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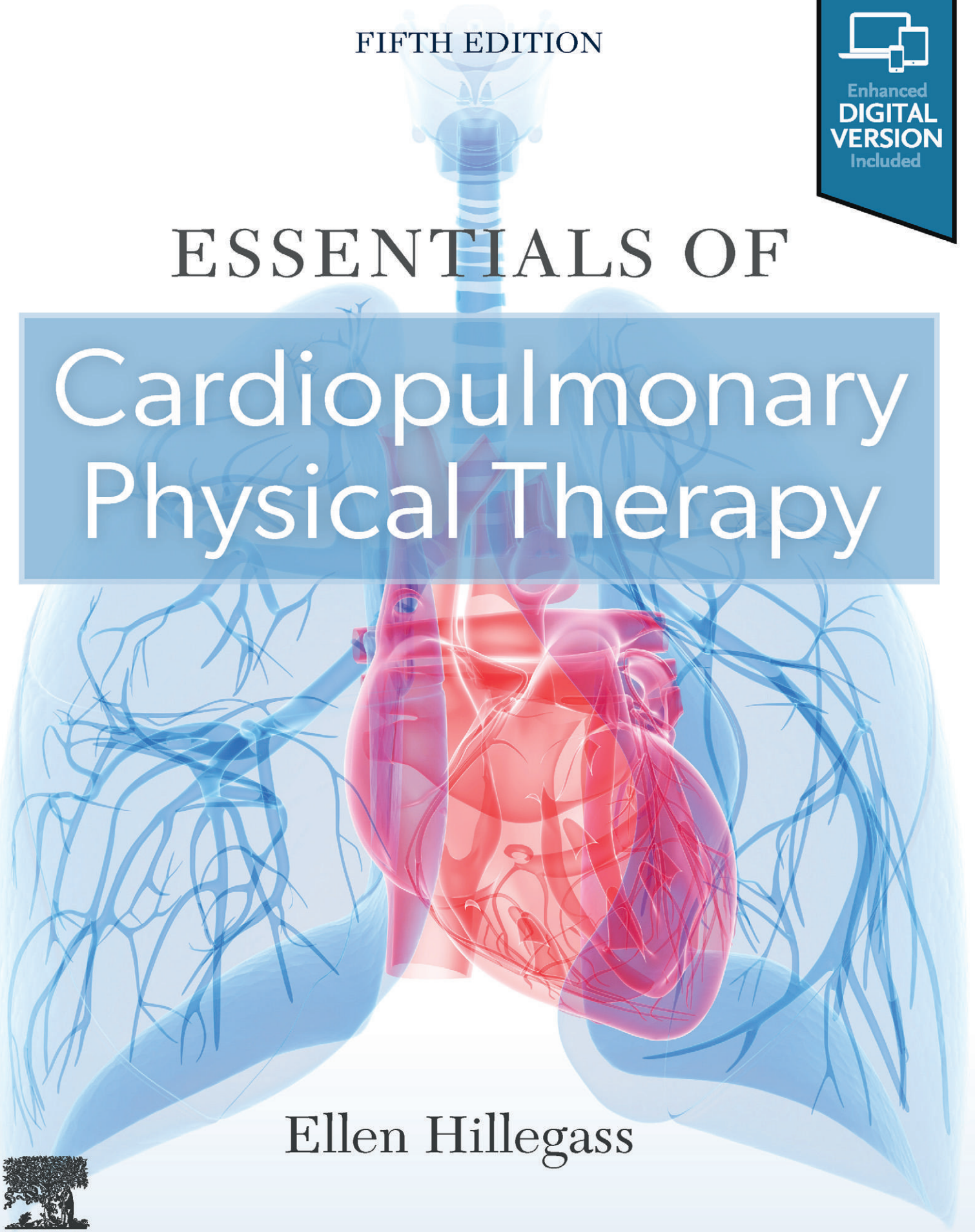

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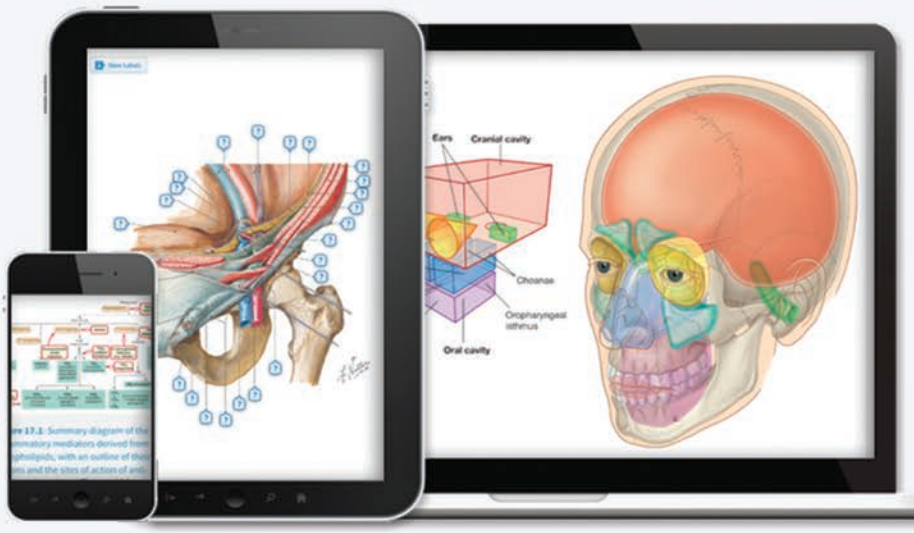

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Essentials of Cardiopulmonary Physical Therapy

FIFTH EDITION

Ellen Hillegass, PT, EdD, CCS, FAPTA
President and CEO, PT Cardiopulmonary Educators
President and CEO, Cardiopulmonary Specialists, Inc.
Adjunct Professor, Mercer University, Atlanta, Georgia

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Senior Content Development Specialist: Maria Broeker
Senior Project Manager: Umarani Natarajan
Publishing Services Manager: Shereen Jameel
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This book is dedicated to my beloved family for all their love and support, as well as their understanding during my endless hours of working on this edition:

To my husband Dan, who is my rock and my biggest supporter whom I could not live without.

To my three wonderful children: Jamie, Christi, and Patrick (and his wife Tiffany) who give me moral support, make me laugh, and who constantly try to keep me up to date on all the modern technologies that have helped me communicate with them, communicate with my colleagues, and write this book! They keep me young with their ideas and assistance and they constantly have a "joie de vivre"; I also need to dedicate this to my three grandchildren: Grae, Grimes, and Brooks, as well as to my future grandchildren. These wonderful children are our future. In addition, my grand dogs give me great pleasure and are part of my full life: Gus, Beans, Darcy, and Roo.

To my dogs: Bear and Ernie (and in loving memory of Sparky) who kept my feet warm while I sat for hours at the computer working on this edition but demanded daily play and provided a wonderful mental break from writing.

And,

In loving memory of my parents, John and Norma Zettler, who kept me busy as their daughter and caregiver while they were alive and were always proud of everything I did.

And, to my brother-in-law George Hillegass, who was an inspiration to everyone he knew and met with his positive attitude and fighting spirit that he had up until the day he died from pancreatic cancer.

In addition, I dedicate this edition:

To my colleagues who keep me informed, give me moral and intellectual support, and who keep me inspired to maintain my passion for the field of cardiovascular and pulmonary physical therapy. I especially rely on the support and inspiration of some very dear friends/colleagues including Angela Campbell, Talia Pollok, Morgan Johanson, Dianne Jewell, Andrew Ries, Claire Rice, and Joanne Watchie.

To my current and all of my former students in DPT programs and from continuing education courses I have presented, as well as my former residents. I have especially enjoyed being a mentor to many rising cardiopulmonary specialists. My former residents will be seen throughout this edition as co-authors and you should expect to see their names as they rise in the profession: Tiffany Haney, Stephen Ramsey, Jenna Floyd Hightower, Liana Geddes, Cydney Nagridge Reilly, Ben Purrington, and Erica Colclough.

And finally, I can never forget my very special friends/mentors to whom I am forever grateful and whose memories and teachings are with me always: Michael Pollock (1937–1998), Linda Crane (1951–1999), and Gary Dudley (1952–2006).

Contributors

Jennifer Adams, FNP
Emergency Medicine
Baylor Scott & White Health
Dallas, Texas

Pamela Bartlo, PT, DPT, CCS
Clinical Associate Professor
Physical Therapy
D'Youville College
Buffalo, New York

Naomi Bauer, DPT
Program Director
Pulmonary Rehab and Cardiopulmonary Therapy
WakeMed Health & Hospitals
Raleigh, North Carolina

Traci Betts, PT, DPT, CCS
Assistant Professor
School of Physical Therapy
University of Texas Southwestern
Dallas, Texas

Benjamin Carrion, PT, DPT
Physical Therapist
Department of Physical Therapy and Occupational Therapy
Duke University Medical Center
Durham, North Carolina

Rohini Krishnan Chandrashekar, PT, MS, CCS
Physical Therapist
Rehabilitation
Nexus Specialty Hospital
Houston, Texas

Physical Therapy Consultant
Vikasid Solutions, LLC
Texas

Meryl I. Cohen, DPT, MS, CCS, FAPTA
Associate Professor
Department of Physical Therapy
University of Miami Miller School of Medicine
Coral Gables, Florida

Kelley Crawford, DPT, CCS
Lead Physical Therapist
Rehabilitation Medicine Department
Maine Medical Center
Portland, Maine

Adjunct Faculty
Physical Therapy
University of New England
Portland, Maine

Rebecca H. Crouch, PT, DPT, MS, CCS, MAACVPR
Assistant Professor
Graduate Program of Physical Therapy
Campbell University, Duke University Medical Center
Chapel Hill, North Carolina

Physical Therapist
Physical Therapy
Duke University Hospital
Durham, North Carolina

Nicole DeLuca, Doctor of Physical Therapy
Physical Therapist - Board Certified Cardiopulmonary Clinical Specialist
Physical Medicine and Rehabilitation
Miami VA Healthcare System
Miami, Florida

Konrad J. Dias, PT, DPT, PhD, CCS
Professor
Physical Therapy
Maryville University of St. Louis
St. Louis, Missouri

Jennifer Edelschick, PT, DPT
Coordinator of Acute Pediatric Physical & Occupational Therapy
Physical & Occupational Therapy
Duke Health
Durham, North Carolina

Germaine Ferreira, PT, DPT, MSPT, BHMS
Assistant Professor
Doctor of Physical Therapy Program
University of St. Augustine for Health Sciences
Austin, Texas

Ann Fick, PT, DPT, MS, CCS
Director of Clinical Education
Physical Therapy
Maryville University
St. Louis, Missouri

Jenna Floyd, PT, DPT, CCS
Critical Care Physical Therapist
Physical Medicine and Rehabilitation
Mayo Clinic
Jacksonville, Florida

Liana Geddes Pt, DPT, CCS
Physical Therapist
Critical Care Physical Therapy
Piedmont Atlanta Hospital
Atlanta, Georgia

Natalie Goldberg, DPT, CCS
Physical Therapist
Department of Rehabilitation
Hartford Hospital
Hartford, Connecticut

Adjunct Professor
Department of Rehabilitation Sciences
University of Hartford
West Hartford, Connecticut

Kate Grimes, PT, MS, DPT, CCS, CCRP
Senior Physical Therapist
Cardiovascular Health Center/Cardiac Rehab
Newton-Wellesley Hospital
Newton, Massachusetts

Meghan Gushurst, DPT
Physical Therapist III
Rehabilitation Services
Advocate Christ Medical Center
Oak Lawn, Illinois

Tiffany Haney, PT, Cardiopulmonary Certified Specialist
Physical Therapist
Rehabilitation Services
Piedmont Healthcare
Atlanta, Georgia

Ellen Hillegass, PT, EdD, CCS, FAPTA
President and CEO, PT Cardiopulmonary Educators
Atlanta, Georgia

Amanda Hollander, DPT
Physical Therapist
Rehabilitation Services
Lucile Packard Children's Hospital Stanford
Palo Alto, California

Morgan Keller Johanson, PT, MSPT, CCS
President
Continuing Education
Good Heart Education
Bellaire, Michigan

Adjunct Faculty
Doctorate of Physical Therapy
University of Toledo
Toledo, Ohio

Physical Therapist
Rehabilitation
Grand Traverse Pavilions
Traverse City

Kristin Lefebvre, PT, PhD
Professor
Physical Therapy
Concordia University St. Paul
St. Paul, Minnesota

Ana Lotshaw, PT, PhD, CCS
Advanced Clinical Specialist
Physical Medicine and Rehabilitation
Baylor Scott and White Institute for Rehabiliation - Baylor University Medical Center
Dallas, Texas

Sean T. Lowers, PT, DPT, CCS
Physical Therapist
Cardiopulmonary Rehab
Duke Health
Durham, North Carolina

Kate MacPhedran, PT, PhD (CCS)
Assistant Professor
Doctor of Physical Therapy Program
Gannon University
Erie, Pennsylvania

Frailty Consultant and Cardiac Researcher
Consultants in Cardiovascular Disease, LLC.
Erie, Pennsylvania

Hannah F. McHugh, PT, DPT, CCS
Senior Physical Therapist
Department of Physical and Occupational Therapy
Duke University Hospital
Durham, North Carolina

Adjunct Faculty
Department of Physical Therapy
Elon University
Elon, North Carolina

Susan Butler McNamara
Portland, Maine

Harold Merriman, PT, PhD, CLT
Associate Professor
Department of Physical Therapy
University of Dayton
Dayton, Ohio

Andrew Mills, PT, DPT, Board Certified Cardiovascular and Pulmonary Specialist
Assistant Professor
Physical Therapy
Touro University Nevada
Henderson, Nevada

Cydney O. Nagridge, PT, DPT, CCS
Physical Therapist
Rehab Therapy–Acute Care
University of Wisconsin Hospitals and Clinics
Madison, Wisconsin

Ashley Parish, PT, DPT, CRT, CCS
Assistant Professor
Department of Physical Therapy
UAB
Birmingham, Alabama

Amy Pawlik, PT, DPT
Co-Owner/Physical Therapist
Vitality Women's Physical Therapy & Wellness
Elmhurst, Illinois

Christiane Perme, PT, CCS, FCCM
Rehabilitation Educations Specialist
Rehabilitation Services
Houston Methodist Hospital
Houston, Texas

President
Perme ICU Rehab Seminars
Houston, Texas

Talia Pollok, PT, DPT, CCS
Physical Therapist III
Therapy Services
University of Virginia Medical Center
Charlottesville, Virginia

Stephen Ramsey, PT, DPT, CCS
Critical Care Physical Therapist
Rehabilitation Services
Piedmont Hospital
Atlanta, Georgia

Adjunct Faculty
College of Health Professions
Mercer University
Atlanta, Georgia

Adjunct Faculty
College of Health & Human Sciences
Western Carolina University
Cullowhee, North Carolina

Debra Seal, PT, DPT, PCS
Department Director
Rehabilitation Services
Lucile Packard Children's Hospital Stanford
Palo Alto, California

Preface

Originally, this text was developed to meet the needs of the physical therapy community because cardiopulmonary was identified as one of the four clinical science components in a physical therapy education program, as well as in clinical practice. Those aspects of physical therapy commonly referred to as "cardiovascular and pulmonary physical therapy" are recognized as fundamental components of the knowledge base and practice base of all entry-level physical therapists. Therefore, this text was developed for entry-level physical therapists, as well as individuals in practice who need more in-depth knowledge of cardiopulmonary content. This text is also used by many clinicians studying for advanced practice board certification, in addition to those involved in residency programs. Although intended primarily for physical therapists, this text has been useful to practitioners in various disciplines who teach students or who work with patients suffering from primary and secondary cardiopulmonary dysfunction. This fifth edition can also be used by all practitioners who teach entry-level clinicians, work with residents, as well as to help in clinical practice of patients with cardiopulmonary dysfunction.

This fifth edition has gone through update and revision from the fourth edition to make the text more user friendly and provide more interactive learning. The same six sections exist: *Anatomy and Physiology; Pathophysiology; Diagnostic Tests and Procedures; Surgical Interventions, Monitoring, and Support; Pharmacology;* and *Cardiopulmonary Assessment and Intervention.* The six sections were maintained because they facilitate the progression of understanding of the material to be able to perform a thorough assessment and provide an optimal intervention, as well as provide measurable outcomes to assess change.

The revisions you should notice include both major and minor changes. Two NEW chapters were added: Chapter 4, *Management of Cardiovascular Disease in Women* and Chapter 8, *Pulmonary Vascular Disease*. Both are very much needed for understanding these special topics in cardiopulmonary disease, and I celebrate their addition!

All chapters have been revised and supplemented with many updated figures and tables; there are also some videos to help the learner visualize the written information. Additional figures, case studies, and resource material can also be found on the Evolve website that accompanies this text. The number of clinical notes was increased to help clinicians and students understand certain clinical findings and help them relate them to the pathophysiology of cardiovascular and pulmonary disease. All chapters were updated with new information, technology, and research.

Each chapter had specific revisions that should be highlighted. Chapters 1 and 2, which explain anatomy and physiology, increased the number of figures to help the learner relate the pathophysiology to the normal anatomy and physiology. Chapter 3, *Ischemic Cardiovascular Conditions and Other Vascular Pathologies,* underwent revision particularly in areas that were lacking such as venous dysfunction including deep vein thrombosis. New material was added, so that you will now find hypertension, peripheral arterial disease, cerebrovascular disease, renal disease, and aortic aneurysm in this chapter, in addition to ischemic disease. Chapter 5, *Cardiac Muscle Dysfunction and Failure,* was restructured and revised to improve the flow and understanding of this important pathologic condition, as well as all new figures and tables to help understand heart dysfunction and failure.

Because of the complexities and number of conditions of restrictive lung dysfunction, many more tables were created in Chapter 6 to separate the material and assist the learner to identify key information quickly. Chapter 7, *Chronic Obstructive Pulmonary Diseases*, was updated and revised to emphasize the importance of this disease and the fact that COPD is the third leading cause of death. Revisions in Chapter 9, *Cardiopulmonary Implications of Specific Diseases*, emphasize information on obesity, diabetes, and metabolic syndrome, as well as cancer and neuromuscular diseases.

New technologies and advancements in diagnostic tests and surgical procedures were added to Chapters 10, 11, 12, and 13. Chapter 13, *Cardiovascular and Thoracic Interventions*, underwent revision with many new figures and text. The advances in transplantation were discussed in Chapter 14 and monitoring and life support (Chapter 15) was revised to increase the depth of information on ventilators, as well as other monitoring equipment found in intensive care units and used by Physical Therapists when mobilizing patients earlier.

As advances in healthcare and diagnostics occur, so do improvements and changes in medications, so both *Cardiovascular Medications* (Chapter 16) and *Pulmonary Medications* (Chapter 17) required updating. Chapter 18, *Examination and Assessment Procedures*, was revised with the addition of new tables to help organize assessments and improve the understanding of this material. Chapter 19, *Interventions for Acute Cardiopulmonary Conditions*, added a greater emphasis on early mobility and Chapter 20, *Interventions and Prevention Measures for Individuals With Cardiovascular Disease, or Risk of Disease* had major updating and revision, new clinical notes, and many new figures and tables. Chapter 21, *Pulmonary Rehabilitation* was revised to correspond with changes in the new pulmonary rehabilitation (PR) definition and in the changing practice since Medicare revised payment for PR. Chapter 22, *Pediatric Cardiopulmonary Physical Therapy* and Chapter 23, the outcomes chapter was totally revamped and provides great information for measurement of improvement in the cardiopulmonary patient population. Finally, Chapter 24, *The Lymphatic System* underwent update and revision.

Whenever possible, case studies are provided to exemplify the material being presented. Additional case studies are found on Evolve.

No matter how well you understand the material in this book, it will not make you a master clinician, skilled in the assessment and treatment of cardiovascular and pulmonary disorders. To become even a minimally competent clinician, you will have to practice physical therapy under the tutelage of an experienced clinician. *Essentials of Cardiopulmonary Physical Therapy* cannot provide you with everything there is to know about the assessment and treatment of cardiovascular and pulmonary disorders. It will provide the essentials as the title indicates. Learning is a continuous process, and technology and treatment are forever improving; therefore, this text provides clinicians, as well as educators, with the most current information at the time of publication.

It is my true hope that you appreciate this edition and are able to learn from all the wealth of information provided by such wonderful contributors. Without heart and breath there is no therapy!

Acknowledgments

"Change is good and change equals opportunity!" This statement explains how I have approached each edition, but most especially this edition! Hopefully, you will gain knowledge and insight from all the changes as there are many excellent contributions from my colleagues, who are THE experts in cardiovascular and pulmonary physical therapy and who poured their passion into their chapters. This edition is what I consider the "Mentoring" edition…. Many of the co-authors in the chapters are newly recognized cardiopulmonary specialists and past Residents of Cardiopulmonary Residency programs and new to writing. They were mentored along the way, and what they provided to this edition was amazing content, figures, videos, and updated material that makes this text stand out. We can all learn from these experts and you will as you dig into the material in the following pages.

This edition was written and published during COVID-19, so of course there are sections throughout the text discussing COVID-19. Speaking of change… COVID-19 has certainly made our lives different and changed everything we do! But, now more than ever, we need to understand the cardiovascular and pulmonary system and evaluate our patients for risk for, or presence of, dysfunction in these important systems!

Learning does not stop with this text. Continuing education is a vital component of lifelong learning so I would also encourage all of my readers to continue their lifelong learning in cardiopulmonary physical therapy by using always updated webinars from PT Cardiopulmonary Educators from their website: www.ptcardioed.com.

During the publication phase of the first edition of the *Essentials of Cardiopulmonary Physical Therapy,* I was always worried about new developments in the field of cardiovascular and pulmonary diagnosis and treatment that were not going to be covered in the book. My very first editor, Margaret Biblis, kept saying "that's what the next edition is for" and that is how I approached the second edition and again the third and fourth edition and now this edition. I have saved comments and suggestions along the way, as well as attended conferences regularly to stay current with new developments in the field. And, with the age of the internet, you have access to the new Evolve site that accompanies this text. Instructional material including PowerPoint presentations and a test bank is available to instructors in the course, as well as updated information.

So, I would like to thank all the amazing experts who have helped with this fifth edition, including each of the wonderful contributors, as well as all those clinicians, students, and faculty members who provided feedback on previous editions and who continue to use this book in their courses and their everyday practice. I would like to especially thank the contributors for their ability to work under my constant nagging to achieve their deadlines and for providing great material including figures, tables, and clinical notes.

Of course, my family and my dogs need to be acknowledged for all the time I spent at the computer working on this edition instead of spending time with them.

Lastly, this edition truly would not be published were it not for my wonderful editor, Maria Broeker. Thanks, Maria!

Contents

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1

Anatomy of the cardiovascular and pulmonary systems

Konrad J. Dias, Germaine Ferreira

CHAPTER OUTLINE

This chapter describes the anatomy of the cardiovascular and pulmonary systems because it is relevant to the physical therapist. Knowledge of the anatomy of these systems provides clinicians with the foundation to perform the appropriate examination and provide optimal treatment interventions for individuals with cardiopulmonary dysfunction. An effective understanding of cardiovascular and pulmonary anatomy allows for comprehension of function and an appreciation of the central components of oxygen and nutrient transport to peripheral tissue. A fundamental assumption is made; namely, that the reader already possesses some knowledge of anatomic terms and cardiopulmonary anatomy.

Thorax

The bony thorax covers and protects the major organs of the cardiopulmonary system. Within the thoracic cavity exists the heart, housed within the mediastinum centrally, and laterally are two lungs. The bony thorax provides a skeletal framework for the attachment of the muscles of ventilation.

The thoracic cage (Fig. 1.1) is conical at both its superior and inferior aspects and somewhat kidney shaped in its transverse aspect. The skeletal boundaries of the thorax are the 12 thoracic vertebrae dorsally, the ribs laterally, and the sternum ventrally.

Sternum

The sternum, or breastbone, is a flat bone with three major parts: manubrium, body, and xiphoid process (see Fig. 1.1). Superiorly located within the sternum, the manubrium is the thickest component articulating with the clavicles and first and second ribs. A palpable jugular notch or suprasternal notch is found at the superior border of the manubrium of the sternum. Inferior to the manubrium lies the body of the sternum, articulating laterally with ribs three to seven. The sternal angle, or "angle of Louis," is the anterior angle formed by the junction of the manubrium and the body of the sternum. This easily palpated structure is in level with the second costal cartilage anteriorly and thoracic vertebrae T4 and T5 posteriorly. The most caudal aspect of the sternum is the xiphoid process, a plate of hyaline cartilage that ossifies later in life.

The sternal angle marks the level of bifurcation of the trachea into the right and left main stem bronchi and provides for the pump-handle action of the sternal body during inspiration.[1]

Pectus excavatum is a common congenital deformity of the anterior wall of the chest in which several ribs and the sternum grow abnormally. This produces a caved-in or sunken appearance of the chest. It is present at birth, but rapidly progresses during the years of bone growth in the early teenage years. These patients have several pulmonary complications, including shortness of breath caused by altered mechanics of the inspiratory muscles on the caved-in sternum and ribs, and often have cardiac complications caused by the restriction (compression) of the heart.[2]

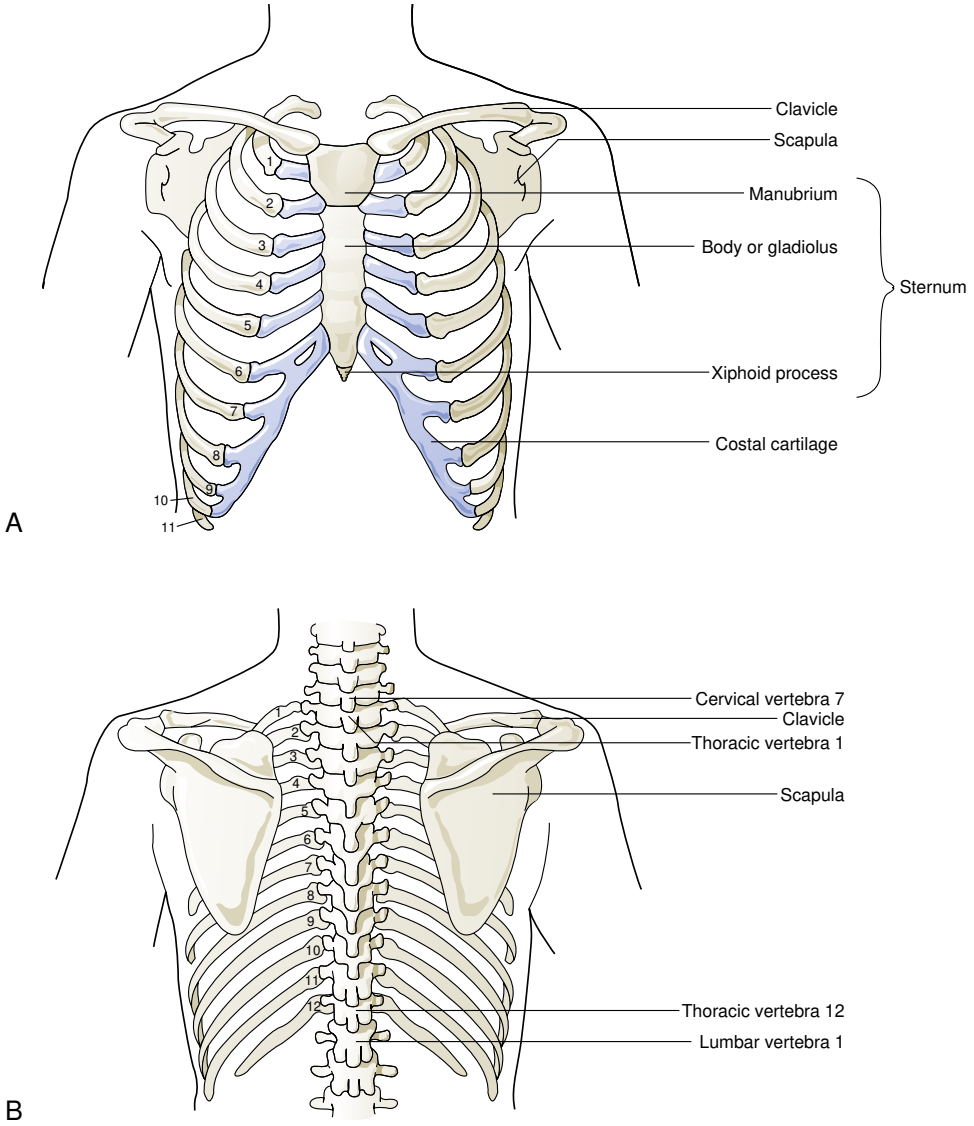

Figure 1.1 (A) Anterior. (B) Posterior views of the bones of the thorax. (From Hicks GH: *Cardiopulmonary anatomy and physiology*, Philadelphia, 2000, Saunders.)

To gain access to the thoracic cavity for surgery, including coronary artery bypass grafting, the sternum is split in the median plane and retracted. This procedure is known as a median sternotomy. Flexibility of the ribs and cartilage allows for separation of the two ends of the sternum to expose the thoracic cavity.[3]

Ribs

The ribs, although considered "flat" bones, curve forward and downward from their posterior vertebral attachments toward their costal cartilages. The first seven ribs attach via their costal cartilages to the sternum and are called the true ribs (also known as the vertebrosternal ribs); the lower five ribs are termed the false ribs—the 8th, 9th, and 10th ribs attach to the rib above by their costal cartilages (the vertebrochondral ribs), and the 11th and 12th ribs end freely (the vertebral ribs; see Fig. 1.1). The true ribs increase in length from above downward, and the false ribs decrease in length from above downward.

Each rib typically has a vertebral end separated from a sternal end by the body or shaft of the rib. The head of the rib (at its vertebral end) is distinguished by a twin-faceted surface for articulation with the facets on the bodies of two adjacent thoracic vertebrae. The cranial facet is smaller than the caudal, and a crest between these permits attachment of the interarticular ligament.

Fig. 1.2 displays the components of typical ribs 3 to 9, each with common characteristics, including a head, neck, tubercle, and body. The neck is the 1-inch long portion of the rib extending laterally from the head; it provides attachment for the anterior costotransverse ligament along its cranial border. The tubercle at the junction of the neck and the body of the rib consists of an articular and a nonarticular portion. The articular part of the tubercle (the more medial and inferior of the two) has a facet for articulation with the transverse process of the inferior-most vertebra to which the head is connected. The nonarticular part of the tubercle provides attachment for the ligament of the tubercle.

The shaft, or body, of the rib is simultaneously bent in two directions and twisted about its long axis, presenting two surfaces (internal and external) and two borders (superior and inferior). A costal groove for the intercostal vessels and nerve extends along the inferior border dorsally but changes to the

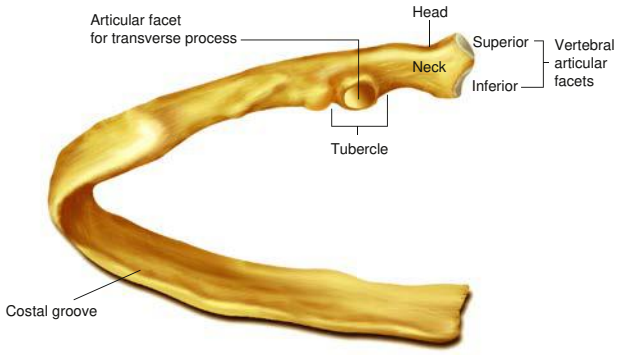

Figure 1.2 Typical middle rib as viewed from the posterior. The head end articulates with the vertebral bones, and the distal end is attached to the costal cartilage of the sternum. (From Wilkins RL: *Egan's fundamentals of respiratory care*, ed 9, St. Louis, 2009, Mosby.)

internal surface at the angle of the rib. The sternal end of the rib terminates in an oval depression into which the costal cartilage makes its attachment.

Although rib fractures may occur in various locations, they are more common in the weakest area where the shaft of the ribs bend—the area just anterior to its angle. The first rib does not usually fracture because it is protected posteroinferiorly by the clavicle. When it is injured, the brachial plexus of nerves and subclavian vessel injury may occur.[4] Lower rib fractures may cause trauma to the diaphragm resulting in a diaphragmatic hernia. Rib fractures are extremely painful because of their profound nerve supply. It is important for all therapists to recommend breathing, splinting, and coughing strategies for patients with rib fractures. Paradoxical breathing patterns and a flail chest may also need to be evaluated in light of multiple rib fractures in adjacent ribs.[3]

Chest tubes are inserted above the ribs to avoid trauma to vessels and nerves found within the costal grove. A chest tube insertion involves the surgical placement of a hollow, flexible drainage tube into the chest. This tube is used to drain blood, air, or fluid around the lungs and effectively allow the lung to expand. The tube is placed between the ribs and into the space between the inner lining and the outer lining of the lung (pleural space).

The 1st, 2nd, 10th, 11th, and 12th ribs are unlike the other, more typical ribs. The first rib is the shortest and most curved of all the ribs. Its head is small and rounded and has only one facet for articulation with the body of the first thoracic vertebra. The sternal end of the first rib is larger and thicker than it is in any of the other ribs. The second rib, although longer than the first, is similarly curved. The body is not twisted. There is a short costal groove on its internal surface posteriorly. The 10th through 12th ribs each have only one articular facet on their heads. The 11th and 12th ribs (floating ribs) have no necks or tubercles and are narrowed at their free anterior ends. The 12th rib sometimes is shorter than the first rib.

The respiratory system

The respiratory system includes the bony thorax, the muscles of ventilation, the upper and the lower airways, and the pulmonary circulation. The many functions of the respiratory system include gas exchange, fluid exchange, maintenance of a relatively low-volume blood reservoir, filtration, and metabolism, and they necessitate an intimate and exquisite interaction of these various components. Because the thorax has already been discussed, this section deals with the muscles of ventilation, the upper and lower airways, and the pulmonary circulation.

Muscles of ventilation

Ventilation, or breathing, involves the processes of inspiration and expiration. For air to enter the lungs during inspiration, muscles of the thoracic cage and abdomen must move the bony thorax to create changes in volume within the thorax and cause a concomitant reduction in the intrathoracic pressure. Inspiratory muscles increase the volume of the thoracic cavity by producing bucket-handle and pump-handle movements of the ribs and sternum, as depicted in Fig. 1.3. The resultant reduced intrathoracic pressure generated is below atmospheric pressure, forcing air into the lungs to help normalize pressure differences. The essential muscles to achieve the active process of inspiration at rest are the diaphragm and internal intercostals. To create a more forceful inspiration during exercise or cardiopulmonary distress, accessory muscles assist with the inspiration. The accessory muscles include the sternocleidomastoid, scalenes, serratus anterior, pectoralis major and minor, trapezius, and erector spinae muscles.

Diaphragm

The diaphragm is the major muscle of inspiration. It is a musculotendinous dome that forms the floor of the thorax and separates the thoracic and abdominal cavities (Fig. 1.4). The diaphragm is divided into right and left hemidiaphragms. Both hemidiaphragms are visible on radiographic studies from the front or back. The right hemidiaphragm is protected by the liver and is stronger than the left. The left hemidiaphragm is more often subject to rupture and hernia, usually because of weaknesses at the points of embryologic fusion. Each hemidiaphragm is composed of three musculoskeletal components, including the sternal, costal, and lumbar portions that converge into the central tendon. The central tendon of the diaphragm is a thin but strong layer of tendons (aponeurosis) situated anteriorly and immediately below the pericardium. There are three major openings to enable various vessels to traverse the diaphragm. These include the vena caval opening for the inferior vena cava; the esophageal opening for the esophagus and gastric vessels; and the aortic opening containing the aorta, thoracic duct, and azygos veins. The phrenic nerve arises from the third, fourth, and fifth cervical spinal nerves (C3–C5) and is involved in contraction of the diaphragm.

The resting position of the diaphragm is an arched position high in the thorax. The level of the diaphragm and the amount of movement during inspiration vary as a result of factors such as body position, obesity, and size of various gastrointestinal organs present below the diaphragm. During normal ventilation or breathing, the diaphragm contracts to pull the central tendon down and forward. In doing so, the resting dome shape of the diaphragm is reversed to a flattening of the diaphragm. Contraction of this muscle increases the dimensions of the thorax in a cephalocaudal, anterior posterior, and lateral direction.[1] The increase in volume decreases pressure

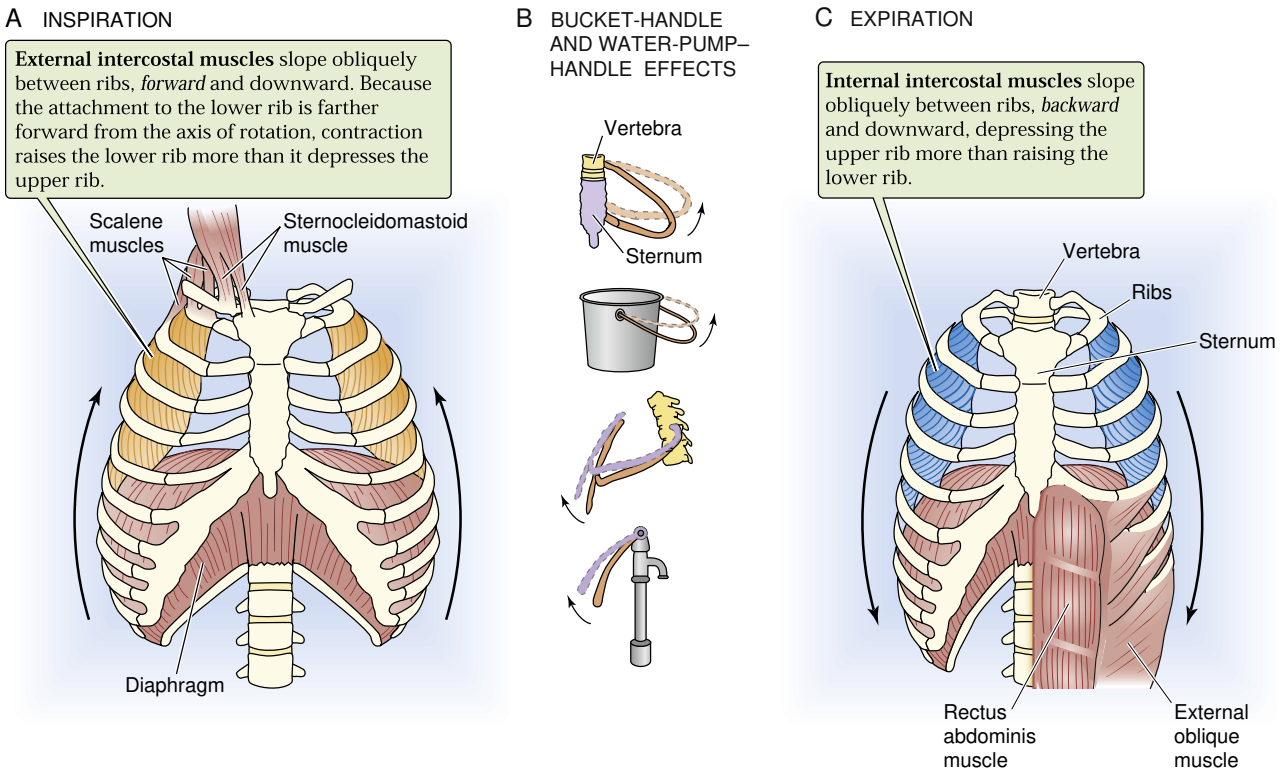

Figure 1.3 **(A–C)** Actions of major respiratory muscles. (From Boron WF: *Medical physiology*, updated ed, St. Louis, 2005, Saunders.)

in the thoracic cavity and simultaneously causes a decrease in volume and an increase in pressure within the abdominal cavity. The domed shape of the diaphragm is largely maintained until the abdominal muscles end their extensibility, halting the downward displacement of the abdominal viscera, essentially forming a fixed platform beneath the central tendon. The central tendon then becomes a fixed point against which the muscular fibers of the diaphragm contract to elevate the lower ribs and thereby push the sternum and upper ribs forward. The right hemidiaphragm meets more resistance than the left during its descent, because the liver underlies the right hemidiaphragm and the stomach underlies the left; it is therefore more substantial than the left.

In patients with chronic obstructive pulmonary disease (COPD), there is compromised ability to expire. This results in a flattening of the diaphragm as a result of the presence of hyperinflated lungs.[1,5] It is essential for therapists to reverse hyperinflation and restore the normal resting arched position of the diaphragm using any exercise aimed at strengthening the diaphragm muscle. A flat and rigid diaphragm cannot be strengthened and will cause an automatic firing of the accessory muscles to trigger inspiration.

Body position in supine, upright, or side lying alters the resting position of the diaphragm, resulting in concomitant changes in lung volumes.[6] In the supine position, without the effects of gravity, the level of the diaphragm in the thoracic cavity rises. This allows for a relatively greater excursion of the diaphragm. Despite a greater range of movement of the diaphragm, lung volumes are low as a consequence of the elevated position of the abdominal organs within the thoracic cavity. In an upright position, the dome of the diaphragm is pulled down because of the effects of gravity. The respiratory excursion is less in this position; however, the lung volumes are larger. In the side-lying position, the hemidiaphragms are unequal in their positions: the uppermost side drops to a lower level and has less excursion than that in the sitting position; the lowermost side rises higher in the thorax and has a greater excursion than in the sitting position. In quiet breathing, the diaphragm normally moves about two-thirds of an inch; with maximal ventilatory effort, the diaphragm may move from 2.5 to 4 inches.[5]

Clinical tip

Stomach fullness, obesity with presence of a large pannus, ascites with increased fluid in the peritoneal space from liver disease, and pregnancy are additional factors affecting the normal excursion of the diaphragm during inspiration.

External intercostal muscles

The external intercostal muscles originate from the lower borders of the ribs and attach to the upper border of the ribs below (Fig. 1.5). There are 11 external intercostal muscles on each side of the sternum. Contraction of these muscles pull the lower rib up and out toward the upper rib, thereby elevating the ribs and expanding the chest.

Accessory muscles

Figs. 1.6 and 1.7 present the anatomy of the accessory muscles.

Sternocleidomastoid muscle

The sternocleidomastoid arises by two heads (sternal and clavicular from the medial part of the clavicle), which unite to extend obliquely upward and laterally across the neck to the mastoid

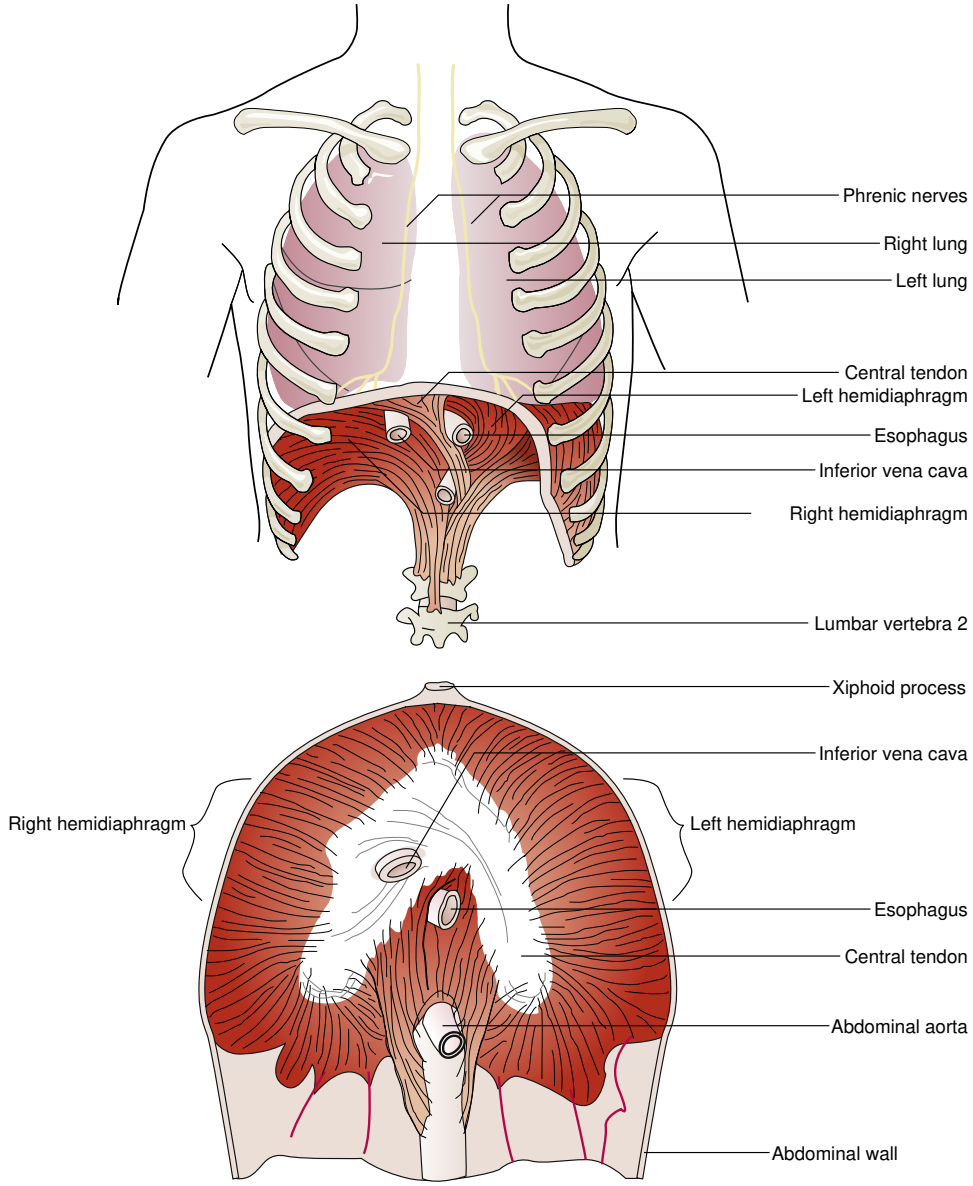

Figure 1.4 The diaphragm originates from the lumbar vertebra, lower ribs, xiphoid process, and abdominal wall and converges in a central tendon. Note the locations of the phrenic nerves and openings for the inferior vena cava, esophagus, and abdominal aorta. (From Hicks GH: *Cardiopulmonary anatomy and physiology*, Philadelphia, 2000, Saunders.)

process. For this muscle to facilitate inspiration, the head and neck must be held stable by the neck flexors and extensors. This muscle is a primary accessory muscle and elevates the sternum, increasing the anteroposterior diameter of the chest.

Scalene muscle

The scalene muscles lie deep to the sternocleidomastoid, but may be palpated in the posterior triangle of the neck. These muscles function as a unit to elevate and fix the first and second ribs:

1. The anterior scalene muscle passes from the anterior tubercles of the transverse processes of the third or fourth to the sixth cervical vertebrae, attaching by tendinous insertion into the first rib.
2. The middle scalene muscle arises from the transverse processes of all the cervical vertebrae to insert onto the first rib (posteromedially to the anterior scalene, the brachial plexus, and subclavian artery pass between the anterior scalene and middle scalene).
3. The posterior scalene muscle arises from the posterior tubercles of the transverse processes of the fifth and sixth cervical vertebrae, passing between the middle scalene and levator scapulae, to attach onto the second or third rib.

Upper trapezius

The trapezius (upper fibers) muscle arises from the medial part of the superior nuchal line on the occiput and the ligamentum nuchae (from the vertebral spinous processes between the skull and the seventh cervical vertebra) to insert onto the distal third of the clavicle. This muscle assists with ventilation by helping to elevate the thoracic cage.

Pectoralis major and minor

The pectoralis major arises from the medial third of the clavicle, from the lateral part of the anterior surface of the

manubrium and body of the sternum, and from the costal cartilages of the first six ribs to insert upon the lateral lip of the crest of the greater tubercle of the humerus. When the arms and shoulders are fixed, by leaning on the elbows or grasping onto a table, the pectoralis major can use its insertion as its origin and pull on the anterior chest wall, lifting the ribs and sternum, and facilitate an increase in the anteroposterior diameter of the thorax.

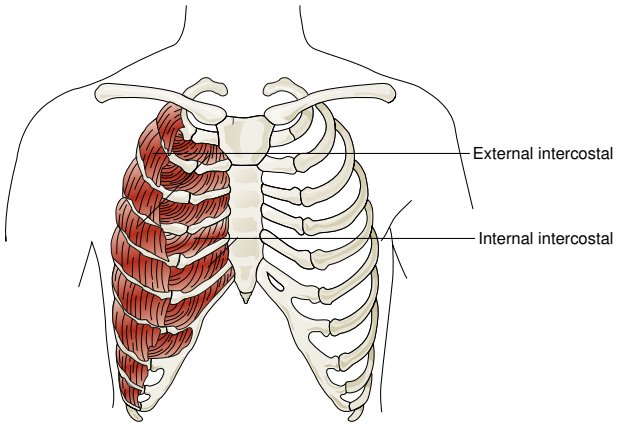

Figure 1.5 The external intercostal muscles lift the inferior ribs and enlarge the thoracic cavity. The internal intercostal muscles compress the thoracic cavity by pulling together the ribs. (From Hicks GH: *Cardiopulmonary anatomy and physiology*, Philadelphia, 2000, Saunders.)

The pectoralis minor arises from the second to fifth or the third to sixth ribs upward to insert into the medial side of the coracoid process close to the tip. This muscle assists in forced inspiration by raising the ribs and increasing intrathoracic volume.

Serratus anterior and rhomboids

The serratus anterior arises from the outer surfaces of the upper eight or nine ribs to attach along the costal aspect of the medial border of the scapula. The primary action of the serratus is to abduct, rotate the scapula, and hold the medial border firmly over the rib cage. The serratus can only be used as an accessory muscle in ventilation, when the rhomboids stabilize the scapula in adduction.[7] The action of the rhomboids fixes the insertion, allowing the serratus to expand the rib cage by pulling the origin toward the insertion.

Latissimus dorsi

The latissimus dorsi arises from the spinous processes of the lower six thoracic, the lumbar, and the upper sacral vertebrae, from the posterior aspect of the iliac crest, and slips from the lower three or four ribs to attach to the intertubercular groove of the humerus.[7] The posterior fibers of this muscle assist in inspiration as they pull the trunk into extension.

Serratus posterior superior

The serratus posterior superior passes from the lower part of the ligamentum nuchae and the spinous processes of

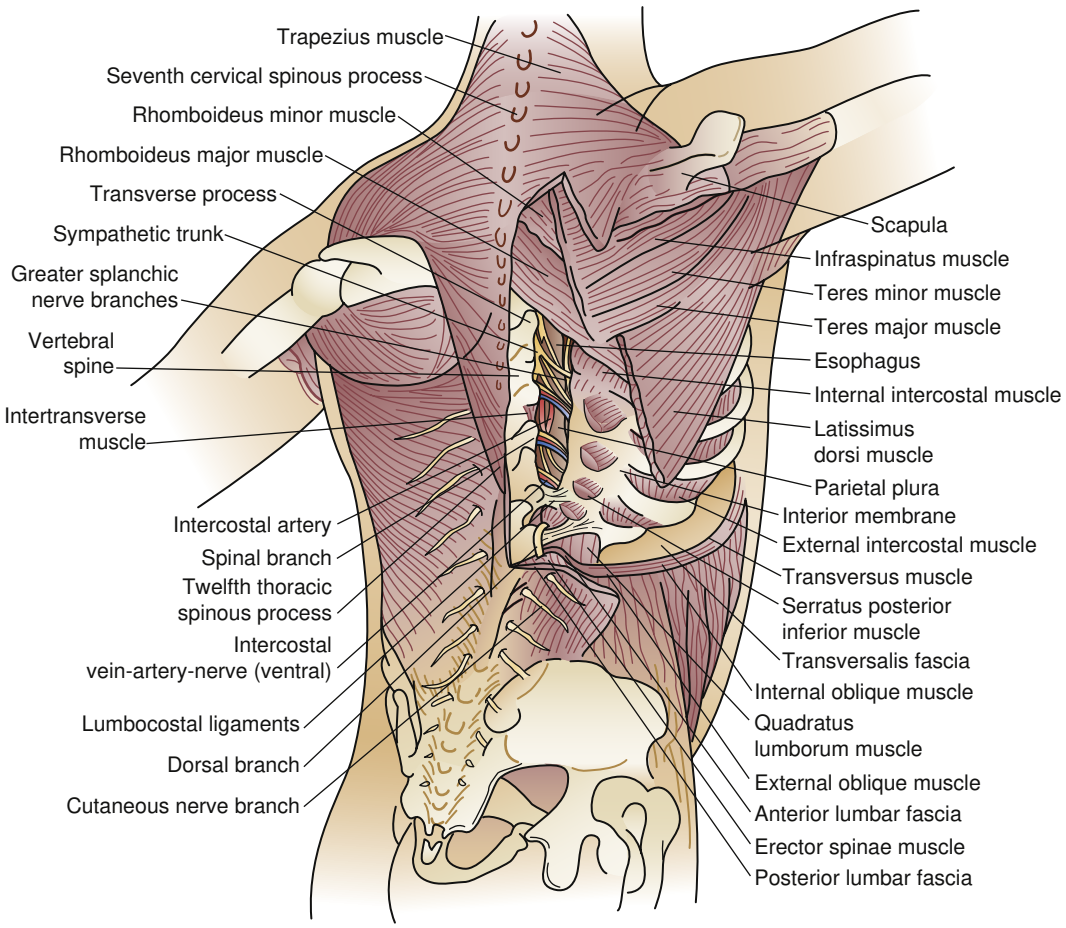

Figure 1.6 Musculature of the chest wall. (From Ravitch MM, Steichen FM: *Atlas of general thoracic surgery*, Philadelphia, 1988, Saunders.)

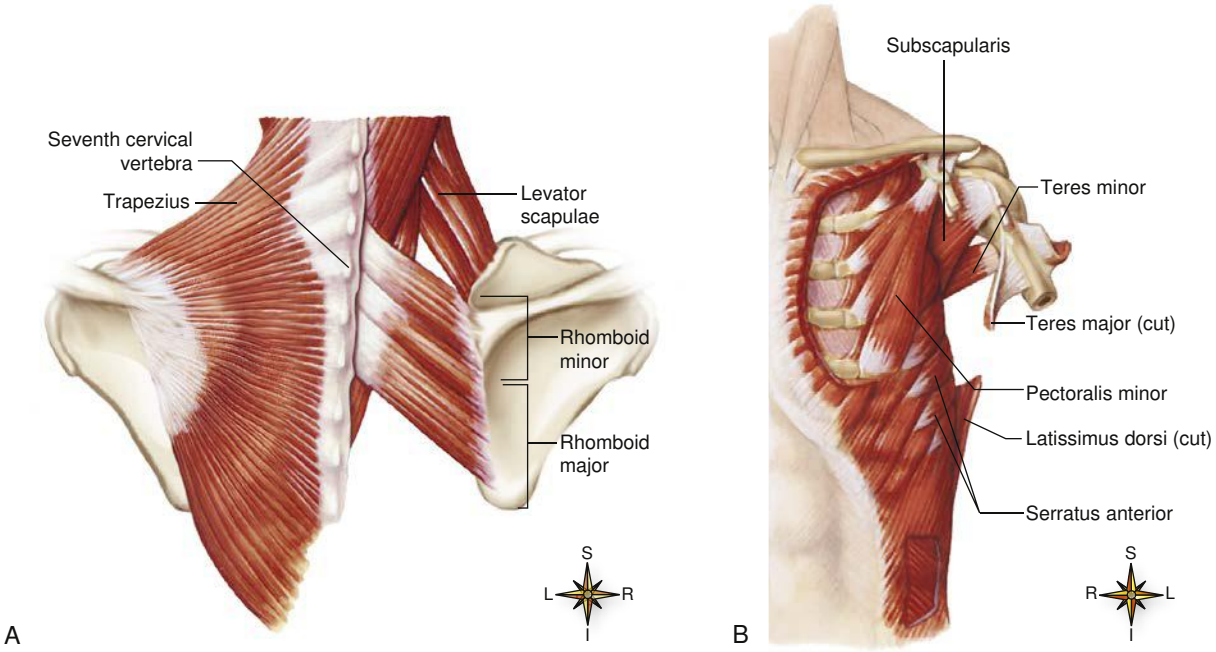

Figure 1.7 Musculature of anterior chest wall. (From Patton K, Thibodeau G, Douglas M: *Essentials of anatomy and physiology*, ed 1, St. Louis, 2011, Elsevier.)

the seventh cervical and first two or three thoracic vertebrae downward into the upper borders of the second to fourth or fifth ribs. This muscle assists in inspiration by raising the ribs to which it is attached and expanding the chest.

Thoracic erector spinae muscles

The erector spinae is a large muscle group extending from the sacrum to the skull. The thoracic erector spinae muscles extend the thoracic spine and raise the rib cage to allow greater expansion of the thorax.

Muscles of expiration

Abdominal muscles

The abdominal muscles include the rectus abdominis, transversus abdominis, and internal and external obliques. These muscles work to raise intraabdominal pressure when a sudden expulsion of air is required in maneuvers, such as huffing and coughing. Pressure generated within the abdominal cavity is transmitted to the thoracic cage to assist in emptying the lungs.

Internal intercostal muscles

Eleven internal intercostal muscles exist on each side of the sternum. These muscles arise on the inner surfaces of the ribs and costal cartilages and insert on the upper borders of the adjacent ribs below (see Fig. 1.5). The posterior aspect on the internal intercostal muscles is termed the interosseus portion and depresses the ribs to aid in a forceful expiration. The intercartilaginous portion of the internal intercostals elevates the ribs and assists in inspiration.

Pulmonary ventilation

Pulmonary ventilation, commonly referred to as breathing, is the process in which air is moved in and out of the lungs. Inspiration, an active process at rest and during exercise, involves contraction of the diaphragm and external intercostal muscles. The muscle that contracts first is the diaphragm, with a caudal movement and resultant increase within the volume of the thoracic cavity. The diaphragm eventually meets resistance against the abdominal viscera, causing the costal fibers of the diaphragm to contract and pull the lower ribs up and out—the bucket-handle movement. The outward movement is also facilitated by the external intercostal muscles. In addition, a pump-handle movement of the upper ribs is achieved through contraction of the external intercostals and the intercartilaginous portion of the internal intercostal muscles. The actions of the inspiratory muscles expand the dimensions of the thoracic cavity and concomitantly reduce the pressure in the lungs (intrathoracic pressure) below the air pressure outside the body. With the respiratory tract being open to the atmosphere, air rushes into the lungs to normalize the pressure difference, allowing inspiration to occur and the lungs to fill with air.

During forced or labored breathing, additional accessory muscles need to be used to increase the inspiratory maneuver. The accessory muscles raise the ribs to a greater extent and promote extension of the thoracic spine. These changes facilitate a further increase in the volume within the thoracic cavity and a subsequent drop in the intrathoracic pressure beyond that caused by the contraction of the diaphragm and external intercostals. This relatively lower intrathoracic pressure will promote a larger volume of air entering the lung.

At rest, expiration is a passive process and achieved through the elastic recoil of the lung and relaxation of the

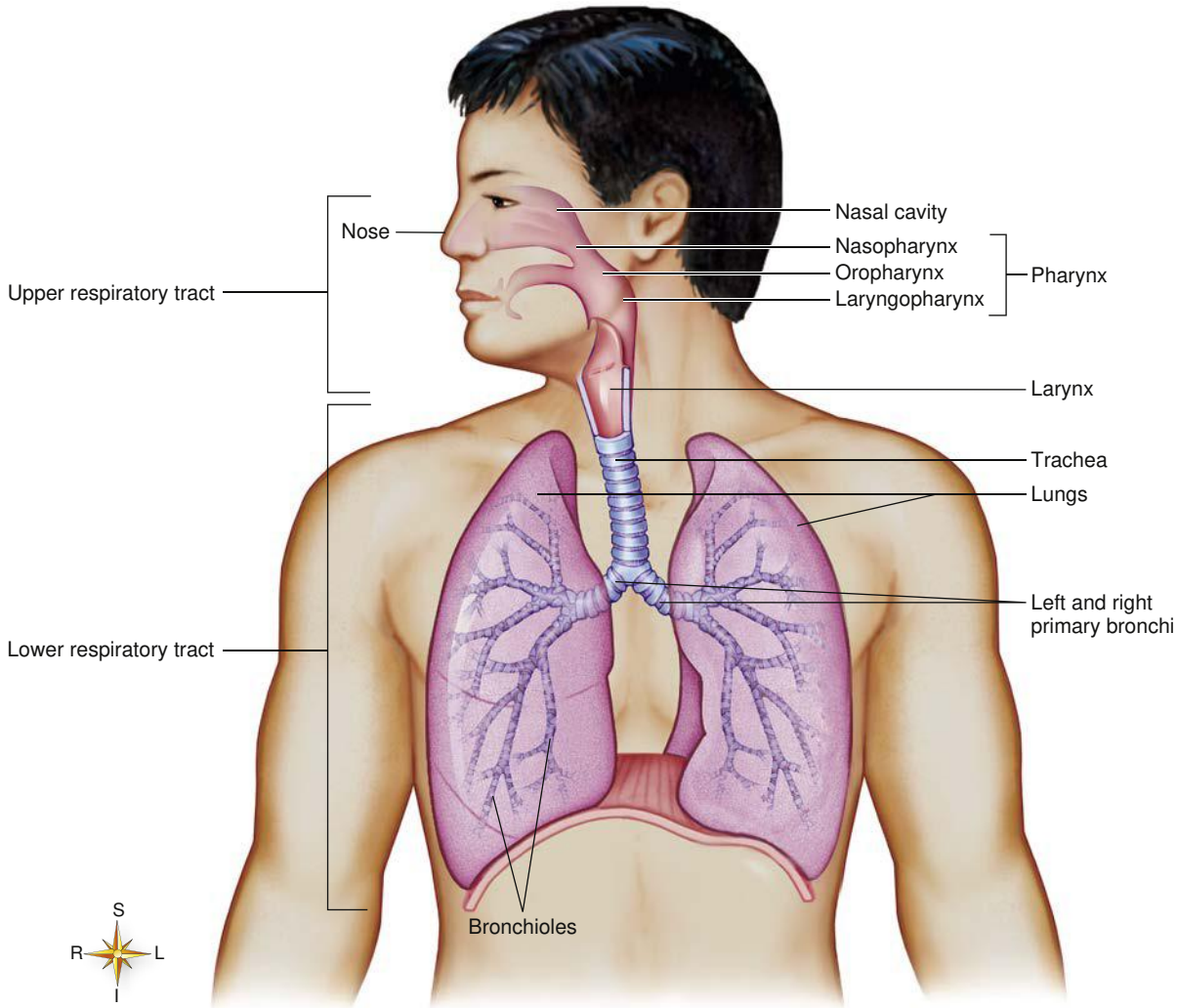

Figure 1.8 Pleurae of the lungs. (From Patton K, Thibodeau G, Douglas M: *Essentials of anatomy and physiology*, ed 1, St. Louis, 2011, Elsevier.)

external intercostal and diaphragm muscle. As the external intercostals relax, the rib drops to its preinspiratory position and the diaphragm returns to its elevated dome position high in the thorax. To achieve a forceful expiration, additional muscles can be used, including the abdominals and internal intercostal muscles. The internal intercostals actively pull the ribs down to help expel air out of the lungs. The abdominals contract to force the viscera upward against the diaphragm, accelerating its return to the dome position.

Clinical tip

The changes in intraabdominal and intrathoracic pressure that occur with forced breathing assist with venous return of blood back to the heart. The drop in pressure allows for a filling of the veins, and the changing pressure within the abdomen and thorax cause a milking effect to help return blood back to the heart.

Pleurae

Two serous membranes, or pleurae, exist that cover each lung (Fig. 1.8). The pleura covering the outer surface of each lung is the visceral pleura and is inseparable from the tissue of the lung. The pleura covering the inner surface of the chest wall, diaphragm, and mediastinum is called the parietal pleura. The parietal pleura is frequently described with reference to the anatomic surfaces it covers: the portion lining the ribs and vertebrae is named the costovertebral pleura; the portion over the diaphragm is the diaphragmatic pleura; the portion covering the uppermost aspect of the lung in the neck is the cervical pleura; and that overlying the mediastinum is called the mediastinal pleura.[8] Parietal and visceral pleurae blend with one another where they come together to enclose the root of the lung. Normally, the pleurae are in intimate contact during all phases of the ventilatory cycle, being separated only by a thin serous film. There exists a potential space between the pleurae called the pleural space or pleural cavity. A constant negative pressure within this space maintains lung inflation. The serous fluid within the pleural space serves to hold the pleural layers together during ventilation and reduce friction between the lungs and the thoracic wall.[6,8]

The parietal pleura receives its vascular supply from the intercostal, internal thoracic, and musculophrenic arteries. Venous drainage is accomplished by way of the systemic veins in the adjacent parts of the chest wall. The bronchial vessels supply the visceral pleura. There exists no innervation

to the visceral pleura and therefore no sensation.[5] The phrenic nerve innervates the parietal pleura of the mediastinum and central diaphragm, whereas the intercostal nerves innervate the parietal pleura of the costal region and peripheral diaphragm.

Irritation of the intercostally innervated pleura may result in the referral of pain to the thoracic or abdominal walls, and irritation of the phrenic-supplied pleura can result in referred pain in the lower neck and shoulder.[9]

Several complications can affect pleural integrity. Infection with resultant inflammatory response within the pleura is termed pleuritis or pleurisy and is best appreciated through the presence of pleural chest pain and an abnormal pleural friction rub on auscultation.[9] A pleural effusion refers to a buildup of fluid in the pleural space commonly seen after cardiothoracic surgery or with cancer. This is evidenced by diminished or absent breath sounds in the area of the effusion, is more likely to be in gravity-dependent areas, and is accompanied by reduced lung volumes. Blood in the pleural space is termed a hemothorax, whereas air in the pleural space from a collapsed lung is termed a pneumothorax. Finally, a bacterial infection with resultant pus in the pleural space is referred to as empyema.

Management for several of these complications of the pleural space is achieved through insertion of a chest tube into the pleural space to drain pleural secretions or to restore a negative pressure within the space and allow for lung inflation. A needle aspiration of fluid from the space, a thoracocentesis, may be performed for patients with large pleural effusions.

Lungs

The lungs are located on either side of the thoracic cavity, separated by the mediastinum. Each lung lies freely within its corresponding pleural cavity, except where it is attached to the heart and trachea by the root and pulmonary ligament. The substance of the lung (the parenchyma) is normally porous and spongy in nature. The surfaces of the lungs are marked by numerous intersecting lines that indicate the polyhedral (secondary) lobules of the lung. The lungs are basically cone shaped and are described as having an apex, a base, three borders (anterior, inferior, and posterior), and three surfaces (costal, medial, and diaphragmatic).

The apex of each lung is situated in the root of the neck, its highest point being approximately 1 inch above the middle third of each clavicle. The base of each lung is concave, resting on the convex surface of the diaphragm. The inferior border of the lung separates the base of the lung from its costal surface; the posterior border separates the costal surface from the vertebral aspect of the mediastinal surface; the anterior border of each lung is thin and overlaps the front of the pericardium. In addition, the anterior border of the left lung presents a cardiac notch. The costal surface of each lung conforms to the shape of the overlying chest wall. The medial surface of each lung may be divided into vertebral and mediastinal aspects. The vertebral aspect contacts the respective sides of the thoracic vertebrae and their intervertebral disks, the posterior intercostal vessels, and nerves. The mediastinal aspect is notable for the cardiac impression; this concavity is larger on the left than on the right lung to accommodate the projection of the apex of the heart toward the left. Just posterior to the cardiac impression is the hilus, where the structures forming the root of the lung enter and exit the parenchyma. The extension of the pleural covering below and behind the hilus from the root of the lung forms the pulmonary ligament.

Hila and roots

The point at which the nerves, vessels, and primary bronchi penetrate the parenchyma of each lung is called the hilus (Fig. 1.9A and B). The structures entering the hila of the lungs and forming the roots of each of the lungs are the principal bronchus, the pulmonary artery, the pulmonary veins, the bronchial arteries and veins, the pulmonary nerve plexus, and the lymph vessels. They lie next to the vertebral bodies of the fifth, sixth, and seventh thoracic vertebrae. The right root lies behind the superior vena cava and a portion of the right atrium, below the end of the azygos vein; the left root lies below the arch of the aorta and in front of the descending thoracic aorta. The pulmonary ligament lies below the root; the phrenic nerve and the anterior pulmonary plexus lie in front of the root; the vagus nerve and posterior pulmonary plexus lie behind the root.

Lobes, fissures, and segments

The right lung consists of three lobes, including the right upper lobe (RUL), right middle lobe (RML), and right lower lobe (RLL). Two fissures separate these three lobes from one another. The upper and middle lobes of the right lung are separated from the lower lobe by the oblique (major) fissure (Fig. 1.10). Starting on the medial surface of the right lung at the upper posterior aspect of the hilus, the oblique fissure runs upward and backward to the posterior border at about the level of the fourth thoracic vertebra; it then descends anteroinferiorly across the anterior costal surface to intersect the lower border of the lung approximately 5 inches from the median plane and then passes posterosuperiorly to rejoin the hilus just behind and beneath the upper pulmonary vein. The RML is separated from the RUL by the horizontal (minor) fissure that joins the oblique fissure at the midaxillary line at about the level of the fourth rib and runs horizontally across the costal surface of the lung to about the level of the fourth costal cartilage; on the medial surface, it passes backward to join the hilus near the upper-right pulmonary vein.

Each lobe of the right lung is further subdivided into segments. The RUL has three segments, including the apical, posterior, and anterior segments. This lobe extends to the level of the fourth rib anteriorly and is adjacent to ribs 3 to 5 posteriorly. The RML is subdivided into the lateral and medial lobes. This lobe is the smallest of the three lobes. Its inferior border is adjacent to the fifth rib laterally and the sixth rib medially. The lowermost lobe, the RLL, consists of four segments (anterior basal, superior basal, lateral basal, and posterior basal). The superior border of the RLL is at the level of the sixth thoracic vertebra and extends inferiorly down to the diaphragm. During maximal inspiration, the inferior border of the RLL may extend to the second lumbar vertebra and superimpose over the superior aspects of the kidney.

The left lung is relatively smaller than the right lung and has only two lobes, including the left upper lobe (LUL) and left lower lobe (LLL). The left lung is divided into upper and lower lobes by the oblique fissure, which is somewhat more vertically oriented than that of the right lung; there is no horizontal fissure. The portion of the left lung that corresponds to the right lung is termed the lingular segment and is a part of

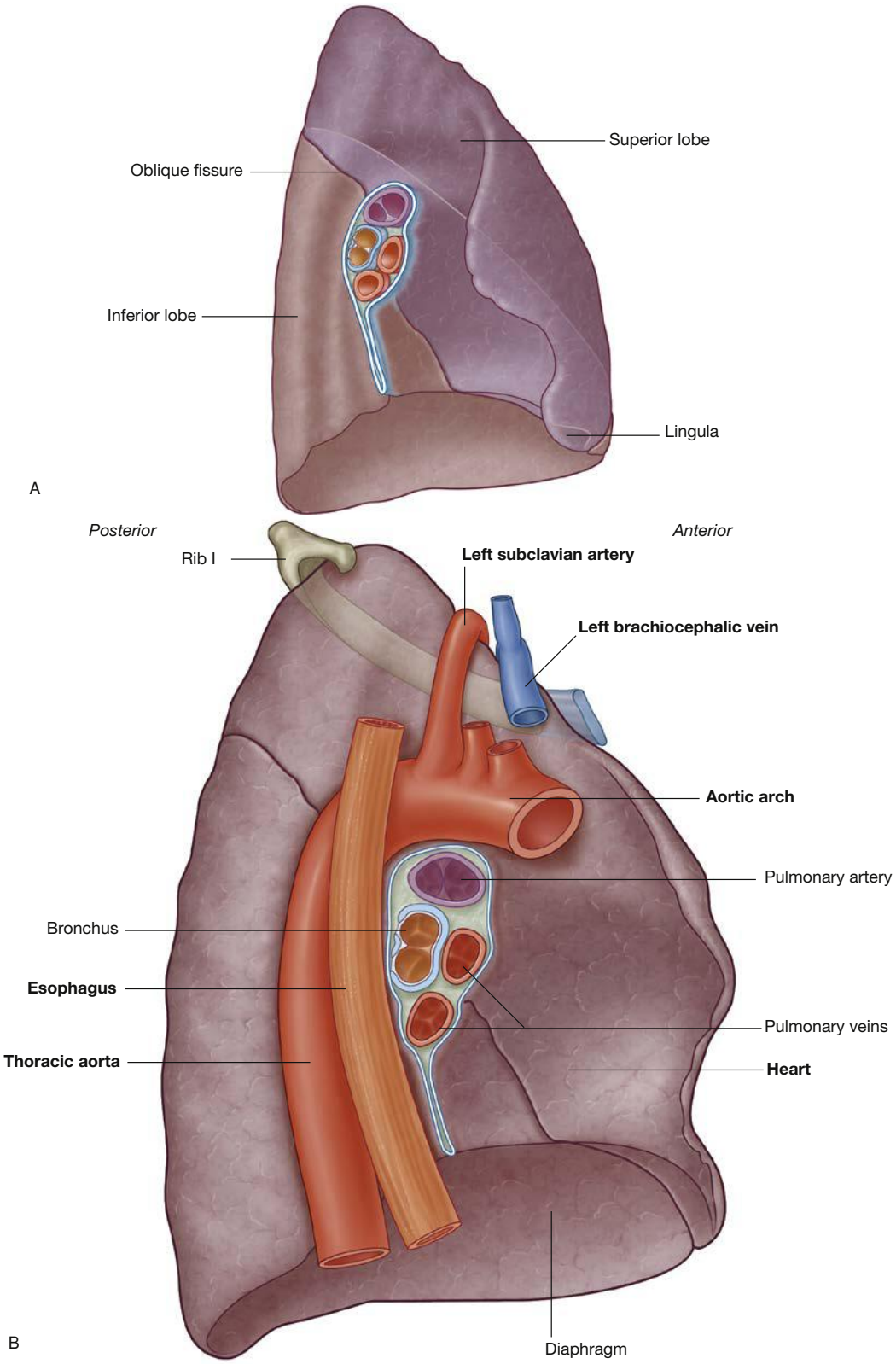

Figure 1.9 (A and B) Hilum area of lungs. (**A,** From Drake RL, Vogl AW, Mitchell A, *Gray's anatomy for students*, ed 4, Philadelphia, 2020, Elsevier; **B,** From Savvas A, Vanhoenacker FM, DeBacker AI. Advances in imaging chest tuberculosis: blurring of differences between children and adults, *Clin Chest Med* 30[4]:717–744, 2009.)

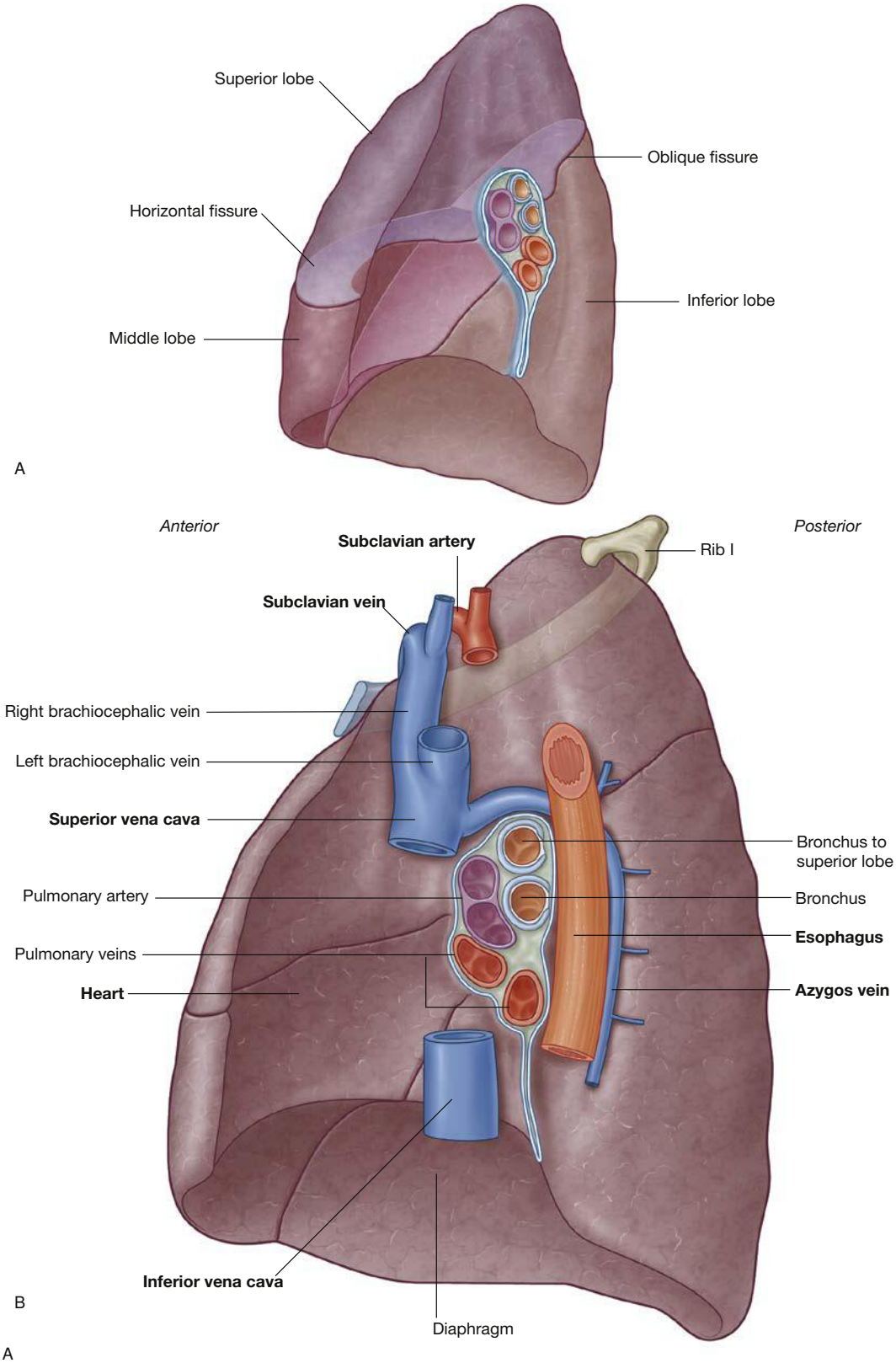

Figure 1.9, cont'd

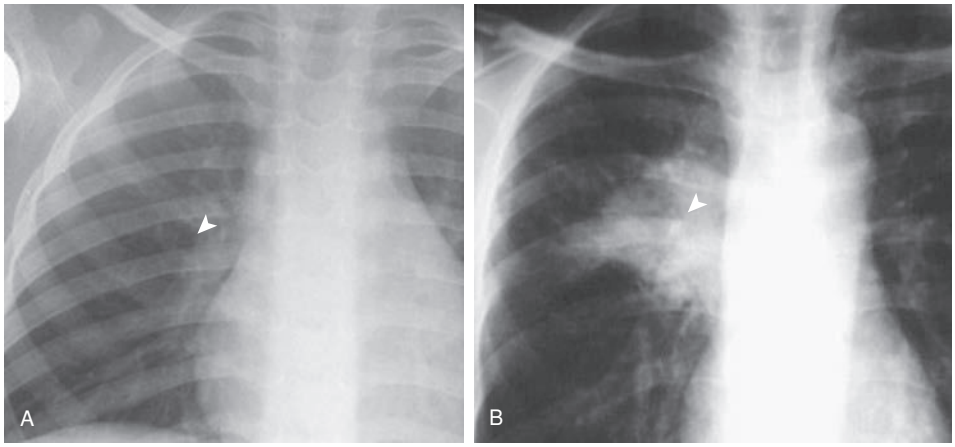

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Figure 1.9, cont'd

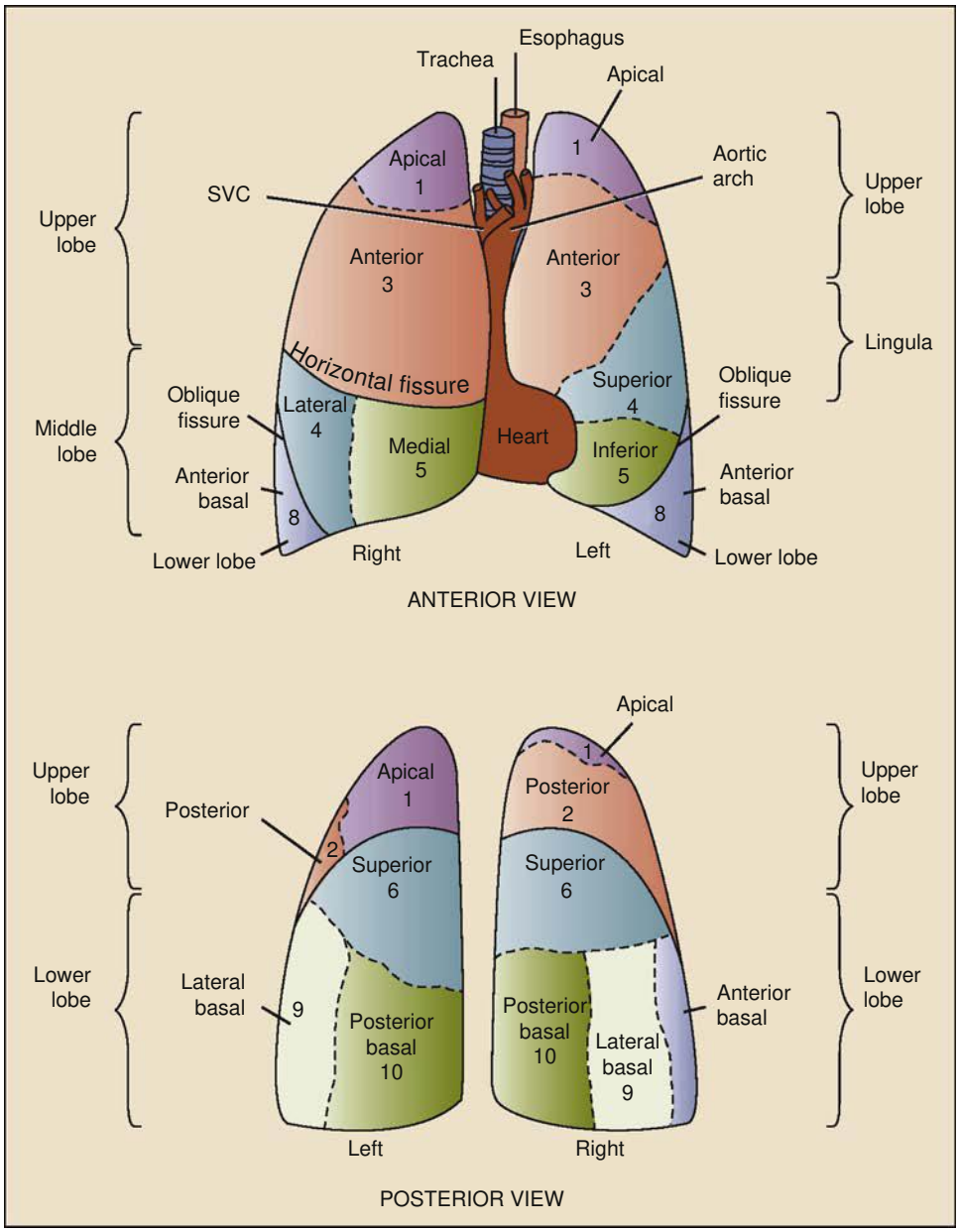


Figure 1.10 Topography of the lung demonstrating the lobes, segments, and fissures. The fissures (or chasms) demarcate the lobes in each lung. Numbers refer to specific bronchopulmonary segments. *SVC*, Superior vena cava. (From Koeppen B, Stanton B: *Berne and Levy physiology,* ed 6, Philadelphia, 2000, Mosby.)

Table 1.1 Topographic Boundaries for the Bronchopulmonary Lung Segments

Lobe	Segment	Borders
Upper lobe	Anterior segment (right or left)	Upper border: clavicle Lower border: a horizontal line at the level of the third intercostal space (ICS), or fourth rib, anteriorly
	Apical segment (R) or apical aspect, apicoposterior segment (L)	Anteroinferior border: clavicle Posteroinferior border: a horizontal line at the level of the upper lateral border of the spine of the scapula
	Posterior segment (R) or posterior aspect, apicoposterior segment (L)	Upper border: a horizontal line at the level of the upper lateral border of the spine of the scapula Lower border: a horizontal line at, or approximately 1 inch below, the inferomedial aspect of the spine of the scapula
Middle lobe (R) or lingula (L)		Upper border: a horizontal line at the level of the third ICS, or fourth rib, anteriorly Lower and lateral borders: the oblique fissure (a horizontal line at the level of the sixth rib anteriorly) extending to the anterior axillary line; from the anterior axillary line, angling upward to approximately the fourth rib at the posterior axillary line The midclavicular line separates the medial and lateral segments of the right middle lobe A horizontal line at the level of the fifth rib, anteriorly, separates the superior and inferior lingular segments
Lower lobe	Superior (basal) segment (right or left)	Upper border: a horizontal line at, or approximately 1 inch below, the inferomedial aspect of the spine of the scapula Lower border: a horizontal line at, or approximately 1 inch above, the inferior angle of the scapula
	Posterior (basal) segment (right or left)	Upper border: a horizontal line at, or approximately 1 inch above, the inferior angle of the scapula Lateral border: a "plumb line" bisecting the inferior angle of the scapula Lower border: a horizontal line at the level of the 10th ICS, posteriorly
	Lateral (basal) segment (right or left)	Upper border: a horizontal line at, or approximately 1 inch above, the inferior angle of the scapula Medial border: a "plumb line" bisecting the inferior angle of the scapula Lateral border: the midaxillary line Lower border: a horizontal line at the level of the 10th ICS, posteriorly
	Anterior (basal) segment (R) or anterior aspect, anteromedial (basal) segment (L)	Upper border: the oblique fissure (a horizontal line at the level of the sixth rib anteriorly, extending to the anterior axillary line; from the anterior axillary line, angling upward to approximately the fifth rib at the midaxillary line Lateral border: the midaxillary line

the LUL. Posteriorly, the inferior border of the LUL is at the level of the 6th rib, and the LLL is at the level of the 11th rib.

Table 1.1 describes the topographic boundaries for the bronchopulmonary segments of each lung. Fig. 1.11 shows the bronchopulmonary segments and Fig. 1.12 shows the bronchopulmonary segment anatomy.

Clinical tip

An understanding of the various lobes and segments and their anatomic orientation is essential for appropriate positioning and removal of secretions from various aspects of the lung during bronchopulmonary hygiene procedures.

Upper respiratory tract

Nose

The nose is a conglomerate of bone and hyaline cartilage. The nasal bones (right and left), the frontal processes of the maxillae, and the nasal part of the frontal bone combine to form the bony framework of the nose. The septal, lateral, and major and minor alar cartilages combine to form the cartilaginous framework of the nose. The periosteal and perichondral membranes blend to connect the bones and cartilages to one another.

Three major muscles assist with movement of the bony framework of the nose. The procerus muscle wrinkles the skin of the nose. The nasalis muscle has two parts, including the transverse and alar portions, and assists in flaring the anterior nasal aperture.[8] Finally, the depressor septi muscle works with the nasalis muscle to flare the nostrils.[8] Skin covers the external nose.

The nasal cavity is a wedge-shaped passageway divided vertically into right and left halves by the nasal septum and compartmentalized by the paranasal sinuses (Fig. 1.13). Opening anteriorly via the nares (nostrils) to the external environment, the nasal cavity blends posteriorly with the nasopharynx. The two halves are essentially identical, having a floor, medial and lateral walls, and a roof divided into three regions: the vestibule, the olfactory region, and the respiratory region.

The primary respiratory functions of the nasal cavity include air conduction, filtration, humidification, and

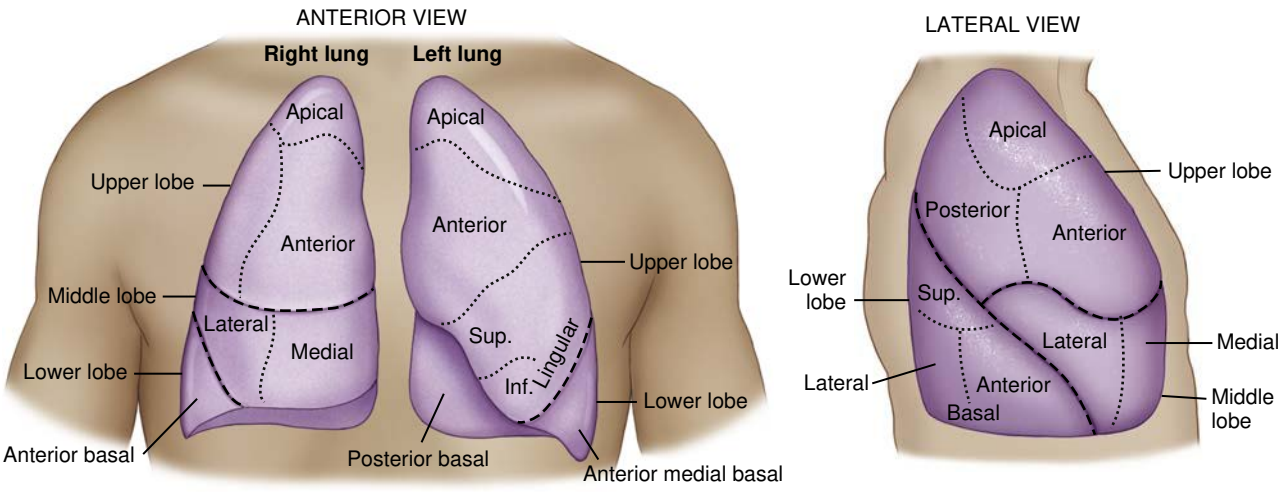
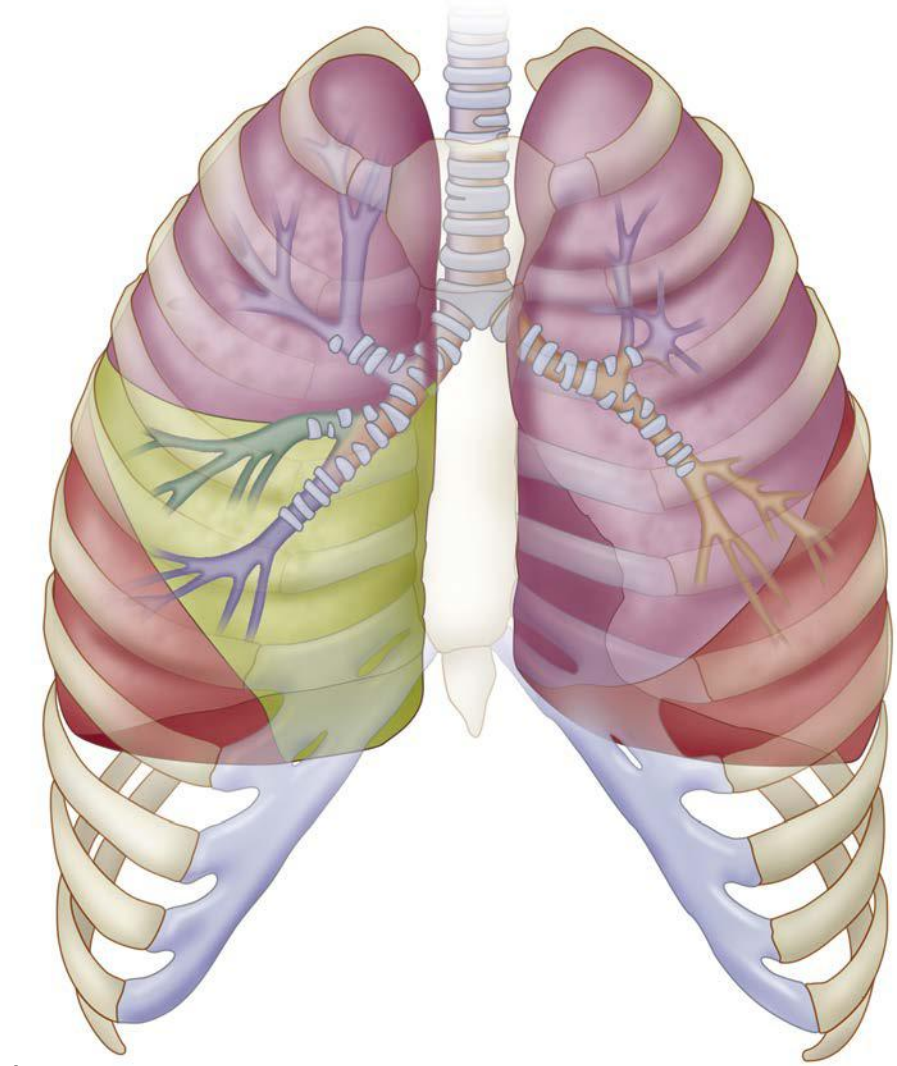


Figure 1.11 Anterior and lateral views of the bronchopulmonary segments as seen projected to the surface of the lungs.



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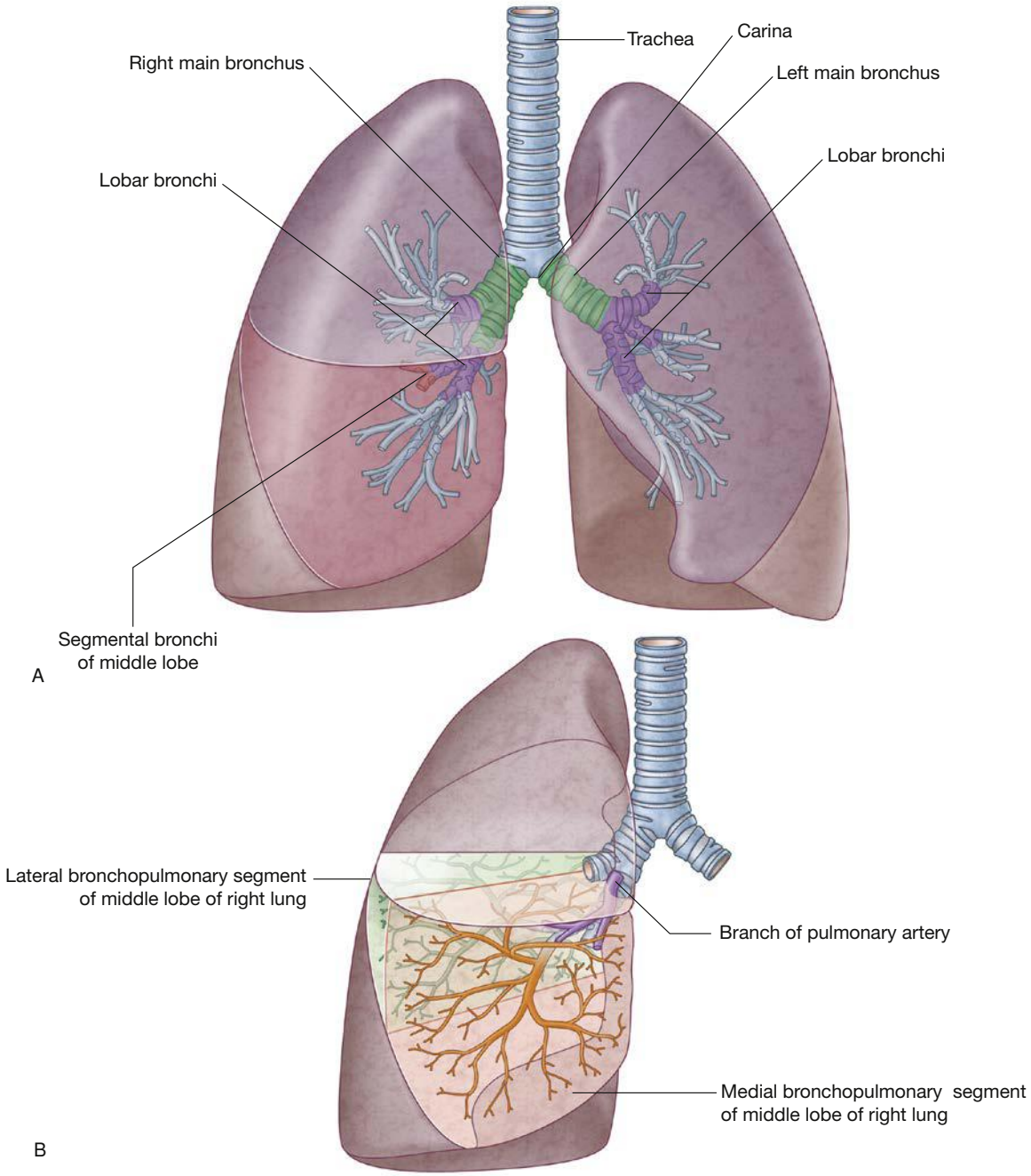

Figure 1.12 (A) Structure of the airways. (B) Bronchopulmonary segmental anatomy. (A, From Patton K, Thibodeau G, Douglas M: *Essentials of Anatomy and Physiology*, ed 1, St. Louis, 2011, Elsevier. (B), From Drake RL, Vogl AW, Mitchell A, *Gray's Anatomy for Students*, ed 4, Philadelphia, 2020, Elsevier.)

temperature control; it also plays a role in the olfactory process. Three nasal conchae project into the nasal cavity from the lateral wall toward the medial wall; they are named the superior, middle, and inferior conchae. The conchae serve to increase the respiratory surface area of the nasal mucous membrane for greater contact with inspired air. The vestibule of the nasal cavity is lined with skin containing many coarse hairs and sebaceous and sweat glands. Mucous membrane lines the remainder of the nasal cavity. Fig. 1.14 depicts examples of some selected types of mucosal coverings in the upper and lower respiratory tracts.

The olfactory region of the nasal cavity is distinguished by specialized mucosa. This pseudostratified olfactory epithelium is composed of ciliated receptor cells, nonciliated sustentacular cells, and basal cells that help to provide a sense of smell.[8] Sniffing increases the volume of inspired air entering the olfactory region, allowing the individual to smell something specific.[4]

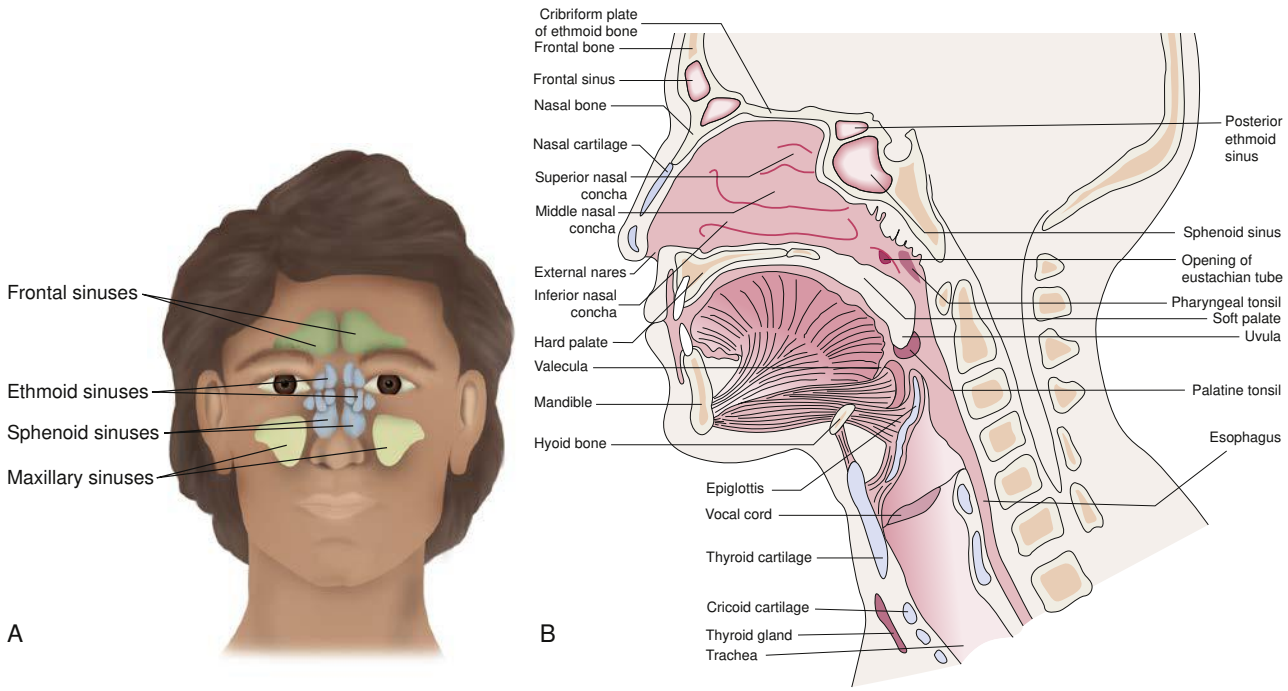

Figure 1.13 (A) Positions of the frontal, maxillary, sphenoid, and ethmoid sinuses; the nasal sinuses are named for the bones in which they occur. (B) Midsagittal section through the upper airway. (From Wilkins RE: *Fundamentals of respiratory care*, ed 9, St. Louis, 2009, Mosby.)

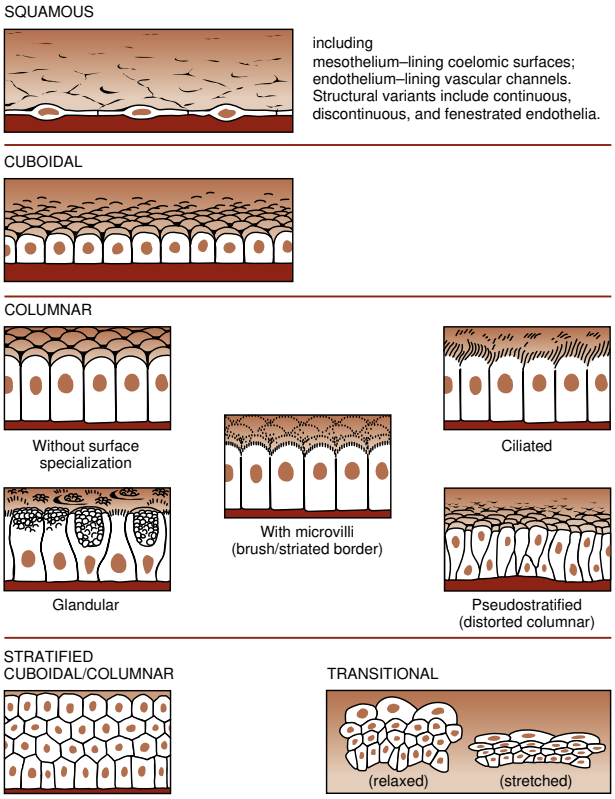

Figure 1.14 Types of cells composing the mucosal lining of the upper and lower respiratory tracts. (Modified from Williams PL, Warwick R, Dyson M, et al., editors: *Gray's anatomy*, ed 37, New York, 1989, Churchill Livingstone.)

The respiratory region is lined with a mixture of columnar or pseudostratified ciliated epithelial cells, goblet cells, nonciliated columnar cells with microvilli, and basal cells. Serous and mucous glands, which open to the surface via branched ducts, underlie the basal lamina of the respiratory epithelium.[10] The submucosal glands and goblet cells secrete an abundant quantity of mucus over the mucosa of the nasal cavity, making it moist and sticky. Turbulent airflow, created by the conchae, causes inhaled dust and other particulate matter larger than approximately 10 μm to "rain out" onto this sticky layer, which is then moved by ciliary action backward and downward out of the nasal cavity into the nasopharynx at an average rate of about 6 mm per minute.[11,12]

Clinical tip

Nasotracheal suctioning must be performed with caution in individuals with low platelet counts because of the likelihood of trauma and bleeding to superficial nasal conchae and cells within the nasal cavity. The placement of a nasopharyngeal airway or nasal trumpet may reduce trauma with recurrent blind suctioning procedures in these patients.

Individuals with seasonal allergies who are prone to developing sinus infections are also prone to developing bronchitis if the infection leaves the sinus cavities and drops down the throat to the bronchioles.

Pharynx

The pharynx is a musculomembranous tube approximately 5 to 6 inches long and located posterior to the nasal cavity. It extends from the base of the skull to the esophagus that corresponds with a line extending from the sixth cervical vertebra to the lower border of the cricoid cartilage. The pharynx consists of three parts: the nasopharynx, the oropharynx, and the laryngopharynx.

Nasopharynx. The nasopharynx is a continuation of the nasal cavity, beginning at the posterior nasal apertures and continuing backward and downward. Its roof and posterior wall are continuous; its lateral walls are formed by the openings of the eustachian tubes; and its floor is formed by the

soft palate anteriorly and the pharyngeal isthmus (the space between the free edge of the soft palate and the posterior wall of the pharynx), which marks the transition to the oropharynx. The epithelium of the nasopharynx is composed of ciliated columnar cells.

Oropharynx. The oropharynx extends from the soft palate and pharyngeal isthmus superiorly to the upper border of the epiglottis inferiorly. Anteriorly, it is bounded by the oropharyngeal isthmus (which opens into the mouth) and the pharyngeal part of the tongue. The posterior aspect of the oropharynx is at the level of the body of the second cervical vertebra and upper portion of the body of the third cervical vertebra. The epithelium in the oropharynx is composed of stratified squamous cells.

Laryngopharynx. The laryngopharynx extends from the upper border of the epiglottis to the inferior border of the cricoid cartilage and the esophagus. The laryngeal orifice and the posterior surfaces of the arytenoid and cricoid cartilages form the anterior aspect of the laryngopharynx. The posterior aspect is at the level of the lower portion of the third cervical vertebra, the bodies of the fourth and fifth cervical vertebrae, and the upper portion of the body of the sixth cervical vertebra. The epithelium in the laryngopharynx is composed of stratified squamous cells.

Larynx

The larynx, or voice box, is a complex structure made up of several cartilages and forms a connection between the pharynx and the trachea. The position of the larynx depends on the age and sex of the individual, being opposite the third to sixth cervical vertebrae in the adult male and somewhat higher in adult females and children.

The larynx consists of the endolarynx and its surrounding cartilaginous structures. The endolarynx is made of two sets of folds, including the false vocal cords (supraglottis) and true vocal cords.[8] Between the true cords are slit-shaped spaces that form the glottis. A space exists above the false vocal cords and is termed the vestibule. Six supporting cartilages, including three large (epiglottis, thyroid, cricoid) and three smaller (arytenoid, corniculate, cuneiform), prevent food, liquids, and foreign objects from entering the airway. Two sets of laryngeal muscles (internal and external) play important roles in swallowing, ventilation, and vocalization. The larynx controls airflow and closes to increase intrathoracic pressure to generate an effective cough. Sounds with speech are created as expired air vibrates over the contracting vocal cords.

Clinical tip

Endotracheal intubation may cause damage to structures within the larynx, producing an inflammatory response—laryngitis—where patients present with hoarseness and pain during speech.

Lower respiratory tract

The lower respiratory tract extends from the level of the true vocal cords in the larynx to the alveoli within the lungs. In general, the lower respiratory tract may be divided into two parts: the tracheobronchial tree, or conducting airways, and the acinar or terminal respiratory units.

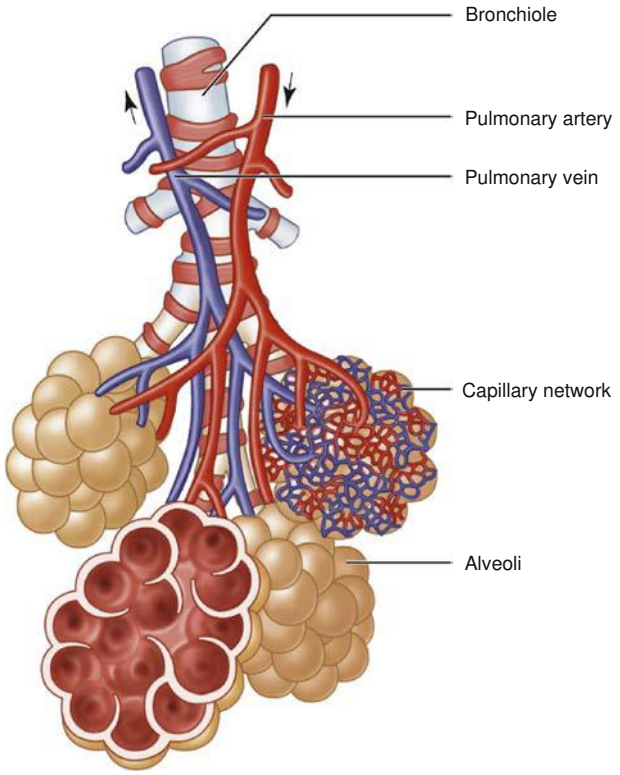

Figure 1.15 A view of the terminal respiratory unit showing the alveolar sac and blood supply surrounding. (From Malamed SF: *Sedation*, ed 4, St. Louis, 2010, Mosby.)

Tracheobronchial tree or conducting airways

The conducting airways are not directly involved in the exchange of gases in the lungs. They simply conduct air to and from the respiratory units. Airway diameter progressively decreases with each succeeding generation of branching, starting at approximately 1 inch in diameter at the trachea and reaching 1 mm or less at the terminal bronchioles. The cartilaginous rings of the larger airways give way to irregular cartilaginous plates, which become smaller and more widely spaced with each generation of branching, until they disappear at the bronchiolar level.[13] There may be as many as 16 generations of branching in the conducting airways from the mainstem bronchi to the terminal bronchioles (Fig. 1.15).[14]

Trachea

The trachea is a tube approximately 4 to 4.5 inches long and approximately 1 inch in diameter, extending downward along the midline of the neck, ventral to the esophagus. As it enters the thorax, it passes behind the left brachiocephalic vein and artery and the arch of the aorta. At its distal end, the trachea deviates slightly to the right of midline before bifurcating into right and left mainstem bronchi. Between 16 and 20 incomplete rings of two or more hyaline cartilages are often joined together along the anterior two-thirds of the tracheal circumference, forming a framework for the trachea. Fibrous and elastic tissues and smooth muscle fibers complete the ring posteriorly. The first and last tracheal cartilages differ somewhat from the others: the first is broader and is attached by the cricotracheal ligament to the lower border of the cricoid cartilage of the larynx. The last is thicker and broader at its

middle, where it projects a hook-shaped process downward and backward from its lower border—the carina—between the two mainstem bronchi. The carina is located at the fifth thoracic vertebra or sternal notch and represents the cartilaginous wedge at the bifurcation of the trachea into the right and left mainstem bronchi.

Clinical tip

During suctioning procedures, the catheter is inserted to the level of the carina. When the catheter is in contact with the carina, a cough ensues along with a strong parasympathetic response. Therapists must monitor for adverse responses in heart rate and provide supplemental oxygen as needed.

Mainstem and lobar bronchi

The right mainstem bronchus is wider and shorter than its left counterpart, and it diverges at approximately a 25-degree angle from the trachea. It passes laterally downward behind the superior vena cava for approximately 1 inch before giving off its first branch—the upper lobe bronchus—and entering the root of the right lung. Approximately 1 inch farther, it gives off its second branch—the middle lobe bronchus—from within the oblique fissure. Thereafter, the remnant of the mainstem bronchus continues as the lower lobe bronchus.

The left mainstem bronchus leaves the trachea at an angle of approximately 40 to 60 degrees and passes below the arch of the aorta and behind the left pulmonary artery, proceeding for a little more than 2 inches before it enters the root of the left lung, giving off the upper lobe bronchus and continuing on as the lower lobe bronchus. The left lung has no middle lobe, which is a major distinguishing feature in the general architecture of the lungs.

Clinical tip

The angulation of the right mainstem bronchus relative to the position of the trachea predisposes foreign objects, food, and fluids to enter the right lung. Consequently, aspiration is relatively more common in the right lung compared with the left lung.

Segmental and subsegmental bronchi

Each of the lobar bronchi gives off two or more segmental bronchi; an understanding of their anatomy is essential to the appropriate assessment and treatment of pulmonary disorders (see Fig. 1.12). The RUL bronchus divides into three segmental bronchi about a half inch from its own origin: the first—the apical segmental bronchus—passes superolaterally toward its distribution in the apex of the lung; the second—the posterior segmental bronchus—proceeds slightly upward and posterolaterally to its distribution in the posteroinferior aspect of the upper lobe; the third—the anterior segmental bronchus—runs anteroinferiorly to its distribution in the remainder of the upper lobe. The RML bronchus divides into a lateral segmental bronchus, which is distributed to the lateral aspect of the middle lobe, and a medial segmental bronchus to the medial aspect. The RLL bronchus first gives off a branch from its posterior surface—the superior segmental bronchus—which passes posterosuperiorly to its distribution in the upper portion of the lower lobe. Then, after continuing to descend posterolaterally, the lower lobe bronchus yields the medial basal segmental bronchus (distributed to a small area below the hilus) from its anteromedial surface. The next offshoots from the lower lobe bronchus are the anterior basal segmental bronchus, which continues its descent anteriorly, and a very small trunk that almost immediately splits into the lateral basal segmental bronchus (distributed to the lower lateral area of the lower lobe) and the posterior basal segmental bronchus (distributed to the lower posterior area of the lower lobe).

The LUL bronchus extends laterally from the anterolateral aspect of the left mainstem bronchus before dividing into correlates of the right upper and middle lobar bronchi. However, these two branches remain within the LUL because there is no left middle lobe. The uppermost branch ascends for approximately one-third of an inch before yielding the anterior segmental bronchus, and then continues its upward path as the apicoposterior segmental bronchus before subdividing further into its subsegmental distribution. The caudal branch descends anterolaterally to its distribution in the anteroinferior area of the LUL, a region called the lingula. This lingular bronchus divides into the superior lingular and inferior lingular segmental bronchi.

The LLL bronchus descends posterolaterally for approximately one-third of an inch before giving off the superior segmental bronchus from its posterior surface (its distribution is similar to that of the RLL superior segmental bronchus). After another one-half to two-thirds of an inch, the lower lobe bronchus splits in two: the anteromedial division is called the anteromedial basal segmental bronchus, and the posterolateral division immediately branches into the lateral basal and posterior basal segmental bronchi. The distributions of these segmental bronchi are similar to those of their right-lung counterparts.

The epithelium of the upper regions of the conducting airways is pseudostratified and, for the most part, ciliated. The epithelium of the terminal and respiratory bronchioles is single layered and more cuboidal in shape, and many of the cells are nonciliated. The lamina propria, to which the epithelial basal lamina is attached, contains longitudinal bands of elastin throughout the length of the tracheobronchial tree that spread into the elastin network of the terminal respiratory units. The framework thus created is responsible for much of the elastic recoil of the lungs during expiration.

The most abundant types of cells in the bronchial epithelium are the ciliated cells. Ciliated cells are found in all levels of the tracheobronchial tree down to the level of the respiratory bronchioles. The cilia projecting from their luminal surfaces are intimately involved in the removal of inhaled particulate matter from the airways via the "mucociliary escalator" mechanism.

Two of the bronchial epithelial cells are mucus secreting: the mucous cells and serous cells.[15] Mucous cells, formerly called goblet cells, are normally more numerous in the trachea and large airways, becoming less numerous with distal progression, until they are infrequently found in the bronchioles. Serous cells are much less numerous than mucous cells and are confined predominantly to the extrapulmonary bronchi. Both types of cells are nonciliated, although both exhibit filamentous surface projections.

Clinical tip

Smoking paralyzes ciliated epithelial cells. These cilia will be paralyzed for 1 to 3 hours after smoking a cigarette, or will be permanently paralyzed in chronic smokers.[16] The inability of the mucociliary escalator to work increases the individual's risk for developing respiratory infections.

Terminal respiratory (acinar) units

The conducting airways terminate in gas-exchange airways made up of respiratory bronchioles, alveolar ducts, and alveoli (see Fig. 1.13). These structures together are termed the *acinus* and participate in gas exchange. The functional unit of the lung is the alveoli, where gas exchange occurs. The acinus is connected to the interstitium through a dense network of fibers. Two major types of epithelial cells exist along the alveolar wall. Squamous pneumocytes (type I) cells are flat and thin and cover approximately 93% of the alveolar surface. Granular pneumocytes (type II) cells are thick, are cuboidal shaped, cover 7% of the alveolar wall, and are involved in the production of surfactant.[13] Surfactant is a lipoprotein that lowers alveolar surface tension at end expiration and thereby prevents the lung from collapsing. The alveoli, like the bronchi, contain cellular components of inflammation and immunity. The alveolar macrophage engulfs and ingests foreign material in the alveoli and provides a protective function against disease.

Capillaries composed of a single layer of endothelial cells deliver blood in close proximity to the alveoli. Capillaries can distend and accommodate to the volume of blood being delivered to the lung. The alveolar capillary interface is where exchange of gases occurs. The thickness of the alveolar capillary membrane is between 0.5 and 1.0 μm.

Innervation of the lungs

The lungs are invested with a rich supply of afferent and efferent nerve fibers and specialized receptors. Parasympathetic fibers are supplied by preganglionic fibers from the vagal nuclei via the vagus nerves to ganglia around the bronchi and blood vessels. Postganglionic fibers innervate the bronchial and vascular smooth muscle, as well as the mucous cells and submucosal bronchial glands. The parasympathetic postganglionic fibers from thoracic sympathetic ganglia innervate essentially the same structures. Posterior and anterior pulmonary plexuses are formed by contributions from the postganglionic sympathetic and parasympathetic fibers at the roots of the lungs. In general, stimulation of the vagus nerve results in bronchial constriction, dilation of pulmonary arterial smooth muscle, and increased glandular secretion.[17] Stimulation of the sympathetic nerves causes bronchial relaxation, constriction of pulmonary arterial smooth muscle, and decreased glandular secretion.[17]

Bronchodilators enhance sympathetic stimulation to the lungs to cause relaxation of bronchial smooth muscle cells and reduce secretions.

The cardiovascular system

Mediastinum

The mediastinum lies between the right and left pleura of the lungs and near the median sagittal plane of the chest. From an anteroposterior perspective, it extends from the sternum in front to the vertebral column behind and contains all the thoracic viscera except the lungs.[8] It is surrounded by the chest wall anteriorly, the lungs laterally, and the spine posteriorly. It is continuous with the loose connective tissue of the neck and extends inferiorly onto the diaphragm. It is the central compartment of the thoracic cavity and contains the heart, the great vessels of the heart, esophagus, trachea, phrenic nerve, cardiac nerve, thoracic duct, thymus, and lymph nodes of the central chest.[8,13]

A shifting of the structures within the mediastinum (mediastinal shift) is appropriate to consider and examine on the chest radiograph in patients who have air trapped in the pleural space (pneumothorax) or after removal of a lung (pneumonectomy).[3] In a tension pneumothorax or pneumonectomy, the mediastinum shift away from the affected or operated side.

Heart

The heart is the primary pump that circulates blood through the entire vascular system. It is closely related to the size of the body and is roughly the size of the individual's closed fist. It lies obliquely (diagonally) in the mediastinum, with two-thirds lying left of the midsagittal plane. The superior portion of the heart formed by the two atria is termed the base of the heart. It is broad and exists at the level of the second intercostal space in adults. The apex of the heart, defined by the tip of the left ventricle, projects into the fifth intercostal space at the midclavicular line.

The heart moves freely and changes its position during its contraction and relaxation phase, as well as during breathing. As the heart contracts, it moves anteriorly and collides with the chest wall. The portion of the heart that strikes the chest wall is the apex of the heart and is termed the point of maximum impulse.[1] Normally, this point is evidenced at the anatomic landmark of the apex, which is the fifth intercostal space at the midclavicular line. In terms of ventilation, quiet resting breathing does not alter the point of maximum impulse because of minimal excursion of the diaphragm. However, with deep inspiration, there is more significant inferior depression of the diaphragm, causing the heart to descend and rotate to the right, displacing the point of maximum impulse away from the normal palpable position.[1]

Clinical tip

The point of maximum impulse is relatively more lateral in patients with left ventricular hypertrophy caused by an increase in left ventricular mass. Also patients with a pneumothorax and resultant mediastinal shift will demonstrate an altered point of maximum impulse away from the normal anatomic position of the apex of the heart.

Tissue layers

Pericardium

The heart wall is made up of three tissue layers (Fig. 1.16). The outermost layer of the heart is a double-walled sac termed the pericardium, anchored to the diaphragm inferiorly and the connective tissue of the great vessels superiorly. The two layers of the pericardium include an outer parietal

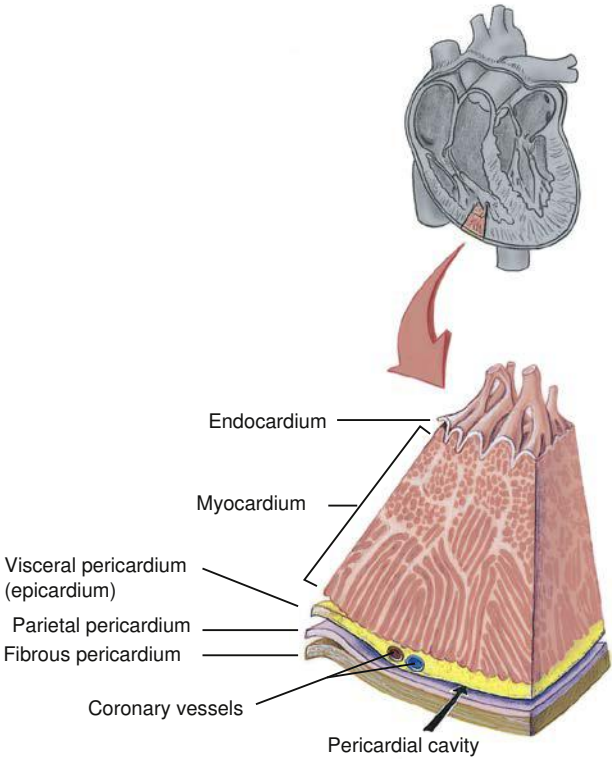

Figure 1.16 Layers of the heart wall. (From Applegate E: *The anatomy and physiology learning system,* ed 3, St. Louis, 2007, Saunders.)

pericardium and an inner visceral pericardium, also referred to as the epicardium.[8] The parietal pericardium is a tough, fibrous layer of dense, irregular connective tissue, whereas the visceral pericardium is a thin, smooth, and moist serous layer. Between the two layers of the pericardium is a closed space termed the pericardial space or pericardial cavity filled with approximately 10 to 20 mL of clear pericardial fluid.[13] This fluid separates the two layers and minimizes friction during cardiac contraction.

In patients with inflammation of the pericardium, fluid may accumulate in the closed pericardial space, producing cardiac tamponade, evidenced as compromised cardiac function and contractility caused by buildup of fluid in the pericardial space. Finally, pericarditis is also commonly noted after a coronary artery bypass grafting procedure.

Myocardium

The middle layer of the heart is termed the myocardium. It is the layer of the heart that facilitates the pumping action of the heart as a result of the presence of contractile elements. Myocardial cells are unique, as they demonstrate three important traits: automaticity (the ability to contract in the absence of stimuli); rhythmicity (the ability to contract in a rhythmic manner); and conductivity (the ability to transmit nerve impulses).[17] Myocardial cells may be categorized into two groups based on their function: mechanical cells contributing to mechanical contraction and conductive cells contributing to electrical conduction.[1,17] Mechanical cells, also termed *myocytes*, are large cells containing a larger number of actin and myosin myofilaments, enabling a greater capacity for mechanical shortening needed for pump action. In addition, these cells have a large number of mitochondria (25% of cellular volume) to provide sufficient energy in the form of

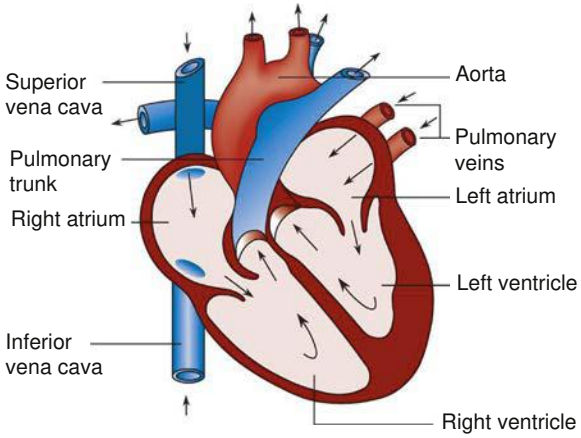

Figure 1.17 View of heart showing all four chambers and forward blood flow through right and left sides. (From Khanna N: *Illustrated synopsis of dermatology and sexually transmitted diseases*, New Delhi, 2005, Peepee Publishers and Distributors.)

adenosine triphosphate to the heart, an organ that can never rest.[5,17] The conducting myocardial cells are joined by intercalated disks forming a structure known as a syncytium. A syncytium characterizes a group of cells in which the protoplasm of one cell is continuous with that of adjacent cells.[8] Intercalated discs contain two junctions: desmosomes attaching one cell to another and connexins that allow the electrical flow to spread from one cell to another. These two junctions work together to move the impulse through a low-resistance pathway.

Clinical tip

Injured myocardial cells cannot be replaced, as the myocardium is unable to undergo mitotic activity. Thus death of cells from an infarction or a cardiomyopathy may result in a significant reduction in contractile function.

Endocardium

The innermost layer of the heart is termed the endocardium. This layer consists of simple squamous endothelium overlying a thin areolar tissue layer.[13] The tissue of the endocardium forms the inner lining of the chambers of the heart and is continuous with the tissue of the valves and the endothelium of the blood vessel.

Because the endocardium and valves share similar tissue, patients with endocarditis must be ruled out for valvular dysfunction. Endocardial infections can spread into valvular tissue, developing vegetations on the valve.[18] Bronchopulmonary hygiene procedures, including percussions and vibrations, are contraindicated for patients with unstable vegetations, as they may dislodge, move as emboli, and cause an embolic stroke.

Chambers of the heart

The heart is divided into right and left halves by a longitudinal septum (Fig. 1.17). The right side of the heart receives deoxygenated venous blood (returning from the body), and the left side of the heart receives oxygenated blood (returning from the lungs). Each half of the heart is made up of two

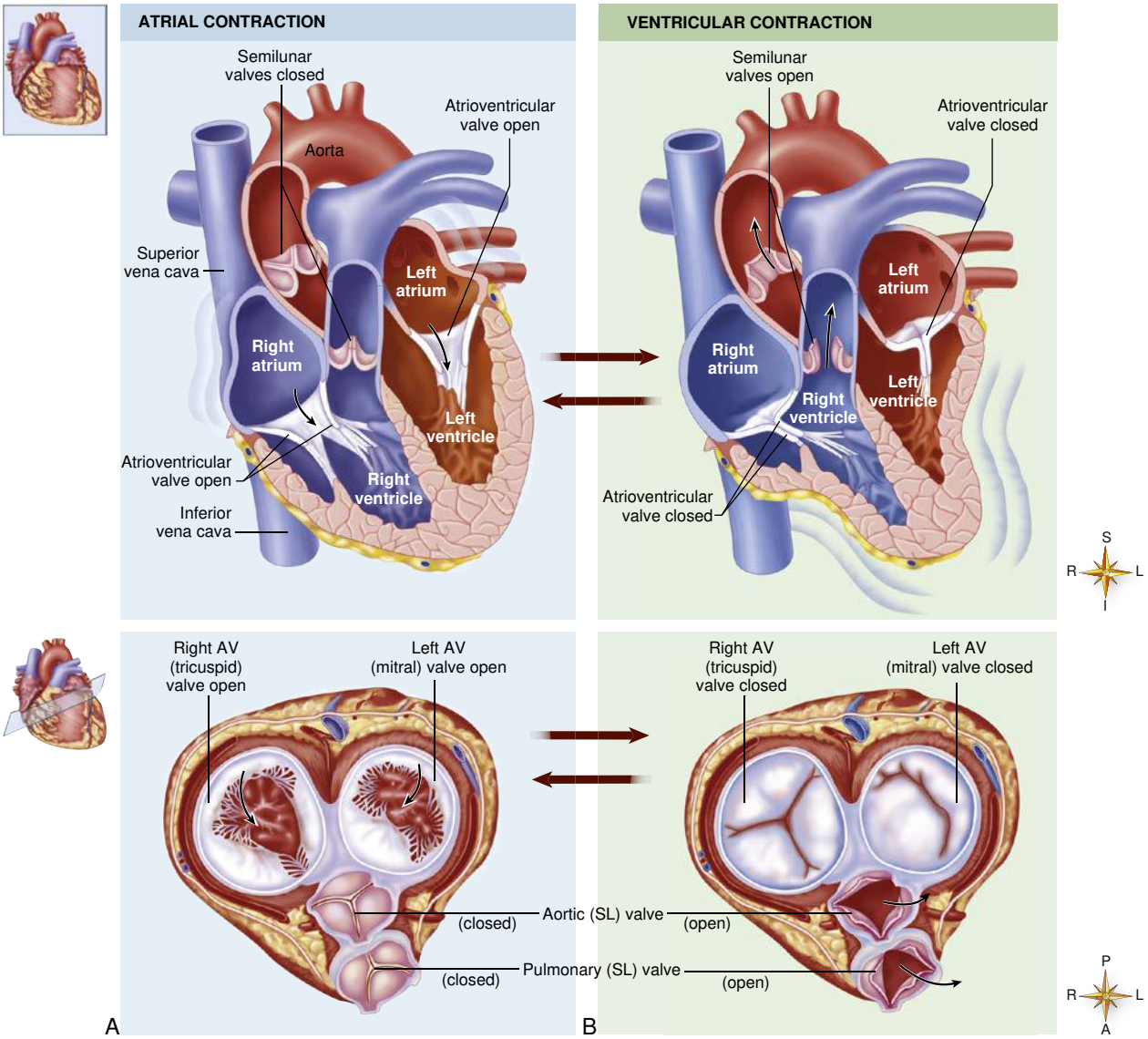

Figure 1.18 Chambers and valves of the heart. (A) The view of the chambers and valves during atrial contraction. (B) The view of ventricular contraction. *AV,* Arterioventricular; *SL,* semilunar. (From Patton K, Thibodeau G, Douglas M: *Essentials of anatomy and physiology*, ed 1, St. Louis, 2011, Elsevier.)

chambers: superiorly the atria and inferiorly the ventricles. Thus the four chambers of the heart include the right atrium (Fig 1.18), right ventricle, left atrium, and left ventricle. The atria receive blood from the systemic and pulmonary veins and eject blood into the ventricles. The ventricles eject blood that is received from the atria into arteries that deliver blood to the lungs and the systemic circulation.

Right atrium

The chamber of the right atrium consists of a smooth posterior and medial inner wall. Parallel muscle bundles known as pectinate muscles exist anteriorly and laterally. Both right and left atria have small earlike extensions called auricles that help to increase volume within the chambers. The right atrium receives deoxygenated blood from three major vessels. The superior vena cava collects venous blood from the head and upper extremities; the inferior vena cava collects blood from the trunk and lower extremities; and the coronary sinus collects venous blood specifically from the heart. The coronary sinus empties into the right atrium above the tricuspid valve. Normal diastolic pressure to enable filling ranges from 0 to 8 mm Hg and is clinically referred to as the central venous pressure.

The effective contraction of the pectinate muscles of the atria accounts for approximately 15% to 20% of cardiac output—the atrial kick.[18] In patients with abnormal electrical conduction causing a quivering of the atria (atrial fibrillation), the mechanical contractile ability of the pectinate muscles is reduced, resulting in a low atrial kick and compromised cardiac output.[1,5]

Right ventricle

The right ventricle is shaped like a crescent or triangle, enabling it to eject large volumes of blood through a small valve into a low-pressure pulmonary system. Blood within the right ventricle is received from the right atrium through a one-way valve present between the atrium and ventricle termed the tricuspid atrioventricular (AV) valve. It ejects blood to the lungs via the pulmonic semilunar valve into the pulmonary artery. The right ventricle, like the right atrium,

may be considered in two parts: (1) a posteroinferior inflow tract, termed the body, which contains the tricuspid valve, chordae tendineae, papillary muscles, and trabeculated myocardium; and (2) an anterosuperior outflow tract, called the infundibulum, from which the pulmonary trunk arises.[8] Four muscular bands separate the inflow and outflow portions of the right ventricle, including the infundibular septum, the parietal band, the septal band, and the moderator band. Pressures within the right ventricle are relatively lower compared with the left ventricle, with diastolic pressures ranging from 0 to 8 mm Hg and systolic pressures ranging from 15 to 30 mm Hg.[17]

During periods of exacerbation, patients with chronic lung pathologies, including COPD and pulmonary fibrosis, often present with hypoxemia and increased pressure within the pulmonary vasculature, termed pulmonary artery hypertension, caused by compromised perfusion capacity to the lung.[18,19] The increased pressure within the pulmonary artery increases the workload on the right ventricle, causing cor pulmonale, or right ventricular hypertrophy, and resultant right ventricular failure.

Left atrium

The left atrium is divided from the right atrium by an interatrial septum. It has a relatively thicker wall compared with the right atrium to adapt to higher pressures of blood entering the chamber from the lung. Oxygenated blood from the lungs enters the left atrium posteriorly via the pulmonary veins. These vessels have no valves; instead, pectinate muscles extend from the atria into the pulmonary veins and exert a sphincter-like action to prevent backflow of blood during contraction of the atria. The normal filling pressure of the left ventricle is between 4 and 12 mm Hg. Oxygenated blood is ejected out of the left atrium through the mitral AV (bicuspid) valve to enter the left ventricle.

Regurgitation, or insufficiency of the mitral valve, causes blood to accumulate in the left atrium and elevate left atrial pressures. These chronically elevated pressures alter the integrity of the atrial wall and predispose the individual to developing a quivering of the atria wall (atrial fibrillation) and potential blood clots within the left atrium.

Left ventricle

The almost conical left ventricle is longer and narrower than the right ventricle. The walls of the left ventricle are approximately 3 times thicker than those of the right, and the transverse aspect of the cavity is almost circular. In contrast to the inflow and outflow orifices of the right ventricle, those of the left are located adjacent to one another, being separated only by the anterior leaflet of the mitral valve and the common fibrous ridge to which it and the left and posterior cusps of the aortic valve are attached. The interventricular septum forms the medial wall of the left ventricle and creates a separation between the left and right ventricle.

This chamber receives oxygenated blood from the left atrium via the mitral valve and ejects blood through the aortic valve and into the aorta to the peripheral systemic vasculature. Normal systolic pressures within the left ventricle are 80 to 120 mm Hg, and diastolic pressures are 4 to 12 mm Hg. Because of the elevated pressures within this chamber, the wall thickness of the left ventricle is the greatest compared with the three other chambers of the heart.

Pathologic thickening of the left ventricular wall is evidenced in patients with various cardiovascular complications, including but not limited to, hypertension, aortic stenosis, and heart failure, as a consequence of an increase in the afterload. This pathologic thickening alters the contractile ability of the ventricle and reduces its filling capacity, causing a reduction in cardiac output.

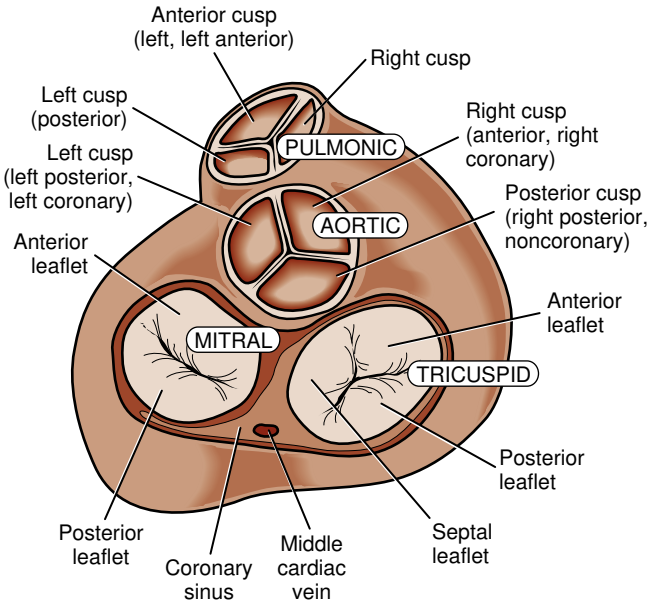

Figure 1.19 Nomenclature for the leaflets and cusps of the principal valves of the heart.

Heart valves

Four heart valves (Fig. 1.19) ensure one-way blood flow through the heart. Two AV valves exist between the atria and the ventricle, including the tricuspid valve on the right and the mitral or bicuspid valve on the left between the left atrium and ventricle. The semilunar valves lie between the ventricles and arteries and are named based on their corresponding vessels: pulmonic valve on the right in association with the pulmonary artery, and aortic valve on the left relating to the aorta.

Flaps of tissue called leaflets or cusps guard the heart valve openings. The right AV valve has three cusps and therefore is termed tricuspid, whereas the left AV valve has only two cusps and hence is termed bicuspid. These leaflets are attached to the papillary muscles of the myocardium by chordae tendineae. The primary function of the AV valves is to prevent backflow of blood into the atria during ventricular contraction or systole, and the semilunar valves prevent backflow of blood from the aorta and pulmonary artery into the ventricles during diastole. Opening and closing of each valve depends on pressure gradient changes within the heart created during each cardiac cycle.

An initial disturbance of valvular function may be picked up through auscultation of the heart sounds and evidenced by variety of murmurs. It must be noted that the identification of a murmur would warrant the need for additional testing, including echocardiography, to accurately diagnose pathology within a particular valve.

Conduction system

In a normal conduction system (Fig. 1.20), electrical impulses arise in the sinoatrial (SA), or sinus, node. The SA node is

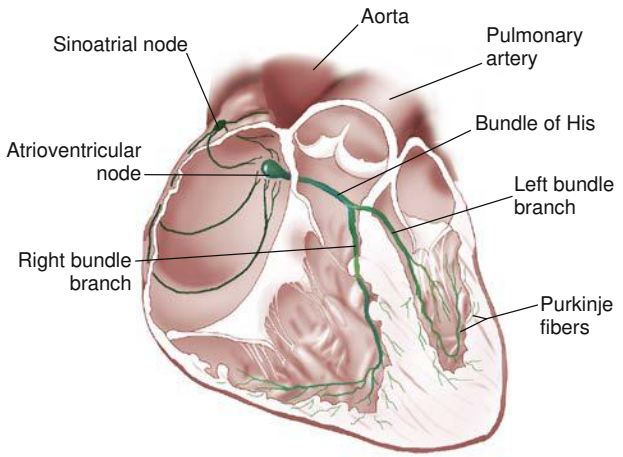

Figure 1.20 Conduction system of the heart. The electrical impulse originates in the heart, and contraction of the heart's chambers is coordinated by specialized heart tissues. (From Leonard PC: *Building a medical vocabulary: with Spanish translations*, ed 7, St. Louis, 2009, Saunders.)

located at the junction of the right atrium and superior vena cava. The P cells of the SA node are the sites for impulse generation; consequently, the SA node is termed the pacemaker of the heart because it makes or creates the impulses that pace the heart.[8] The normal pacing ability of the SA node is between 60 and 100 beats per minute at rest. The impulse generated at the SA node travels down one of three internodal tracts to the AV node. The three conduction pathways that exist between the SA and AV node include an anterior tract of Bachman, a middle tract of Wenckebach, and a posterior tract of Thorel.[8]

The AV node is located at the inferior aspect of the right atrium, near the opening of the coronary sinus and above the tricuspid valve. Posterior to the AV node are several parasympathetic autonomic ganglia that serve as receptors for the vagus nerve and cause slowing of the cardiac cycle. The major function of the AV node during each cardiac cycle is to slow down the cardiac impulse to mechanically allow time for the ventricles to fill.

Conducting fibers from the AV node converge to form the bundle of His to carry the impulse into the ventricles. The bundle of His appears as a triangle of nerve fibers within the posterior border of the interventricular septum. The bundle bifurcates to give rise to the right and left bundle branches carrying the impulse to the right and left ventricles, respectively. The right bundle branch is thin, with relatively fewer branches proceeding inferiorly to the apex of the right ventricle. The left bundle branch arises perpendicularly and divides into two branches or fascicles.[8] The left anterior bundle branch crosses the left anterior papillary muscle and proceeds along the base of the left ventricle toward the aortic valve. The left posterior bundle branch advances posteriorly through the posterior papillary muscle toward the posterior inferior left ventricular wall.

Both bundles terminate into a network of nerve fibers called the Purkinje fibers. These fibers extend from the apex of each ventricle and penetrate the heart wall to the outer myocardium. Electrical stimulation of the Purkinje fibers causes mechanical contraction of the ventricles. It may be important to appreciate that normal electrical conduction through the heart allows for appropriate mechanical activity and maintenance of cardiac output to sustain activity. An alteration in the conduction pathway subsequently alters the mechanical activity of the heart and reduces cardiac output.

Clinical tip

An evaluation of electrocardiographic (ECG) changes is necessary to help a clinician recognize and differentially diagnose reduced exercise tolerance caused from an electrical disturbance producing mechanical alterations that reduce cardiac output and exercise tolerance and not a true mechanical problem within the heart.

Innervation

Although the SA node and conduction pathway have an intrinsic rate of depolarization causing contraction of the myocardium, the autonomic nervous system influences the rate of impulse generation, contraction, relaxation, and strength of contraction.[6,17] Thus autonomic neural transmission creates changes in the heart rate and contractility to allow adjustments in cardiac output to meet metabolic demands. The central and peripheral components of the autonomic nervous system exert control over the heart. The cortex and medulla oblongata are the central components that regulate the autonomic control of the heart.[20] The peripheral components of the autonomic nervous system form a cardiac plexus.[21] The cardiac plexus contains both sympathetic (via the sympathetic chain) and parasympathetic (via the right and left vagus nerve) nerve fibers.[17] The cardiac plexus is divided into a superficial and deep portion. The superficial part of the cardiac plexus lies beneath the aortic arch and anterior to the right pulmonary artery.[21] The deep part of the is located anterior to the tracheal bifurcation cardiac plexus (Fig. 1.21).[17]

Subsequently, nerves branch off the plexus, follow the coronary vessels, and innervate the SA node and other components of the conduction system. There is relatively less parasympathetic innervation to the ventricles, resulting in a sympathetic dominance on ventricular function. Vagal stimulation is inhibitory on the cardiovascular system and is evidenced by decreased heart rate and blood pressure.[6] The neurohormone involved with parasympathetic stimulation is acetylcholine.

The sympathetic input to the plexus arises from the sympathetic trunk in the neck. Cardiac nerves from the cervical and upper four to five thoracic ganglia feed into the cardiac plexus.[6,17] Sympathetic stimulation releases catecholamines (epinephrine and norepinephrine) that interact with β-adrenergic receptors on the cardiac cell membrane, causing an excitation of the cardiovascular system. This is evidenced by an increase in heart rate, increased contractility through a greater influx of calcium into myocytes, increased blood pressure, a shortening of the conduction time through the AV node, and an increase in rhythmicity of the AV pacemaker fibers.

Sympathetic nervous system stimulation is cardioexcitatory and increases heart rate and contractility—the fight-or-flight response. Conversely, parasympathetic stimulation is cardioinhibitory and slows down heart rate and contractility.

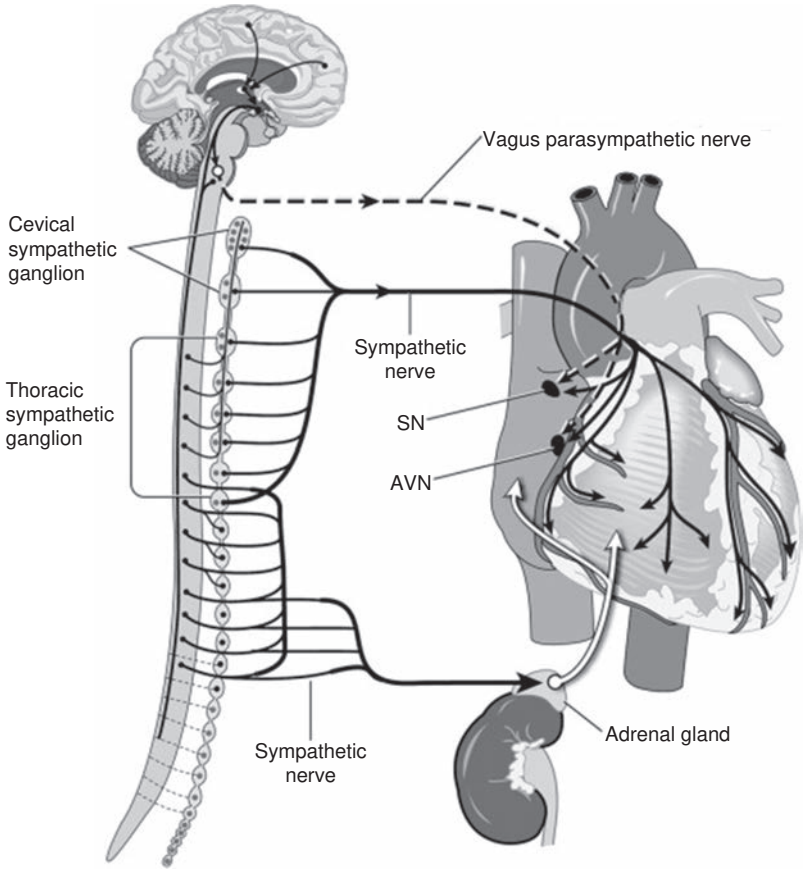

Figure 1.21 Cardiac sympathetic and parasympathetic nerves. The vagus nerves to the heart are parasympathetic nerves. *AVN,* Atrioventricular node; *SN,* sinoatrial node. (From Kobayashi M, Massiello A: Cardiac autonomic nerve stimulation in the treatment of heart failure, *Ann Thorac Surg* 96(1):339–345, 2013.)

Clinical tip

During a heart transplant, there is denervation of the sympathetic and parasympathetic fibers causing these fibers to be separated from the central control while the postganglionic fibers are left intact. This denervation leads to an increase in basal heart rate as the transplanted heart relies on the intrinsic rather than neurologic input from the recipient. The decentralized heart will respond to catecholamines as the α and β receptors are still present on the heart but will not respond to anticholinergic drugs because of disruption in the parasympathetic fibers.[22]

Cardiac and pulmonary vessels

Aorta

The ascending aorta begins at the base of the left ventricle and is approximately 2 inches long. From the lower border of the third costal cartilage at the left of the sternum, it passes upward and forward toward the right as high as the second right costal cartilage. The aorta exhibits three dilations above the attached margins of the cusps of the aortic valve at the root of the aorta—the aortic sinuses (of Valsalva). The coronary arteries (Fig. 1.22) open near these aortic sinuses of Valsalva. Three branches typically arise from the upper aspect of the arch of the aorta: the brachiocephalic trunk (innominate artery), the left common carotid artery, and the left subclavian artery. During systole, the aortic valve is open and the leaflets of the aortic valve block blood from entering the openings of the coronary arteries. Therefore the part of the cardiac cycle when the coronary arteries receive their blood is during diastole, when the aortic valves are closed.

Right coronary artery

The right coronary artery arises from the right anterolateral surface of the aorta and passes between the auricular appendage of the right atrium and the pulmonary trunk, typically giving off a branch to the sinus node and yielding two or three right anterior ventricular rami as it descends into the coronary sulcus to come around the right (acute) margin of the heart into the posterior aspect of the sulcus. As the right coronary artery crosses the right margin of the heart, it gives off the right (acute) marginal artery before continuing as far as the posterior interventricular sulcus, where it usually turns to supply the diaphragmatic surfaces of the ventricles as the posterior interventricular (posterior descending) artery. In approximately 70% of hearts, an AV nodal artery is given off just before the posterior interventricular artery.[23]

Left coronary artery

The left coronary artery originates from the left anterolateral aspect of the aorta and splits into two major branches: the anterior interventricular and circumflex arteries. The anterior interventricular, or left anterior descending (LAD) artery, traverses the anterior interventricular groove to

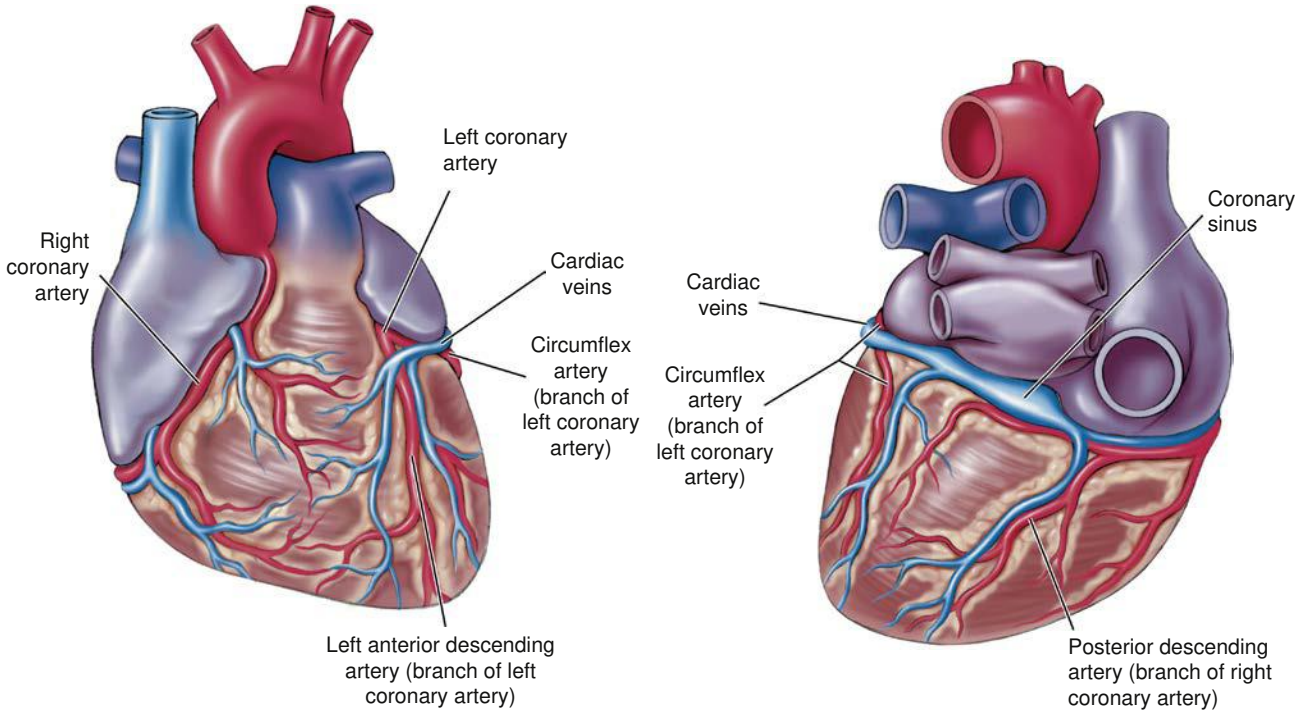

Figure 1.22 Typical distributions of the right and left coronary arteries and veins.

supply sternocostal aspects of both ventricles. In its course, the anterior interventricular artery gives off right and left anterior ventricular and anterior septal branches. The larger left anterior ventricular branches vary in number from two to nine, with the first being designated the diagonal artery. Approximately 70% of the left ventricle is fed by the LAD artery. The circumflex artery runs in the coronary sulcus between the left atrium and ventricle, crosses the left margin of the heart, and usually continues to its termination, just short of the junction of the right coronary and the posterior interventricular arteries. In many instances, as the circumflex artery crosses the left margin of the heart, it gives off a large branch that supplies this area—the left marginal (obtuse) artery.

The right coronary artery is the primary supply route for blood to the majority of the right ventricle and the inferior and posterior portions of the left ventricle. In addition, specialized conduction tissue within the right atrium, including the SA node and AV node, are nourished by the right coronary artery. The LAD supplies blood to the anterior and septal aspects of the left ventricle, and the circumflex artery supplies blood to the lateral aspect of the left ventricle.

Occlusion of a coronary artery produces an infarction in a defined region within the heart. Right coronary artery occlusions cause inferior or posterior infarctions and affect the functioning of the SA node in the right atrium. Left anterior descending artery occlusions produce anterior septal infarctions, also termed the widow maker, whereas circumflex occlusions are responsible for generating lateral infarctions.

Distribution of blood supply within the heart is variable from one individual to another because of the presence of collateral circulation involving the formation of new blood vessels (angiogenesis) in areas of the heart that are partially occluded.

Pulmonary artery

The pulmonary trunk runs upward and backward (first in front of and then to the left of the ascending aorta) from the base of the right ventricle; it is approximately 2 inches in length. At the level of the fifth thoracic vertebra, it splits into right and left pulmonary arteries. The right pulmonary artery runs behind the ascending aorta, superior vena cava, and upper pulmonary vein, but in front of the esophagus and right primary bronchus to the root of the lung. The left pulmonary artery runs in front of the descending aorta and the left primary bronchus to the root of the left lung. It is attached to the arch of the aorta by the ligamentum arteriosum.

Clinical tip

A saddle embolus is life-threatening and involves an embolus dislodged at the bifurcation of the right and left pulmonary arteries.

Pulmonary veins

The pulmonary veins, unlike the systemic veins, have no valves. They originate in the capillary networks and join together to ultimately form two veins—a superior and an inferior pulmonary vein—from each lung, which open separately into the left atrium (see Fig. 1.22).

Vena cava and cardiac veins

The superior vena cava is approximately 3 inches long from its termination in the upper part of the right atrium opposite the third right costal cartilage to the junction of the two brachiocephalic veins. The inferior vena cava extends from the

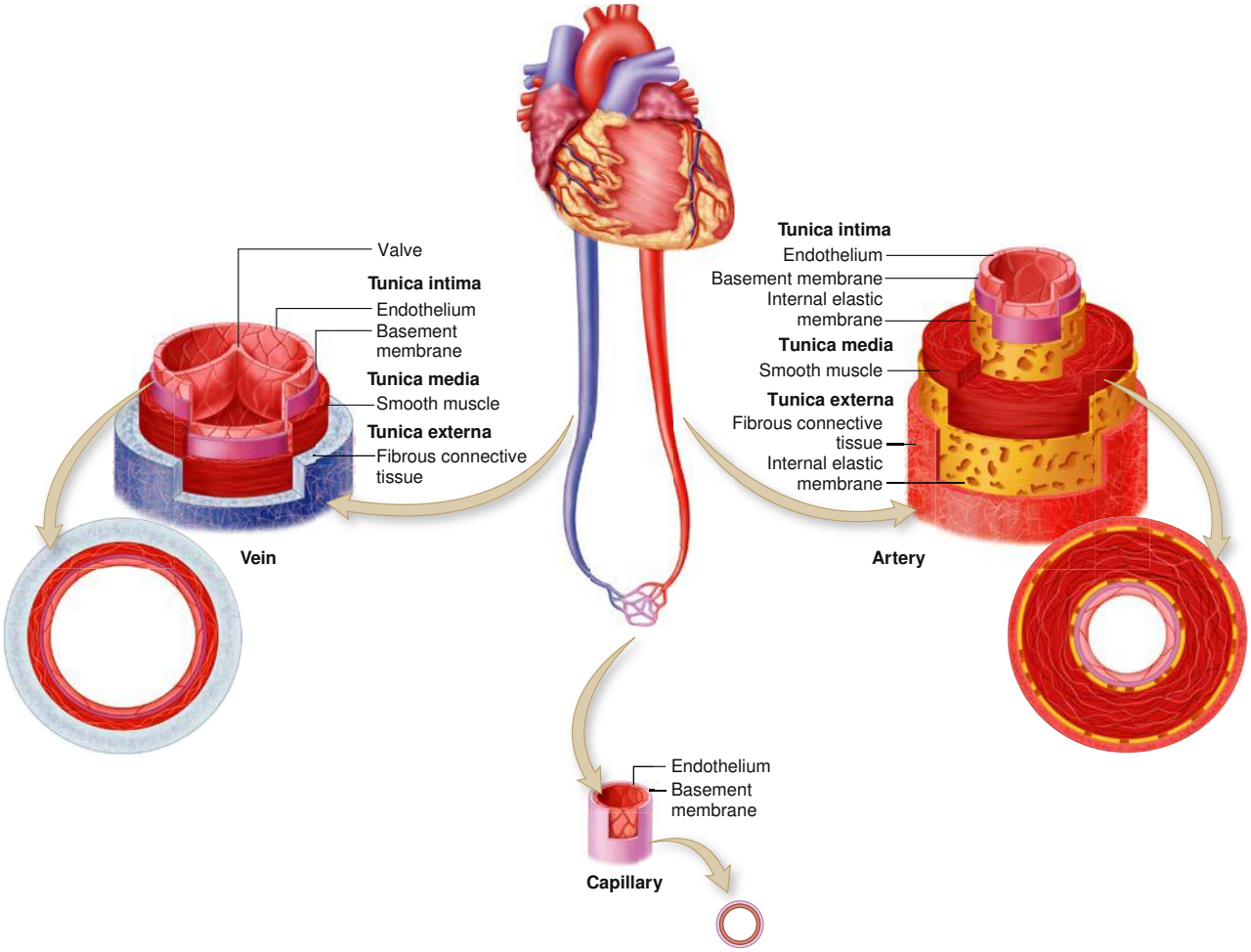

Figure 1.23 Anatomy of artery. (From Patton K, Thibodeau G, Douglas M: *Essentials of anatomy and physiology*, ed 1, St. Louis, 2011, Elsevier.)

junction of the two common iliac veins, in front of the fifth lumbar vertebra, passing through the diaphragm to open into the lower portion of the right atrium. The vena cavae have no valves.

The cardiac veins can be categorized into three groups: the coronary sinus and its supplying veins, the anterior cardiac veins, and the thebesian veins. Most of the veins of the heart drain into the coronary sinus, which runs into the posterior aspect of the coronary sulcus and empties through the valve of the coronary sinus, a semilunar flap, into the right atrium between the opening of the inferior vena cava and the tricuspid valve. As Fig. 1.22 shows, the small and middle cardiac veins, the posterior vein of the left ventricle, the left marginal vein, and the great cardiac vein feed the coronary sinus.

The anterior cardiac veins are fed from the anterior part of the right ventricle. They originate in the subepicardial tissue, crossing the coronary sulcus as they terminate directly into the right atrium. The right marginal vein runs along the right border of the heart and usually opens directly into the right atrium. Occasionally, it may join the small cardiac vein.

The thebesian veins (venae cordis minimae) vary greatly in their number and size. These tiny veins open into all the cavities of the heart, but are most numerous in the right atrium and ventricle, are found occasionally in the left atrium, and are rare in the left ventricle.

Systemic circulation

Oxygenated blood ejected out of the heart flows through the aorta into systemic arteries. These arteries branch into smaller vessels called arterioles, which further branch into the smallest vessels, the capillaries primarily involved in the exchange of nutrients and gases. Deoxygenated blood from the capillaries enters venules that join together to form larger veins that return blood back to the right heart and lungs. Blood vessels have three layers: the innermost tunica intima, middle tunica media, and outermost tunica adventitia.

Arteries

The wall of the artery is composed of elastic and fibrous connective tissue and smooth muscle (Fig. 1.23). Anatomically, arteries can be categorized into two types depending on the structural components along their wall. Elastic arteries, including the aorta and pulmonary trunk, have a thick tunica media with more elastic fibers than smooth muscle cells, allowing for a greater stretch as blood is ejected out of the heart. During diastole, the elasticity of the vessel promotes recoil of the artery and maintains blood pressure within the vessel. Muscular arteries are present in medium and small arteries and contain more smooth muscle cells within the middle tunica media layer.

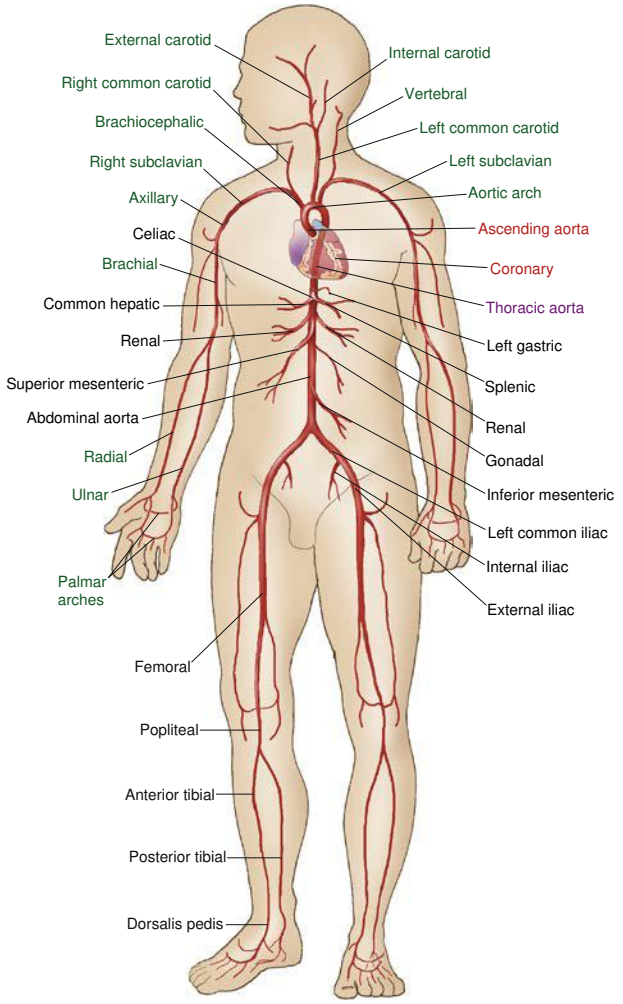

Figure 1.24 Anterior view of the aorta and its principal arterial branches. Labels for the ascending, arch, thoracic, and abdominal aorta and their corresponding arteries are shown. (From Leonard PC: *Building a medical vocabulary: with Spanish translations*, ed 7, St. Louis, 2009, Saunders.)

These arteries have the ability of vasoconstriction and vasodilation as a result of the presence of smooth muscles cells to control the amount of blood flow to the periphery.[6] These smooth muscle cells are under autonomic nervous system influence through the presence of α-receptors. As the artery becomes more distal, a greater amount of smooth muscle is evidenced. Arterioles have primarily smooth muscle along their walls, enabling their diameter to alter significantly as needed. Arterioles empty into capillary beds. The density of capillaries within a capillary bed is greater in active tissue, including the muscle. Exchange of nutrients and gases occur within the capillary bed. Fig. 1.24 depicts the major arterial tree within the human body.

Endothelium

Endothelial cells form the endothelium, or endothelial lining, of the blood vessel. These cells have the ability to adjust their number and arrangement to accommodate local requirements. Endothelial cells serve several important functions, including filtration and permeability, vasomotion, clotting, and inflammation.[18] Atherosclerosis is initiated through endothelial dysfunction, evidenced by endothelial cells that are extensively permeable to fat cells and white blood cells.

Veins

Compared with arteries, veins have thinner walls and a larger diameter. Veins also have less elastic tissue and hence are not as distensible. In the lower extremity, veins have valves to assist with unidirectional flow of blood back to the heart. Blood is transferred back to the heart through muscle pump activity, which causes a milking effect on the veins.

Patients with incompetent valves in their veins develop varicosities in their lower extremities. Also patients on prolonged bed rest are likely to develop deep vein thrombosis from a lack of muscle activity, resulting in a pooling of blood and clot formation within the venous vasculature.

Summary

This chapter provides the reader with an understanding of the anatomy of the cardiovascular and pulmonary systems and its relevance for the therapist. This content provides the basis for an understanding of the pathophysiology of these systems and lays a foundation for the development of relevant examination and treatment strategies to use when managing patients with cardiopulmonary dysfunction. A comprehensive understanding of anatomy is fundamental to the knowledge base of the therapist in understanding the central components involved in the delivery of oxygen and nutrients to peripheral tissue.

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2

Physiology of the cardiovascular and pulmonary systems

Stephen Ramsey, Konrad J. Dias, Ellen Hillegass

CHAPTER OUTLINE

This chapter reviews concepts relating to the physiology of the cardiovascular and pulmonary systems and its relevance in physical therapy practice. The cardiopulmonary systems not only share a close spatial relationship in the thoracic cavity, but also have a close functional relationship to maintain homeostasis. Physiologically, these systems must work collaboratively to provide oxygen required for energy production and assist in removing carbon dioxide manufactured as a waste product. A disorder affecting the lungs has a direct effect on the heart and vice versa. An understanding of normal physiology helps the reader better appreciate pathophysiologic changes associated with diseases and dysfunction of these systems that will be discussed in subsequent chapters.

The pulmonary system

The pulmonary system has several important functions. The most important function of the pulmonary system is to exchange oxygen and carbon dioxide between the environment, blood, and tissue. Oxygen is necessary for the production of energy. If a cell has oxygen, a single molecule of glucose can undergo aerobic metabolism and produce 36 adenosine triphosphates (ATP). However, if a cell is devoid of oxygen, each molecule of glucose undergoes anaerobic metabolism, yielding only two ATP. Thus pathology of the pulmonary system will result in reduced energy production because of decreased oxygen within the tissue and a concomitant reduction in the exercise tolerance of the individual. Carbon dioxide is another gas that must be effectively exchanged at the cellular level of the lung. Through the release of carbon dioxide from the body, the pulmonary system plays an important role in regulating the acid–base balance and maintaining normal blood pH. The second function of the pulmonary system is temperature homeostasis, which is achieved through evaporative heat loss from the lungs. Finally, the pulmonary system helps to filter and metabolize toxic substances, as it is the only organ that receives all blood coming from the heart.

To facilitate comprehension of the physiology of the pulmonary system, three major physiologic components are discussed in this chapter, including: (1) the process of ventilation or breathing; (2) the process of gas exchange or respiration; and (3) the transport of gases to peripheral tissue.

Ventilation

Ventilation, or breathing, often misnamed respiration, involves the mechanical movement of gases into and out of the lungs.[1] At rest, an adult breathes at a rate of 10 to 15 breaths per minute, termed the ventilatory rate or respiratory rate. Approximately 350 to 500 mL of air is inhaled or exhaled at rest with each breath and is termed the tidal volume (TV or VT). The amount of effective ventilation, termed the minute ventilation, represents the total volume of air that is inhaled or exhaled in 1 minute. The minute ventilation, expressed in liters per minute, is calculated by multiplying the respiratory rate and tidal volume. At rest, the minute ventilation is approximately 5 L/min, whereas at maximum exercise, it increases to a level between 70 and 125 L/min.[2]

Additional lung volumes

Before considering the mechanical properties of the lungs during ventilation or breathing, it is helpful to consider the static volumes of the lungs measured via spirometry studies (Fig. 2.1).[2–4] We can effectively categorize these spirometry values as either volumes or capacities, where a capacity is simply the sum of two or more lung volumes. As mentioned earlier, the volume of air normally inhaled and exhaled with each breath during quiet breathing is called the TV. The additional volume of air that can be taken into the lungs beyond the normal tidal inhalation is called the inspiratory reserve volume. The additional volume of air that can be let out beyond the normal tidal exhalation is called the expiratory reserve volume. The volume of air that remains in the lungs after a forceful expiratory effort is called the residual volume (RV). The inspiratory capacity is the sum of the tidal and inspiratory

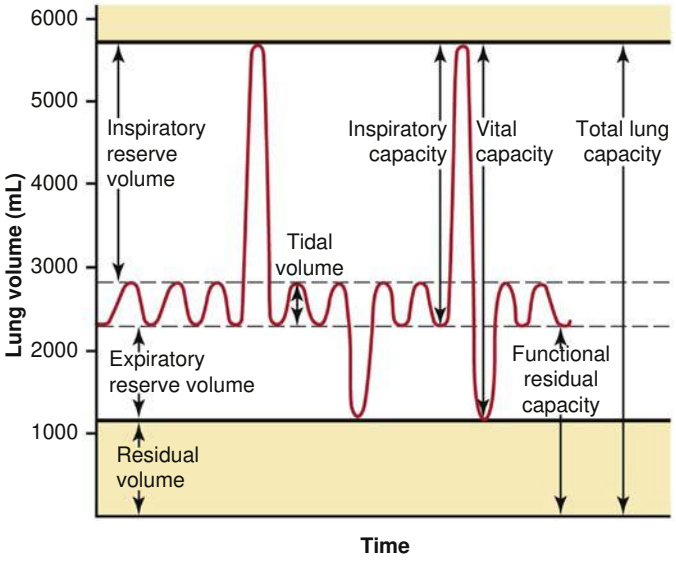

Figure 2.1 Lung volumes and capacities as displayed by a time-versus-volume spirogram. Values are approximate. The tidal volume is measured under resting conditions. (From Seeley RR, Stephens TD, Tate P: *Anatomy & physiology*, ed 3, New York, 1995, McGraw-Hill.)

reserve volumes; it is the maximum amount of air that can be inhaled after a normal tidal exhalation. The functional residual capacity (FRC) is the sum of the expiratory reserve and RV; it is the amount of air remaining in the lungs at the end of a normal tidal exhalation. The importance of FRC cannot be overstated; it represents the point at which the forces tending to collapse the lungs are balanced against the forces tending to expand the chest wall. The vital capacity is the sum of the inspiratory reserve, tidal, and expiratory reserve volumes; it is the maximum amount of air that can be inhaled following a maximum exhalation. The total lung capacity (TLC) is the maximum volume to which the lungs can be expanded; it is the sum of all the lung volumes.

Control of ventilation

Breathing requires repetitive stimulation from the brain, as skeletal muscles required for ventilation are unable to contract without nervous stimulation.[5] Although breathing usually occurs automatically and involuntarily, there are circumstances when individuals hold their breath, take deep breaths, or change ventilation, such as when singing or laughing. In light of this, it is important to review the mechanisms involved in helping to control breathing.

This section describes the neural mechanisms that regulate ventilation. Neurons in parts of the brainstem, including the medulla oblongata and pons, provide control for automatic breathing and adjust ventilatory rate and tidal volume for normal gas exchange (Fig. 2.2).[5] The medulla oblongata contains inspiratory neurons that produce inspiration and expiratory neurons that are triggered with forced expiration. Inspiratory neurons are located in the inspiratory center, or dorsal respiratory group, of the medulla. An enhanced frequency of firing of these neurons increases the motor units recruited and results in a deeper breath.[6] An elongation in the time of firing prolongs each breath and results in a slower respiratory rate.[6] A cessation of neural stimulation of these neurons causes elastic recoil of the lungs and passive expiration.

The expiratory center, or ventral respiratory group, in the medulla contains inspiratory neurons in the midregion and expiratory neurons in the anterior and posterior zones. Neural stimulation of the expiratory neurons causes inhibition of the inspiratory center when a deeper expiration is warranted.

The pons has two major centers that assist with ventilation, including the pneumotaxic center in the upper pons and the apneustic center in the lower pons.[5] The pneumotaxic center maintains the rhythm of ventilation, balancing the time periods of inspiration and expiration by inhibiting the apneustic center or the inspiratory center of the medulla. The apneustic center facilitates sustained or prolonged breathing patterns when it is uninhibited from the pneumotaxic center.

Breathing concerning a conscious change in pattern involves control from the motor cortex of the frontal lobe of the cerebrum.[6] Here, impulses are sent directly down to the corticospinal tracts to the respiratory neurons in the spinal cord, bypassing the respiratory centers in the brainstem to trigger changes in ventilation.

Afferent connections to the brainstem

The respiratory centers of the brainstem receive afferent input from various locations, including the limbic system, hypothalamus, chemoreceptors, and lungs.[5]

Hypothalamic and limbic influence

Sensations of pain and alterations in emotion alter ventilation through input coming to the brainstem from the limbic system and hypothalamus.[7] For example, anxiety triggers hyperventilation and a concomitant reduction in carbon dioxide levels in blood, as the rate of carbon dioxide elimination out of the lungs exceeds the rate of carbon dioxide production in the body.

Clinical tip

Patients with injuries within the central nervous system from an acute brain injury or stroke demonstrate altered ventilatory patterns following neurologic insult. These patients lose the normal response to breathing, resulting in altered ventilatory rates and volumes.

Chemoreceptors

Chemoreceptors are located in the brainstem and peripheral arteries. These receptors are responsible for sensing alterations in blood pH, carbon dioxide, and oxygen levels.[7] There primarily exist two types of chemoreceptors, including central and peripheral. The receptors found along the anterior lateral surfaces of the upper medulla of the brainstem are called central chemoreceptors. These receptors are stimulated when carbon dioxide concentrations rise in the cerebrospinal fluid. Central chemoreceptors facilitate an increased depth and rate of ventilation so as to restore normal carbon dioxide levels and pH in the body.[5,7] Peripheral chemoreceptors are found within the carotid artery and aortic arch. These receptors help to increase ventilation in response to increasing levels of carbon dioxide in blood (hypercapnia), as well as low oxygen levels in blood (hypoxia).[5,7] Normal ventilation is driven mostly by the levels of carbon dioxide and very little by oxygen levels.

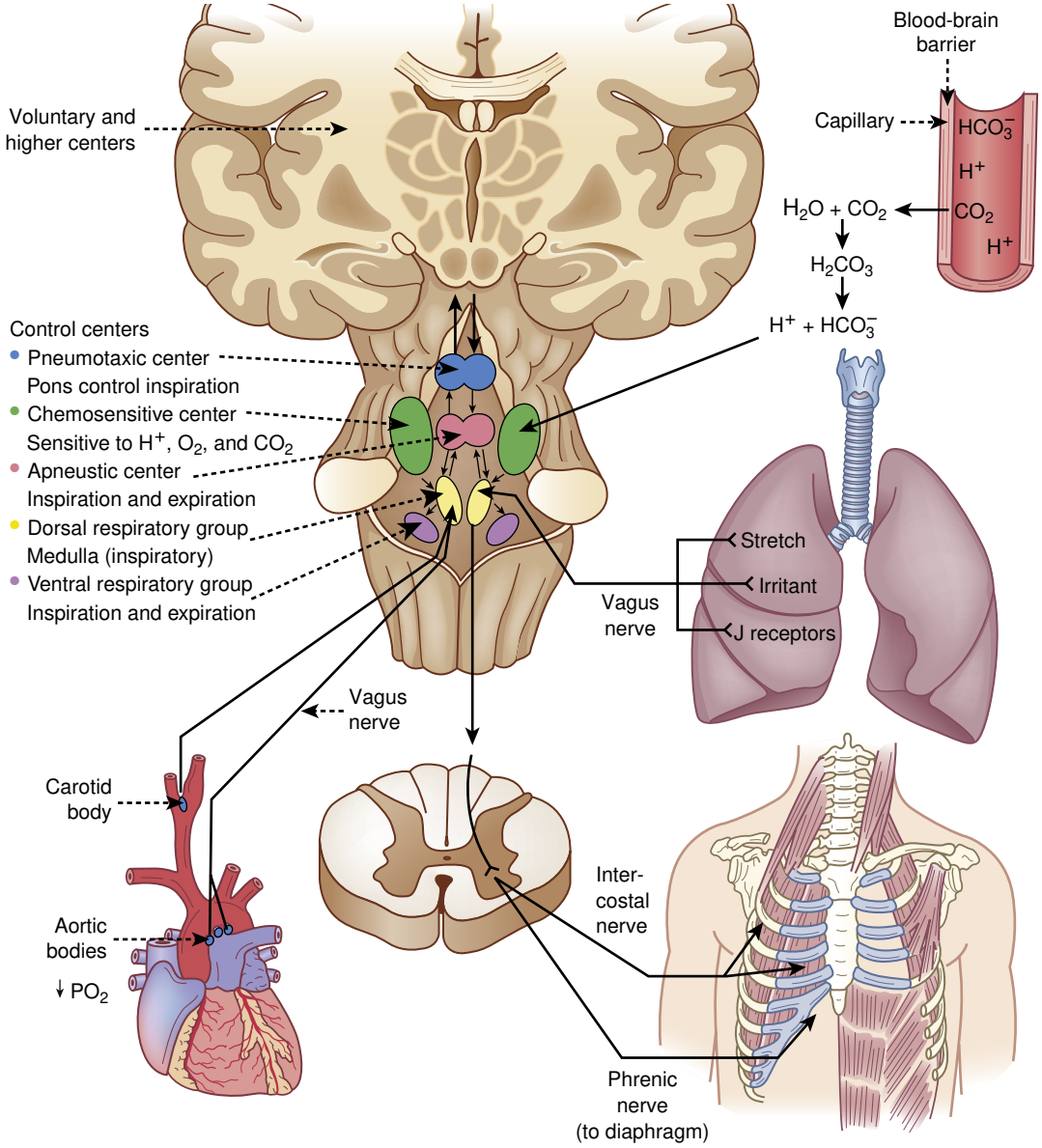

Figure 2.2 Neurochemical respiratory control system. (From McCance KL, Huether SE, Brashers VL, et al., editors: *Pathophysiology: the biologic basis for disease in adults and children,* ed 6, St. Louis, 2010, Mosby.)

In a small percentage of patients with chronically high carbon dioxide levels in blood, such as in patients with severe chronic obstructive pulmonary disease, the body must begin to use other mechanisms to regulate breathing. The hypoxic drive to breathe is a form of respiratory drive in which the body uses oxygen receptors instead of carbon dioxide receptors to regulate the respiratory cycle.[8] For these patients, oxygen supplementation must be prudently administered, as an increase in oxygen within blood (hyperoxemia) suppresses the hypoxic drive and results in a reduced drive to breathe.[8]

Lung receptors

There exist three types of receptors on the lung that send signals to the respiratory centers within the brainstem:

1. Irritant receptors: These receptors are found within the epithelial layer of the conducting airways and respond to various noxious gases, particulate matter, and irritants, causing them to initiate a cough reflex. When stimulated, these receptors also cause bronchial constriction and increase ventilatory rate.[5]
2. Stretch receptors: These receptors are located along the smooth muscles lining the airways and are sensitive to increasing size and volume within the lung.[6] Hering and Breuer discovered that ventilatory rate and volume was reduced following distention of anesthetized animal lungs. This stimulation of the ventilatory changes in response to increased volume and size is termed the Hering–Breuer reflex and is more active in newborns. In adults, this reflex is only active with large increases in the tidal volume, which is especially seen during exercise, and protects the lung from excessive inflation.[6]
3. J receptor: The juxtapulmonary receptors (J receptors) are located near the pulmonary capillaries and are sensitive to increased pulmonary capillary pressures. On stimulation, these receptors initiate a rapid, shallow

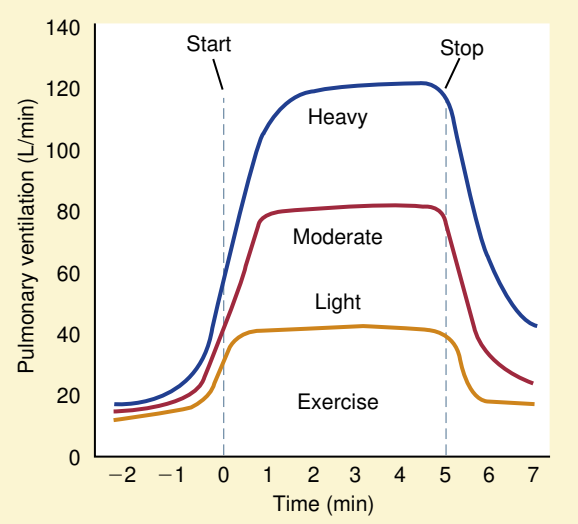

Figure 2.3 Ventilatory response during exercise.

breathing pattern.[7] In addition, the interstitial J receptors produce a cough reflex with fluid accumulation within the lung in patients with pulmonary edema and pleural effusions.

Clinical tip

In patients with acute left-side congestive heart failure and resultant pulmonary edema, the interstitial J receptors within the lung are stimulated. The firing of these receptors causes the patient to breathe in a shallow, tachypneic pattern. This breathing pattern causes a milking of the lymphatic vasculature to facilitate a removal of fluid out of the lungs.[9]

Joint and muscle receptors

Receptors within peripheral joints and muscles of the extremities respond to changes in movement and increase ventilation. During exercise, a twofold increase in minute ventilation is noted—an initial abrupt increase in ventilation followed by a secondary gradual increase in ventilation (Fig. 2.3).[10] The initial abrupt increase in ventilation is a result of sensory input conveyed from receptors within peripheral joints and muscles, whereas the secondary gradual increase in ventilation is a result of changes in pH within the blood caused by increased lactic acid production. This is conveyed to the brainstem by the chemoreceptors.

Mechanics of breathing

Movement of air into and out of the lungs occurs as a result of pressure differences between the two ends of the airway. Airflow through the conducting airway is directly proportional to the pressure difference created between the ends of the airway and inversely proportional to the resistance within the airway. In addition, ventilation is affected by various physical properties of the lungs, including compliance, elasticity, and surface tension. This section focuses on pressure changes that allow breathing to occur and explains how lung compliance, elasticity, and surface tension affect breathing. The physiologic importance for pulmonary surfactant is also discussed.

Intrapulmonary and atmospheric pressures

Inspiration is always an active process and involves contraction of the respiratory muscles. When the diaphragm and external intercostals contract they increase the volume of the thoracic cavity and lungs. This in turn causes a concomitant reduction in the intrapulmonary pressure, or pressure within the lung (Fig. 2.4).[11] The pressure within the lung is reduced in accordance with Boyle's law, which states that the pressure of a given quantity of gas is inversely proportional to its volume. During inspiration, intrapulmonary pressures decrease below atmospheric levels which is termed a subatmospheric or negative intrapulmonary pressure. This difference in pressure between the atmosphere and the lungs facilitates the flow of air into the lungs to normalize pressure differences. Conversely, expiration occurs when the intrapulmonary pressure exceeds the atmospheric pressure, allowing the lungs to recoil inward and expel air into the atmosphere.

There exists a primary difference between normal ventilation and mechanical ventilation. In normal ventilation, air is pulled into the lungs because of a negative pressure created through activation of the respiratory muscles. Patients placed on mechanical ventilation lack the ability to generate an effective negative or subatmospheric pressure. In light of this, the mechanical ventilator forces air into the lungs through creation of a positive pressure greater than the atmospheric pressure that exists within the lung.

Note that patients on mechanical ventilation often demonstrate reduced strength of the inspiratory muscles (including the diaphragm), as the ventilator assists with breathing. These patients may benefit from breathing exercises, positioning, and the use of an inspiratory muscle trainer to improve functioning of the inspiratory muscles.

Intrapleural and transmural pressures

Two layers cover each lung, including the outer parietal pleura and inner visceral pleura, separated by an intrapleural space containing a thin layer of viscous fluid. A small amount of viscous fluid within the intrapleural space serves as a lubricant and allows for the lungs to slide relative to the chest during breathing. With ventilation, there exist two opposing forces, including an inward pull from the elastic tension of the lung tissue trying to collapse the lung and an outward pull of the thoracic wall trying to expand the lungs.[1,2,5] These two opposing forces give rise to a subatmospheric (negative) pressure within the intrapleural space, termed the intrapleural pressure. This intrapleural pressure is normally lower than the intrapulmonary pressure developed during both inspiration and expiration. In light of these two pressure differences, a transpulmonary or transmural pressure is developed across the wall of the lung.[1,2,5] The inner intrapulmonary pressure is relatively greater than the outer intrapleural pressure, allowing the difference in pressure (the transmural pressure) to maintain the lung near the chest wall. It is the transmural or transpulmonary pressure that allows changes in lung volume to parallel changes in thoracic excursion during inspiration and expiration.

When changes in lung volume do not parallel the normal outward and inward pull during inspiration and expiration and are, in fact, opposite, the breathing pattern is said to be paradoxical. This breathing pattern is often seen in patients with multiple rib fractures and a resultant flail chest.

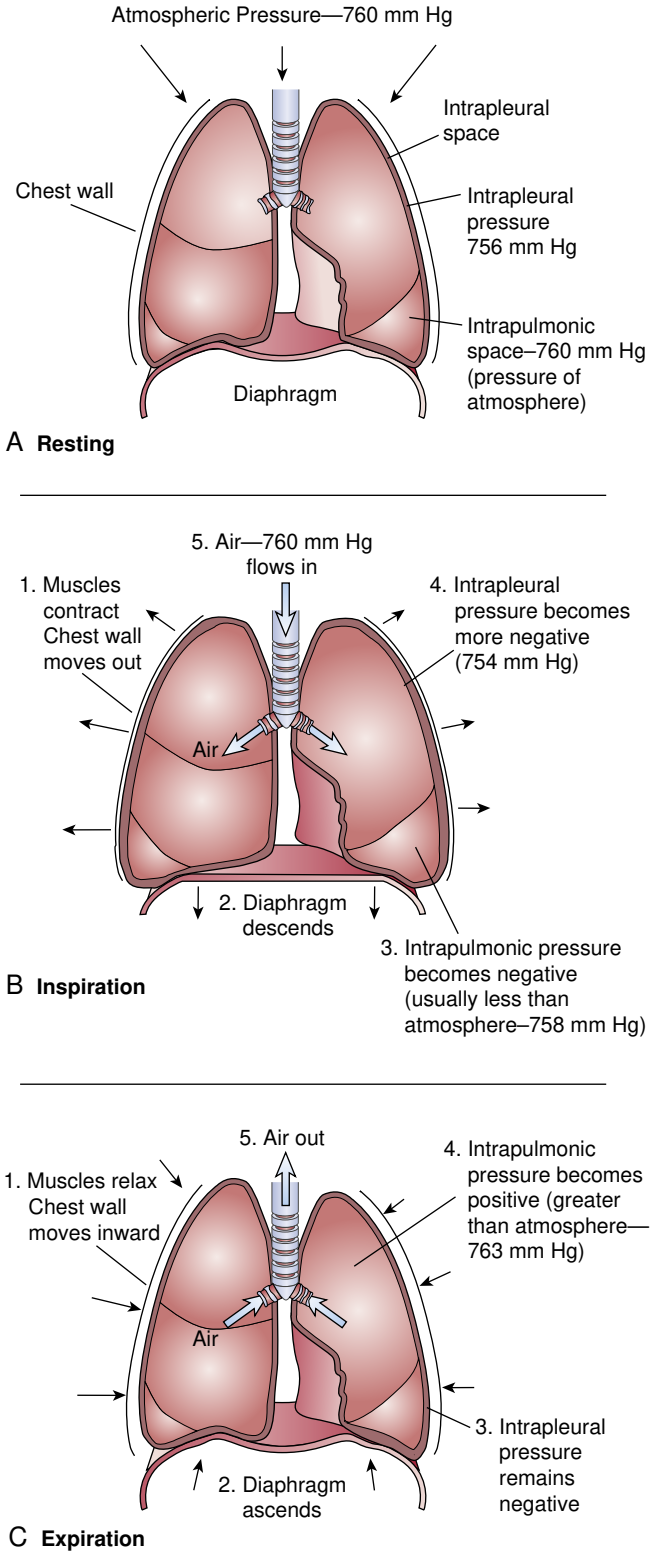

Figure 2.4 (A–C) Ventilation: changes in pressure with inspiration and expiration. (From Gould BE: *Pathophysiology for the health professions*, ed 3, St. Louis, 2007, Saunders.)

Physical properties of lungs

The processes of inspiration and expiration are facilitated by three physical properties of lung tissue. Compliance allows lung tissue to stretch during inspiration; the elastic recoil of the lung allows passive expiration to occur; and surface tension forces with the alveoli allow the lung to get smaller during expiration.

Compliance

The lung can be compared with a balloon during inspiration, where there exists a tendency to collapse or recoil while inflated. To maintain inflation, the transmural pressure, or pressure difference between the intrapulmonary pressure and intrapleural pressure, must be maintained. A distending force is needed to overcome the inward recoil forces of the lung. This outward force is provided by the elastic properties of the lung and through the action of the inspiratory muscles.

Compliance describes the distensibility of lung tissue. It is defined as the change in lung volume per change in transmural or transpulmonary pressure, expressed symbolically as $\Delta V/\Delta P$ (Fig. 2.5).[12] In other words, a given transpulmonary pressure will cause a greater or lesser degree of lung expansion, depending on the distensibility or compliance of the lung. The total compliance of both lungs together in the normal adult human averages about 200 milliliters of air per centimeter of water transpulmonary pressure. That is, every time the transpulmonary pressure increases 1 centimeter of water, the lung volume, after 10 to 20 seconds, will expand 200 milliliters. The compliance of the lung is reduced by factors that produce a resistance to distension. Also the compliance is reduced as the lung approaches its TLC, where it becomes relatively stiffer and less distensible.

In patients with emphysema, the chronicity of the disease leads to progressive destruction of the elastic recoil, making the compliance high.[13] A reduced inward pull from low recoil allows small changes in transmural pressure to cause large changes in lung volumes and resultant hyperinflation of the lung. The changes seen in individuals with emphysema include a barrel chest and flattened diaphragms. These negative sequelae result in less diaphragm use with breathing, more accessory muscle, and an increase in the work of breathing.

In patients with pulmonary fibrosis, the lung is fibrotic and stiff and thereby has reduced compliance.[13] In these patients, despite large changes in transmural pressure, only small changes in lung volume will occur as a result of the stiffness or lack of distensibility of lung tissue. Consequently, in the clinical setting, one sees these individuals with increased respiratory rates and accessory muscle use because of decreased lung volumes. The work of breathing with activity is greatly increased as a consequence of the inability to increase lung volumes.

Elasticity

Elasticity refers to the tendency of a structure to return to its initial size after being distended. A network of elastin and collagen fibers within the alveolar wall and surrounding bronchi and pulmonary capillaries provides for the elastic properties of the lung.

Surface tension

Although the elastic characteristics of the lung tissue itself play a role in resisting lung distension or compliance, the surface tension at the air–liquid interface on the alveolar surface has a greater influence. Anyone who has ever attempted to separate two wet microscope slides by lifting (not sliding) the top slide from the bottom has firsthand experience with the forces of surface tension. In the lung,

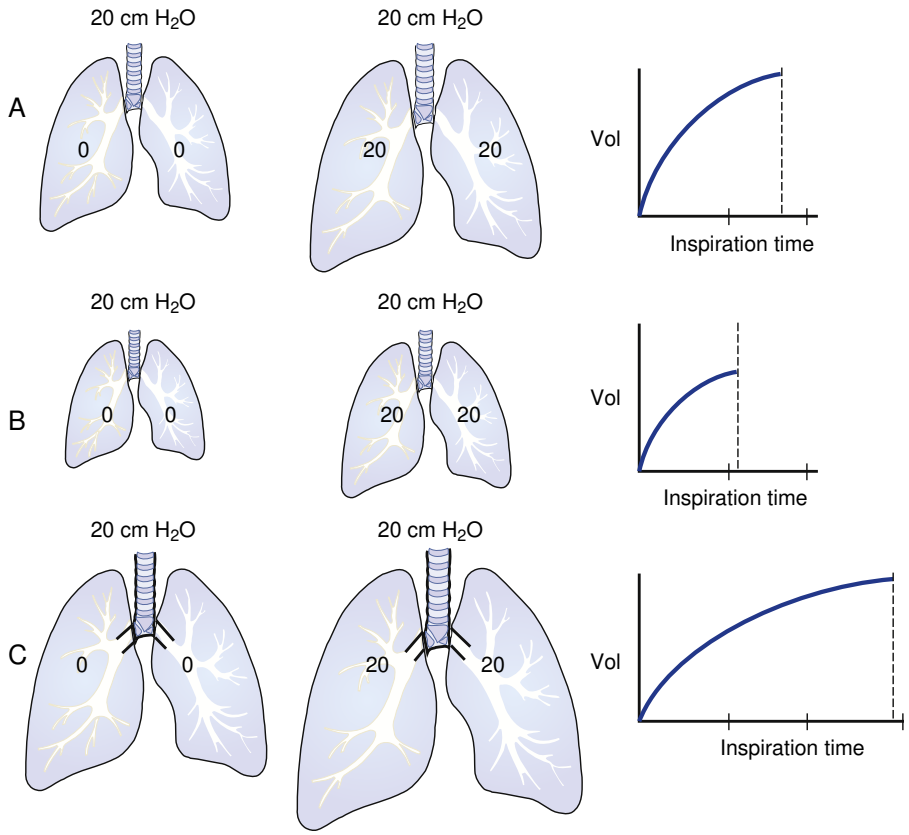

Figure 2.5 (A–C) Lung compliance changes associated with disease. (A) Normal lung; (B) depicts restrictive lung disease; (C) depicts obstructive lung disease.

a thin film of fluid on the alveolus has a surface tension, which is caused by water molecules at the surface being relatively more attracted to other water molecules than to air. This surface tension acts to collapse the alveolus and increase the pressure of air within the alveolus. The law of Laplace states that the pressure created within the alveolus is directly proportional to the surface tension and inversely proportional to the radius of the alveolus.

For example, consider two alveoli of different sizes, one at either end of a bifurcated respiratory bronchiole (Fig. 2.6); because of the size difference, the smaller alveolus must have a higher pressure than the larger alveolus if the surface tension of each is the same. To keep the air in the smaller alveolus from emptying into the larger, a surface-active agent is needed to decrease the overall surface tension of the alveoli so as to lower wall tension in proportion to the radius of the alveolus (see Fig. 2.6). Moreover, it must do so almost in anticipation of diminishing alveolar size. Only if such a surface-active agent were present could alveoli with different radii coexist in the lungs.

The surface-active agent in the human lung that performs this function is called surfactant.[14,15] Pulmonary surfactant is not composed of a single class of molecules, but, rather, is a collection of interrelated macromolecular lipoprotein complexes that differ in composition, structure, and function.[14,15] Nonetheless, the principal active ingredient of surfactant is dipalmitoyl phosphatidylcholine. The structure of the surfactant molecule is such that it presents a nonpolar end of fatty acids (two palmitate residues) that is insoluble in water, and a smaller, polar end (a phosphatidylcholine group) that dissolves readily in water. Thus surfactant orients itself perpendicularly to the surface in the alveolar fluid layer, with its nonpolar end projecting toward the lumen. If surfactant were uniformly dispersed throughout the alveoli, its concentration at the air–fluid interface would vary in accordance with the surface area of any individual alveolus. Thus the molecules would be compressed in the smaller alveoli, as depicted in Fig. 2.6. Compressing the surfactant molecules increases their density and builds up a film pressure that counteracts much of the surface tension at the air–fluid interface. The rate of change in the surface tension resulting from compression of the surfactant molecules as the alveolus gets smaller is faster than the rate of change of the decreasing alveolar radius, so that a point is rapidly reached in which the pressure in the small alveolus equals the pressure in the big alveolus.

Surfactant begins to develop in late fetal life. Premature babies may be born with less surfactant, resulting in collapsed alveoli and respiratory distress. In an effort to reduce complications in women likely to go into premature labor (<7 months' gestation), the fetus is injected with amniotic fluid from the placenta of an infant born through cesarean section to mature the lungs of the fetus. Note that even under normal circumstances, the first breath of life is more challenging because the newborn must overcome greater surface tension forces to inflate its partially collapsed alveoli.

Resistance to airflow

The ability to inflate the lungs with air depends on pressure differences and resistance to flow within the airways.[2,5] Poiseuille's law states that flow through a vessel or airway is directly proportional to the pressure difference and radius and inversely proportional to the length of the airway and viscosity of the gas. In addition, note that the radius is raised to

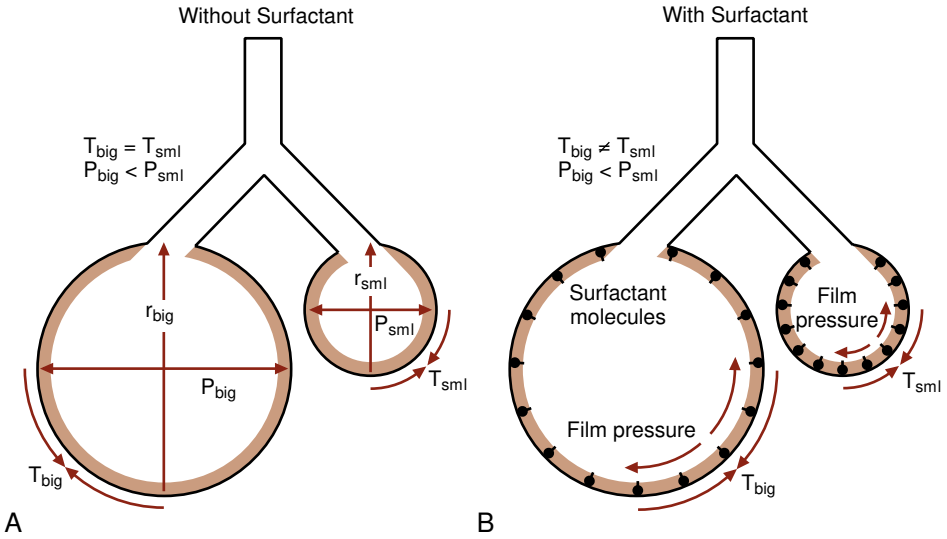

Figure 2.6 Two pairs of unequally filled alveoli arranged in parallel illustrate the effect of a surface-active agent. One pair of alveoli is shown without surfactant (A) and the other is shown with surfactant (B). In the alveoli without surfactant, if T_{sml} were the same as T_{big}, P_{sml} would have to be many times greater than P_{big}; otherwise, the smaller alveolus would empty into the larger one. In the alveoli with surfactant, T_{sml} is reduced in proportion to the radius of the alveolus, which permits P_{sml} to equal P_{big}. Thus alveoli of different radii can coexist. Refer to the text for details. *P,* Pressure; *T,* temperature.

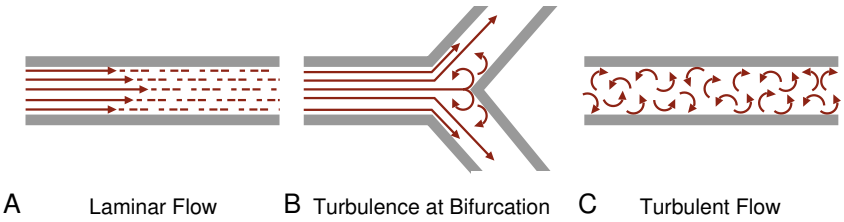

Figure 2.7 Laminar and turbulent airflow in the airways. (A) At low flow rates, air flows in a laminar pattern, and the resistance to airflow is proportional to the flow rate. (B) At airway bifurcation, eddy formation creates a transitional flow pattern. (C) At high flow rates, when a great deal of turbulence is created, the resistance to airflow is proportional to the square of the flow rate. (Modified from West JB: *Respiratory physiology: the essentials*, ed 9, Baltimore, 2012, Lippincott Williams & Wilkins.)

the fourth power, and so small changes in the radius account for large changes in airflow through the airway.[5,7]

The upper airways are responsible for most of the airway resistance. The lower airways play a much smaller role in influencing airway resistance because of the irregularity of the branching patterns, as well as variations in the diameter of the lumen of the distal airway.[6] In addition, resistance in smaller airways is lower because flow is laminar. This involves only slight resistance between the sides of the airway and resistance caused by collision of air molecules. In the upper airway, airflow has a relatively high turbulence involving increased resistance as a result of frequent molecular collisions in addition to the resistance along the sides of the tube (Fig. 2.7).

Resistance to airflow is also affected by the diameter of the airway, which is influenced by changes in the transmural pressure within the lung during ventilation. With inspiration, the pressure outside the airway within the lung (transmural pressure) is relatively more negative than the airway pressure, thereby increasing the radius of the airway and reducing resistance to airflow. Conversely, with expiration, the transmural pressure is greater than the airway pressure, reducing the radius and increasing airway resistance.[2]

Finally, resistance to airflow may also be affected by autonomic nervous system control. Increases in parasympathetic nervous system activation cause constriction of the smooth muscle cells of the bronchi and increase airway resistance, whereas sympathetic influence decreases airway resistance. Also mucus and edema in the airway as a consequence of inflammation increase airway resistance and reduce airflow.[1,2]

Status asthmaticus, an acute asthma attack, is marked by severe airway resistance caused by constriction of bronchial smooth muscle cells and mucus production within the airway (Fig. 2.8). These patients use accessory muscles to increase transmural pressures to increase airway radius. In addition, they benefit from bronchodilators to relieve smooth muscle cell constriction and from steroids to reduce the inflammatory process and mucus production.

Respiration

Respiration refers to the process of gas exchange in the lungs facilitated through the process of simple diffusion. This process serves two major functions, including the replenishment of the blood's oxygen supply used for oxidative energy production and the removal of carbon dioxide returning from venous blood manufactured as a waste product. For diffusion to occur, there are two requirements: air bringing in oxygen to the lungs (alveolar ventilation) and blood to receive the oxygen and give up carbon dioxide (pulmonary perfusion). Air is delivered to the distal alveolus for gas exchange via the process of pulmonary ventilation; blood is brought to the lungs from the right side of the heart through the pulmonary artery

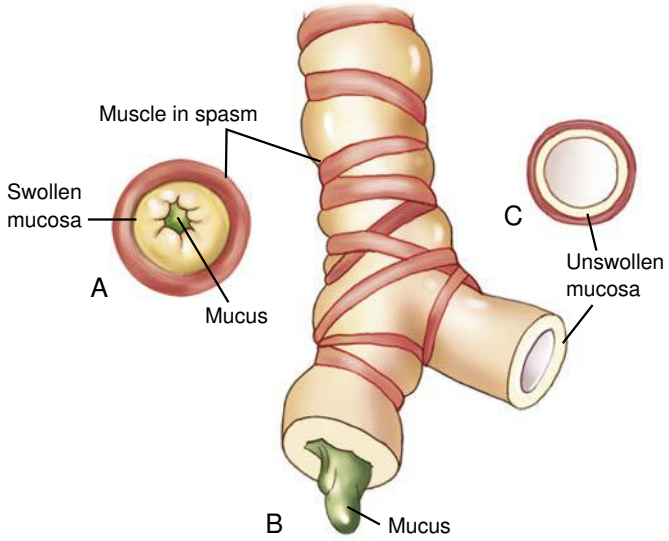

Figure 2.8 Factors causing expiratory obstruction in asthma. (A) Cross-section of a bronchiole occluded by muscle spasm, swollen mucosa, and mucus. (B) Longitudinal section of an obstructed bronchiole. (C) Cross-section of a clear bronchiole. (From Shiland BJ: *Mastering healthcare terminology*, ed 3, St. Louis, 2010, Mosby.)

and branching pulmonary capillaries. Gas exchange or respiration between the alveoli and pulmonary capillary occurs across the semipermeable alveolar–capillary membrane, also referred to as the respiratory membrane.

Partial pressures of gases

The air delivered to distal alveoli is a mixture of gases. It is important to understand that each gas exerts its own pressure in proportion to its concentration in the gas mixture. The amount of individual pressure exerted by each gas within the mixture is termed the partial pressure of that individual gas. According to Dalton's law, the total pressure of a mixture of gases equals the sum of the individual gases within the mixture. Atmospheric air is a mixture of gases containing 79.04% nitrogen, 20.93% oxygen, and 0.03% carbon dioxide.[7] At sea level, the barometric or atmospheric pressure is 760 mm Hg. This is considered the total pressure of the mixture of the three gases present in the atmosphere. Therefore the partial pressure of nitrogen in the atmosphere is 600 mm Hg (79.04% of 760), of oxygen is 159 mm Hg (20.83% of 760), and of carbon dioxide is 0.2 mm Hg (0.03% of 760). Once gases enter the body, Henry's law explains how gases in the body are dissolved in fluids. According to this law, gases dissolve in liquids in proportion to their partial pressure.[16]

Diffusion

To allow for effective gas exchange to occur between the alveoli and pulmonary capillary, different partial pressures of oxygen and carbon dioxide must exist in each of the two areas. The differences in partial pressure of each gas within the alveoli and pulmonary capillary create a pressure gradient across the alveolar capillary interface. This gradient will enable gases to diffuse from areas of high concentration to areas of low concentration across the semipermeable respiratory membrane.

Clinical tip

The diffusing capacity of lung for carbon monoxide (DLCO) is a test of the integrity of the alveolar–capillary surface area for gas transfer.[13] It may be reduced in disorders that damage the alveolar walls (septa), such as emphysema, leading to a loss of effective surface area. The DLCO is also reduced in disorders that thicken or damage the alveolar walls, such as pulmonary fibrosis.

Perfusion

Perfusion refers to blood flow to the lungs available for gas exchange. The driving pressure in the pulmonary circulation is much less than the systemic circulation, yet flow rates in both circulation systems are similar because of reduced vascular resistance within the pulmonary circulation. Thus the pulmonary circulation system is considered a low-resistance, low-pressure pathway. The low pressure allows for relatively lower filtration pressures compared with the systemic vascular system, thereby protecting the lung from pulmonary edema, a dangerous condition where fluid accumulates in the lung, hindering alveolar ventilation and gas exchange.

In addition, perfusion of the lung is affected by alterations in the partial pressures of oxygen within the alveoli.[16] Pulmonary arterioles constrict when partial pressures of oxygen in alveoli are low and dilate when alveolar partial pressures for oxygen increase. These vasomotor changes of the pulmonary and systemic vasculature help to reduce blood flow to areas in the lung that are poorly ventilated and increase blood flow to peripheral tissue that needs more oxygen. The vasoconstriction of the pulmonary vasculature to low oxygen levels is automatic and improves the ability for gas exchange. This phenomenon prevents blood from poorly ventilated alveoli (with low partial pressures of oxygen) from mixing with blood from well-ventilated alveoli (with relatively higher partial pressures of oxygen). If blood did have to mix, then the overall oxygen concentration for blood leaving the lungs and returning to the heart would be lower because of the dilution effect.

Finally, alterations in the pH of blood affect vasomotor tone of the pulmonary vasculature, thereby affecting perfusion required for gas exchange.[6] A low pH, or acidemia, causes pulmonary vasoconstriction. The lung is significantly involved in regulating the acid–base balance in blood. When the pH of blood is reduced as a result of lung pathology, vasoconstriction of the pulmonary vessels is potentiated in response to the altered pH, which, in turn, affects the gas exchange and exacerbates the problem to a higher degree.

Ventilation and perfusion matching

For optimal respiration or gas exchange to occur, the distribution of gas (ventilation [V]) and blood (perfusion [Q]) at the level of the alveolar capillary interface must be matched. Position plays a vital role in the distribution of V/Q to different aspects of the lung. In the upright position, gravity allows for a greater amount of blood flow or perfusion to the base of the lung relative to the apices.[16] In addition, alveoli in the upper portions or apices of the lung have greater RV of gas and are subsequently larger.[16] The larger alveoli have greater surface tension and have relatively more difficulty inflating because of less compliance than the smaller alveoli toward the base

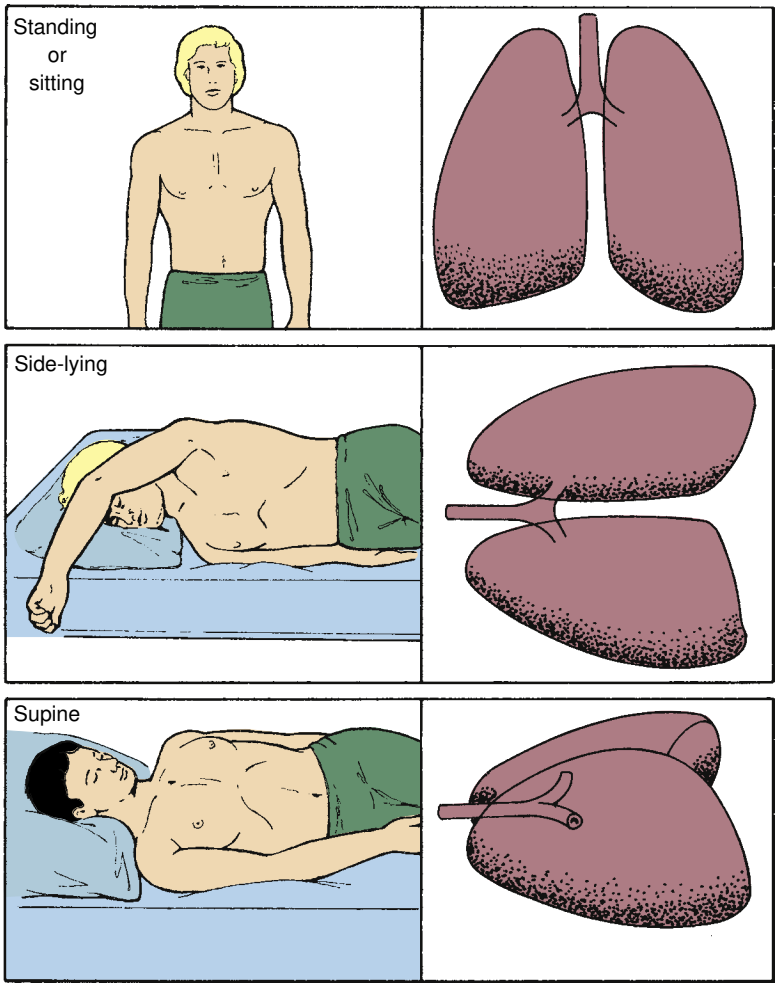

Figure 2.9 Pulmonary blood flow and gravity. The greatest volume of pulmonary blood flow will normally occur in the gravity-dependent areas of the lungs. Body position has a significant effect on the distribution of pulmonary blood flow. (From McCance KL, Huether SE, Brashers VL, et al., editors: *Pathophysiology: the biologic basis for disease in adults and children,* ed 6, St. Louis, 2010, Mosby.)

of the lung. In light of this, V/Q is relatively greater toward the base of the lung, favoring better matching and resultant respiration or gas exchange.[16] A change in the position of the patient changes areas of V/Q. In general, greater perfusion occurs, and greater ventilation in the upper lung. Therefore to improve ventilation to the posterior bases, one must position an individual prone (Fig. 2.9).

Often, V/Q ratios are not uniform within the lung, which compromises gas exchange. Regions of the lung with relatively greater amounts of perfusion compared with ventilation act as shunts. Conversely, regions of the lung with relatively greater amounts of ventilation compared with perfusion act as dead space. Alterations in the V/Q matching lead to hypoxia and reduced oxygen to peripheral tissue.

An effective noninvasive tool to measure respiration is the pulse oximeter. It is important for clinicians to monitor pulse oximeter readings and observe for signs of distress when changing patient positions that alter the V/Q matching. Abnormal V/Q ratios cause concomitant reductions in pulse oximetry that are noted in patients with pneumonia, pulmonary embolus, edema, emphysema, bronchitis, acute respiratory distress syndrome (ARDS), and other pulmonary disorders.

In patients with ARDS, for example, prone positioning has been an effective way to enhance V/Q matching, effectively improving oxygenation and decreasing mortality. Fig. 2.10 demonstrates the changes in alveolar aeration and secretion mobilization with prone positioning.

Transport of oxygen and carbon dioxide

Following the previous discussions of how air is brought into the lungs through the process of pulmonary ventilation and exchanged within the lungs via respiration, it is important to consider the mechanisms for the delivery of oxygen to peripheral tissue and the removal of carbon dioxide that the tissue produces as a waste product. This section reviews transport of each gas individually.

Transport of oxygen

A majority of oxygen (98%) is transported to the peripheral tissue bound to hemoglobin within red blood cells of blood. A very small portion of oxygen (<2%) is dissolved in plasma within blood.

Hemoglobin

A hemoglobin molecule consists of four protein chains called globins and four iron-containing organic molecules called hemes.[2,5] The protein component of the molecule contains two identical α and two identical β protein chains. The α

Figure 2.10 (A) Team performing manual prone positioning. (B) Patient in prone position. (A, From Stryker, Kalamazoo, MI. In: Dickinson S, Park PK, Napolitano LM: Prone-positioning therapy in ARDS. *Crit Care Clin* 27[3]:511–523, 2011, Copyright © 2011 Elsevier Inc. (B) From Benson AB, Albert RK: Prone positioning for acute respiratory distress syndrome. *Clin Chest Med* 35[4]:743–775, 2014.)

chains contain 141 amino acids, and the β chains contain 146 amino acids.[7] The four-polypeptide chains are connected to each heme molecule. Each heme molecule has a central iron atom that can combine with a single molecule of oxygen. Consequently, as a result of the presence of four heme molecules, one hemoglobin has the ability to carry four oxygen molecules to the peripheral tissue.

Hemoglobin molecules within blood can exist in one of four conditions, depending on the molecule that binds to, unloads from, or is unable to bind to the iron atom within heme. Oxyhemoglobin represents a hemoglobin molecule bound to oxygen, because iron in heme is in its reduced state. Deoxyhemoglobin refers to the oxyhemoglobin molecule that has released its oxygen molecule to peripheral tissue. Because methemoglobin has iron in its oxidized state, it is unable to bind to oxygen and participate in oxygen transport. Blood contains a very small amount of this molecule. Carboxyhemoglobin is another abnormal form of hemoglobin; it involves the binding of heme to carbon monoxide instead of oxygen. Because the bond with carbon monoxide is 210 times stronger than oxygen, it displaces oxygen and inhibits oxygen's binding capacity.

Clinical tip

Carbon monoxide poisoning results from smoke inhalation and suicide attempts in its severe form and is treated by placing patients in a hyperbaric oxygen environment. This strategy enables the patient to breathe 100% oxygen at 2 to 3 atmospheres pressure. In a milder form, carboxyhemoglobin is generated in blood from breathing smoggy air and through chronic cigarette smoking.

The percentage of oxyhemoglobin to total hemoglobin provides an indication of how well the blood has been oxygenated by the lungs. This is termed the percent oxyhemoglobin saturation. In the systemic arteries, at a partial pressure of 100 mm Hg, the percent hemoglobin is 97%, indicating that 97% of hemoglobin molecules in blood are bound to oxygen. The remaining 3% reflects deoxyhemoglobin, methemoglobin, and carboxyhemoglobin concentrations. The gold standard for measuring oxyhemoglobin saturation is through an analysis of arterial blood gases (ABGs). However, pulse oximeter can also be used to obtain this number.

The oxygen-carrying capacity of the body is determined by the concentration of hemoglobin. Normal levels of hemoglobin are between 12 and 16 g/dL for women and 13 and 18 g/dL for men.[17] A below-normal level of hemoglobin occurs with anemia and compromises the ability to carry oxygen. Conversely, an increase in hemoglobin concentrations, a condition called *polycythemia*, increases oxygen-carrying capacity within the system.

Clinical tip

In patients with chronic lung disease, there may be a compensatory increase in red blood cells, termed secondary polycythemia, to compensate for chronically low levels of oxygen in blood.

Oxyhemoglobin dissociation curve

The oxyhemoglobin dissociation curve (Fig. 2.11) describes the relation between the amount of O_2 bound to hemoglobin (Hb), clinically referred to as the percentage of saturation of hemoglobin, and the partial pressure of O_2 (PO_2) with which the Hb is in equilibrium.[18–20] Under ideal conditions (blood pH = 7.4, body temperature = 37°C, Hb = 147 g/L), less than 10% of the O_2 dissociates from the Hb as PO_2 falls 40 mm Hg from 100 to 60 mm Hg. However, nearly 60% of the O_2 is dissociated from the Hb as PO_2 falls another 40 mm Hg from 60 to 20 mm Hg. Decreasing the pH (increasing acidemia) of the blood from the normal value of 7.40 to 7.30 shifts the hemoglobin dissociation curve downward and to the right an average of 7% to 8%; in contrast, alkalemia shifts the curve to the left. Increasing the concentration of CO_2 in the tissue capillary beds displaces oxygen from the hemoglobin, delivering the O_2 to the tissues at a higher PO_2 than would otherwise occur. In conditions of prolonged hypoxemia (lasting longer than a few hours), the amount of 2,3-diphosphoglycerate in the blood is increased, resulting in a rightward shift in the hemoglobin dissociation curve. 2,3-Bisphosphoglycerate is present in human red blood cells and binds with greater affinity to deoxygenated hemoglobin than it does to oxygenated hemoglobin. In bonding to partially deoxygenated hemoglobin, it allosterically upregulates the release of the remaining oxygen molecules bound

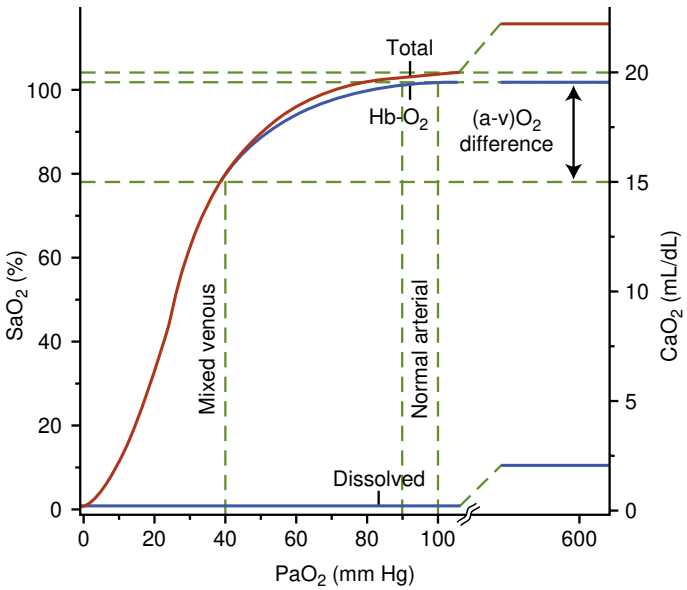

Figure 2.11 The oxyhemoglobin dissociation curve. Note that in the "flat" portion of the curve (80 mm Hg and above), a change in the partial pressure of arterial oxygen (PaO_2) of as much as 20 mm Hg does not appreciably alter the hemoglobin saturation. However, in the "steep" portion of the curve (<60 mm Hg), relatively small changes in saturation result in large changes in the PaO_2.

to the hemoglobin, thus enhancing the ability of red blood cells to release oxygen near tissues that need it most. Furthermore, increasing the temperature of the tissue, as happens normally in exercising muscle, also results in a shift of the hemoglobin dissociation curve to the right. The result of these rightward shifts is a decreased hemoglobin affinity for oxygen. Although a rightward shift in the hemoglobin dissociation curve can be beneficial, the reader is cautioned that the range of variability normally tolerated by the body is relatively narrow: rapid fluctuations in pH or core temperature are not at all well tolerated.

Carbon dioxide transport

Carbon dioxide released from metabolically active cells is carried by blood in one of three ways:

1. Dissolved in plasma
2. Bound to the protein component of hemoglobin (carbaminohemoglobin)
3. As bicarbonate ion

Dissolved carbon dioxide

Carbon dioxide released from tissue may get dissolved in blood plasma and transported through the system. A very small percentage of carbon dioxide, approximately only 7% to 10%, is transported in this manner.

Carbaminohemoglobin

Carbon dioxide can also be transported by binding to the hemoglobin molecule in blood. The carbon dioxide molecule binds to the protein chains rather than the heme component of the hemoglobin molecule. The complex formed from the binding of carbon dioxide and hemoglobin is termed carbaminohemoglobin. About one-fifth of the total blood carbon dioxide is carried in this manner.

Bicarbonate ions and the chloride and reverse chloride shifts

The majority of carbon dioxide combines with water to form a compound called carbonic acid. This reaction is facilitated through the action of the carbonic anhydrase enzyme under conditions of high partial pressure of carbon dioxide at the level of the tissue.[2,7] This enzyme is confined to the red blood cell, thereby allowing most of the carbonic acid to be produced within the red blood cell. A small amount of carbonic acid is also produced spontaneously within the plasma of blood.

$$CO_2 + H_2O - \text{carbonic anhydrase} \rightarrow H_2CO_3$$

Carbonic acid that is built up within the red blood cell dissociates into positively charged hydrogen ions (protons) and negatively charged bicarbonate ions.

$$H_2CO_3 \rightarrow H^+ + HCO_3^-$$

The hydrogen ions released from carbonic acid combine with deoxyhemoglobin molecules within red blood cells. As a result, fewer hydrogen ions move out of red blood cells, causing the negatively charged bicarbonate ions to leak out of the blood cell into plasma. The trapping of hydrogen ions within the red blood cell results in a net positive charge within the blood cell and a compensatory shift of negative chloride ions into the red blood cell as bicarbonate moves out. This exchange of anions as blood travels through tissue capillaries is termed the chloride shift (Fig. 2.12).

It is important to appreciate that deoxyhemoglobin bonds more strongly to hydrogen ions than to oxyhemoglobin. In light of this, the unloading of oxygen to peripheral tissue is increased (the Bohr effect) as more hydrogen ions are released from carbonic acid to produce a greater amount of deoxyhemoglobin for the hydrogen ions to bind. In summary, increased carbon dioxide production increases oxygen unloading into the tissue, which, in turn, improves carbon dioxide transport out of the tissue.

At the level of the lung, deoxyhemoglobin is converted to oxyhemoglobin. As mentioned earlier, oxyhemoglobin has a weaker affinity for hydrogen ions. With the partial pressure of oxygen being high at the level of the lung, free hydrogen ions are released from hemoglobin within the red blood cell. The free hydrogen ions attract bicarbonate ions from the plasma and join to form carbonic acid.

$$H^+ + HCO_3^- \rightarrow H_2CO_3$$

Under conditions of low partial pressures of carbon dioxide within the lung, carbonic anhydrase facilitates the breakdown of carbonic acid into carbon dioxide and water. The carbon dioxide is then released from the lungs through the process of expiration.

$$H_2CO_3 - \text{carbonic anhydrase} \rightarrow CO_2 + H_2O$$

A reverse chloride shift occurs at the level of the lungs to facilitate the entry of bicarbonate into the red blood cell. As bicarbonate ions leave the plasma to enter the blood cell, chloride ions shift out of the red blood cell and enter the plasma. These processes are vital in maintaining acid–base regulation and normal pH of blood.

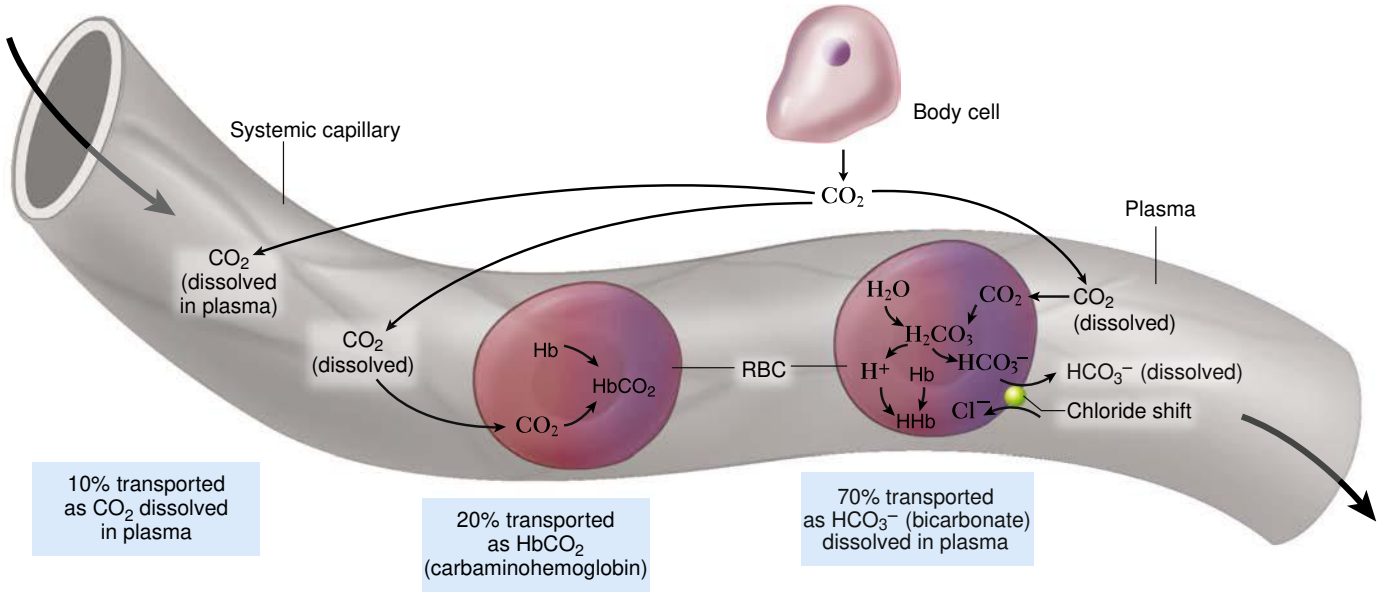

Figure 2.12 Transport of carbon dioxide and the chloride shift.

Acid–base balance

Metabolically produced acids are largely eliminated from the body via the lungs in the form of CO_2 because the major blood acid, carbonic acid (H_2CO_3), is volatile; that is, it can chemically vary between a liquid and gaseous state. The other blood acids (dietary acids, lactic acids, and ketoacids) are regulated by the kidneys and the liver. The measurement of arterial oxygen or carbon dioxide tension and hydrogen ion concentration for assessment of acid–base balance and oxygenation status are commonly accomplished by means of laboratory analysis of ABGs. In general, an ABG report contains the pH, the partial pressure of arterial carbon dioxide ($PaCO_2$), the partial pressure of arterial oxygen (PaO_2), and the HCO_3^-, and base excess (BE) values for the sample analyzed. Chapter 12 provides a detailed discussion of acid–base balance and arterial blood gases.

The cardiovascular system

The primary function of the cardiovascular (circulatory) system is the transportation and distribution of essential substances to the tissues of the body and the removal of the byproducts of cellular metabolism (Fig. 2.13). The heart provides the principal force that pushes blood through the vessels of the pulmonary and systemic circuits. In the case of the systemic circuit, the forward movement of blood is also facilitated by the recoil of the arterial walls during diastole, skeletal muscle compression of veins during exercise, and changes to thoracic pressure during breathing.

The cardiac cycle

The period from the beginning of one heartbeat to the beginning of the next is called the cardiac cycle. Figs. 2.14 and 2.15 depict selected events during the cardiac cycle. Beginning with an action potential in the sinoatrial (SA) node, a depolarization wave is spread through both atria to the atrioventricular (AV) node and then, through the His–Purkinje complex, into the ventricles. However, because of the nature of the specialized conduction system, the impulse is delayed for about 0.1 second in the upper two-thirds of the AV node. This allows the atria to contract (a result of excitation–contraction coupling) and pump an additional volume of blood into the ventricles—an atrial "kick."[21] The ventricles then provide the primary force to move blood through the vascular system.

Clinical tip

In patients with atrial fibrillation and a resultant quivering of the atria, the atrial kick is lost, losing 15% to 20% of the cardiac output, especially when the heart rate increases.[22] When managing patients with atrial fibrillation and rapid ventricular rates, signs and symptoms of compromised cardiac output must be continually assessed during treatment.

The cardiac cycle may be further divided into two periods: systole and diastole. Systole is the period of ventricular contraction; diastole is the period of ventricular relaxation. Fig. 2.15 illustrates left-sided pressure and volume, electrocardiogram (ECG), and phonocardiographic events associated with the cardiac cycle. Closure of the tricuspid and mitral valves generates the first heart sound (S_1), signaling the onset of ventricular systole, and is shown on the phonocardiographic tracing just after the peak of the R wave on the ECG tracing. In early ventricular systole, the ventricular volume remains unchanged despite a rapid rise in ventricular pressure. This isovolumic contraction occurs until the aortic valve opens, at which time the ventricular ejection phase begins. The retrograde bulging of the mitral valve into the left atrium is responsible for the rise in atrial pressure seen during the isovolumic ventricular contraction, the c wave. Ventricular ejection continues until the aortic valve closes, terminating systole and generating the second heart sound (S_2). Immediately following aortic valve closure, there is a phase of isovolumic relaxation that continues until the mitral valve opens

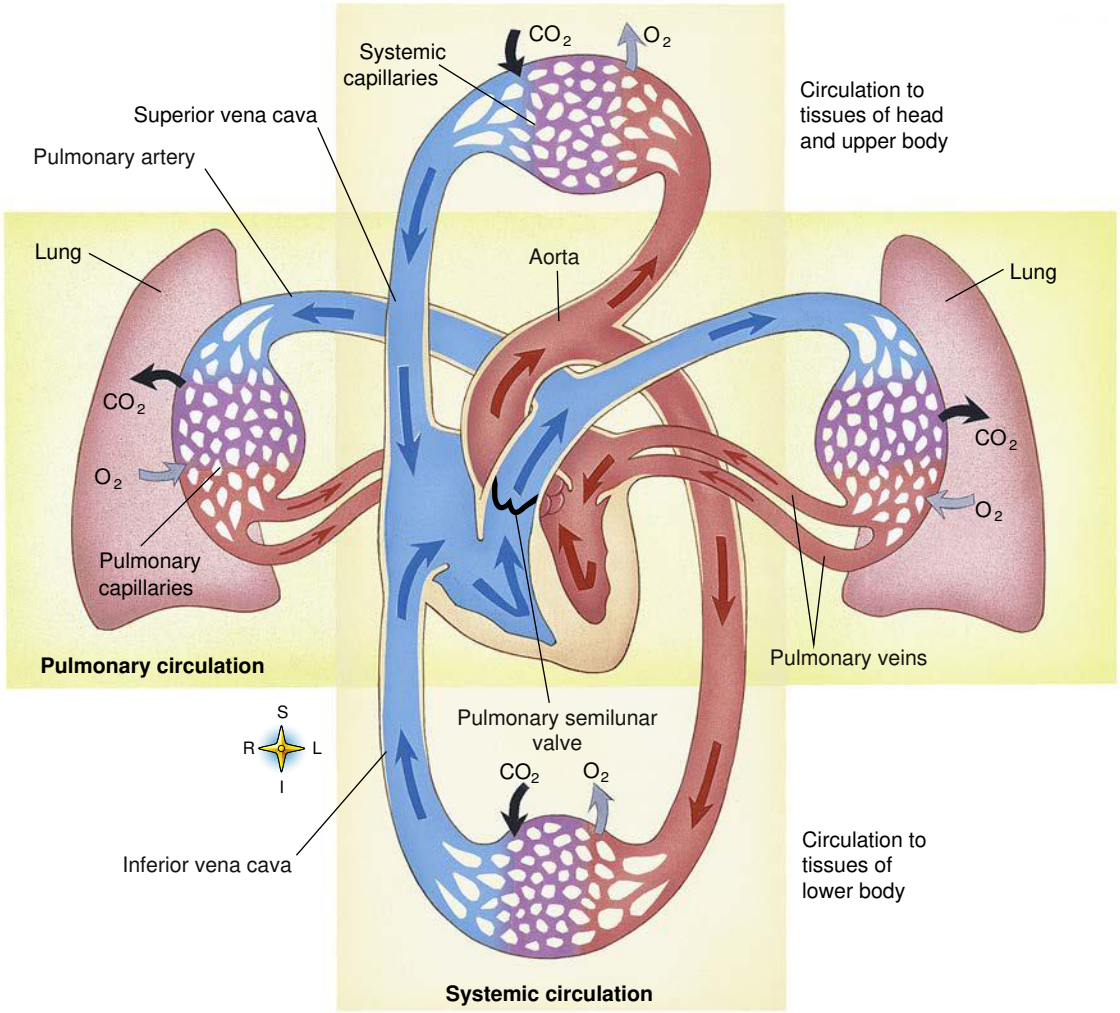

Figure 2.13 Blood flow through the circulatory system. In the pulmonary circulatory route, blood is pumped from the right side of the heart to the gas-exchange tissues of the lungs. In the systemic circulation, blood is pumped from the left side of the heart to all other tissues of the body. (From Thibodeau GA: *The human body in health & disease,* ed 4, St. Louis, 2006, Mosby.)

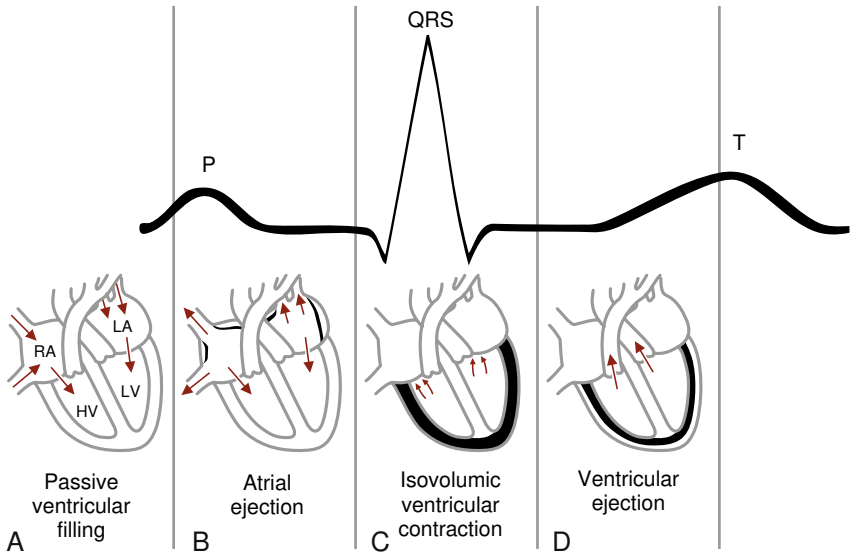

Figure 2.14 The mechanical events of the cardiac cycle shown in relation to the electrical events of the electrocardiogram. In late diastole, just before the P wave, the ventricles fill passively (A). At about the time that the P wave ends, the atria contract to eject up to 20% of the end-diastolic ventricular volume (B). A period of isovolumic ventricular contraction begins very shortly after the onset of the QRS complex (C). Ventricular ejection coincides with the early portion of the ST segment (D).

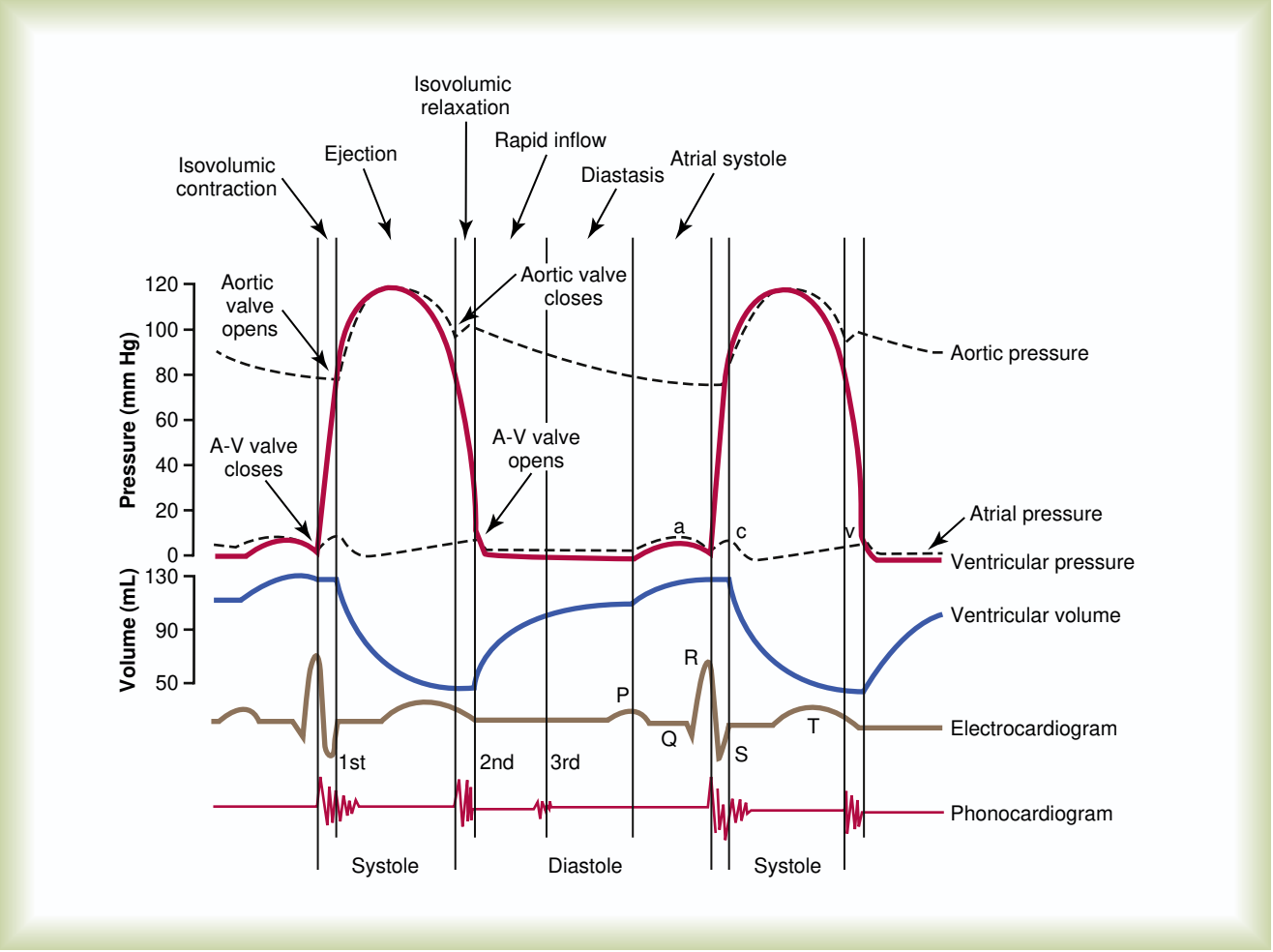

Figure 2.15 Events of the cardiac cycle for left ventricular function, showing changes in left atrial pressure, left ventricular pressure, aortic pressure, ventricular volume, the electrocardiogram, and the phonocardiogram. (From Guyton AC, Hall JE: *Textbook of medical physiology,* ed 11, St. Louis, 2006, Saunders.)

when ventricular pressure falls below atrial pressure. The rise in atrial pressure indicated by the v wave of the atrial pressure tracing is probably brought about by the relative negative pressure resulting from ventricular relaxation. Once the mitral valve opens, ventricular volume begins rising as the ventricle passively fills during the rapid-filling phase. Immediately following the rapid-filling phase is the slow-filling phase, also called diastasis, which continues until atrial systole. Atrial systole is indicated on the atrial pressure tracing as a wave. These same events are essentially mirrored on the right side of the heart.

Physiology of cardiac output

The previous section outlines the sequence of events to allow the heart to function as an efficient pump, with the end product being an ejection of blood out of the heart. An adequate volume of blood must be ejected out of the heart to sustain life and activity. The cardiac output reflects the volume of blood ejected out of the left ventricle into the systemic vasculature per minute. It is a function of the number of heart beats per minute (heart rate) and the volume of blood ejected per beat (stroke volume). On average, the cardiac output at rest is between 4 and 6 L/min to allow for sufficient tissue perfusion.

Cardiac output = Heart rate × Stroke volume

It is also interesting to note that the average total blood volume is approximately 5.5 L.[7] This indicates that the ventricle pumps an amount of blood equivalent to the total blood volume each minute. Therefore it takes approximately 1 minute for a given volume of blood (a drop of blood) to complete the systemic and pulmonary circuits. With exercise, an increase in cardiac output is warranted to meet increases in oxygen consumption/demand by the working muscles. Fig. 2.16 demonstrates the parallel rise in cardiac output and oxygen consumption with increases in workload during exercise. To allow for this increase in cardiac output, an increase in blood volume must also exist. The following section reviews factors that regulate heart rate and stroke volume to accomplish an increase in blood volume and cardiac output.

Regulation of heart rate

As mentioned in Chapter 1, the heart continues to beat automatically between 60 and 100 beats per minute, as long as myocardial cells are alive as a consequence of spontaneous depolarization of the pacemaker cells in the SA node. Sympathetic and parasympathetic nerve fibers to the heart are activated to alter this intrinsic pacing rate of the SA node.[23] Epinephrine from the adrenal medulla of the adrenal gland

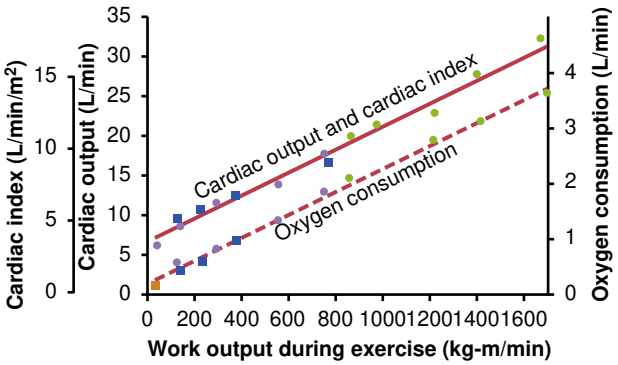

Figure 2.16 Relationship of cardiac output and cardiac index with oxygen consumption of task. (Modified from Guyton AC, Jones CE, Coleman TB: *Circulatory Physiology: Cardiac Output and Its Regulation*,Philadelphia, 1973, WB Saunders; and Hall J, Hall M: *Textbook of medical physiology*, ed 14, Philadelphia, 2021, Elsevier.)

and norepinephrine from the sympathetic axons open channels of the pacemaker cells of the SA node and increase the rate of depolarizations, resulting in an increase in heart rate.[1,5,7] Conversely, parasympathetic influence is achieved through the release of acetylcholine released by vagus nerve endings that bind to acetylcholine receptors, slowing down the rate of action potential production at the level of the SA node, thereby depressing heart rate.[1,5,7] It is important to understand that the actual pacing rate of the SA node is because of the net effect of these antagonistic influences. Mechanisms that alter the cardiac rate are said to have a chronotropic effect. Influences that increase heart rate are said to have a positive chronotropic effect, whereas those that decrease heart rate are said to produce negative chronotropic effects.

Autonomic nervous system influences not only affect the firing of the SA node, but also affect sympathetic endings (β-adrenergic receptors) in the myocardial wall of the atria and ventricles.[2,5] Sympathetic stimulation vasodilates coronary arteries to increase blood flow to the heart and increases myocardial contraction. Conversely, parasympathetic influence vasoconstricts coronary arteries, reducing blood flow to the myocardium and depressing myocardial contractility.[2,5] Mechanisms that affect the contractility of the myocardium are said to have an ionotropic effect. Influences that increase contractility have a positive ionotropic effect, and those that reduce contractility have a negative ionotropic effect.

Clinical tip

A common goal in the pharmacologic management of patients with heart failure is to provide medications that cause a positive ionotropic effect to increase the pumping ability of the failing heart. These medications include phosphodiesterase inhibitors, such as milrinone, and adrenergic agonists, such as epinephrine or dobutamine, that allow the heart to pump stronger and increase cardiac output.

A bout of aerobic exercise causes a linear increase in heart rate with increasing intensity as a result of decreased vagus nerve inhibition and increased sympathetic nerve stimulation. In addition, the slow resting heart rate, or bradycardic responses seen in endurance-trained athletes, is often thought to occur as a consequence of enhanced parasympathetic input to the heart.

Patients on β blockers have a blunted heart rate response during exercise, as β receptors on the myocardial wall are unable to respond to sympathetic stimulation and appropriately increase heart rate. In light of this, when treating a patient who is taking a β blocker, it may be more effective to use a subjective assessment of intensity, such as a Borg rate of perceived exertion, because measures of exercise intensity examined through heart rate responses will be inaccurate.

Regulation of stroke volume

The stroke volume, or volume of blood ejected out of the heart per beat, is affected by three variables:

1. Preload
2. Contractility
3. Afterload

Preload

The preload is a reflection of the volume of blood returning to the heart from systemic circulation. It is often correlated with the end-diastolic volume (EDV), which is the maximum amount of blood that can be in the ventricles at the end of diastole, immediately before contraction. In normal cardiovascular physiology, the preload is directly proportional to the stroke volume. In other words, as more blood returns to the heart, a greater volume of blood leaves the heart with every contraction. This physiologic principle is known as the Frank-Starling mechanism, whereby the strength of ventricular contraction increases as the precontractile myocardial cell length increases.[6,12] This length is influenced by the volume of blood returned to the ventricles before contraction, or the preload (Fig. 2.17). Clinically, the term preload, directly influenced by the EDV, refers to the amount of stretch, or load, on the myocardial wall before contraction (precontraction).

It is worth noting that in patients with congestive heart failure as a result of reduced systolic function (a weaker contraction), an increase in the EDV does not produce an increase in the preload and subsequent increase in stroke volume through the Frank–Starling mechanism. In fact, an increase in the EDV puts additional stress on the failing heart. Therefore much of the medical management of congestive heart failure secondary to systolic dysfunction is geared toward reducing the preload or stretch on the myocardial wall.

Clinical tip

Patients with heart failure secondary to a weakened left ventricle will often not tolerate a supine or recumbent position as this position will lead to a greater preload, causing blood to back up into the lungs and exacerbate signs and symptoms of heart failure. Therefore one aim of physical therapy management should be encouraging upright position, using the effects of gravity to decrease blood return to the heart, creating less congestion and stress on the failing heart.

Contractility

Myocardial contractility is influenced by intrinsic and extrinsic factors. The intrinsic control of contraction strength is a result of the degree of myocardial stretch caused by changes in the EDV.[12,13] This is discussed more comprehensively in the preceding section. In addition, force–frequency relationships cause an