$
Neutrophils (seg)	$6.5 \times 10^3/\mu L$	$2.4–11.3 \times 10^3/\mu L$
Neutrophils (band)	0	$0.0–0.1 \times 10^3/\mu L$
Lymphocytes	$5.0 \times 10^3/\mu L$	$0.5–6.4 \times 10^3/\mu L$
Monocytes	$0.4 \times 10^3/\mu L$	$0.0–0.6 \times 10^3/\mu L$
Eosinophils	$0.6 \times 10^3/\mu L$	$0.0–1.4 \times 10^3/\mu L$
Basophils	$0.2 \times 10^3/\mu L$	$0.0–0.2 \times 10^3/\mu L$
Serum Chemistry		
Total Protein	7.2 g/dL	6.5–7.8 g/dL
Albumin	3.3 g/dL	3.0–3.9 g/dL
Globulin	3.9 g/dL	2.8–4.7 g/dL
Glucose	104 mg/dL	78–143 mg/dL
BUN	36 mg/dL	19–36 mg/dL
Creatinine	1.5 mg/dL	1.0–2.3 mg/dL
ALT	75 U/L	25–76 U/L
AST	34 U/L	14–36 U/L
ALP	48 U/L	13–48 U/L
Total bilirubin	0.1 mg/dL	0.1–0.3 mg/dL
Amylase	957 U/L	476–1779 U/L
Ca^{++}	10.3 mg/dL	9.1–10.7 mg/dL
Cl^-	114 mmol/L	110–123 mmol/L
K^+	3.8 mmol/L	3.8–5.4 mmol/L
Mg^+	2.2 mg/dL	1.8–2.5 mg/dL
Na^+	152 mmol/L	145–155 mmol/L
Phos	5.1 mg/dL	2.7–5.7 mg/dL
Iron	123 µg/dL	45–157 µg/dL
CK	275 U/L	46–490 U/L
Chol	149 mg/dL	72–248 mg/dL
Serum Thyroid		
TT3 (total thyroxine)	91 nmol/L **H**	10–47 nmol/L
TT4 (total triiodothyronine)	2.0 nmol/L **H**	0.6–1.4 nmol/L
FT3 (free thyroxine)	82 pmol/L **H**	10–53 pmol/L
FT4 (free triiodothyronine)	6.3 pmol/L **H**	0.3–2.9 pmol/L

Based on the laboratory data, Dr. Lang made the diagnosis of hyperthyroidism. She called the owner to inform her of the diagnosis. She also informed the owner that she ordered medication to treat the condition from a compounding pharmacy. She faxed the following prescription to the compounding pharmacy:

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Veterinary Medical Center, P.C.
2500 S. Fork Road, Anytown, MI 01234
(012) 345-6789 Fax (012) 345-6790
N.M. Lang, DVM T.L. Jones, DVM E.L. English, DVM

Patient: Bridget
MR# 695318 Feline, DSH, FS, 15 yrs, dilute calico
Owner: Doris Allen
563 E. Paris Ave., Anytown, MI 01234
(012) 345-1212

R_x: Methimazole 5mg/mL susp. (60mL, chicken flavor)

Sig: 0.5mL PO q12h

Refills: 6

Signature: *Nyssa M. Lang*, DVM DEA#: _____

Date: 03-15-19

Bridgēt was rechecked after 6 weeks of methimazole therapy. Her owner reported that Bridgēt's appetite was still very good, but not as ravenous as before. Bridgēt only vomits on rare occasions now. At the time of the recheck, Bridgēt's body weight was 4.6 kg. Blood was drawn for repeated serum chemistry and thyroid tests. Both appeared within normal limits. So, Dr. Lang called Bridgēt's owner, telling her to reduce Bridgēt's dose to 0.4 mL PO q12h. She also told the owner to bring Bridgēt back in 6 weeks for a recheck.

Case Study Questions

1. Due to the patient's persistent vomiting, the owner requested an _____ agent to inhibit the vomiting.
2. Bridgēt was not cooperative for her abdominal ultrasound. So, the veterinarian ordered dexmedetomidine for sedation. How should the entire prescription, including the drug and the administration orders, be recorded in the patient's medical record? (write on a separate piece of paper)
3. Following the abdominal ultrasound, atipamezole was

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given to reverse the effects of the dexmedetomidine. The medical term for a reversal agent like this is _____.

4. In the hematology report, the MCV value was 46 fL. What is the medical term for the abbreviation fL?

   _____

5. In the hematology report, the MCH value was 16 pg. What is the medical term for the abbreviation pg?

   _____

6. In the hematology report, the WBC was reported as 12.7 × 10^3/μL. What is the medical term for the abbreviation μL? _____

7. In the serum chemistry report, the BUN was 36 mg/dL. What is the medical term for the abbreviation dL?

   _____

8. In the serum chemistry report, the calcium value was 10.3 mg/dL. What is the medical term for the abbreviation mg? _____

9. In the written prescription that Dr. Lang faxed to the compounding pharmacy, what is the concentration of the methimazole? _____

10. In the written prescription for methimazole, what is the form of the drug that has been prescribed, abbreviated susp.? ____

11. How should the instructions for administration of the drug be written on the medication label for the owner? (write on a separate piece of paper)

12. Bridget's body weight was recorded in kg, which is the abbreviation for _____.

13. What portion of a liter is a mL? _____

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14. What portion of a gram is a mcg? _____

15. How many micrograms are in 1 mg? _____

16. BONUS: What is the apothecary equivalent of 1 kg?

_____

The Answer Key to these case study questions may be found in Appendix B.

[a] www.WorldAtlas.comAt the time of this writing, all but three countries around the world use the metric system as their standard unit of measure. The United States is among the three countries that do not.

[b] Cinderella: a popular folk tale told and written many times by many people. The most popular version of the story was written by French author, Charles Perrault, in 1697. The Brother's Grimm from Germany put their twist on the tale and included it in their collection of Grimm's Fairy Tales in 1812. Numerous theatrical productions and movies have also captured the tale.

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APPENDIX A

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Chemical Symbol–Element Cross Reference

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Atomic No.	Symbol	Name
1	H	hydrogen
2	He	helium
3	Li	lithium
4	Be	beryllium
5	B	boron
6	C	carbon
7	N	nitrogen
8	O	oxygen
9	F	fluorine
10	Ne	neon
11	Na	sodium
12	Mg	magnesium
13	Al	aluminum
14	Si	silicon
15	P	phosphorus
16	S	sulfur
17	Cl	chlorine
18	Ar	argon
19	K	potassium
20	Ca	calcium
21	Sc	scandium
22	Ti	titanium
23	V	vanadium
24	Cr	chromium
25	Mn	manganese
26	Fe	iron
27	Co	cobalt
28	Ni	nickel
29	Cu	copper
30	Zn	zinc
31	Ga	gallium
32	Ge	germanium
33	As	arsenic
34	Se	selenium
35	Br	bromine
36	Kr	krypton
37	Rb	rubidium
38	Sr	strontium
39	Y	yttrium
40	Zr	zirconium
41	Nb	niobium
42	Mo	molybdenum
43	Tc	technetium
44	Ru	ruthenium
45	Rh	rhodium
46	Pd	palladium
47	Ag	silver
48	Cd	cadmium
49	In	indium
50	Sn	tin
51	Sb	antimony
52	Te	tellurium

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Atomic No.	Symbol	Name
53	I	iodine
54	Xe	xenon
55	Cs	cesium
56	Ba	barium
57	La	lanthanum
58	Ce	cerium
59	Pr	praseodymium
60	Nd	neodymium
61	Pm	promethium
62	Sm	samarium
63	Eu	europium
64	Gd	gadolinium
65	Tb	terbium
66	Dy	dysprosium
67	Ho	holmium
68	Er	erbium
69	Tm	thulium
70	Yb	ytterbium
71	Lu	lutetium
72	Hf	hafnium
73	Ta	tantalum
74	W	tungsten
75	Re	rhenium
76	Os	osmium
77	Ir	iridium
78	Pt	platinum
79	Au	gold
80	Hg	mercury
81	Tl	thallium
82	Pb	lead
83	Bi	bismuth
84	Po	polonium
85	At	astatine
86	Rn	radon
87	Fr	francium
88	Ra	radium
89	Ac	actinium
90	Th	thorium
91	Pa	protactinium
92	U	uranium
93	Np	neptunium
94	Pu	plutonium
95	Am	americium

Table Continued

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Atomic No.	Symbol	Name
96	Cm	curium
97	Bk	berkelium
98	Cf	californium
99	Es	einsteinium
100	Fm	fermium
101	Md	mendelevium
102	No	nobelium
103	Lr	lawrencium
104	Rf	rutherfordium
105	Ha	hahnium
106	Sg	seaborgium
107	Bh	bohrium
108	Hs	hassium
109	Mt	meitnerium

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APPENDIX B

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Case Study and Practice Exercise Answer Keys

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Chapter 1

Case Study Answer Key

1. Mediolateral
2. Lateral
3. Right lateral recumbency
4. Signalment
5. Dorsopalmar
6. Dorsoplantar
7. Anteroposterior
8. Lateral recumbency
9. Craniocaudal
10. Proximal
11. Ventrodorsal
12. Sternal recumbency
13. Sonography
14. Medial
15. Bilateral

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Chapter 2

Case Study Answer Key

1. Hypotensive
2. Hypokalemia
3. Hypoglycemia
4. Hydration
5. Isotonic
6. Hypertonic
7. Homeostasis
8. Dehydration
9. Hyponatremia
10. Acidosis
11. Electrolytes
12. Symbiotic
13. Alkalosis
14. Pathogenic
15. Phagocytosis

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Chapter 3

Case Study Answer Key

1. Icterus
2. Splenomegaly
3. Anisocytosis
4. Anemia
5. Lymphopenia
6. Leukocytosis
7. Metarubricyte
8. Monocytosis
9. Polychromasia
10. Agglutination
11. Autoimmune
12. Anticoagulant
13. Thrombosis
14. Thrombocytosis
15. Immunosuppressive
16. Immunoglobulins
17. Pyrexia
18. Splenectomy
19. Hemorrhage
20. Neutrophilia

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Chapter 4

Case Study Answer Key

1. Musculoskeletal
2. Bilateral
3. Femorotibial
4. Tarsus
5. Cruciate ligament
6. Osteoarthritis
7. Osteotomy
8. Condyles
9. Articular
10. Arthritis

Bovine Musculoskeletal Challenge Answer Key

1. Distal interphalangeal joints (right rear)
2. Metatarsophalangeal joints (right)
3. Cannon bone (right rear)
4. Tarsus (right)
5. Calcaneus (right)
6. Tibia (right)
7. Femorotibial joint (right)
8. Femur (right)
9. Coxofemoral joint (right)
10. Greater trochanter of femur (right)
11. Ischiatic tuberosity (right)
12. Sacrum
13. Tuber coxae (right)
14. Lumbar vertebrae
15. Rib
16. Costochondral junction
17. Xiphoid (xyphoid) process

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18. Olecranon (right)
19. Knee (right)
20. Third and fourth metacarpals (right)
21. Proximal sesamoid bones (right front)
22. Proximal interphalangeal joints (right front)
23. Fetlock (right front)
24. Mandible
25. Maxilla
26. Orbit
27. Atlantooccipital joint
28. Cervical vertebrae
29. Ligamentum nuchae
30. Scapula (right)
31. Scapulohumeral joint (right)
32. Humerus (right)

Canine Musculoskeletal Challenge Answer Key

1. Maxilla
2. Mandible
3. Cervical vertebrae
4. Thoracic vertebrae
5. Lumbar vertebrae
6. Sacrum
7. Coccygeal vertebrae
8. Ilium (left)
9. Ribs
10. Costochondral junction
11. Xiphoid (xyphoid) process
12. Sternum
13. Scapula (left)
14. Scapulohumeral joint (left)
15. Humerus (left)
16. Humeroradioulnar joint (left)
17. Radius (left)
18. Ulna (left)

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19. Carpus (left)
20. Metacarpals (left) or metacarpal bones
21. Metacarpophalangeal joints (left)
22. Distal interphalangeal joints (left)
23. Distal phalanx, 3rd phalanx, or ungual process (left)
24. Coxofemoral joint (left)
25. Femur (left)
26. Ischiatic tuberosity (left)
27. Femorotibial joint (left)
28. Fibula (left)
29. Tibia (left)
30. Patella (left)
31. Metatarsals (left) or metatarsal bones
32. Phalanges (left rear)
33. Lumbar epaxial muscles
34. Semimembranosus-semitendinosus muscles

Equine Musculoskeletal Challenge Answer Key

1. Ligamentum nuchae
2. Cervical vertebrae
3. Thoracic vertebrae
4. Lumbar vertebrae
5. Sacrum
6. Scapula (left)
7. Shoulder (left)
8. Humerus (left)
9. Elbow (left)
10. Radius (and ulna, left)
11. Knee (left)
12. Third metacarpal bone (left)
13. Metacarpophalangeal joint (left)
14. First phalanx, or proximal phalanx (left front)
15. Short pastern (left front)
16. Coffin joint (left front)
17. Third phalanx, or distal phalanx (left front)

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18. Distal sesamoid bone (left front); *note: "navicular bone" could be acceptable, as the name is derived from the Latin navicula. Students may perceive this as "scientific."*
19. Proximal interphalangeal joint (left front)
20. Proximal sesamoid bones (left front)
21. Tuber coxae (left)
22. Hip (left)
23. Greater trochanter of femur (left)
24. Ischiatic tuberosity (left)
25. Femur (left)
26. Stifle (left)
27. Tibia (left and fibula)
28. Cannon bone (left rear)
29. Fetlock (left rear)
30. Pastern joint (left rear)
31. Distal interphalangeal joint (left rear)
32. Coffin bone (left rear)
33. Second phalanx (left rear)
34. Long pastern (left rear)
35. Lateral neck muscles (left)
36. Gluteal muscles (left)
37. Semimembranosus-semitendinosus muscles (left)
38. Triceps muscles (left)

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Chapter 5

Case Study Answer Key

1. Dyspnea
2. Orthopnea
3. Tachycardic
4. Cyanotic
5. Hypoxia
6. Hypercapnic
7. Electrocardiography
8. Intranasal
9. Intravenous
10. Bronchoalveolar lavage
11. Pneumonia
12. Antimycotic
13. Nebulization
14. Coupage
15. Oximetry or oxymetry

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Chapter 6

Case Study Answer Key

1. Urolithiasis
2. Hematuria
3. Pyuria
4. Bacteriuria
5. Cystitis
6. Urolith
7. Cystotomy
8. Dysuria
9. Pollakiuria
10. Cystourethrogram

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Chapter 7

Case Study Answer Key

1. Hypotension
2. Tympany
3. Trocarization
4. Gastric dilatation volvulus
5. Laparotomy
6. Gastrectomy
7. Splenectomy
8. Necrosis
9. Gastropexy
10. Orogastric
11. Postprandial
12. Peritoneal

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Chapter 8

Case Study Answer Key

1. Dermatitis
2. Otitis
3. Pruritus
4. Pyoderma
5. Pododermatitis
6. Purulent
7. Urticaria
8. Atopy
9. Intradermal
10. Allergen
11. Antihistamine
12. Chronic

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Chapter 9

Case Study Answer Key

1. Gestation
2. Dystocia
3. Hypocalcemia
4. Pseudocyesis
5. Hypoglycemia
6. Polydipsia
7. Polyuria
8. Pyometra
9. Peritonitis or septic peritonitis
10. Ovariohysterectomy

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Chapter 10

Case Study Answer Key

1. Hypoglycemia
2. Hyperkalemia
3. Hyponatremia
4. Polyuria
5. Hypotension
6. Acidosis
7. Adrenocorticotropic hormone
8. Hypoadrenocorticism
9. Adenohypophysis
10. Pancreatitis
11. Hyperglycemic
12. Insulin

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Chapter 11

Case Study Answer Key

1. Quadriparesis or tetraparesis
2. Myelopathy
3. Encephalopathy
4. Anisocoria
5. Nystagmus or horizontal nystagmus
6. Olfactory
7. Auditory
8. Ataxia
9. Mesencephalon
10. Otitis
11. Idiopathic
12. Vestibular

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Chapter 12

Case Study and Rx Practice Exercise Answer Keys

1. Antiemetic
2. VO per Dr. Lang; Rx dexmedetomidine 0.1 mg/mL; Sig: 4 mcg/kg IV (the date, time, and your initials should also be included)
3. Antagonist
4. Femtoliter(s)
5. Picogram(s)
6. Microliter
7. Deciliter
8. Milligram
9. 5 mg/mL
10. Suspension
11. Give 0.5 mL by mouth every 12 hours (mL may be abbreviated to match the notations on the syringe)
12. Kilograms
13. One thousandth or 1/1000 or 0.001
14. One millionth or 1/1,000,000 or 0.000,001
15. 1000
16. 2.2 pounds

Rx Practice #1 Answer Key

VO Dr. Jones, 02:00 a.m., Rx Methadone 10 mg/mL, Sig: 0.5 mg/kg IV q4h (the date and your initials should also included)

Rx Practice #2 Answer Key

VO Dr. Jones, 05:00 a.m., Rx Fentanyl 50 mcg/mL, Sig: 2–5 mcg/kg/h IV CRI (the date and your initials should also be included)

Rx Practice #3 Answer Key

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Veterinary Medical Center, 2500 S. Fork Rd., Anytown, MI 01234
(012) 345-6789

R_x # 120052 Jones, Pat M. DVM 06-25-19 (DEC)

Fuzzball Canine

Ally Pruitt, 95 W. Putnam St., Anytown, MI 01234

Give 1 capsule by mouth 4 times per day for 10 days

Ampicillin 250mg cap Qty 40 cap

Refills – 0

Veterinary Medical Center, 2500 S. Fork Rd., Anytown, MI 01234
(012) 345-6789

R_x # 120053 Jones, Pat M. DVM 06-25-19 (DEC)

Fuzzball Canine

Ally Pruitt, 95 W. Putnam St., Anytown, MI 01234

Give 1 tablet by mouth every 12 hours after meals

Carprofen 25mg tab Qty 30 tab

Refills – 0

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Veterinary Medical Center, 2500 S. Fork Rd., Anytown, MI 01234
(012) 345-6789

R_x # 120054 Jones, Pat M. DVM 06-25-19 (DEC)

Fuzzball Canine

Ally Pruitt, 95 W. Putnam St., Anytown, MI 01234

Give 1 tablet by mouth 2 to 3 times per day, as needed for pain

Tramadol 50 mg tab Qty 60 tab

Refills – 0

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APPENDIX C

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Tables of Introductory Terms

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Medical Terms Introduced in Chapter 1

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Chapter 1 Introductory Terms			
Term	**Phonetics**	**Division**	**Basic Definition**
Anechoic	an”ĕ-ko’ik	**an/echo/ic**	**Pertaining to no/absent echo**
Anterior	an-te’re-or	**anter/i/or**	**Pertaining/referring to the front**
Caudal	kaw’dal	**caud/al**	**Pertaining to the tail**
Caudocranial	kaw’do-kra”ne-al	**caud/o/crani/al**	**Pertaining to tail to head**
Contralateral	kon”trah-lat’er-al	**contra/later/al**	**Pertaining to opposite side**
Cranial	kra’ne-al	**crani/al**	**Pertaining to the head**
Cranium	kra’ne-um	**crani/um**	**The head**
Craniocaudal	kra’ne-o-kaw”dal	**crani/o/caud/al**	**Pertaining to head to tail**
Distal	dis’tal	**dist/al**	**Pertaining to farthest**
Dorsal	dor’sal	**dors/al**	**Pertaining to the back (dorsum)**
Dorsopalmar	dor’so-pal”mar	**dors/o/palm/ar**	**Pertaining to back to palm**
Dorsoplantar	dor’so-plan”tar	**dors/o/plant/ar**	**Pertaining to back to sole**
Dorsoventral	dor’so-ven”tral	**dors/o/ventr/al**	**Pertaining to back to belly**
Echogenic	ek”o-jen’ik	**echo/gen/ic**	**Pertaining to echo producing**
Echogenicity	ek”o-jen-is’ĭ-te	**echo/gen/icity**	**Characteristic of echo production**
Heterogenous	het”er-oj’ĕ-nes	**heter/o/gen/ous**	**Pertaining to difference producing**
Homogenous	hōm”oj’ĕ-nes	**homo/gen/ous**	**Pertaining to sameness producing**
Hyperechoic	hi”per-ĕ-ko’ik	**hyper/echo/ic**	**Pertaining to excessive echo**
Hypoechoic	hi-po”ĕ-ko’ik	**hypo/echo/ic**	**Pertaining to decreased echo**
Isoechoic	i-so”ĕ-ko’ik	**iso/echo/ic**	**Pertaining to same echo**
Lateral	lat’er-al	**later/al**	**Pertaining to the side**
Lateromedial	lat’er-o-me”de-al	**later/o/medi/al**	**Pertaining to side to middle**
Medial	me’de-al	**medi/al**	**Pertaining to the middle**
Mediolateral	me’de-o-lat”er-al	**medi/o/later/al**	**Pertaining to middle to side**
Midsagittal	mid”saj’ĭ-tal	**mid/sagitt/al**	**Pertaining to middle “arrow”/plane**
Palmar	pal’mar	**palm/ar**	**Pertaining to the “palm”**
Plantar	plan’tar	**plant/ar**	**Pertaining to the sole**
Posterior	pos-tēr’e-or	**poster/i/or**	**Pertaining to the rear**
Proximal	prok’sĭ-mal	**proxim/al**	**Pertaining to nearest**
Rostral	ros’tral	**rostr/al**	**Pertaining to the nose/beak**
Sagittal	saj’ĭ-tal	**sagitt/al**	**Pertaining to [straight as] an “arrow”**
Transect	tran-sekt’	**trans/sect**	**To cut across (cross cut)**
Ventral	ven’tral	**ventr/al**	**Pertaining to the “belly”**
Ventrodorsal	ven”tro-dor’sal	**ventr/o/dors/al**	**Pertaining to belly to back**

∗Color legend: **root word**, **prefix**, **suffix**, combining vowel

This table contains most but not all terms introduced in this chapter.

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Medical Terms Introduced in Chapter 2

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Chapter 2 Introductory Terms			
Term	**phonetics**	**Division**	**Basic Definition**
Anaphase	an'ah-fāz	**ana/phase**	**A, the up, back, again stage**
Atomic	ă-tom'ik	**atom/ic**	**Pertaining to atoms**
Biochemistry	bi"o-kem'is-tre	**bi/o/chemistry**	**A life chemistry**
Biologic	bi-o-loj'ik	**bi/o/log/ic**	**Pertaining to life study, knowledge**
Cellular	sel'u-lar	**cellul/ar**	**Pertaining to cells**
Centriole	sen'tre-ōl	**centr/i/ole**	**A, the small center**
Centromere	sen'tro-mēr	**centr/o/mere**	**A, the central part**
Chromosomal	kro'mo-so"mal	**chrom/o/som/al**	**Pertaining to colored bodies**
Covalent	co-va'lent	**co/val/ent**	**One that is together strong**
Cytology	si-tol'o-je	**cyt/o/logy**	**The study of cells**
Cytoplasmic	si"to-plaz'mik	**cyt/o/plasm/ic**	**Pertaining to cell matter**
Dehydration	de"hi-dra'shun	**de/hydr/a/tion**	**A condition away from/reduced water**
Electron	e-lek'tron	**electr/on**	**A unit (of) electricity**
Electrolyte	e-lek'tro-līt"	**electr/o/lyte**	**An electron / that may be dissolved**
Electrovalent	e-lek'tro-va"lert	**electr/o/val/ent**	**One that is electron/electrically strong**
Endoplasmic	en"do-plaz'mik	**endo/plasm/ic**	**Pertaining to within matter**
Exocytosis	eks"o-si-to'sis	**exo/cyt/o/sis**	**The process of outing/ousting (by) cells**
Extracellular	eks"trah-sel'u-lar	**extra/cellul/ar**	**Pertaining to outside cells**
Homeostasis	ho"me-o-sta'sis	**home/o/stasis**	**State of standing unchanged**
Hydration	hi-dra'shun	**hydr/a/tion**	**The state of having water**
Hypertonic	hi"per-ton'ik	**hyper/ton/ic**	**Pertaining to excessive tension**
Hypoglycemia	hi"po-gli-se'me-uh	**hypo/glyc/emia**	**A blood condition of low sugar**
Hypokalemia	hi"po-ka-le'me-uh	**hypo/kal/emia**	**A blood condition of low potassium**
Hyponatremia	hi"po-na-tre'me-uh	**hypo/natr/emia**	**A blood condition of low sodium**
Hypotension	hi"po-ten'shun	**hypo/tens/ion**	**A state of low tension (i.e., pressure)**
Hypotonic	hi-po"ton'ik	**hypo/ton/ic**	**Pertaining to under, less, low tension**
Inorganic	in"or-gan'ik	**in/organ/ic**	**Pertaining to not carbon-containing**
Intercellular	in"ter-sel'u-lar	**inter/cellul/ar**	**Pertaining to between cells**
Intracellular	in"trah-sel'u-lar	**intra/cellul/ar**	**Pertaining to within cells**
Ionic	i-on'ik	**ion/ic**	**Pertaining to ions**
Isotonic	i-so"ton'ik	**iso/ton/ic**	**Pertaining to equal tension**
Lysosomal	li"so-so'mal	**lys/o/som/al**	**Pertaining to a dissolving body**
Macrophage	mak'ro-fahj"	**macr/o/phage**	**A, the large eater**
Metaphase	met'ah-fāz	**meta/phase**	**A, the after stage**
Microscopic	mi"kro-skop'ik	**micro/scop/ic**	**Pertaining to small view**
Mitosis	mi-to'sis	**mit/o/sis**	**Condition/Process of "threads"**
Molecular	mo-lek'u-lar	**molecul/ar**	**Pertaining to molecules**
Neutron	nu'tron	**neutr/on**	**A unit (of) neither**
Nuclear	nu'kle-ar	**nucle/ar**	**Pertaining to a nucleus**
Nucleolus	nu"kle-o'lus	**nucleol/us**	**A, the small nucleus**
Nucleus	nu'kle-us	**nucle/us**	**A, the [dim. L. *nux* "nut"] nucleus**
Organelle	or"gan-el'	**organ/elle**	**A small organ**
Organic	or-gan'ik	**organ/ic**	**Pertaining to carbon-containing**
Osmosis	oz-mo'sis	**osm/o/sis**	**Process or condition of impulsion**
Osmotic	oz-mah'tik	**osm/o/tic**	**Pertaining to impulsion (i.e., osmosis)**
Pathogenic	path"o-jen'ik	**path/o/gen/ic**	**Pertaining to disease producing**
Pathologic	păth-o-loj'ik	**path/o/log/ic**	**Pertaining to disease study/knowledge**

Table Continued

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Chapter 2 Introductory Terms			
Term	**phonetics**	**Division**	**Basic Definition**
Phagocytosis	fag”o-si-to’sis	**phag/o/cyt/o/sis**	**The process of eating** (by) **cells**
Physiology	fiz”e-ol’o-je	**physi/o/logy**	**The study of nature** (i.e., natural processes)
Prophase	pro’fāz	**pro/phase**	**A, the before stage**
Proton	pro’ton	**prot/on**	**A unit first or primitive**
Reticular	rĕ-tik’u-lar	**reticul/ar**	**Pertaining to a net, network**
Reticulum	rĕ-tik’u-lum	**reticul/um**	**A, the net, network**
Ribosomal	ri’bo-so”mal	**rib/o/som/al**	**Pertaining to RNA bodies**
Subatomic	sub”a-tom’ik	**sub/atom/ic**	**Pertaining to under atoms**
Symbiotic	sim”bi-ot’ik	**sym/bi/o/tic**	**Pertaining to together, with life/living**
Telophase	te’lo-fāz	**telo/phase**	**A, the end stage**
Vacuole	vak’u-ōl	**vacu/ole**	**A small “emptiness”**

*Color legend: **root word**, **prefix**, **suffix, combining vowel**

This table contains most but not all terms introduced in this chapter.

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Medical Terms Introduced in Chapter 3

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Chapter 3 Introductory Terms			
Term	**Phonetics**	**Division**	**Basic Definition**
Acanthocyte	a-kan'tho-sīt	**acanth/o/cyte**	**A thorn cell**
Agglutination	uh-gloo"tĭ-na'shun	**agglutin/a/tion**	**Process of gluing**
Agranulocyte	a-gran'u-lo-sīt"	**a/granul/o/cyte**	**A without granules cell**
Allergen	al'er-jen	**aller/gen**	**An allergy producer**
Allergenic	al"er-jen'ik	**aller/gen/ic**	**Pertaining to allergy production**
Anamnestic	an"am-nes'tik	**anamnes/tic**	**Pertaining to recalling/memory**
Anemia	uh-ne'me-ah	**an/em/ia**	**Condition without** (deficient) **blood**
Anisocytosis	an-e"so-si-to'sis	**anis/o/cyt/o/sis**	**Condition of varied cells**
Anticoagulant	an"te-ko-ag'u-lant; an"ti-ko-ag'u-lant	**anti/coagul/ant**	**One that is against clotting**
Antigen	an'tĭ-jen	**anti/gen**	**Antibody producer**
Antigenic	an"tĭ-jen'ik	**anti/gen/ic**	**Pertaining to antibody producer**
Antihistamine	an"te-his'tuh-mēn an"ti-his'tuh-mēn	**anti/histamine**	**Against histamine**
Anuclear	a-noo'kle-ar	**a/nucle/ar**	**Pertaining to without a nucleus**
Autoantibody	aw"to-an'tĭ-bod"e	**auto/antibody**	**Self antibody**
Autoimmune	aw"to-ĭ-mūn'	**auto/immune**	**Self exemption/protection**
Axillary	ak'sĭ-lar"e	**axill/ary**	**Pertaining to the axilla**
Basophil	ba'so-fil	**bas/o/phil**	**Blue loving/lover**
Basophilia	ba"so-fil'e-uh	**bas/o/phil/ia**	**Condition of blue loving**
Basophilic	ba"so-fil'ik	**bas/o/phil/ic**	**Pertaining to blue loving**
Bilirubinemia	bil"e-roo"bĭ-ne'me-uh, bil"ĭ-roo"bĭ-ne'me-uh	**bilirubin/emia**	**A blood condition of "bile"**
Carboxyhemoglobin	kar-bok"se-he'mo-glo"bin	**carb/oxy/hem/o/glob/in**	**The carbon oxygen blood "stuff"**
Centrifuge	sen'trĭ-fūj	**centr/i/fuge**	**A putting to flight/driving away (from) center**
Cervical	sur'vĭ-kal	**cervic/al**	**Pertaining to the neck**
Chemotaxis	ke"mo-tak'sis	**chem/o/taxis**	**Movement stimulated (by) chemicals**
Coagulation	ko-ag"u-la'shun	**coagul/a/tion**	**Process of clotting**
Coagulopathy	ko-ag"u-lop'uh-the	**coagul/o/pathy**	**A disease of clotting**
Cytotoxic	si"to-tok'sik	**cyt/o/tox/ic**	**Pertaining to cell poison**

Table Continued

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Chapter 3 Introductory Terms			
Term	**Phonetics**	**Division**	**Basic Definition**
Deoxyhemoglobin	de-ok"se-he'mo-glo"bin	**deoxy/hem/o/glob/in**	**The reduced oxygen blood "stuff"**
Dermatitis	der"mah-ti'tis	**dermat/itis**	**Inflammation of the skin**
Echinocyte	e-ki'no-sit	**echin/o/cyte**	**A hedgehog cell**
Edematous	ĕ-dem'uh-tus	**edema/tous**	**Pertaining to swelling**
Eosinophil	e-o-sin'o-fil	**eosin/o/phil**	**Red loving/lover**
Eosinophilia	e-o-sin"o-fil'e-uh	**eosin/o/phil/ia**	**Condition of eosinophils (excess)**
Erythematous	ĕr"uh-them'ŭ-tus	**erythema/tous**	**Pertaining to redness**
Erythrocyte	e-rith'ro-sit	**erythr/o/cyte**	**A red cell**
Erythropoictin	e-rith"ro-poi'ĕ-tin	**erythr/o/poie/tin**	**The red producer**
Extravascular	ek"strŭ-vas'ku-lar	**extra/vascul/ar**	**Pertaining to outside vessels**
Fibrinogen	fi-brin'o-gen	**fibrin/o/gen**	**A fiber producer**
Granulocyte	gran'u-lo-sit	**granul/o/cyte**	**A granule cell**
Hematocrit	he-mă'to-krit"	**hemat/o/crit**	**Blood separation**
Hematology	hĕm"ah-tol'o-je	**hemat/o/logy**	**Study of blood**
Hematoma	he"mah-to'mah	**hemat/oma**	**A "tumor"/"swelling"of blood**
Hematopoietic	hem"uh-to-poi-et' ik	**hemat/o/poie/tic**	**Pertaining to blood production**
Hemoglobin	he'mo-glo"bin	**hem/o/glob/in**	**The blood glob/"stuff"**
Hemolysis	he-mol'uh-sis	**hem/o/lysis**	**Process of breaking blood**
Hemolytic	he-mo-lit'ik	**hem/o/lytic**	**Pertaining to breaking blood**
Hemolyze	he'mo-liz	**hem/o/lyze**	**Act of breaking blood**
Hemorrhage	hem'or-ij	**hem/o/rrhage**	**Escaping blood** (i.e., bleeding)
Hemostasis	he"mo-sta'sis	**hem/o/stasis**	**Process of stopping blood/bleeding**
Hyperbilirubinemia	hi"per-bil"e-roo"bĭ-ne'me-uh	**hyper/bilirubin/emia**	**A blood condition of excess bilirubin**
Hypersensitivity	hi"per-sen'sĭ-tiv"ĭ-te	**hyper/sensitiv/ity**	**State of being overly sensitive**
Hypoallergenic	hi"po-al"er-jen'ik	**hypo/allerg/gen/ic**	**Pertaining to low allergy producing**
Hypoproteinemia	hi"po-pro"tĕ-ne'me-uh	**hypo/protein/emia**	**A blood condition of low protein**
Hyposensitization	hi"po-sen"si-tĭ-za'shun	**hypo/sensitiz/a/tion**	**Process of lowering sensitivity**
Icteric	ik'ter-ik	**icter/ic**	**Pertaining to jaundice**
Icterus	ik'ter-us	**icter/us**	**The jaundice** (yellowing)
Immunization	im"u-nĭ-za'shun	**immuniz/a/tion**	**Process of giving protection**
Immunodeficient	im"u-no-dĕ-fish'ent	**immun/o/deficient**	**Protection low/deficient**
Immunoglobulin	im"u-no-glob'u-lin	**immun/o/globul/in**	**The protection "blob"/globule**
Immunology	im"u-nol'o-je	**immun/o/logy**	**Study of exemption/protection**
Immunosuppressant	im"u-no-sŭ-pres'ant	**immun/o/suppress/ant**	**One that is protection reducing**
Immunosuppressive	im"u-no-sŭ-pres'iv	**immun/o/suppress/ive**	**Pertaining to protection suppression/reduction**
Inflammation	in"fluh-ma'shun	**inflamm/a/tion**	**Process/condition of fire**
Inflammatory	in-flam'uh-tor"e	**inflamm/a/tory**	**Pertaining to inflammation/"fire"**
Inguinal	ing'gwĭ-nal	**inguin/al**	**Pertaining to the groin**
Interleukin	in"ter-loo'kin	**inter/leuk/in**	**The between white**
Interstitial	in"ter-stish'ul	**inter/stiti/al**	**Pertaining to between tissues**
Interstitium	in"ter-stish'um	**inter/stiti/um**	**The between tissues**
Intravascular	in"trŭ-vas'ku-lar	**intra/vascul/ar**	**Pertaining to inside vessels**
Keratocyte	ker-at'o-sit	**kerat/o/cyte**	**A horn cell**
Leukocyte	loo'ko-sit	**leuk/o/cyte**	**A white cell** (i.e., white blood cell)
Leukocytosis	loo"ko-si-to'sis	**leuk/o/cyt/o/sis**	**Condition of white cells** (excess)
Leukopenia	loo"ko-pe'ne-uh	**leuk/o/penia**	**A deficiency of white** (cells)
Leukopoiesis	loo"ko-poi-e'sis	**leuk/o/poie/sis**	**Process of white** (cell) **production**
Lipemia	li-pe'me-uh	**lip/emia**	**A blood condition of fat**
Lipid	lip'id	**lip/id**	**A fat**
Lymphadenectomy	lim-fad"en-ek'tŭ-me	**lymph/aden/ectomy**	**To cut out lymph glands**

Table Continued

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Term	Phonetics	Division	Basic Definition
Lymphadenopathy	lim-fad"en-op'ŭ-the	lymph/aden/o/pathy	A disease of lymph glands
Lymphocyte	lim'fo-sit	lymph/o/cyte	The lymph cell
Lymphocytosis	lim"fo-si-to'sis	lymph/o/cyt/o/sis	Condition of lymphocytes (excess)
Lymphoid	lim'foid	lymph/oid	Resembling lymph
Lymphopenia	lim"fo-pe'ne-uh	lymph/o/penia	A deficiency of lymphocytes
Macrocytic	mak"ro-sit'ik	macr/o/cyt/ic	Pertaining to large cells
Macrophage	mak'ro-fahj"	macr/o/phage	A large eater
Mandibular	man-dib'u-lar	mandibul/ar	Pertaining to the mandible
Mediastinal	me"de-uh-sti'nal	mediastin/al	Pertaining to the mediastinum
Megakaryocyte	meg'uh-kar'e-o-sit	mega/kary/o/cyte	A large nucleus cell
Mesenteric	mes"en-tār'ik, mez"en-tār'ik	mesenter/ic	Pertaining to the mesentery
Metamyelocyte	met'uh-mi'ĕ-lo-sit	meta/myel/o/cyte	An after marrow cell
Metarubricyte	met"ah-roo'brĭ-sit	meta/rubri/cyte	An after red cell
Microcytic	mi"kro-sit'ik	micro/cyt/ic	Pertaining to small cells
Monocyte	mon'o-sit	mono/cyte	The one/single cell
Monocytopenia	mon"o-si"to-pe'ne-uh	mono/cyt/o/penia	A deficiency of monocytes
Monocytosis	mon"o-si-to'sis	mono/cyt/o/sis	Condition of monocytes (excess)
Morphology	mor-fol'o-je	morph/o/logy	Study of shape/form
Myeloblast	mi'ĕ-lo-blast	myel/o/blast	A marrow germ/shoot
Myelocyte	mi'ĕ-lo-sit	myel/o/cyte	A marrow cell
Myelopathy	mi"ĕ-lop'uh-the	myel/o/pathy	A disease of marrow
Neutropenia	noo"tro-pe'ne-uh	neutr/o/penia	A deficiency of neutrophils
Neutrophil	noo'tro-fil	neutr/o/phil	Neither loving/lover
Neutrophilia	noo"tro-fil'e-uh	neutr/o/phil/ia	Condition of neutrophils (excess)
Normocytic	nor"mo-sit'ik	norm/o/cyt/ic	Pertaining to normal cells
Oxyhemoglobin	ok"se-he'mo-glo"bin	oxy/hem/o/glob/in	The oxygen blood "stuff"
Pancytopenia	pan'si-to-pe'ne-uh	pan/cyt/o/penia	Deficiency of all (blood) cells
Pathogen	path'o-jen	path/o/gen	A disease producer
Pathogenic	path"o-jen'ik	path/o/gen/ic	Pertaining to disease production
Pathologist	path-ol'o-jist	path/o/log/ist	A specialist of disease knowledge/study
Pathology	path-ol'o-je	path/o/logy	Study of disease
Phagocytosis	fag"o-si-to'sis	phag/o/cyt/o/sis	Process of eating (by) cells
Pleomorphic	ple"o-mor'fik	ple/o/morph/ic	Pertaining to more shapes
Poikilocytosis	poi"kĭ-lo-si-to'sis	poikil/o/cyt/o/sis	Condition of varied/irregular cells
Polychromasia	pol"e-kro-ma'zhuh	poly/chrom/asia	Condition of many colors
Polycythemia	pol"e-si-the'me-uh	poly/cyt/hem/ia	Condition of many cells of blood
Polymorphonuclear	pol"e-mor"fo-noo'kle-ar	poly/morph/o/nucle/ar	Pertaining to many shaped nucleus
Popliteal	pop-lit'e-al	poplit/e/al	Pertaining to the "ham" [L. *poples*]
Prescapular	pre-skap'u-lar	pre/scapul/ar	Pertaining to before/in front of the scapula
Promyelocyte	pro-mi'ĕ-lo-sit	pro/myel/o/cyte	A before marrow cell
Prorubricyte	pro-roo'brĭ-sit	pro/rubri/cyte	A before red cell
Prothrombin	pro-throm'bin	pro/thromb/in	The before clotter
Pruritic	proo-rit'ik	prurit/ic	Pertaining to itching
Pruritus	proo-ri'tus	prurit/us	An itch
Pyrexia	pi-rek'se-uh	pyrex/ia	A condition of fever
Refractometer	re"frak-tom'ĕ-ter	refract/o/meter	A breaking(bending) measurer
Reticulocyte	rĕ-tik'u-lo-sit"	reticul/o/cyte	A "net" (reticulum) cell
Retropharyngeal	ret"ro-fah-rin'je-al	retro/pharyng/e/al	Pertaining to behind the throat

Table Continued

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Chapter 3 Introductory Terms			
Term	**Phonetics**	**Division**	**Basic Definition**
Rubriblast	roo'brĭ-blast	**rubri/blast**	**A red germ/shoot**
Rubricyte	roo'brĭ-sīt	**rubri/cyte**	**A red cell**
Schistocyte	shis'to-sīt; skis'to-sīt	**schist/o/cyte**	**A split/divided cell**
Schizocyte	skiz'o-sīt; shi'zo-sīt	**schiz/o/cyte**	**A split/divided cell**
Serology	ser-ol'o-je, sēr-ol'o-je	**ser/o/logy**	**Study of serum**
Serous	sēr'us	**ser/ous**	**Pertaining to serum**
Spherocyte	sfēr'o-sīt	**spher/o/cyte**	**A ball cell**
Splenectomy	splen-ek'tŭ-me	**splen/ectomy**	**To cut out the spleen**
Splenic	splen'ik	**splen/ic**	**Pertaining to the spleen**
Splenomegaly	splen"o-meg'uh-le	**splen/o/megaly**	**Enlargement of the spleen**
Stomatocyte	sto-mat'o-sīt	**stomat/o/cyte**	**A "mouth" cell**
Thoracic	tho-ras'ik	**thorac/ic**	**Pertaining to the chest**
Thrombin	throm'bin	**thromb/in**	**The clotter** (activated form of prothrombin, Clotting Factor II)
Thrombocyte	throm'bo-sīt	**thromb/o/cyte**	**A clot cell**
Thrombocytopenia	throm"bo-si"to-pe'ne-uh	**thromb/o/cyt/o/penia**	**A deficiency of clot cells**
Thromboplastin	throm"bo-plas'tin	**thromb/o/plast/in**	**A clot former** (Clotting Factor III)
Thrombosis	throm-bo'sis	**thromb/o/sis**	**A condition/process of a clot/clotting**
Thrombus	throm'bus	**thromb/us**	**A clot**
Tonsillectomy	ton"sil-ek'tŭ-me	**tonsill/ectomy**	**To cut out tonsils**
Tonsillitis	ton"sĭ-li'tis	**tonsill/itis**	**Inflammation of tonsils**
Vascular	vas'ku-lar	**vascul/ar**	**Pertaining to vessels**

∗Color legend: **root word**, **prefix**, **suffix**, combining vowel

This table contains most but not all terms presented in this chapter.

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Medical Terms Introduced in Chapter 4

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Chapter 4 Introductory Terms			
Term	**Phonetics**	**Division**	**Basic Definition**
Abduction	ab-duk'shun	**ab/duc/tion**	**Act of away drawing/leading**
Abductor	ab-duk'ter	**ab/duct/or**	**An away drawer/leader**
Adduction	ad-duk'shun	**ad/duc/tion**	**Act of toward drawing/leading**
Adductor	ad-duk'ter	**ad/duct/or**	**A toward drawer/leader**
Ankylosis	ang"kuh-lo'sis	**ankyl/o/sis**	**Condition of**
Antebrachium	an"te-bra'ke-um	**ante/brachi/um**	**The [be] fore arm**
Arthritis	ahr-thri'tis	**arthr/itis**	**Inflammation of joints**
Arthrocentesis	ahr-thro"sen-te'sis	**arthr/o/centesis**	**Puncture/tapping of a joint**
Arthrodesis	ahr-thro"de'sis	**arthr/o/desis**	**Fusion of a joint**
Arthropathy	ahr-throp'uh-the	**arthr/o/pathy**	**A disease of joints**
Arthroscopy	ahr-thros'kuh-pe	**arthr/o/scopy**	**Viewing of a joint**
Articular	ahr-tik'u-lar	**articul/ar**	**Pertaining to joints**
Atrophy	at'ro-fe	**a/trophy**	**A state without development**
Biceps	bi'seps	**bi/ceps**	**Two heads**
Brachium	bra'ke-um	**brachi/um**	**The "arm"**
Cancellous	kan'sel-us	**cancell/ous**	**Pertaining to "lattice"**
Cardiac	kar'de-ak	**cardi/ac**	**Pertaining to the heart**
Carpus	kar'pus	**carp/us**	**The "wrist"**
Caudal	kaw'dul	**caud/al**	**Pertaining to the tail**
Cervical	ser'vĭ-kul	**cervic/al**	**Pertaining to the neck**
Cholinergic	ko"lin-er'jik	**cholin/erg/ic**	**Pertaining to acetylcholine work/working**

Table Continued

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Term	Phonetics	Division	Basic Definition
Chondral	kon'drul	chondr/al	Pertaining to cartilage
Chondrocyte	kon'dro-sit	chondr/o/cyte	A cartilage cell
Chondrodystrophic	kon'dro-dis-tro"fik	chondr/o/dys/troph/ic	Pertaining to cartilage poor/bad development
Circumduction	ser"kum-duk'shun	circum/duc/tion	Act of around drawing/leading
Coccygeal	kok-sij'e-ul	coccyg/e/al	Pertaining to the tail
Condylar	kon'dil-ăhr	condyl/ar	Pertaining to "knuckle"
Cortical	kor'tik-ul	cortic/al	Pertaining to the cortex
Costal	kos'tul	cost/al	Pertaining to rib
Costochondral	kos"to-kon'drul	cost/o/chondr/al	Pertaining to rib & cartilage
Coxofemoral	kok'so-fem'or-ul	cox/o/femor/al	Pertaining to hip & femur
Cranial	kra'ne-ul	crani/al	Pertaining to the head
Diaphysis	di-ă'fĭ-sis	dia/physis	(the) between growth
Digital	dij'ĭ-tul	digit/al	Pertaining to a toe/finger
Dysplasia	dis-pla'zhuh	dys/plas/ia	A condition of poor/bad formation
Endosteal	en-dos'te-ul	endo/ste/al	Pertaining to within bone
Epaxial	ep-ak'se-ul	ep/axi/al	Pertaining to upon the axis
Epicondyle	ep"ĭ-kon'dil	epi/condyle	Upon (the) knuckle
Epiphyseal	e-pif"ĭ-se"ul	epi/phys/e/al	Pertaining to upon growth
Extension	eks-ten'shun	ex/ten/sion	Act of out stretching
Extensor	eks-ten'sor	ex/tens/or	An out stretcher
Femorotibial	fem"or-o-tib'e-ul	femor/o/tibi/al	Pertaining to the femur & tibia
Fibroblast	fi'bro-blast	fibr/o/blast	A fiber germ/shoot
Fibrocartilage	fi"bro-kar'til-ej	fibr/o/cartilage	The fiber cartilage
Flexion	flek'shun	flex/ion	Act of bending
Flexor	fleks'or	flex/or	A bender
Gastrocnemius	gas-trok-ne'me-us	gastr/o/cnem/i/us	The "stomach" leg
Gluteal	gloo'te-ul	glute/al	Pertaining to the "butt"
Glycogen	gli'ko-jen	glyc/o/gen	A sugar producer
Humeroradiculnar	hu'mer-o-ra"de-o-ul'nar	humer/o/radi/o/uln/ar	Pertaining to the humerus, radius, & ulna
Hypertrophy	hi-pur'truh-fe	hyper/trophy	A state of excess development
Iliac	il'e-ak	ili/ac	Pertaining to the ilium
Infraspinous	in-fruh-spi'nus	infra/spin/ous	Pertaining to below the spine
Intercalated	in"ter-ka'la-ted	inter/calat/ed	Being between "calls"
Intercostal	in"ter-kos'tul	inter/cost/al	Pertaining to between ribs
Intervertebral	in"ter-ver-te'bral	inter/vertebr/al	Pertaining to between vertebrae
Intramedullary	in"truh-med'u-lăr-e	intra/medull/ary	Pertaining to within marrow
Intramuscular	in"truh-mus'ku-lar	intra/muscul/ar	Pertaining to within muscle
Intraosseous	in"truh-os'e-us	intra/osse/ous	Pertaining to within bone
Ischiatic	ish"e-at'ik	ischi/a/tic	Pertaining to the ischium "hip"
Ligamentous	lig"uh-men'tus	ligament/ous	Pertaining to ligaments
Lumbar	lum'bar	lumb/ar	Pertaining to the "loin"
Lumbosacral	lum"bo-sa'krul	lumb/o/sacr/al	Pertaining to the "loin" and sacrum
Mandibular	man-dib'u-lar	mandibul/ar	Pertaining to the mandible
Maxillary	mak'sil-ar-e	maxill/ary	Pertaining to the maxilla
Medullary	med"u-lar'e	medull/ary	Pertaining to marrow
Metacarpal	met"uh-kar'pul	meta/carp/al	Pertaining to beyond/after the carpus
Metacarpophalangeal	met"uh-kar"po-fuh-lan'je-ul	meta/carp/o/phalang/e/al	Pertaining to beyond/after the carpus and phalanges
Metaphysis	me-taf'ĭ-sis	meta/physis	(the) beyond/after growth
Metatarsal	met"uh-tar'sul	meta/tars/al	Pertaining to beyond/after the tarsus

Table Continued

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Chapter 4 Introductory Terms			
Term	**Phonetics**	**Division**	**Basic Definition**
Metatarsophalangeal	met"uh tar"so fuh lan'je-ul	**meta/tars/o/phalang/e/al**	**Pertaining to beyond/after the tarsus & phalanges**
Myasthenia	mi"us-the'ne-uh	**my/asthen/ia**	**A condition of muscle weakness**
Myocyte	mi-o-sīt'	**my/o/cyte**	**A muscle cell**
Myofibril	mi"-o-fi'bril	**my/o/fibr/il**	**A muscle fiber**
Myoglobin	mi-o-glo'bin	**my/o/glob/in**	**The muscle glob/"stuff"**
Myopathy	mi-op'uh-the	**my/o/pathy**	**A disease of muscle**
Myositis	mi"o-si'tis	**myos/itis**	**Inflammation of muscle**
Neurologic	nur-o-loj'-ik	**neur/o/log/ic**	**Pertaining to nerve knowledge/study**
Neurology	nur-ol'o-je	**neur/o/logy**	**The study of nerves**
Neuromuscular	ner"o-mus'ku-lar	**neur/o/muscul/ar**	**Pertaining to nerve & muscle**
Orthopedic	or-tho-pe'dik	**orth/o/ped/ic**	**Pertaining to straightening "a child"**
Osseous	os'e-us	**osse/ous**	**Pertaining to bone**
Ostectomy	os"tek'to-me	**ost/ectomy**	**To cut out (remove) bone**
Osteoarthritis	os"te-o-ahr-thri'tis	**oste/o/arthr/itis**	**Inflammation of bone & joint**
Osteoblast	os"te-o-blast	**oste/o/blast**	**A bone germ/shoot**
Osteoclast	os"te-o-klast	**oste/o/clast**	**A bone breaker**
Osteomyelitis	os"te-o-mi"-uh-li'tis	**oste/o/myel/itis**	**Inflammation of bone marrow**
Osteopathy	os"te-op'ah-the	**oste/o/pathy**	**A disease of bone**
Osteoplasty	os"te-o-plas'te	**oste/o/plasty**	**A reforming of bone**
Osteosarcoma	os"te-o-sar-ko'mah	**oste/o/sarc/oma**	**A tumor of bone & flesh**
Osteotomy	os"te-ah-to'me	**oste/o/tomy**	**To cut bone**
Pectoral	pek'tor-ul	**pector/al**	**Pertaining to the chest**
Periosteum	pār"e-os'te-um	**peri/oste/um**	**The around bone**
Quadriceps	kwad'rī-seps	**quadri/ceps**	**Four heads**
Quadruped	kwad'rū-ped	**quadru/ped**	**Four feet/footed**
Rotation	ro-ta'shun	**rota/tion**	**The act of turning**
Sacroiliac	sa"kro-il'e-ak	**sacr/o/ili/ac**	**Pertaining to the sacrum & ilium**
Sarcolemma	sahr"ko-lem'uh	**sarc/o/lemma**	**A sheath of flesh**
Sarcomere	sahr'ko-mēr	**sarc/o/mere**	**A flesh part**
Sarcoplasmic	sahr"ko-plaz'mik	**sarc/o/plasm/ic**	**Pertaining to flesh matter**
Scapulohumeral	skap'u-lo-hu"mer-ul	**scapul/o/humer/al**	**Pertaining to the scapula & humerus**
Semimembranosus	sem"e-mem'bren-o"sus	**semi/membran/o/sus**	**The partial membrane**
Semitendinosus	sem"e-ten'din-o"sus	**semi/tendin/o/sus**	**The partial tendon**
Sesamoid	ses'uh-moid	**sesam/oid**	**Resembling sesame (seed)**
Spondylitis	spon"dil-i'tis	**spondyl/itis**	**Inflammation of vertebrae**
Spondylopathy	spon"dil-op'uh-the	**spondyl/o/pathy**	**A disease of vertebrae**
Spondylosis	spon"dil-o'sis	**spondyl/o/sis**	**A condition of vertebrae**
Sternal	ster'nul	**stern/al**	**Pertaining to the sternum**
Supraspinous	soo"pruh-spi'nus	**supra/spin/ous**	**Pertaining to above the spine**
Symphysis	sim'fih-sis	**sym/physis**	**Together growing**
Synaptic	sin-ap'tik	**synap/tic**	**Pertaining to a connection**
Synovial	sin-o've-ul	**syn/ov/i/al**	**Pertaining to with "egg"**
Tarsal	tar'sul	**tars/al**	**Pertaining to the tarsus**
Tendinous	ten'din-us	**tendin/ous**	**Pertaining to tendons**
Thoracic	thor-ā'sik	**thorac/ic**	**Pertaining to the chest**
Triceps	trī'seps	**tri/ceps**	**Three heads**
Tubercle	too'ber-kul	**tuber/cle**	**A small "bump"**
Tuberosity	too"ber-os'i-te	**tuber/os/ity**	**State of "bumpy" bone**

∗Color legend: **root word**, **prefix**, **suffix**, combining vowel

This table contains most but not all terms presented in this chapter.

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Medical Terms Introduced in Chapter 5

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Chapter 5 Introductory Terms			
Term	**Phonetics**	**Division**	**Basic Definition**
Achondral	a-kon'drul	a/chondr/al	Pertaining to without cartilage
Alveolar	al-ve'uh-lur	alveol/ar	Pertaining to alveolus
Antitussive	an"te-tus'iv	anti/tuss/ive	Pertaining to against coughing
Aortic	a-or'tik	aort/ic	Pertaining to the aorta
Apnea	ap'ne-uh	a/pnea	Without breathing
Arrhythmia	a-rith'me-uh	a/rrhythm/ia	A condition without rhythm
Arterial	ahr-tēr'e-ul	arteri/al	Pertaining to arteries
Arteriole	ahr-tēr'e-ōl"	arteri/ole	A small artery
Asystole	a-sis'tuh-le	a/systole	Without contraction
Atelectasis	at"uh-lek'tuh-sis	atel/ectasis	Incomplete expansion (i.e., collapse)
Atrial	a'tre-ul	atri/al	Pertaining to the atrium
Atrioventricular	a"tre-o-ven-trik'u-lur	atri/o/ventricul/ar	Pertaining to the atrium and ventricle
Brachycephalic	bra"ke-suh-fal'ik	brachy/cephal/ic	Pertaining to short head
Bradycardia	brād"uh-kar'de-uh	brady/cardia	A slow heart
Bradypnea	brād-ip'ne-uh	brady/pnea	Slow breathing
Bronchial	brong'ke-ul	bronch/i/al	Pertaining to a bronchus
Bronchiole	brong'ke-ōl	bronch/i/ole	A small bronchus
Bronchospasm	brong"ko-spaz'um	bronch/o/spasm	Bronchus violent contraction
Bronchostenosis	brong"ko-stuh-no'sis	bronch/o/stenosis	Bronchus narrowing
Capnograph	kap'nuh-graf	capn/o/graph	A carbon dioxide recorder
Carbaminohemoglobin	kahr-bam"e-no-he"mo-glo'bin	carbamin/o/hemoglobin	Carbon dioxide and hemoglobin
Cardiac	kahr'de-ak	cardi/ac	Pertaining to the heart
Cardiologist	kahr"de-ol'uh-jist	cardi/o/log/ist	One who specializes in heart study
Cardiomegaly	kahr"de-o-meg'uh-le	cardi/o/megaly	Enlargement of the heart
Cardiomyopathy	kahr"de-o-mi-op'uh-the	cardi/o/my/o/pathy	A disease of heart muscle
Cardiopulmonary	kahr"de-o-pul'mun-ar-e	cardi/o/pulmon/ary	Pertaining to the heart and lungs
Cardiovascular	kahr"de-o-vas'ku-lur	cardi/o/vascul/ar	Pertaining to the heart and vessels
Chondral	kon'drul	chondr/al	Pertaining to cartilage
Chondromalacia	kon"dro-muh-la'shuh	chondr/o/malac/ia	A condition of cartilage softening
Cyanosis	si"uh-no'sis	cyan/o/sis	A condition of blue
Cyanotic	si"uh-not'ik	cyan/o/tic	Pertaining to blue
Deoxyhemoglobin	de-ok"se-he"mo-glo'bin	deoxy/hemoglobin	Reduced oxygen hemoglobin
Diastolic	di"uh-stol'ik	diastol/ic	Pertaining to diastole
Dyspnea	disp'ne-uh	dys/pnea	Difficulty breathing
Dysrhythmia	dis-rith'me-uh	dys/rhythm/ia	A state of bad rhythm
Echocardiography	ek"o-kahr"de-og'ruh-fe	echo/cardi/o/graphy	Recording echoes of the heart
Electrocardiography	e-lek"tro-kahr"de-og'ruh-fe	electr/o/cardi/o/graphy	Recording electricity of the heart
Embolism	em'buh-liz-um	embol/ism	State of plugging
Endocarditis	en"do-kahr-di'tis	endo/card/itis	Inflammation inside the heart
Endocardium	en"do-kahr'de-um	endo/cardi/um	The inside (of) the heart
Endotracheal	en"do-tra'ke-ul	endo/trache/al	Pertaining to within the trachea
Epicardium	ep-ĭ-kahr'de-um	epi/cardi/um	The upon the heart
Etiology	et"e-ol'uh-je	eti/o/logy	The study of cause
Expiration	ek"spĭ-ra'shun	ex/pira/tion	The act of out breathing (i.e., exhaling)
Hemiplegia	hem"e-ple'juh	hemi/pleg/ia	A condition of half paralysis
Hemoptysis	he-mop'tuh-sis	hem/o/ptysis	To spit blood
Holodiastolic	ho"lo-di"uh-stol'ik	hol/o/diastol/ic	Pertaining to the entire diastole

Table Continued

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Term	Phonetics	Division	Basic Definition
Holosystolic	ho"lo-sis-tol'ik	hol/o/systol/ic	Pertaining to the entire systole
Hypercapnia	hi"per-kap'ne-uh	hyper/capn/ia	Condition of excess carbon dioxide
Hyperpnea	hi"purp'ne-uh	hyper/pnea	Excessive breathing
Hypertrophic	hi"per-tro'fik	hyper/troph/ic	Pertaining to excess development
Hypotension	hi"po-ten'shun	hypo/tens/ion	State of low pressure
Hypoventilation	hi"po-ven"tuh-la'shun	hypo/ventila/tion	State of under breathing
Hypovolemia	hi"po-vo-le'me-uh	hypo/vol/emia	A blood condition of low volume
Hypoxia	hi-pok'se-uh	hyp/ox/ia	Condition of low oxygen
Inspiration	in"spi-ra'shun	in/spira/tion	Act of in breathing (i.e., inhaling)
Interatrial	in-ter-a'tre-ul	inter/atri/al	Pertaining to between the atria
Intercostal	in-ter-kos'tul	inter/cost/al	Pertaining to between ribs
Interventricular	in-ter-ven-trik'u-lar	inter/ventricul/ar	Pertaining to between the ventricles
Intranasal	in-truh-na'zul	intra/nas/al	Pertaining to within the nose
Intravenous	in-truh-ve'nus	intra/ven/ous	Pertaining to within veins
Ischemia	is-ke'me-uh	isch/emia	A blood condition of suppression
Laryngeal	luh-rin'je-ul	laryng/e/al	Pertaining to the larynx
Lobectomy	lo-bek'tuh-me	lob/ectomy	To cut out a lobe
Myocardial	mi"o-kahr'de-ul	my/o/cardi/al	Pertaining to heart muscle
Myocardium	mi"o-kahr'de-um	my/o/cardi/um	The heart muscle
Nasopharynx	na"zo-far'inks	nas/o/pharynx	The nose and throat
Oropharynx	or"o-far'inks	or/o/pharynx	The mouth and throat
Orthopnea	or-thop'ne-uh	orth/o/pnea	Straight breathing
Oximetry	ok-sim'uh-tre	oxi/metry	Measuring oxygen
Oxyhemoglobin	ok"se-he'mo-glo"bin	oxy/hemoglobin	Oxygenated hemoglobin
Oxymeter	ok-sim'uh-ter	oxy/meter	Oxygen measurer
Palatoplasty	pal"uh-to-plas'te	palat/o/plasty	Reconstruction of the palate
Pericardial	per"i-kahr'de-ul	peri/cardi/al	Pertaining to around the heart
Perivascular	per"i-vas'ku-lahr	peri/vascul/ar	Pertaining to around vessels
Phlebitis	fleb-i'tis	phleb/itis	Inflammation of a vein
Phlebotomy	fleb-ot'uh-me	phleb/o/tomy	To "cut" a vein
Pleural	ploo'rul	pleur/al	Pertaining to the pleura
Pneumothorax	noo"mo-thor'aks	pneum/o/thorax	Air of the chest
Pulmonary	pul'mun-ar-e	pulmon/ary	Pertaining to the lungs
Pulmonic	pul-mon'ik	pulmon/ic	Pertaining to the lungs
Pyothorax	pi"o-thor'aks	py/o/thorax	Pus of the chest
Respiratory	re-spi'ruh-tor-e	re/spira/tory	Pertaining to again breathing
Rhinitis	ri-ni'tis	rhin/itis	Inflammation of the nose
Rhinoplasty	ri'no-plas'te	rhin/o/plasty	Reconstruction of the nose
Rhinoscopy	ri-nos'kuh-pe	rhin/o/scopy	Viewing of the nose
Sinusitis	si"nuh-si'tis	sinus/itis	Inflammation of the sinus
Stenosis	sten-o'sis	sten/o/sis	Condition of narrowing
Systolic	sis-tol'ik	systol/ic	Pertaining to systole
Tachycardia	tak"uh-kahr'de-uh	tachy/card/ia	Condition of a fast heart
Tachypnea	tak-ip'ne-uh	tachy/pnea	Fast breathing
Thoracentesis	thor"un-ser-te'sis	thora/centesis	Puncture of the chest
Thoracotomy	thor"uh-kot'uh-me	thorac/o/tomy	To cut (into) the chest
Thromboembolism	throm"bo-em'bo-liz-um	thromb/o/embol/ism	Act of a clot thrown in
Thrombosis	throm-bo'sis	thromb/o/sis	Condition of clotting
Tracheitis	tra"ke-i'tis	trache/itis	Inflammation of the trachea

Table Continued

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Chapter 5 Introductory Terms			
Term	**Phonetics**	**Division**	**Basic Definition**
Tracheobronchitis	tra”ke-o-brong-ki’tis	**trache/o/bronch/itis**	**Inflammation of the trachea and bronchus**
Tracheostomy	tra”ke-os’tuh-me	**trache/o/stomy**	**Creation of a “mouth” of the trachea**
Vasoconstriction	va”zo-kon-strik’shun	**vas/o/constrict/ion**	**Act of vessel constricting**
Vasodilation	va”zo-di-la’shun	**vas/o/dilat/ion**	**Act of vessel dilating**
Venous	ve’nus	**ven/ous**	**Pertaining to veins**
Ventricular	ven-trik’u’lur	**ventricul/ar**	**Pertaining to the ventricle**
Venule	ven’ül	**ven/ule**	**A small vein**

∗Color legend: **root word**, **prefix**, **suffix**, combining vowel

This table contains most but not all terms presented in this chapter.

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Medical Terms Introduced in Chapter 6

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Chapter 6 Introductory Terms			
Term	**Phonetics**	**Division**	**Basic Definition**
Angiotensin	an"je-o-ten'sin	**angi/o/tens/in**	**The vessel tension** (hormone)
Antidiuretic	an"ti-di-ūr-et'ik	**anti/diure/tic**	**Pertaining to against urination**
Anuria	an-u're-uh	**an/ur/ia**	**A condition without urine**
Azotemia	a-zo-te'me-uh	**azot/emia**	**A blood condition of nitrogen**
Bacteriuria	bak-tēr"e-u're-uh	**bacteri/ur/ia**	**A condition of bacteria** (in) **urine**
Cystalgia	sis-tal'juh	**cyst/alg/ia**	**A condition of bladder pain**
Cystitis	sis-ti'tis	**cyst/itis**	**Inflammation of the bladder**
Cystocentesis	sis"to-sen-te'sis	**cyst/o/centesis**	**Puncture of the bladder**
Cystography	sis-tog'ruh-fe	**cyst/o/graphy**	**Recording of the bladder**
Cystotomy	sis-tot'uh-me	**cyst/o/tomy**	**To cut the bladder**
Cystourethrogram	sis"to-u-re'thro-gram	**cyst/o/urethr/o/gram**	**A recording of the bladder and urethra**
Diuretic	di"u-ret'ik	**diure/tic**	**Pertaining to urination**
Dysuria	dis-u're-uh	**dys/ur/ia**	**A condition of difficult urination**
Glomerular	glo-mār'u-lar	**glomerul/ar**	**Pertaining to the glomerulus**
Glycosuria	gli"ko-su're-uh	**glycos/ur/ia**	**A condition of sugary** (glucose) **urine**
Hematuria	he"muh-tu're-uh	**hemat/ur/ia**	**A condition of bloody urine**
Homeostasis	ho"me-o-sta'sis	**home/o/stasis**	**State of standing unchanged/the same**
Hydronephrosis	hi"dro-nĕ-fro'sis	**hydr/o/nephr/o/sis**	**A condition of water kidney**
Hypertonic	hi"per-ton'ik	**hyper/ton/ic**	**Pertaining to excess tonicity**
Hypotonic	hi"-po-ton'ik	**hypo/ton/ic**	**Pertaining to low tonicity**
Intravenous	in"truh-ve'nus	**intra/ven/ous**	**Pertaining to within a vein**
Isosthenuric	i"sos-thĕ-nu'rik	**iso/sthen**/ur/**ic**	**Pertaining to equal strength urine**
Isotonic	i"so-ton'ik	**iso/ton/ic**	**Pertaining to equal tonicity**
Nephritis	nef-ri'tis	**nephr/itis**	**Inflammation of the kidney**
Nephron	nef'ron	**nephr/on**	**A/the kidney** (functional unit of)
Nephrotoxicity	nef"ro-tok-sis'it-e	**nephr/o/tox/i/city**	**A state of kidney poisoning**
Oliguria	o"lig-u're-uh	**olig/ur/ia**	**A condition of small urine** (volume)
Peritubular	pār"ĭ-too'bu-lar	**peri/tubul/ar**	**Pertaining to around a tubule**
Pneumocystogram	noo"mo-sis'to-gram	**pneum/o/cyst/o/gram**	**A record of air** (in the) **bladder**
Pollakiuria	puh-lak"ĭ-u"re-uh	**pollaki/ur/ia**	**A condition of frequent urination**
Polydipsia	pol"e-dip'se-uh	**poly/dips/ia**	**A condition of much thirst**
Polyuria	pol"e-u're-uh	**poly/ur/ia**	**A condition of much urine**
Postrenal	pōst-re'nul	**post/ren/al**	**Pertaining to after the kidney**
Prerenal	pre-re'nul	**pre/ren/al**	**Pertaining to before the kidney**

Table Continued

Chapter 6 Introductory Terms			
Term	**Phonetics**	**Division**	**Basic Definition**
Proteinuria	pro"ten-u're-uh	**protein/ur/ia**	**A condition of protein urine**
Pyelogram	pi'uh-lo-gram	**pyel/o/gram**	**A record of the** (renal) **pelvis**
Pyelonephritis	pi"uh-lo-nef-ri'tis	**pyel/o/nephr/itis**	**Inflammation of the pelvis and kidney**
Pyuria	pi-u're-uh	**py/ur/ia**	**A condition of pus** (WBCs in) **urine**
Renal	re'nul	**ren/al**	**Pertaining to the kidney**
Renin	ren'in	**ren/in**	**The kidney** (hormone)
Retrograde	ret'ro-grād	**retro/grade**	**A backward step**
Retroperitoneal	ret"ro-pār"ĭ-to-ne'ul	**retro/peritone/al**	**Pertaining to backward/behind the peritoneum**
Uremia	u-re'me-uh	**ur/emia**	**A blood condition of urea**
Ureteral	u-re'ter-ul	**ureter/al**	**Pertaining to the ureter**
Urethritis	u"re-thri'tis	**urethr/itis**	**Inflammation of the urethra**
Urethrocystogram	u-re"thro-sis'to-gram	**urethr/o/cyst/o/gram**	**A record of the urethra and bladder**
Urethrostomy	u-re-thros'tuh-me	**urethr/o/stomy**	**Creation of a "mouth"** (in) **the urethra**
Urinary	u"rin-ār'e	**urin/ary**	**Pertaining to urine**
Urolithiasis	u"ro-lith-i'uh-sis	**ur/o/lith/i/asis**	**A condition of urinary stones**

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∗Color legend: **root word**, **prefix**, **suffix**, combining vowel

This table contains most but not all terms presented in this chapter.

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Medical Terms Introduced in Chapter 7

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Chapter 7 Introductory Terms			
Term	**Phonetics**	**Division**	**Basic Definition**
Abdominocentesis	ab-dom”in-o-sen-te’sis	**abdomin/o/centesis**	**To puncture the abdomen**
Abomasopexy	ab-o-ma”so-pek’se	**abomas/o/pexy**	**Fixation of the abomasum**
Amylase	am’uh-lās	**amyl/ase**	**An enzyme of starch**
Anorexia	an”o-rek’se-uh	**an/orex/ia**	**A state without appetite**
Antidiarrheal	an”ti-di”uh-re’ul	**anti/diarrhe/al**	**Pertaining to against diarrhea**
Antiemetic	an”te-uh-met’ik	**anti/eme/tic**	**Pertaining to against vomiting**
Biliary	bil’e-ār-e	**bili/ary**	**Pertaining to bile**
Buccal	buk’ul	**bucc/al**	**Pertaining to the cheek**
Carnivorous	kahr-niv’uh-rus	**carnivor/ous**	**Pertaining to flesh eating**
Cholecystectomy	ko”le-sis-tek’tuh-me	**chole/cyst/ectomy**	**To cut out the gallbladder**
Cholecystitis	ko”le-sis-ti’tis	**chole/cyst/itis**	**Inflammation of the gallbladder**
Chylothorax	ki”lo-tho’raks	**chyl/o/thorax**	**Chyle of the chest**
Chylous	ki’lous	**chyl/ous**	**Pertaining to chyle**
Colectomy	ko-lek’tuh-me	**col/ectomy**	**To cut out the colon**
Colitis	ko-li’tis	**col/itis**	**Inflammation of the colon**
Coprophagia	kop”ro-fa’juh	**copr/o/phag/ia**	**Process of feces eating**
Defecation	def”ĕ-ka’shun	**defecat/ion**	**The act of defecating**
Duodenal	doo-od’uh-nul	**duoden/al**	**Pertaining to the duodenum**
Dysphagia	dis-fa’je-uh	**dys/phag/ia**	**Process of difficult eating**
Enterectomy	en”ter-ek’tuh-me	**enter/ectomy**	**To cut out intestines**
Enteric	en-tār’ik	**enter/ic**	**Pertaining to intestines**
Enteritis	en”ter-i’tis	**enter/itis**	**Inflammation of intestines**
Enterotomy	en”ter-ot’uh-me	**enter/o/tomy**	**To cut** (into) **the intestines**
Eructation	uh-ruk-ta’shun	**eructat/ion**	**The act of belching**
Esophageal	e-sof”uh-je’ul	**esophag/e/al**	**Pertaining to the esophagus**
Esophagitis	e-sof”uh-ji’tis	**esophag/itis**	**Inflammation of the esophagus**

Table Continued

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Chapter 7 Introductory Terms			
Term	**Phonetics**	**Division**	**Basic Definition**
Gastrectomy	gas-trek'tuh-me	**gastr/ectomy**	**To cut out stomach**
Gastric	gas'trik	**gastr/ic**	**Pertaining to the stomach**
Gastritis	gas-tri'tis	**gastr/itis**	**Inflammation of the stomach**
Gastroenteritis	gas"tro-en-ter-i'tis	**gastr/o/enter/itis**	**Inflammation of the stomach and intestines**
Gastroesophageal	gas"tro-e-sof"uh-je'ul	**gastr/o/esophag/e/al**	**Pertaining to the stomach and esophagus**
Gastropexy	gas'tro-pek"se	**gastr/o/pexy**	**Fixation of the stomach**
Gastroscopy	gas-tros'kuh-pe	**gastr/o/scopy**	**Viewing of the stomach**
Gastrostomy	gas-tros'tuh-me	**gastr/o/stomy**	**"Mouth" creation of the stomach**
Gastrotomy	gas-trot'uh-me	**gastr/o/tomy**	**To cut (into) the stomach**
Gingival	jin'jī-vul	**gingiv/al**	**Pertaining to the gingiva (i.e., "gums")**
Gingivitis	jin"jī-vi'tis	**gingiv/itis**	**Inflammation of the gingiva**
Glossal	glos'ul	**gloss/al**	**Pertaining to the tongue**
Hematemesis	hē-mat"em'uh-sis	**hemat/emesis**	**Bloody vomiting**
Hematochezia	he"muh-to-ke'zhuh	**hemat/o/chez/ia**	**A condition of bloody feces**
Hepatic	hē-pat'ik	**hepat/ic**	**Pertaining to the liver**
Hepatomegaly	hē-pat"o-meg'uh-le	**hepat/o/megaly**	**Enlargement of the liver**
Hepatopathy	hep"uh-top'uh-the	**hepat/o/pathy**	**Disease of the liver**
Hepatoportal	hep"uh-to-por"tul	**hepat/o/port/al**	**Pertaining to the liver entrance**
Herbivorous	ur-biv'uh-rus	**herbivor/ous**	**Pertaining to plant eating**
Hyperbilirubinemia	hī"per-bil"e-roo"bī-ne'me-uh	**hyper/bilirubin/emia**	**A blood condition of excess bilirubin**
Hypersialosis	hī"per-si"uh-lo'sis	**hyper/sial/o/sis**	**A condition of excess saliva**
Ieteric	ik'ter-ik	**icter/ic**	**Pertaining to icterus (jaundice)**
Ileocecal	il"e-o-se'kul	**ile/o/cec/al**	**Pertaining to the ileum and cecum**
Intussusception	in"tuh-suh-sep'shun	**intus/suscep/tion**	**The act of within receiving**
Jejunal	jē-joo'nul	**jejun/al**	**Pertaining to the jejunum**
Labial	la'be-ul	**labi/al**	**Pertaining to the lip**
Lacteal	lak'te-ul	**lact/e/al**	**Pertaining to milky**
Laparoscopy	lap"uh-ros'kuh-pe	**lapar/o/scopy**	**Viewing of the abdomen**
Laparotomy	lap"uh-rot'uh-me	**lapar/o/tomy**	**To cut (into) the abdomen**
Lingual	ling'gwul	**lingu/al**	**Pertaining to the tongue**
Lipase	lī'pās	**lip/ase**	**An enzyme of fat**
Lipemia	li-pe'me-uh	**lip/emia**	**A blood condition of fat**
Mandibular	man-dib'u-lar	**mandibul/ar**	**Pertaining to the mandible**
Mastication	mas"tĭ-ka'shun	**mastica/tion**	**The act of chewing**
Maxillary	mak'sil-ar-e	**maxilla/ary**	**Pertaining to the maxilla**
Megaesophagus	meg"uh-e-sof'uh-gus	**mega/esophagus**	**Enlarged esophagus**
Melenic	muh-le'nik	**melen/ic**	**Pertaining to black**
Mesenteric	mes"en-tār'ik	**mesenter/ic**	**Pertaining to the mesentery**
Mesial	me'ze ul	**mesi/al**	**Pertaining to the middle**
Monogastric	mon"o-gas'trik	**mono/gastr/ic**	**Pertaining to one stomach**
Mucoid	mu'koid	**muc/oid**	**Resembling mucus**
Mucosal	mu-ko'sul	**mucos/al**	**Pertaining to mucosa**
Mucous	mu'kus	**muc/ous**	**Pertaining to mucus**
Mucus	mu'kus	**muc/us**	**The slime**
Nasogastric	na"zo-gas'trik	**nas/o/gastr/ic**	**Pertaining to the nose and stomach**
Necrosis	nĕ-kro'sis	**necr/o/sis**	**A condition of death**
Necrotic	nĕ-krot'ik	**necr/o/tic**	**Pertaining to death**
Odontoclastic	o-don"to-klas'tik	**odont/o/clast/ic**	**Pertaining to teeth breaking**
Omentopexy	o-men'to-pek"se	**oment/o/pexy**	**Fixation of the omentum**

Table Continued

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Chapter 7 Introductory Terms			
Term	**Phonetics**	**Division**	**Basic Definition**
Omnivorous	om-niv'uh-rus	**omnivor/ous**	**Pertaining to all eating**
Orogastric	or"o-gas'trik	**or/o/gastr/ic**	**Pertaining to the mouth and stomach**
Oropharynx	or"o-făr'inks	**or/o/pharynx**	**The mouth and throat**
Palatal	pal'uh-tul	**palat/al**	**Pertaining to the palate**
Palatoplasty	pal"uh-to-plas'te	**palat/o/plasty**	**Reconstruction of the palate**
Palatoschisis	pal"uh-to-ske'sis	**palat/o/schisis**	**A cleft palate**
Pancreatic	pan"kre-at'ik	**pancreat/ic**	**Pertaining to the pancreas**
Pancreatitis	pan"kre-uh-ti'tis	**pancreat/itis**	**Inflammation of the pancreas**
Parietal	puh-ri'uh-tul	**pariet/al**	**Pertaining to the wall**
Periodontal	per"e-o-don'tul	**peri/odont/al**	**Pertaining to around teeth**
Peristalsis	per"ĭ-stal'sis	**peri/stalsis**	**Around/near contraction**
Peritoneal	per"ĭ-tuh-ne'ul	**periton/e/al**	**Pertaining to the peritoneum**
Peritonitis	per"ĭ-tuh-ni'tis	**periton/itis**	**Inflammation of the peritoneum**
Pharyngeal	fuh-rin'je-ul	**pharyng/e/al**	**Pertaining to the throat**
Posthepatic	post"huh-pat'ik	**post/hepat/ic**	**Pertaining to after the liver**
Postprandial	pōst-pran'de-ul	**post/prandi/al**	**Pertaining to after a meal**
Prehepatic	pre"huh-pat'ik	**pre/hepat/ic**	**Pertaining to before the liver**
Proteolytic	pro"te-o-lit'ik	**prote/o/lytic**	**Pertaining to breaking protein**
Pyloric	pi-lor'ik	**pylor/ic**	**Pertaining to the pylorus**
Regurgitation	re-gur"jĭ-ta'shun	**re/gurgit/a/tion**	**The act of back flooding**
Rumenostomy	roo"mun-os'tuh-me	**rumen/o/ostomy**	**Creation of an opening to the rumen**
Sialocele	si-al'o-sēl	**sial/o/cele**	**A swelling of saliva**
Steatorrheic	ste"at-o-re'ik	**steat/o/rrhe/ic**	**Pertaining to fatty flow**
Stomatitis	sto-muh-ti'tis	**stomat/itis**	**Inflammation of the mouth**
Sublingual	sub-ling'gwul	**sub/lingu/al**	**Pertaining to below the tongue**
Submucosal	sub"mu-ko'sul	**sub/mucos/al**	**Pertaining to below the mucosa**
Visceral	vis'er-ul	**viscer/al**	**Pertaining to organs**

∗Color legend: **root word**, **prefix**, **suffix**, combining vowel

This table contains most but not all terms presented in this chapter.

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Medical Terms Introduced in Chapter 8

Chapter 8 Introductory Terms			
Term	**Phonetics**	**Division**	**Basic Definition**
Allergen	al'er-jen	**allerg/gen**	**An allergy producer**
Carcinoma	kar"sĭ-no'muh	**carcin/oma**	**A tumor of cancer**
Circumoral	ser"kum-o'rul	**circum/or/al**	**Pertaining to around the mouth**
Cornuectomy	kor"nu-ek'tuh-me	**cornu/ectomy**	**To cut out the horn** (i.e., dehorning)
Coronary	kor'uh-nār"e	**coron/ary**	**Pertaining to a "crown"**
Dermatitis	der"muh-ti'tis	**dermat/itis**	**Inflammation of the skin**
Dermatomycosis	der"mat-o-mi-ko'sis	**dermat/o/myc/o/sis**	**A condition of skin fungus**
Dermatophytosis	der"mat-o-fi-to'sis	**dermat/o/phyt/o/sis**	**A condition of skin "plants"** (i.e., fungus)
Epidermal	ep"ĭ-der'mul	**epi/derm/al**	**Pertaining to on/upon the dermis**
Erythematous	er"ĭ-them'uh-tus	**erythema/tous**	**Pertaining to redness**
Fibroblast	fi'bro-blast	**fibr/o/blast**	**A fiber germ/shoot**
Fibroplasia	fi"bro-pla'zhuh	**fibr/o/plas/ia**	**A process of fiber forming**
Hyperkeratosis	hi"per-ker"uh-to'sis	**hyper/kerat/o/sis**	**A condition of excess keratin**

Table Continued

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Chapter 8 Introductory Terms			
Term	**Phonetics**	**Division**	**Basic Definition**
Hypersensitivity	hi"per-sen"sĭ-tiv'ĭ-te	**hyper/sensitiv/ity**	**A state of being overly sensitive**
Hypodermic	hi"po-der'mik	**hypo/derm/ic**	**Pertaining to below the skin**
Interdigital	in"ter-dij'ĭ-tul	**inter/digit/al**	**Pertaining to between toes**
Intradermal	in"truh-der'mul	**intra/derm/al**	**Pertaining to within the skin**
Laminitis	lam"in-i'tis	**lamin/itis**	**Inflammation of the lamina**
Melanocyte	mĕ-lan'o-sīt	**melan/o/cyte**	**A black cell**
Melanoma	mel"uh-no'muh	**melan/oma**	**A tumor of black**
Onychectomy	on"ĭ-kek'tuh-me	**onych/ectomy**	**To cut out the nail**
Perianal	pār"e-a'nul	**peri/an/al**	**Pertaining to around the anus**
Piloerection	pi"lo-uh-rek'shun	**pil/o/erect/ion**	**The act of hair raising**
Pododermatitis	po"do-der"muh-ti'tis	**pod/o/dermat/itis**	**Inflammation of foot skin**
Polydactyly	pol"e-dak-tuh-le	**poly/dactyly**	**A state of many toes**
Pruritus	proo-ri'tus	**prurit/us**	**The state of itching**
Pyoderma	pi"o-der'muh	**py/o/derma**	**Pus skin**
Seborrhea	seb"o-re'uh	**seb/o/rrhea**	**Flow of sebum** (fat)
Subcutaneous	sub"ku-ta'ne-us	**sub/cutan/e/ous**	**Pertaining to below the skin**
Subcutis	sub-ku'tis	**sub/cutis**	**Under the skin**
Tenotomy	ten-ot'uh-me	**ten/o/tomy**	**To cut a tendon**
Transdermal	trans-der'mul	**trans/derm/al**	**Pertaining to across the skin**
Urticaria	ur"tĭ-kār'e-uh	**urticar/ia**	**A condition of hives**

∗Color legend: **root word**, **prefix**, **suffix**, combining vowel

This table contains most but not all terms presented in this chapter.

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Medical Terms Introduced in Chapter 9

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Chapter 9 Introductory Terms			
Term	**Phonetics**	**Division**	**Basic Definition**
Anestrus	an-es'trus	**an/estrus**	**Without estrus**
Cryptorchidism	kript-or'kid-izm	**crypt/orchid/ism**	**A state of hidden testicles**
Diestrous	di-es'trus	**di/estr/ous**	**Pertaining to two estrus** (cycles)
Diestrus	di-es'trus	**di/estrus**	**Two estrus**
Dystocia	dis-to'shuh	**dys/toc/ia**	**A condition of difficult birth**
Endometrium	en"do-me'tre-um	**endo/metr/i/um**	**The inside** (of the) **uterus**
Epididymis	ep"ĭ-did'uh-mis	**epi/didymis**	**Upon the testis**
Episioplasty	uh-pēz'e-o-plas"te	**episi/o/plasty**	**Surgical repair/reconstruction of the vulva**
Episiorrhaphy	uh-pēz"e-or'uh-fe	**episi/o/rrhaphy**	**Suturing of the vulva**
Estrogen	es'truh-jen	**estr/o/gen**	**Estrus producing (hormone)**
Estrous	es'trus	**estr/ous**	**Pertaining to estrus**
Genitourinary	jen"ĭ-to-u'rĭ-nār-e	**genit/o/urin/ary**	**Pertaining to reproductive & urinary** (organs)
Gestation	jes-ta'shun	**gest/a/tion**	**The state of bearing** (i.e., pregnancy)
Hypocalcemia	hi"po-kal-se'me-uh	**hypo/calc/emia**	**A blood condition of low calcium**
Lactation	lak-ta'shun	**lact/a/tion**	**The state of milking**
Mammary	mam'ar-e	**mamm/ary**	**Pertaining to the breast**
Mastectomy	mas-tek'tuh-me	**mast/ectomy**	**To cut out the breast**
Mastitis	mas-ti'tis	**mast/itis**	**Inflammation of the breast**
Metestrus	met-es'trus	**met/estrus**	**After (meta-) estrus**
Metritis	me-tri'tis	**metr/itis**	**Inflammation of the uterus**

Table Continued

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Chapter 9 Introductory Terms			
Term	**Phonetics**	**Division**	**Basic Definition**
Monestrous	mon-es'trus	**mon/estr/ous**	**Pertaining to one estrus** (cycle)
Neonatal	ne"o-na'tul	**ne/o/nat/al**	**Pertaining to newly born**
Oocyte	o'o-sīt	**oo/cyte**	**An egg cell**
Oophorectomy	o"fuh-rek'tuh-me	**oophor/ectomy**	**To cut out the ovary**
Orchiectomy	or"ke-ek'tuh-me	**orchi/ectomy**	**To cut out the testis** (i.e., castration; also orchidectomy)
Ovariohysterectomy	o-vār"e-o-his"ter-ek'tuh-me	**ovari/o/hyster/ectomy**	**To cut out the ovaries & uterus** (i.e., spay)
Ovulation	ov"u-la'shun	**ovul/a/tion**	**Process of egg** (release)
Oxytocin	ok"se-to'sin	**oxy/toc/in**	**The fast birth** (hormone)
Parabasal	pahr-uh-ba'sul	**para/bas/al**	**Pertaining to near** (the) **base**
Paraphimosis	par"uh-fĭ-mo'sis	**para/phimosis**	**Beside muzzling**
Parturition	pahr"tu-rĭ'shun	**parturi/tion**	**The process/state of birthing**
Periparturient	pār-e-pahr-tu're-ent	**peri/parturi/ent**	**Pertaining to around birth**
Polyestrous	pol"e-es'trus	**poly/estr/ous**	**Pertaining to many estrus** (cycles)
Postparturient	pōst-pahr-tu're-ent	**post/parturi/ent**	**Pertaining to after birth**
Preparturient	pre-pahr-tu're-ent	**pre/parturi/ent**	**Pertaining to before birth**
Proestrus	pro-es'trus	**pro/estrus**	**Before estrus**
Progesterone	pro-jes'ter-ōn	**pro/gester/one**	**A hormone for bearing** (i.e., pregnancy)
Prolactin	pro-lak'tin	**pro/lact/in**	**The for milk** (hormone)
Prostatitis	pros"tuh-ti'tis	**prostat/itis**	**Inflammation of the prostate**
Pseudocyesis	soo"do-si-e'sis	**pseud/o/cyesis**	**False pregnancy**
Pyometra	pi"o-me'truh	**py/o/metra**	**Pus** (in) **the uterus**
Scrotal	skro'tul	**scrot/al**	**Pertaining to a sac** (scrotum)
Seminiferous	sem"ĭ-nif'er-us	**semin/i/ferous**	**Pertaining to producing seed** (semen)
Spermatic	spur-mat'ik	**spermat/ic**	**Pertaining to sperm**
Spermatogenesis	spur"mat-uh-jen'uh-sis	**spermat/o/gen/e/sis**	**Process of sperm production**
Testicular	tes-tik'u-lahr	**testicul/ar**	**Pertaining to the testis**
Testosterone	tes-tos'ter-ōn	**test/o/sterone**	**A testis steroid** (hormone)
Urogenital	u"ro-jen'ĭ-tul	**ur/o/genit/al**	**Pertaining to urinary & reproductive** (organs)

∗Color legend: **root word**, **prefix**, **suffix**, combining vowel

This table contains most but all terms presented in this chapter.

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Medical Terms Introduced in Chapter 10

Chapter 10 Introductory Terms			
Term	**Phonetics**	**Division**	**Basic Definition**
Adenohypophysis	ad”ĕ-no-hi-pof’ĭ-sis	**aden/o/hypo/physis**	**(the) gland below growth**
Adrenal	uh-dre’nul	**ad/ren/al**	**Pertaining to near the kidney**
Adrenergic	ad”ren-er’jik	**adren/erg/ic**	**Pertaining to adrenal working**
Adrenocortical	uh-dre”no-kor’tĭ-kul	**adren/o/cortic/al**	**Pertaining to the adrenal cortex**
Adrenocorticotropic	uh-dre”no-kor”tĭ-ko-tro’pik	**adren/o/cortic/o/trop/ic**	**Pertaining to adrenal cortex changing/influencing**
Adrenomedullary	uh-dre”no-med’u-lār”e	**adren/o/medull/ary**	**Pertaining to the adrenal medulla**
Androgenous	an”droj’en-us	**andr/o/gen/ous**	**Pertaining to male producing**
Angiotensin	an”je-o-ten’sin	**angi/o/tens/in**	**The vessel tension (hormone)**
Antidiuretic	an”ti-di-ūr-et’ik	**anti/diure/tic**	**Pertaining to against urination**
Endocrine	en’do-krin	**endo/crine**	**An inside separation/secretion** (i.e., hormones)
Endocrinologist	en”do-krĭ-nol’uh-jist	**endocrin/o/log/ist**	**One specializing in hormone study**

Table Continued

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Chapter 10 Introductory Terms			
Term	**Phonetics**	**Division**	**Basic Definition**
Endocrinology	en"do-krĭ-nol'uh-je	**endocrin/o/logy**	**Study of hormones**
Endocrinopathy	en"do-krin-op'uh-the	**endocrin/o/pathy**	**Disease of hormones**
Endogenous	en-doj'en-us	**endo/gen/ous**	**Pertaining to inside production**
Erythropoietin	ĕ-rith"ro-poi'e-tin	**erythr/o/poie/tin**	**The red producer (hormone)**
Estrogen	es'truh-jen	**estr/o/gen**	**Estrus producing (hormone)**
Etiology	et"e-ol'uh-je	**eti/o/logy**	**The study of cause**
Exogenous	ek-soj'uh-nus	**exo/gen/ous**	**Pertaining to outside produced/production**
Gestation	jes-ta'shun	**gest/a/tion**	**State of bearing** (i.e., pregnancy)
Gluconeogenesis	gloo"ko-ne"o-jen'ĕ-sis	**gluc/o/ne/o/gen/e/sis**	**Process of sugar newly produced**
Glycosuria	gli"ko-su're-uh	**glycos/ur/ia**	**Condition of sugary urine**
Gonadotropic	go"nad-o-tro'pik	**gonad/o/trop/ic**	**Pertaining to gonad changing/influencing**
Homeostasis	ho"me-o-sta'sis	**home/o/stasis**	**State of standing unchanged/the same**
Hormonal	hor-mo'nul	**hormon/al**	**Pertaining to hormones**
Hyperadrenocorticism	hi"per-uh-dre"no-kor'tĭ-siz-um	**hyper/adren/o/cortic/ism**	**Condition of excess adrenal cortex (hormones)**
Hypercalcemia	hi"per-kal-se'me-uh	**hyper/calc/emia**	**Blood condition of excess calcium**
Hyperglycemia	hi"per-gli-se'me-uh	**hyper/glyc/emia**	**Blood condition of excess sugar** (glucose)
Hyperkalemia	hi"per-ka-le'me-uh	**hyper/kal/emia**	**Blood condition of excess potassium**
Hyperparathyroidism	hi"per-par-uh-thi'roid-iz-um	**hyper/parathyroid/ism**	**State/condition of excess parathyroid (hormones)**
Hyperthyroidism	hi"per-thi'roid-iz-um	**hyper/thyroid/ism**	**State/condition of excess thyroid (hormones)**
Hypoadrenocorticism	hi"po-uh-dre"no-kor'tĭ-siz-um	**hypo/adren/o/cortic/ism**	**Condition of insufficient adrenal cortex (hormones)**
Hypocalcemia	hi"po-kal-se'me-uh	**hypo/calc/emia**	**Blood condition of low calcium**
Hypoglycemia	hi"po-gli-se'me-uh	**hypo/glyc/emia**	**Blood condition of low sugar**
Hyponatremia	hi"po-na-tre'me-uh	**hypo/natr/emia**	**Blood condition of low sodium**
Hypophysis	hi-pof'ĭ-sis	**hypo/physis**	**(the) under growth**
Hypothalamus	hi"po-thal'uh-mus	**hypo/thalamus**	**Below the inner chamber**
Hypothyroidism	hi"po-thi'roid-iz-um	**hypo/thyroid/ism**	**Condition/state of low thyroid (hormones)**
Neurohypophysis	noor"o-hi-pof'ĭ-sis	**neur/o/hypo/physis**	**(the) nerve below growth**
Oxytocin	ok"se-to'sin	**oxy/toc/in**	**The fast birth (hormone)**
Pancreatic	pan"kre-at'ik	**pancreat/ic**	**Pertaining to the pancreas**
Pancreatitis	pan"kre-uh-ti'tis	**pancreat/itis**	**Inflammation of the pancreas**
Parathyroid	par"uh-thi'roid	**para/thyroid**	**Near the thyroid**
Parathyroidectomy	par"uh-thi"roid-ek'tuh-me	**parathyroid/ectomy**	**To cut out the parathyroid**
Polydipsia	pol"e-dip'se-uh	**poly/dips/ia**	**State of much thirst**
Polyuria	pol"e-u're-uh	**poly/ur/ia**	**State of much urine**
Progesterone	pro-jes'ter-ōn	**pro/gester/one**	**A hormone for bearing** (i.e., pregnancy)
Prolactin	pro-lak'tin	**pro/lact/in**	**The for milk** (hormone)
Renin	ren'in	**ren/in**	**The kidney** (hormone)
Somatotropic	so"mat-o-tro'pik	**somat/o/trop/ic**	**Pertaining to body changing/influencing**
Sympathomimetic	sim-path"o-mĭ-met'ik	**sympath/o/mime/tic**	**Pertaining to sympathetic imitation**
Testosterone	tes-tos'ter-ōn	**test/o/sterone**	**A testes steroid (hormone)**
Tetraiodothyronine	tet"ruh-i"o-do-thi'ro-nēn	**tetra/iod/o/thyronine**	**A four-iodine thyroid substance**
Thyroidectomy	thi"roid-ek'tuh-me	**thyroid/ectomy**	**To cut out the thyroid**
Thyroiditis	thi"roid-i'tis	**thyroid/itis**	**Inflammation of the thyroid**
Triiodothyronine	tri-i"o-do-thi'ro-nēn	**tri/iod/o/thyronine**	**A three-iodine thyroid substance**
Vasopressin	va-zo-pres'in	**vas/o/press/in**	**The vessel pressure (hormone)**

∗Color legend: **root word**, **prefix**, **suffix**, combining vowel

This table contains most but not all terms presented in this chapter.

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Medical Terms Introduced in Chapter 11

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Chapter 11 Introductory Terms			
Term	**Phonetics**	**Division**	**Basic Definition**
Acoustic	uh-koos'tik	**acoust/ic**	**Pertaining to sound**
Adrenergic	ad"ren-er'jik	**adren/erg/ic**	**Pertaining to adrenal work**
Afferent	af'uhr-unt	**af/ferent**	**To/toward carrying**
Analgesia	an"ul-je'zhuh	**an/alges/ia**	**A state without pain**
Anesthesia	an"es-the'zhuh	**an/esthes/ia**	**A state without sensation**
Anisocoria	an-e"so-kor'e-uh	**anis/o/cor/ia**	**Condition of unequal pupils**
Anticholinergic	an"te-ko"lin-ur'jik	**anti/cholin/erg/ic**	**Pertaining to against acetylcholine work**
Auditory	aw'di-tor"e	**audi/tory**	**Pertaining to hearing**
Aural	aw'rul	**aur/al**	**Pertaining to the ear**
Autonomic	aw"tuh-nom'ik	**auto/nom/ic**	**Pertaining to self-law** (i.e., control)
Axonal	ak'so-nul	**axon/al**	**Pertaining to an axon**
Blepharorrhaphy	blef"uh-ror'uh-fe	**blephar/o/rrhaphy**	**Suturing of the eyelids**
Blepharospasm	blef'uh-ro-spaz'um	**blephar/o/spasm**	**Eyelid violent contraction**
Brachial	bra'ke-ul	**brachi/al**	**Pertaining to the "arm"**
Cerebellar	ser"uh-be'lur	**cerebell/ar**	**Pertaining to the cerebellum**
Cerebral	suh-re'brul	**cerebr/al**	**Pertaining to the cerebrum**
Cerebrospinal	ser"e-bro-spi'nul	**cerebr/o/spin/al**	**Pertaining to the cerebrum and spine**
Ceruminal	suh-roo'mĭ-nul	**cerumin/al**	**Pertaining to wax**
Cholinergic	ko"lin-ur'jik	**cholin/erg/ic**	**Pertaining to acetylcholine work**
Cochlear	kŏk'e-ur	**cochle/ar**	**Pertaining to the "snail shell"** (cochlea)
Conjunctival	kon-junk'tĭ-vul	**conjunctiv/al**	**Pertaining to the conjunctiva**
Corneal	kor'ne-ul	**corne/al**	**Pertaining to the cornea**
Dendritic	den-drit'ik	**dendr/i/tic**	**Pertaining to a "tree"** (dendrite)
Diencephalon	di"un-sef'uh-lon	**dia/encephal/on**	**The through brain**
Ectropion	ek-tro'pe-on	**ec/trop/i/on**	**An out turning**
Efferent	ef"uhr-unt	**ef/ferent**	**Out/outward carrying**
Electroencephalography	e-lek"tro-en-sef"uh-log'raf-e	**electr/o/encephal/o/graphy**	**Recording of electricity of the brain**
Electroretinogram	e-lek"tro-ret'in-o-gram	**electr/o/retin/o/gram**	**A record of electricity of the retina**
Encephalitis	en-sef"uh-li'tis	**encephal/itis**	**Inflammation of the brain**
Encephalomyelitis	en-sef"uh-lo-mi"uh-li'tis	**encephal/o/myel/itis**	**Inflammation of the brain and spinal cord**
Encephalopathy	en-sef"uh-lop'uh-the	**encephal/o/pathy**	**A disease of the brain**
Entropion	en-tro'pe-on	**en/trop/i/on**	**An in turning**
Epidural	ep"ĭ-doo'rul	**epi/dur/al**	**Pertaining to upon the dura** (mater)
Etiology	e"te-ol'uh-je	**eti/o/logy**	**The study of cause**
Exophthalmos	eks"of-thal'mos	**ex/ophthalmos**	**(an) out eye**
Extraocular	eks"truh-ok'u-lar	**extra/ocul/ar**	**Pertaining to outside the eye**
Glossopharyngeal	glos"o-fuh-rin'je-ul	**gloss/o/pharyng/e/al**	**Pertaining to the tongue and throat**
Hemiparesis	hem"e-puh-re'sis	**hemi/paresis**	**Half weakness**
Hemiplegia	hem"e-ple'juh	**hemi/pleg/ia**	**Condition of half paralysis**
Hemisphere	hem'ĭ-sfēr	**hemi/sphere**	**Half a ball**
Hydrocephalus	hi"dro-sef'uh-lus	**hydr/o/cephal/us**	**A water head**
Hyperesthesia	hi"per-es-the'zhuh	**hyper/esthes/ia**	**A condition of excess sensation**
Hyperkalemia	hi"per-ka-le'me-uh	**hyper/kal/emia**	**A blood condition of excess potassium**
Hypermetric	hi"per-met'rik	**hyper/metr/ic**	**Pertaining to excess "measure"** (gait)
Hyperthermia	hi"per-thur'me-uh	**hyper/therm/ia**	**Condition of excess temperature**
Hypertrophy	hi"per'truh-fe	**hyper/trophy**	**A state of excess development**
Hypocalcemia	hi"po-kal-se'me-uh	**hypo/calc/emia**	**A blood condition of low calcium**

Table Continued

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Chapter 11 Introductory Terms			
Term	**Phonetics**	**Division**	**Basic Definition**
Hypoglossal	hi"po-glos'ul	**hypo/gloss/al**	**Pertaining to below the tongue**
Hypokalemia	hi"po-ka-le'me-uh	**hypo/kal/emia**	**A blood condition of low potassium**
Hyponatremia	hi"po-na-tre'me-uh	**hypo/natr/emia**	**A blood condition of low sodium**
Hypoplasia	hi"po-pla'zhuh	**hypo/plas/ia**	**A condition of under-development**
Hypothalamus	hi"po-thal'uh-mus	**hypo/thalamus**	**Below the inner chamber**
Hypothermia	hi"po-thur'me-uh	**hypo/therm/ia**	**Condition of low temperature**
Idiopathic	id"e-o-path'ik	**idi/o/path/ic**	**Pertaining to one's own disease**
Intervertebral	in"ter-ver-te'bral	**inter/vertebr/al**	**Pertaining to between vertebrae**
Intraocular	in"truh-ok'u-lar	**intra/ocul/ar**	**Pertaining to within the eye**
Iridocorneal	ir"i do kor'ne ul	**irid/o/corne/al**	**Pertaining to the iris and cornea**
Keratitis	ker"uh-ti'tis	**kerat/itis**	**Inflammation of the cornea**
Keratoconjunctivitis	ker"uh-to-kun-junk"ti-vi'tis	**kerat/o/conjunctiv/itis**	**Inflammation of the cornea and conjunctiva**
Lacrimal	lak'ri-mul	**lacrim/al**	**Pertaining to tears**
Lumbosacral	lum"bo-sa'krul	**lumb/o/sacr/al**	**Pertaining to the "loin" and sacrum**
Meningeal	meh-nin'je-ul	**mening/e/al**	**Pertaining to the meninges**
Meningitis	men"in-ji'tis	**mening/itis**	**Inflammation of the meninges**
Mesencephalon	mez"en-sef'uh-lon	**mes/encephal/on**	**The mid-brain**
Microphthalmia	mi'krof-thal'me-uh	**micr/ophthalm/ia**	**A condition of small eyes**
Miotic	mi-ot'ik	**mi/o/tic**	**Pertaining to smaller (pupils)**
Monoparesis	mon"o-puh-re'sis	**mono/paresis**	**Single (limb) weakness**
Monoplegia	mon"o-ple'juh	**mono/pleg/ia**	**A condition of single (limb) paralysis**
Mydriatic	mid"re-at'ik	**mydria/tic**	**Pertaining to dilated (pupils)**
Myelopathy	mi'uh-lop'uh-the	**myel/o/pathy**	**Disease of the spinal cord**
Nasolacrimal	na"zo-lak'ri-mul	**nas/o/lacrim/al**	**Pertaining to the nose and tears**
Neuritis	noo-ri'tis	**neur/itis**	**Inflammation of nerves**
Neurologist	noo-rol'uh-jist	**neur/o/log/ist**	**A specialist in nerve study**
Neurology	noo-rol'uh-je	**neur/o/logy**	**The study of nerves**
Neurotropic	noo"ro-tro'pik	**neur/o/trop/ic**	**Pertaining to nerve influencing**
Nociceptor	no"si-sep'tur	**noci/cept/or**	**An injury (pain) receptor**
Oculomotor	ok"u-lo-mo'tur	**ocul/o/motor**	**Eye movement**
Olfactory	ōl-fak'tuh-re	**olfact/ory**	**Pertaining to smell**
Oligodendroglial	ol"i-go-den-dro'gle-ul	**olig/o/dendr/o/gli/al**	**Pertaining to small tree "glue"**
Ophthalmic	of-thal'mik	**ophthalm/ic**	**Pertaining to the eye**
Ophthalmologist	of"thal-mol'uh-jist	**ophthalm/o/log/ist**	**A specialist in eye study**
Ophthalmology	of"thal-mol'uh-je	**ophthalm/o/logy**	**The study of eyes**
Ophthalmoscopy	of"thal-mos'kuh-pe	**ophthalm/o/scopy**	**Viewing of the eyes**
Optic	op'tik	**opt/ic**	**Pertaining to vision**
Osteotomy	os"te-ah-to'me	**oste/o/tomy**	**To cut bone**
Otic	o'tik	**ot/ic**	**Pertaining to the ear**
Otitis	o-ti'tis	**ot/itis**	**Inflammation of the ear**
Otodectes	o"to-dek'tēz	**ot/o/dectes**	**Ear biter**
Otolith	o'to-lith	**ot/o/lith**	**Ear stone**
Otoscope	o'to-skōp	**ot/o/scope**	**An ear viewer**
Ototoxic	o"to-tok'sik	**ot/o/tox/ic**	**Pertaining to ear poison**
Palpebral	pal'pe-brul	**palpebr/al**	**Pertaining to the eyelid**
Paraparesis	par"uh-puh-re'sis	**para/paresis**	**"Near" (i.e., hindlimb) weakness**
Paraplegia	par"uh-ple'je	**para/pleg/ia**	**A condition of "near" (i.e., hindlimb) paralysis**
Parasympatholytic	par"uh-sim"path-o-lit'ik	**parasympath/o/lytic**	**Pertaining to breaking the parasympathetic (system)**
Parasympathomimetic	par"uh-sim"path-o-mi-met'ik	**parasympath/o/mimetic**	**Pertaining to mimicking the parasympathetic (system)**

Table Continued

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Chapter 11 Introductory Terms			
Term	**Phonetics**	**Division**	**Basic Definition**
Periocular	per"e-ok'u-lar	**peri/ocul/ar**	**Pertaining to around the eye**
Photophobia	fo"to-fo'be-uh	**phot/o/phobia**	**Light "fear"** (i.e., aversion)
Proprioceptive	pro"pre-o-sep'tiv	**propri/o/cept/ive**	**Pertaining to one's own receptor**
Quadriplegia	kwod"rĭ-ple'juh	**quadri/pleg/ia**	**A condition of four paralysis** (i.e., 4 limbs)
Retinopathy	ret"in-op'uh-the	**retin/o/pathy**	**A disease of the retina**
Retrograde	ret'ro-grād	**retro/grade**	**A backward step**
Scleral	sklār'ul	**scler/al**	**Pertaining to the sclera**
Semicircular	sem"e-ser'ku-lur	**semi/circul/ar**	**Pertaining to a partial circle**
Somatic	so-mat'ik	**somat/ic**	**Pertaining to the body**
Subarachnoid	sub"uh-rak'noid	**sub/arachnoid**	**Beneath the arachnoid**
Subdural	sub-doo'rul	**sub/dur/al**	**Pertaining to beneath the dura** (mater)
Sympatholytic	sim"path-o-lit'ik	**sympath/o/lytic**	**Pertaining to breaking the sympathetic** (system)
Sympathomimetic	sim"path-o-mĭ-met'ik	**sympath/o/mimetic**	**Pertaining to mimicking the sympathetic** (system)
Synaptic	sin-ap'tik	**synap/tic**	**Pertaining to a connection** (i.e., synapse)
Tetraplegia	tet"ruh-ple'juh	**tetra/pleg/ia**	**A condition of four paralysis** (i.e., 4 limbs)
Tonometry	to-nom'uh-tre	**ton/o/metry**	**Measurement of pressure**
Tympanic	tim-pan'ik	**tympan/ic**	**Pertaining to a drum**
Uveitis	u"ve-i'tis	**uve/itis**	**Inflammation of the uvea**
Vagal	va'gul	**vag/al**	**Pertaining to the vagus** (nerve)
Vagosympathetic	va"go-sim"puh-thet'ik	**vag/o/sympathet/ic**	**Pertaining to the vagus and sympathetic** (nerves)
Vestibular	ves-tib'u-lur	**vestibul/ar**	**Pertaining to the vestibule**
Vestibulocochlear	ves-tib"u-lo-kok'le-ur	**vestibul/o/cochle/ar**	**Pertaining to the vestibule and cochlea**
Visceral	vis'er-ul	**viscer/al**	**Pertaining to organs**

∗Color legend: **root word**, **prefix**, **suffix**, combining vowel

This table contains most but not all terms presented in this chapter.

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Medical Terms Introduced in Chapter 12

Chapter 12 Introductory Terms			
Term	**Phonetics**	**Division**	**Basic Definition**
Agonist	ag'uh-nist"	**agon/ist**	**One that competes**
Analgesic	an"ul-je'zik	**an/alges/ic**	**Pertaining to without pain**
Anesthesia	an"es-the'zhuh	**an/esthes/ia**	**Condition without sensation**
Anesthetic	an" es-thĕ'tik	**an/esthet/ic**	**Pertaining to no sensation**
Antagonist	an-tag'uh-nist	**ant/agon/ist**	**One that opposes competition**
Anthelmintic	ant"hel-min'tik	**ant/helmin/tic**	**Pertaining to against worms**
Antibiotic	an"ti-bi-ot'ik; an"te-bi-ot'ik	**anti/bi/o/tic**	**Pertaining to against life**
Antiemetic	an"te-ĕ-met'ik	**anti/eme/tic**	**Pertaining to against vomiting**
Antimicrobial	an"ti-mi-kro'be-ul	**anti/microb/i/al**	**Pertaining to against microbes**
Antimycotic	an"te-mi-kot'ik an"ti-mi-kot'ik	**anti/myc/o/tic**	**Pertaining to against fungus**
Antipyretic	an"te-pi-rĕ'tik	**anti/pyr/e/tic**	**Pertaining to against fever**
Antiseptic	an"tĭ-sep'tik	**anti/sep/tic**	**Pertaining to against infection**
Antitussive	an"te-tus'iv; an"ti-tus'iv	**anti/tuss/ive**	**Pertaining to against coughing**
Asepsis	a-sep'sis	**a/sep/sis**	**A state without infection**
Aseptic	a-sep'tik	**a/sep/tic**	**Pertaining to no infection**

Table Continued

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Chapter 12 Introductory Terms			
Term	**Phonetics**	**Division**	**Basic Definition**
Bactericidal	bak-ter"ĭ-sī'dul	**bacteri/cid/al**	**Pertaining to bacteria killing**
Bacteriostatic	bak-tēr"e-o-stat'ik	**bacteri/o/static**	**Pertaining to inhibiting bacteria**
Biological	bi-o-loj'ĭ-kul	**bi/o/log/i/cal**	**Pertaining to life knowledge**
Biotransformation	bi"o-trans"for-ma'shun	**bi/o/transform/a/tion**	**Process of life changing**
Centimeter	sen'tĭ-me"ter	**centi/meter**	**Hundredth measure**
Contraindication	kon"truh-in"dĭ-ka'shun	**contra/indica/ion**	**To make against advice**
Cytotoxic	si'to-tok"sik	**cyt/o/tox/ic**	**Pertaining to cell poison**
Deciliter	des'ĭ-le"ter	**deci/liter**	**Tenth volume**
Disinfectant	dis"in-fek'tant	**dis/infect/ant**	**One that reverses infection**
Emetic	e-met'ik	**eme/tic**	**Pertaining to vomiting**
Enteral	en'ter-ul	**enter/al**	**Pertaining to intestines**
Enteric	en-tār'ik	**enter/ic**	**Pertaining to intestines**
Hepatotoxicity	hĕ-pat"o-tok-sis'ĭ-te	**hepat/o/toxic/ity**	**State of liver poisoning**
Hydrophilic	hi"dro-fil'ik	**hydr/o/phil/ic**	**Pertaining to water loving**
Iatrogenic	i-at"ro-gen'ik	**iatr/o/gen/ic**	**Pertaining to physician produced**
Intradermal	in"truh-der'mul	**intra/derm/al**	**Pertaining to within skin**
Intramuscular	in"truh-mus'ku-lahr	**intra/muscul/ar**	**Pertaining to within muscle**
Intranasal	in"truh-na'zul	**intra/nas/al**	**Pertaining to within the nose**
Intraosseous	in"truh-os'e-us	**intra/osse/ous**	**Pertaining to within bone**
Intraperitoneal	in"truh-pār"ĭ-tuh-ne'ul	**intra/periton/e/al**	**Pertaining to within the abdomen**
Intratracheal	in"truh-tra'ke-ul	**intra/trache/al**	**Pertaining to within the trachea**
Intravenous	in"truh-ve'nus	**intra/ven/ous**	**Pertaining to within veins**
Kilogram	kil'o-gram; ke'lo-gram	**kilo/gram**	**Thousand weights**
Lipophilic	lip"o-fil'ik	**lip/o/phil/ic**	**Pertaining to fat loving**
Microgram	mi'kro-gram	**micro/gram**	**Small (millionth) weight**
Microliter	mi"kro-le'ter	**micro/liter**	**Small (millionth) volume**
Milligram	mil'ĭ-gram	**milli/gram**	**Thousandth weight**
Milliliter	mil'ĭ-le-ter	**milli/liter**	**Thousandth volume**
Millimeter	mil'ĭ-me"ter	**milli/meter**	**Thousandth measure**
Nephrotoxicity	nef"ro-tok-sis'ĭ-te	**nephr/o/toxic/ity**	**State of kidney poisoning**
Parenteral	pahr-en'ter-ul	**par/enter/al**	**Pertaining to around intestines**
Percent	per-sent'	**per/cent**	**For each hundred**
Pesticide	pes'tĭ-sīd	**pest/i/cide**	**A pest killer**
Pharmaceutical	fahr"muh-soo-tĭ-kul	**pharmaceutic/al**	**Pertaining to drugs**
Pharmacodynamic	fahr"muh-ko-di-nam'ik	**pharmac/o/dynam/ic**	**Pertaining to drug power**
Pharmacokinetic	fahr"muh-ko-kĭ-net'iks	**pharmac/o/kinet/ic**	**Pertaining to drug movement**
Pharmacology	fahr"muh-kol'uh-je	**pharmac/o/logy**	**The study of drugs**
Probiotic	pro"bi-ot'ik	**pro/bi/o/tic**	**Pertaining to for life**
Subcutaneous	sub"ku-ta'ne-us	**sub/cutan/e/ous**	**Pertaining to beneath the skin**
Transdermal	tranz-dur'mul	**trans/derm/al**	**Pertaining to across the skin**
Virucidal	vi"ruh-si'dul	**viru/cid/al**	**Pertaining to virus killing**
Zoonosis	zo"o-no'sis	**zo/o/nos/is**	**Condition of animal disease (i.e., in people)**

*Color legend: **root word**, **prefix**, **suffix**, combining vowel

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