VOLUME 3 MEDICAL EMERGENCIES

PARAMEDIC CARE Principles & Practice

Fifth Edition

Meets National EMS Education Standards

Bledsoe
Porter
Cherry

Paramedic Care: Principles & Practice

Fifth Edition

Volume 3

Medical Emergencies

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This text is respectfully dedicated to all EMS personnel
who have made the ultimate sacrifice. Their memory
and good deeds will forever be in our thoughts and prayers.

BEB, RAC

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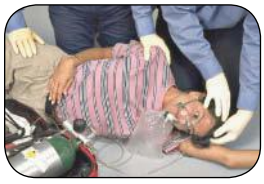
Contents



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Preface to Volume 3

Advanced life support (ALS) and paramedic care were initially developed to treat cardiac problems in the field, specifically sudden death. Many people were suffering acute coronary events and dying before reaching the hospital. Physicians with foresight believed that rapid prehospital intervention could mean the difference between life and death for many people. The earliest origins of EMS were this initial emphasis on prehospital cardiac care, which developed simultaneously with the early advances in trauma care. Over the years, EMS proved effective in treating different types of cardiac emergencies. As EMS evolved, prehospital care was expanded to many other types of medical emergencies, including diabetic emergencies and respiratory emergencies.

Under the current *National EMS Education Standards* and the accompanying *Paramedic Instructional Guidelines*, paramedics are responsible for a much more detailed understanding of medical emergencies. Those Standards and Instructional Guidelines, on which this book is based, address in detail the various types of medical emergencies. Cardiac emergencies still represent the most common reason EMS is summoned, and the discussion of cardiac emergencies remains the most comprehensive in the text and is consistent with the *2015 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care*.

Paramedics are also expected to have a high level of understanding of emergencies involving other body systems. In this volume, we briefly review important anatomy and physiology as it applies to each emergency, followed by a discussion of the relevant pathophysiology. Finally, we present focused prehospital assessment and treatment for each type of medical emergency. *Paramedic Care: Principles & Practice*, Volume 3, *Medical Emergencies*, provides a detailed discussion of virtually all types of medical emergencies likely to be encountered in the prehospital setting.

This volume follows a systems approach that also parallels the various subspecialties of internal medicine.

Overview of the Chapters . . . and What's New in the 5th Edition?

CHAPTER 1 **Pulmonology** introduces the paramedic student to commonly encountered respiratory system emergencies. Emphasis is on the recognition and treatment of reactive airway diseases such as asthma.

New in the 5th Edition: Emphasis on administering just enough oxygen to achieve **normoxia** if hypoxia is determined. (Dangers of hyperoxia are stressed.) There is a new section on **Middle Eastern Respiratory Syndrome (MERS)**.

CHAPTER 2 **Cardiology** presents the material crucial to advanced prehospital cardiac care. The first part of the chapter reviews essential anatomy and physiology and introduces electrophysiology. The second part of the chapter deals with cardiac emergencies and peripheral vascular system emergencies. The third part addresses 12-lead ECG interpretation and prehospital application of 12-lead ECG diagnostics and monitoring.

New in the 5th Edition: A detailed new section on **cardiac arrest in pregnancy** has been added. This chapter (and all chapters in all five volumes) updated as needed for consistency with the 2015 AHA guidelines.

CHAPTER 3 **Neurology** reviews the anatomy and physiology of the central and peripheral nervous systems. This is followed by a detailed discussion of neurologic emergencies.

CHAPTER 4 **Endocrinology** is a detailed discussion of the endocrine system, which, along with the nervous system, is an alternative control system for the body. Emphasis is placed on diabetic emergencies, as they are by far the most common endocrine emergency encountered by paramedics.

CHAPTER 5 Immunology reviews the immune system, with particular emphasis on hypersensitivity reactions (allergic reactions). The chapter emphasizes prehospital recognition and treatment of allergic reactions, particularly the severe reactions known as anaphylaxis.

CHAPTER 6 Gastroenterology is a detailed discussion of emergencies arising within the gastrointestinal system. The chapter initially reviews the relevant anatomy and physiology and follows this with a discussion of assessment and treatment of gastroenterologic emergencies.

New in the 5th Edition: A new section on **cyclical vomiting syndrome (CVS)** and a new section on **irritable bowel disease (IBS)** have been added.

CHAPTER 7 Urology and Nephrology presents an overview of emergencies that arise from the genitourinary system and the male reproductive system. This includes a discussion of infectious emergencies, renal failure, and other problems.

CHAPTER 8 Toxicology and Substance Abuse provides a detailed description of basic toxicology as it applies to prehospital care. The chapter reviews both common and uncommon causes of poisoning. In addition to accidental poisoning, there is a detailed discussion of the various drugs of abuse that are frequently seen in prehospital care.

New in the 5th Edition: Tables listing **toxic syndromes** and **drugs of abuse** have been updated.

CHAPTER 9 Hematology is a comprehensive chapter covering the blood and the reticuloendothelial system. The beginning of the chapter provides a detailed discussion of the blood and blood-forming organs. This is followed by a discussion of hematologic emergencies seen in emergency care.

CHAPTER 10 Infectious Diseases and Sepsis addresses an important but often overlooked aspect of prehospital care. Infectious diseases pose a risk to both the paramedic and the patient. This chapter reviews the basics of infectious disease, including disease transmission. This is followed by a discussion of infectious diseases likely to be encountered in prehospital care.

New in the 5th Edition: The following have been added to update this chapter's information on infectious diseases: New information on the **brain-eating amoeba** (*Naegleria fowler*)*;* a new section on **Middle East respiratory syndrome (MERS)**; a new section on **Ebola virus disease**; a new section on **Zika virus disease**; a new section on **Chikungunya virus disease**; an extensive new section on the **Centers for Disease Control and Prevention Enhanced Precautions** to protect EMS personnel at risk of exposure to Ebola or other highly contagious disease; information on **antiviral drugs to treat hepatitis C**; information on the **increase in the occurrence of measles related to "anti-vaccine" movement** among some parents.

CHAPTER 11 Psychiatric and Behavioral Disorders provides an overview of psychiatric and behavioral problems. Paramedics are often the first health care professionals encountered by a patient with a psychiatric disorder. Because of this, paramedics must be ready to recognize and manage these emergencies appropriately.

New in the 5th Edition: Information added regarding increasing use of **ketamine as a chemical restraint** in the prehospital management of excited delirium.

CHAPTER 12 Diseases of the Eyes, Ears, Nose, and Throat presents an overview of the relevant anatomy and physiology and specific medical conditions that may affect each element that makes up this category of disease–the eyes, the ears, the nose, and the throat.

CHAPTER 13 Nontraumatic Musculoskeletal Disorders begins with a review of the anatomy and physiology of the skeletal system and the muscular system, then discusses relevant assessment and findings and details specific nontraumatic (medical) disorders that may affect these systems.

Acknowledgments

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We wish to acknowledge the remarkable talents of the following people who contributed to this five volume series. Individually, they worked with extraordinary commitment. Together, they form a team of highly dedicated professionals who have upheld the highest standards of EMS instruction.

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BEB, RAC

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The reviewers of this edition of ***Paramedic Care: Principles & Practice*** have provided many excellent suggestions and ideas for improving the text. The quality of the reviews has been outstanding, and the reviews have been a major aid in the preparation and revision of the manuscript. The assistance provided by these EMS experts is deeply appreciated.

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Dr. Bledsoe is active in emergency medicine and EMS research. He is a popular speaker at state, national, and international seminars and writes regularly for numerous EMS journals. He is active in educational endeavors with the United States Special Operations Command (USSOCOM) and the University of Nevada at Las Vegas. Dr. Bledsoe is the author of numerous EMS textbooks and has in excess of 1 million books in print. Dr. Bledsoe was named a "Hero of Emergency Medicine" in 2008 by the American College of Emergency Physicians as a part of their 40th anniversary celebration and was named a "Hero of Health and Fitness" by *Men's Health* magazine as part of their 20th anniversary edition in November of 2008. He is frequently interviewed in the national media. Dr. Bledsoe is married and divides his time between his residences in Midlothian, TX, and Las Vegas, NV.

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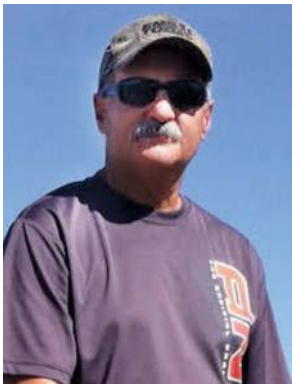

Richard Cherry is a Training Consultant for Northern Onondaga Volunteer Ambulance (NOVA) in Liverpool, New York, a suburb of Syracuse. He is also a program reviewer for The Continuing Education Coordinating Board for Emergency Medical Services (CECBEMS). He formerly held positions in the Department of Emergency Medicine at Upstate Medical University as Director of Paramedic Training, Assistant Emergency Medicine Residency Director, Clinical Assistant Professor of Emergency Medicine, and Technical Director for Medical Simulation. His experience includes years of classroom teaching and emergency fieldwork. A native of Buffalo, Mr. Cherry earned his bachelor's degree at nearby St. Bonaventure University in 1972. He taught high school for the next ten years while he earned his master's degree in education from Oswego State University in 1977. He holds a permanent teaching license in New York State.

Mr. Cherry entered the emergency medical services field in 1974 with the DeWitt Volunteer Fire Department, where he served his community as a firefighter and EMS provider for more than 15 years. He took his first EMT course in 1977 and became an ALS provider two years later. He earned his paramedic certificate in 1985 as a member of the area's first paramedic class. He then worked both as a paid and volunteer paramedic for the next 15 years.

Mr. Cherry has authored several books for Brady. Most notable are *Paramedic Care: Principles & Practice, Essentials of Paramedic Care, Intermediate Emergency Care: Principles & Practice,* and *EMT Teaching: A Common Sense Approach.* He has made presentations at many state, national, and international EMS conferences on a variety of EMS clinical and teaching topics. He and his wife, Sue, reside in Sun City West, Arizona. In addition to riding horses, hiking, and playing softball, they volunteer their time at Banner Del Webb Medical Center. Mr. Cherry also plays lead guitar in a Christian band.

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A GUIDE TO KEY FEATURES

Emphasizing Principles

Chapter 1

Introduction to Paramedicine

Bryan Bledsoe, DO, FACEP, FAAEM

STANDARD

Preparatory (EMS Systems)

COMPETENCY

Integrates comprehensive knowledge of EMS systems, the safety and well-being of the paramedic, and medical–legal and ethical issues, which is intended to improve the health of EMS personnel, patients, and the community.

Learning Objectives

Terminal Performance Objective: After reading this chapter your should be able to discuss the characteristics of the profession of paramedicine.

Enabling Objectives: To accomplish the terminal performance objective, you should be able to:

1. Define key terms introduced in this chapter.
2. Compare and contrast the four nationally recognized levels of EMS providers in the United States.
3. Describe the requirements that must be met for EMS professionals to function at the paramedic level.
4. Discuss the traditional and emerging roles of the paramedic in health care, public health, and public safety.
5. List and describe the various health care settings paramedics may practice in with an expanded scope of practice.

KEY TERMS

Advanced Emergency Medical Technician (AEMT), p. 3
community paramedicine, p. 4
critical care transport, p. 7
Emergency Medical Responder (EMR), p. 3
Emergency Medical Services (EMS) system, p. 2
Emergency Medical Technician (EMT), p. 3
mobile integrated health care, p. 4
National Emergency Medical Services Education Standards: Paramedic Instructional Guidelines, p. 5
Paramedic, p. 3
paramedicine, p. 4

LEARNING OBJECTIVES

Terminal Performance Objectives and a separate set of Enabling Objectives are provided for each chapter.

KEY TERMS

Page numbers identify where each key term first appears, boldfaced, in the chapter.

more rapid are the pulse and respiratory rates.

3.0 and 3.5 kg. Because of the excretion of extracellular

As newborns make the transition from fetal to pulmonary circulation in the first few days of life, several important

Table 11-1 Normal Vital Signs

	Pulse (Beats per Minute)	Respiration (Breaths per Minute)	Blood Pressure (Average mmHg)	Temperature	
Infancy:					
At birth:	100–180	30–60	60–90 systolic	98–100°F	36.7–37.8°C
At 1 year:	100–160	30–60	87–105 systolic	98–100°F	36.7–37.8°C
Toddler (12 to 36 months)	80–110	24–40	95–105 systolic	96.8–99.6°F	36.0–37.5°C
Preschool age (3 to 5 years)	70–110	22–34	95–110 systolic	96.8–99.6°F	36.0–37.5°C
School-age (6 to 12 years)	65–110	18–30	97–112 systolic	98.6°F	37°C
Adolescence (13 to 18 years)	60–90	12–26	112–128 systolic	98.6°F	37°C
Early adulthood (19 to 40 years)	60–100	12–20	120/80	98.6°F	37°C
Middle adulthood (41 to 60 years)	60–100	12–20	120/80	98.6°F	37°C
Late adulthood (61 years and older)	*	*	*	98.6°F	37°C

*Depends on the individual's physical health status.

TABLES

A wealth of tables offers the opportunity to highlight, summarize, and compare information.

components of the rule of threes. Whenever BVM ventilation is difficult, however, the rule of threes should be employed.

CONTENT REVIEW

- The Rule of Threes for Optimal BVM Ventilation
  - Three providers
  - Three inches
  - Three fingers
  - Three airways
  - Three PSI
  - Three PEEP

- ***Three providers.*** One provider on the mask, one on the bag, and one for cricoid pressure.
- ***Three inches.*** A reminder to place the patient in the sniffing position (elevate the head three inches) if not contraindicated.
- ***Three fingers.*** Three fingers on the cricoid cartilage to perform cricoid pressure.
- ***Three airways.*** In a worst-case scenario, the airway can be maintained, if necessary, with an orophrayngeal airway and two nasopharyngeal airways (one in each nostril).

CONTENT REVIEW

Content review boxes set off from the text are interspersed throughout the chapter. They summarize key points and serve as a helpful study guide—in an easy format for quick review.

index, and middle finger of one hand. If a lesser-trained provider is performing the maneuver, you should confirm that they are in the correct position (Figure 15-47).

Use caution not to apply so much pressure as to deform and possibly obstruct the trachea; this is a particular danger in infants. The necessary pressure has been estimated as the amount of force that will compress a capped 50-mL syringe from 50 mL to the 30 mL marking. In the event that the patient actively vomits, it is imperative to release the pressure to avoid esophageal rupture. Similarly, if cricoid pressure is being performed during intubation, reduce or release the pressure if the intubator is having difficulty visualizing the vocal cords.

Optimal BVM Ventilation Using the Rule of Threes

The *rule of threes* was developed to help providers recall the components of optimal BVM ventilation. Many patients can be easily oxygenated and ventilated without using all

- ***Three PSI.*** A gentle reminder to use the lowest pressure necessary to see the chest rise.
- ***Three seconds.*** A reminder to ventilate slowly and allow time for adequate exhalation.
- ***Three PEEP.*** Or up to 15 cm/H_2O positive-end expiratory pressure (PEEP) as needed to improve oxygen saturations.

Bag-Valve Ventilation of the Pediatric Patient

The differences in the pediatric patient's anatomy require some variation in ventilation technique. First, the child's relatively flat nasal bridge makes achieving a mask seal more difficult. Pressing the mask against the child's face to improve the seal can actually obstruct the airway, which is more compressible than an adult's. You can best achieve the mask seal with the two-person BVM technique, using a jaw-thrust to maintain an open airway.

For BVM ventilation, the bag size depends on the child's age. Full-term neonates and infants will require a pediatric BVM with a capacity of at least 450 mL. For children up to 8 years of age, the pediatric BVM is preferred, although for patients in the upper portion of that age range you can use an adult BVM with a capacity of 1,500 mL if you do not maximally inflate it. Children older than 8 years require an adult BVM to achieve adequate tidal volumes. Additionally, be

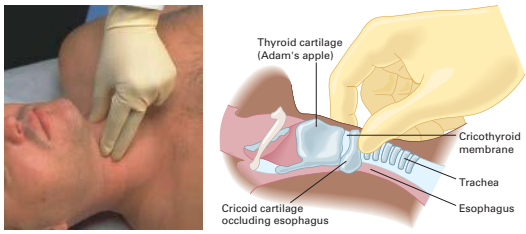

FIGURE 15-47 Cricoid pressure.

PHOTOS AND ILLUSTRATIONS

Carefully selected photos and a unique art program reinforce content coverage and add to text explanations.

Summary

The scene size-up is the initial step in the patient care process. Sizing up the scene and situation begins at your initial dispatch and does not end until you are clear of the call. As the call unfolds, you should be making constant observations and adjustments to your plan of action. Remember that your safety and the safety of your partner are paramount—it is hard to effectively treat both yourself and others.

Scene size-up should be practiced so much that it becomes second nature to you. It is like noticing veins on people in public after you begin starting IVs. (You have all done it—looked across the room at the back of someone's hand and noticed what nice veins they had.) Sizing up a scene is no different. After a while, you begin to notice mechanisms of injury and other important details almost subconsciously. But be careful and do not get complacent! Always make it a point to pause for just a few seconds and consciously look around the scene before proceeding into any situation.

Scene size-up is not a step-by-step process, but a series of decisions you make when confronted with a variety of circumstances that are often beyond your control. It is a way to make order out of chaos, keep yourself and your crew safe, and ensure that all necessary resources are focused on patient care and outcomes. With time and experience, you will learn to perform a scene size-up quickly and focus on important issues. Your careful size-up lays the foundation for an organized and timely approach toward patient care and scene management. And always remember that scene size-up is not a one-time occurrence. It is an ongoing process.

SUMMARY

This end-of-chapter feature provides a concise review of chapter information.

airway management in every patient, you should learn and use advanced skills such as intubation, RSI, and cricothyrotomy. You must maintain proficiency in all airway skills, especially the more advanced techniques, through ongoing continuing education, physician medical direction, and testing with each EMS service. If you cannot do this, it is in the patient's best interest to focus on less sophisticated airway skills. If you anticipate that every airway will be complicated, apply basic airway skills before using advanced procedures, and perform frequent reassessments, you will give the patient his best chance for meaningful survival.

You Make the Call

You and your paramedic partner, Preston Connelly, are assigned to District 4, a quiet suburban neighborhood, on a warm Saturday in June. At 2:00 P.M., you are dispatched to care for a choking child at the Happy Hotdog Restaurant on Main Street. On your way to the location, the dispatcher advises you that they are currently giving prearrival choking instructions to the bystanders at the scene. On arrival, you find a frantic mother who tells you that her 6-year-old son was eating a hot dog and drinking a soda when he started coughing and gasping for air. She keeps yelling for you to do something. Bystanders surround the child and are attempting to perform the Heimlich maneuver without success. On your primary assessment, you find a 6-year-old boy lying on the floor, unconscious and apneic, with a pulse rate of 130. There is cyanosis surrounding his lips and fingernail beds, with a moderate amount of secretions coming from his mouth. There are no signs of trauma. You and Preston immediately start management of this child.

1. What is your primary assessment and management of this child?
2. What are your first actions?
3. What are your options for managing the airway after the obstruction is relieved?
4. What are the major anatomic differences between pediatric and adult patients in terms of airway management?

See Suggested Responses at the back of this book.

YOU MAKE THE CALL

A scenario at the end of each chapter promotes critical thinking by requiring students to apply principles to actual practice.

REVIEW QUESTIONS

These questions ask students to review and recall key information they have just learned.

6. Which radio frequencies may be used by cities and municipalities for their ability to better transmit through concrete and steel?
   a. UHF
   b. VHF
   c. 800-mHz
   d. none of the above
7. Which frequency band is typically used by county and suburban agencies due to its ability to transmit over various terrains and longer distances?
   a. UHF
   b. VHF
   c. 800-mHz
   d. none of the above
8. What is the name of the basic communications system that uses the same frequency to both transmit and receive?
   a. Multiplex
   b. Duplex
   c. Simplex
   d. Complex
9. A communications system that uses a different transmit and receive frequency allowing for simultaneous communications between two parties is called _____
   a. multiplex.
   b. duplex.
   c. simplex.
   d. complex.
10. _____ communications systems are capable of transmitting both voice and electronic patient data simultaneously.
    a. Multiplex
    b. Duplex
    c. Simplex
    d. Complex

See answers to Review Questions at the back of this book.

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Further Reading

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CASE STUDY

This feature at the start of each chapter draws students into the reading and creates a link between text content and real-life situations.

Review Questions

1. When you couple the physical assessment findings with the patient's medical history, you are able to derive a list of _____
   a. clinical diagnostics.
   b. field prognoses
   c. chief complaints
   d. differential field diagnoses.
2. The pain, discomfort, or dysfunction that caused your patient to request help is known as the _____
   a. primary problem.
   b. nature of the illness.
   c. differential diagnosis.
   d. chief complaint.
3. You are assessing a patient who complains of cardiac-type chest pain that is felt in the jaw and down the left arm. This pattern of pain is known as _____
   a. sympathetic pain.
   b. tenderness.
   c. referred pain.
   d. associated pain.
4. Your patient has smoked 2 packs of cigarettes each day for the past 35 years. He is a _____ pack/year smoker.
   a. 35
   b. 70
   c. 730
   d. 25,550
5. The CAGE questionnaire is used as an evaluation tool to assess a patient with what type of history?
   a. Alcoholism
   b. Lung disease
   c. Allergies
   d. Pregnancy
6. What interviewing mnemonic should be used for each presenting problem a patient has?
   a. SAMPLE
   b. DCAP–BTLS
   c. OPQRST–ASPN
   d. AEIOU–TIPS
7. The mnemonic GPAL is used to evaluate a patient's _____
   a. alcoholism.
   b. allergies.
   c. pregnancy history.
   d. endocrine dysfunction.

Match the following elements of the present illness of the patient with a chief complaint of chest pain with their respective examples:

1. O	**a.** Pain is 6 on a scale of 1–10
2. P	**b.** Patient also complains of shortness of breath and nausea
3. Q	**c.** Pain had a sudden onset
4. R	**d.** Pain began 2 hours ago
5. S	**e.** Pain worsens while lying down
6. T	**f.** Patient denies dizziness
7. AS	**g.** Pain goes through to the back
8. PN	**h.** Pain is heavy and vise-like

See Answers to Review Questions at the back of this book.

REFERENCES

This listing is a compilation of source material providing the basis of updated data and research used in the preparation of each chapter.

FURTHER READING

This list features recommendations for books and journal articles that go beyond chapter coverage.

cleaning, p. 70
Code Green Campaign, p. 78
disinfection, p. 70
exposure, p. 70
isotonic exercise, p. 61
pathogens, p. 65
personal protective equipment (PPE), p. 66
sterilization, p. 70
stress, p. 74
stressor, p. 74
Tema Conter Memorial Trust, p. 78

Case Study

Howard is a 15-year veteran of a high-volume, inner-city EMS service. When he first started his career, Howard thought he knew what he was getting into, but the years have taught him differently.

Right now, Howard is in the spotlight for saving the life of a police officer who was shot in a hostage situation. "That call forced me to reflect on a few important things," he says. "Two years ago, I had a minor heart problem, and it was a good wake-up call. Since then I've been lifting weights and running, so I was able to get to the officer with enough strength to carry him to safety.

"Another thing is that I always use personal protective equipment. I never go to work without steel-toed boots and I never leave the ambulance without a pair of disposable gloves. Can you believe there are still paramedics who knock the concept of infection control? If any one of my partners sticks a needle into the squad bench in my ambulance, they know I'll speak up."

Howard, a mild-mannered, nondescript man, doesn't realize that his young colleagues regard him as a role model. They've seen him handle himself at chaotic scenes as well as when a situation demands sensitivity, patience, and gentleness. "Howard is the man I'd want to tell bad news to my mother," one of his partners says. "He can handle people involved in just about any circumstance—death situations, panicked parents, lonely elderly people, and even hostile drunks. I've never seen anyone treat others with such dignity and respect. He's the best partner anyone could want, especially when we have to manage patients who are thrashing around. But that was not always so, was it, Howard?"

"No, it wasn't," Howard replies. "There was a time when no one wanted to work with me. I was a rebel, and I figured there was only one way to do things: my way. But an incident that occurred a few years ago changed all that. It's a long story. But the upshot is that when I recovered from the stress, my outlook had been altered. I realized that though I couldn't save the world, I could save myself. That's when I learned how to deal with the effects of a stressful job. I started eating right, lost a lot of weight, and adopted a new attitude. Anyway, if I can maintain my own well-being, I can do a lot more to help others. Right? Isn't that what we're about?"

Introduction

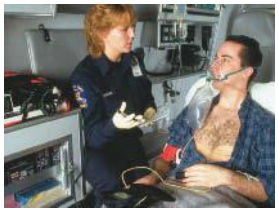
The safety and well-being of the workforce is a fundamental aspect of top-notch performance in EMS.[1] As a paramedic, it includes your physical well-being as well as your mental and emotional well-being. If your body is fed well and kept fit, if you use the principles of safe lifting, observe safe driving practices, and avoid potentially addictive and and insidious infections. If you let your spirit appreciate the fear and sadness on other faces, you will find ways to combat your prejudices and treat people with dignity and respect. By doing all these things, you will also be able to promote the benefits of well-being to your EMS colleagues.

Death, dying, stress, injury, infection, fear—all these threaten your wellness and conspire to interfere with your good intentions. However, you can do something about

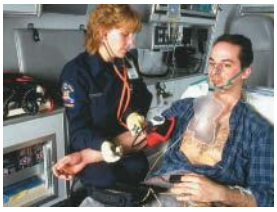
PROCEDURE SCANS

Visual skill summaries provide step-by-step support in skill instruction.

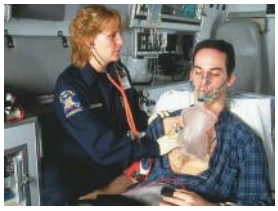
Procedure 7-4 Reassessment



7-4a Reevaluate the ABCs.



7-4b Take all vital signs again.



7-4c Perform your focused assessment again.

7-4d Evaluate your interventions' effects.

laryngospasm may be occurring. Airway and breathing management requires constant reevaluation.

oxygenation. Lip cyanosis indicates central hypoxia (overall oxygen status), whereas peripheral cyanosis indicates decreased oxygen to the tissues. Pallor and coolness sug-

Special Features

the present illness. Common sense and clinical experience will determine how much of the following history to use.

Preliminary Data

For documentation, always record the date and time of the physical exam. Determine your patient's age, sex, race, birthplace, and occupation. This provides a starting point for the interview and establishes you as the interviewer. Who is the source of the information you receive about your patient? Is it the competent patient himself, his spouse, a friend, or a bystander? Are you receiving a report from a first responder, the police, or another health care worker? Do you have the medical record from a transferring facility?

After you have gathered the information, you should establish its reliability, which will vary according to the source's knowledge, memory, trust, and motivation. Again, reconfirm the information with the patient, if possible. This is a judgment call based on your experience. For example, if the patient information you received from a particular EMT first responder has been accurate in the past, you probably will trust it again. On the other hand, if the nurse at a physician's office has repeatedly provided you with erroneous information, you probably will doubt its accuracy.

scious patient, the chief complaint becomes what someone else identifies or what you observe as the primary problem. In some trauma situations, for instance, the chief complaint might be the mechanism of injury, such as "a penetrating wound to the chest" or "a fall from 25 feet."

Patho Pearls

The renowned Canadian physician Sir William Osler said, "Listen to the patient, and he will tell you what is wrong." This advice is as true today as it was 100 years ago. A great deal of information can be determined from a skillful history taking. As you listen to a patient's medical history, try to understand the underlying pathophysiologic processes that might cause the symptoms the patient describes. This will help you to fully comprehend the disease process or processes affecting the patient.

For example, consider the following case. Mrs. J. Franklin is a 72-year-old pensioner, twice widowed, who lives in an older section of town. She summons EMS with what initially seem like vague complaints. She reports to the dispatcher, when queried, that she is "just sick." You arrive and begin an assessment, starting with a pertinent history. The patient reports that her symptoms began about two weeks ago after several family members came to her house with dinner, which included a baked ham. Since that time, she has developed some fatigue, progressive dyspnea, and occasional chest pain. She now reports that she often wakes up at 3:00 A.M. with breathing trouble that resolves when she walks around the room or

PATHO PEARLS

Offer a snapshot of pathological considerations students will encounter in the field.

LEGAL CONSIDERATIONS

Offer a snapshot of pathological considerations students will encounter in the field.

FIGURE 2-11 Patients may be transported by ground or air. Medical helicopter transport was introduced in the 1950s during the Korean War. *(© Ed Effron)*

Vietnam, and success of military evacuation procedures led to their use in civilian ambulance systems. In 1970, the Military Assistance to Safety and Traffic (MAST) program was established. This demonstration project set up 35 helicopter transportation programs nationwide to test the feasibility of using military helicopters and paramedics in

Legal Considerations

Emergency Department Closures. Numerous factors have resulted in emergency department closures and ambulance diversions. This can have a significant impact on the EMS system. All systems must address this situation so that patient care does not suffer.

In 1974, in response to a request from the DOT, the General Services Administration (GSA) developed the "KKK-A-1822 Federal Specifications for Ambulances." This was the first attempt at standardizing ambulance design to permit intensive life support for patients en route to a definitive care facility. The act defined the following basic types of ambulance:

- ***Type I* (Figure 2-13).** This is a conventional cab and chassis on which a module ambulance body is mounted, with no passageway between the driver's and patient's compartments.
- ***Type II* (Figure 2-14).** A standard van, body, and cab form an integral unit. Most have a raised roof.

An important part of patient assessment is gathering information that is accurate, complete, and relevant to the present emergency. To begin, you must identify the patient's chief complaint. Although dispatch probably will have given you an idea of what the emergency is about, it is

Cultural Considerations

Eye contact is a major form of nonverbal communication. Short eye contact is often seen as friendly, whereas prolonged eye contact may be interpreted as threatening. Thus, timing is an important factor in how a person interprets eye contact.

One's culture also influences how eye contact is interpreted. Eye contact can mean respect in one culture and disrespect in another. Often, Asians will avoid eye contact even when they have nothing to hide. Eye contact between people of different sexes is problematic in Muslim cultures, in which a prolonged look in the face of a member of the opposite sex might be misinterpreted. Because of this, people in Middle Eastern countries might look a person of the same sex in the eye and not look into the eyes of a person of the opposite sex.

If you work in a culturally diverse community, you should learn the customs of eye contact and other forms of nonverbal communication of those you might encounter during the course of your work.

unexpected but important facts. For example, instead of asking your patient with abdominal pain, "Did you have breakfast today?" which can be answered with either a "yes" or a "no," ask: "What have you eaten today?"

- ***Use direct questions when necessary.*** Direct questions, or **closed questions**, ask for specific information. ("Did you take your pills today?" or "Does the abdominal pain come and go like a cramp, or is it constant?") These questions are good for three reasons: They fill in information generated by open-ended questions. They help to answer crucial questions when time is limited. And they can help to control overly talkative patients, who might want to tell you about their gallbladder surgery in 1969 when their chief complaint is a sprained ankle.
- ***Ask only one question at a time, and allow the patient to complete his answers.*** If you ask more than one question, the patient may not know which one to answer and may leave out portions of information or become confused. Equally important is having one person do the interview. Don't force your patient to discern questions from multiple interviewers.
- ***Listen to the patient's complete response before asking the next question.*** By doing so, you might find that

CULTURAL CONSIDERATIONS

Provide an awareness of beliefs that might affect patient care.

ASSESSMENT PEARLS

Offer tips, guidance, and information to aid in patient assessment.

Provocation/Palliation

What provokes the symptom (makes it worse)? Does anything palliate the symptom (make it better)? In many

Assessment Pearls

Chest pain is a common reason that people summon EMS. However, the causes of chest pain are numerous. In emergency medicine or EMS, we often look to exclude the most serious causes before determining whether chest pain is of a benign origin. Internal organs do not have as many pain fibers as do such structures as the skin and other areas. Pain arising from an internal organ tends to be dull and vague. This is because nerves from various spinal levels innervate the organ in question. The heart, for example, is innervated by several thoracic spinal nerve segments. Thus, cardiac pain tends to be dull and is sometimes described as pressure. It also tends to cause referred pain (i.e., pain in an area somewhat distant to the organ), such as pain in the left arm and jaw. Dull pain that is hard to localize (or to reproduce with palpation) may be due to cardiac disease. One sign often seen with patients suffering cardiac disease is Levine's sign. With Levine's sign, the patient will subconsciously clench his fist when describing the chest pain. Levine's sign is associated with pain of a cardiac origin (e.g., angina or acute coronary syndrome).

Ask about any activity, medication, or other circumstance that either alleviates or aggravates the chief complaint.

Quality

How does your patient perceive the pain or discomfort? Ask him to explain how the symptom feels, and listen carefully to his answer. Does your patient call his pain crushing, tearing, oppressive, gnawing, crampy, sharp, dull, or otherwise? Quote his exact descriptors in your report.

Region/Radiation

Where is the symptom? Does it move anywhere else? Identify the exact location and area of pain, discomfort, or dysfunction. Does your patient complain of pain "here," while holding a clenched fist over the sternum, or does he grasp the entire abdomen with both hands and moan? If your patient has not done so, ask him to point to the painful area. Identify the specific location, or the boundary of the pain if it is regional.

Determine whether the pain is truly pain (occurring independently) or **tenderness** (pain on palpation). Also determine whether the pain moves or radiates. Localized pain occurs in one specific area, whereas radiating pain

the result of a head injury, hypothermia, severe hypoxia, or drug overdose. Bradycardia is a common finding in the well-conditioned athlete, but it may be found in almost anyone. Treat bradycardia only if it compromises your patient's cardiac output and general circulatory status.

Tachycardia usually indicates an increase in sympathetic nervous system stimulation as the body compensates for another problem, such as blood loss, fear, pain, fever, drug overdose, or hypoxia. It is an early indicator of shock and may indicate ventricular tachycardia, a life-threatening cardiac dysrhythmia.

The pulse's quality can be weak, strong, or bounding. Weak, thready pulses indicate a decreased circulatory status, such as shock. Strong, bounding pulses may indicate high blood pressure, heat stroke, or increasing intracranial pressure. The pulse location may be another indicator of your patient's clinical status. The presence of a carotid pulse generally means that his systolic blood pressure is at least 60 mmHg. The presence of peripheral pulses indicates a higher blood pressure; their absence suggests circulatory collapse. Practice locating each of the pulse locations (Figure 5-12). As with other vital signs, take your patient's pulse frequently in the emergency setting and note any trends.

To take the pulse of a conscious adult or large child, the most accessible and commonly used location is the radial artery. With the pads of your first two or three

Pediatric Pearls

In infants and small children, use the brachial artery or auscultate for an apical pulse. Remember that auscultating an apical pulse does not provide information about your patient's hemodynamic status. To locate the brachial artery, feel just medial to the biceps tendon. Auscultate the apical pulse just below the left nipple.

fingers, compress the radial artery onto the radius, just below the wrist on the thumb side (Procedure 5-1b). In the unconscious patient, begin by checking his carotid pulse. To locate the carotid pulse, palpate medial to and just below the angle of the jaw. Locate the thyroid cartilage (Adam's apple) and slide your fingers laterally until they are between the thyroid cartilage and the large muscle in the neck (sternocleidomastoid).

First, note your patient's pulse rate by counting the number of beats in 1 minute. If his pulse is regular, you can count the beats in 15 seconds and multiply that number by 4. If his pulse is irregular, you must count it for a full minute to obtain an accurate total. Also note the pulse's rhythm and quality.

Blood Pressure

Blood pressure is the force of blood against the arteries' walls as the heart contracts and relaxes. It is equal to cardiac output times the systemic vascular resistance. Any

PEDIATRIC PEARLS

Offer tips, guidance, and information on how to deal with pediatric patients encountered in the field.

CUSTOMER SERVICE MINUTE

Shows how extending extra kindness and compassion can make an important difference to patients and families coping with an emergency.

Customer Service Minute

Following Up. Last week, a man took his dog to the vet for an upper respiratory infection. The dog was pretty sick, but the vet assured the owner that she was not critical, and with antibiotics she would be better in a few days, so he brought her home. The next day, the veterinarian called to find out how the dog was doing. She called every day until the dog was back to normal. Needless to say, the man was delighted in the service he received from that vet.

Physicians' offices, dentists' offices, and veterinary offices often call their patients a few days following a visit to see how things are going. Why don't we? Before you leave your patient and the family, why not ask them for permission to call the next day or in a few days to see how they're doing? If they say no or are hesitant to give permission, drop it. If they give permission, call them and see if there is anything you can do for them.

The follow-up has many benefits. You get to reconnect with the people in your community. It is great for public relations. It is educational because you can see whether your diagnosis was accurate. It's a winner from every angle. When they hang up, they'll be thinking, *"Wow!"*

Introduction

Patient assessment means conducting a problem-oriented evaluation of your patient and establishing priorities of your patient en route to the hospital to detect changes in patient condition.

Your proficiency in performing a systematic patient assessment will determine your ability to deliver the highest quality of prehospital **advanced life support** (ALS) to sick and injured people. Paramedic patient assessment is a straightforward skill, similar to the assessment you might have performed as an EMT. It differs, however, in depth and in the kind of care you will provide as a result.

Your assessment must be thorough, because many ALS procedures are potentially dangerous. Safely and appropriately performing advanced procedures such as administration of drugs, defibrillation, synchronized cardioversion, needle decompression of the chest, or endotracheal intubation will depend on your assessment and correct field diagnosis. If your assessment does not reveal your patient's true problem, the consequences can be devastating.

As always, common sense dictates how you proceed in the field. When you assess the responsive medical patient, the history reveals the most important diagnostic information and takes priority over the physical exam. For the trauma patient and the unresponsive medical patient, the reverse is true. However, trauma may cause a medical emergency, and, conversely, a medical emergency may cause trauma. Only by performing a thorough patient assessment can you discover the true cause of your patient's problems. This chapter provides problem-oriented patient assessment examples based on the information and techniques presented in the previous six chapters.

IN THE FIELD

Provides extra tips that can help ensure success in real-life emergency situations.

In the Field

The Tools of Your Trade: *The Ophthalmoscope*

An **ophthalmoscope** (Figure 5-27) is a medical instrument used to examine the internal eye structures, especially the retina, located at the back of the eye. Although it is most often used to diagnose eye conditions, you can discover information that may be relevant to other medical and traumatic events.

The ophthalmoscope is basically a light source with lenses and mirrors. It has a handle, which houses the batteries, and a head, which includes a window through which you visualize the internal eye; an aperture dial, which changes the width of the light beam; a lens dial to bring the eye into focus; and a lens indicator, which identifies the lens magnification number (i.e., 0 to +40 or 0 to –20). You examine the eye by looking through a monocular eyepiece into the eye of your patient. You can view different depths of the eye at different magnifications by rotating a disk of varying lenses within the instrument itself.

FIGURE 5-27 An ophthalmoscope is used to visualize the interior of your patient's eyes.

eye while the patient continues to fix his gaze on an object in the distance. Adjust the lens disk as needed to focus on the retina. Farsighted patients will require more "plus" diopters (black or green numbers), whereas nearsighted patients will require more "minus" diopters (red numbers) to keep the retina in focus.

Try to keep both your eyes open and relaxed. The optic disk should come into view when you are about 1.5 to 2 inches from the eye while you are still aiming your light 15 to 25 degrees nasally. If you are having difficulty finding the disk, look for a branching (bifurcation) in a retinal blood vessel. Usually the bifurcation will point toward the disk.

Follow the vessel in the direction of the bifurcation and you should arrive at the optic disk. The disk should appear as a yellowish-orange to pink round structure. Within the center of the disk there should be a central physiologic cup, which normally appears as a smaller, paler circle. The cup should be less than half the diameter of the disk. An enlarged cup may indicate chronic open-angle glaucoma. Indistinct borders or elevation of the optic disk may indicate papilledema, which is a marker of increased intracranial pressure.

Next, look at the arteries and veins of the retina. The arteries are usually brighter and smaller than the veins. Spontaneous venous pulsations are normal. Abnormalities of the retina such as hemorrhages, arteriovenous (AV) nicking, and cotton wool spots may indicate local or systemic disease such as retinal vein occlusion, hypertension, or many other conditions.

Finally, look at the fovea and surrounding macula. This area is where vision is most acute. It is located about two disk diameters temporal to the optic disk. You may also find the macula by asking the patient to look directly into the light of your ophthalmoscope. Prepare for a fleeting glimpse as this area is very sensitive to light and may be uncomfortable for your patient to maintain. A "cherry red" macula with surrounding pallor of tissue in the setting of acute painless monocular visual loss indicates a central retinal artery occlusion. Irreversible damage occurs


Image by Christof VanDerWalt

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ALWAYS LEARNING PEARSON

Chapter 1
Pulmonology

Bryan Bledsoe, DO, FACEP, FAAEM, EMT-P

STANDARD
Medicine (Respiratory)

COMPETENCY
Integrates assessment findings with principles of epidemiology and pathophysiology to formulate a field impression and implement a comprehensive treatment/disposition plan for a patient with a medical complaint.

Learning Objectives

Terminal Performance Objective: After reading this chapter, you should be able to integrate patient assessment findings, patient history, and knowledge of anatomy, physiology, pathophysiology, and basic and advanced life support interventions to recognize and manage patients with pulmonary disorders.

Enabling Objectives: To accomplish the terminal performance objective, you should be able to:

1. Define key terms introduced in this chapter.
2. Identify risk factors that increase the likelihood of developing a respiratory disease.
3. Review the anatomy and physiology of the pulmonary system.
4. Describe pathophysiological changes that lead to disruption of ventilation, diffusion, and perfusion as they relate to the pulmonary system.
5. Integrate the scene size-up, primary assessment, patient history, secondary assessment, and use of monitoring technology to arrive at field impressions and differentials for pulmonary patients.
6. Recognize signs and symptoms of airway compromise, respiratory distress, and respiratory failure.
7. Explain the pathophysiology of respiratory disorders commonly seen in the prehospital environment by the paramedic.
8. Use a process of clinical reasoning to guide and interpret the patient assessment findings and develop a management plan for patients with pulmonary disorders in the prehospital environment.
9. Given a variety of scenarios, discuss the integration of assessment and management guidelines as they relate to pulmonary emergencies.

KEY TERMS

acute respiratory distress syndrome (ARDS), p. 26
apnea, p. 13
asphyxia, p. 16
bradypnea, p. 20
carbaminohemoglobin, p. 12
carboxyhemoglobin, p. 42
carina, p. 5
chronic obstructive pulmonary disease (COPD), p. 3
cor pulmonale, p. 30
crepitus, p. 18
cyanosis, p. 15
deoxyhemoglobin, p. 11
diaphoresis, p. 15
diffusion, p. 11
dyspnea, p. 16
flail chest, p. 13
free radicals, p. 25
hemoglobin, p. 11
hemoptysis, p. 16
hemothorax, p. 13
hyperoxia, p. 25
hypoxia, p. 25
nasal flaring, p. 16
normoxia, p. 25
orthopnea, p. 16
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Case Study

Paramedics Tony Alvarez and Lee Smith are just finishing their barbecue lunch when they are toned out for a "medical emergency." They quickly go to the ambulance for the rest of the dispatch information. The emergency communications center dispatches them to 423 Black Champ Road, where a male patient is reportedly having difficulty breathing. The dispatcher also informs the crew that first responders from the Maypearl Fire Department are already en route. The paramedics are familiar with this area. It is a rural part of the county with mainly cotton farms. The response time is approximately 12 minutes. Upon arrival at the farmhouse, Alice Swenson, an emergency medical responder from the Maypearl Volunteer Fire Department, meets the paramedics. Alice reports that they have a 55-year-old white male who is having difficulty breathing. She further states that oxygen is already being administered.

The paramedics grab the drug box, monitor/defibrillator, airway kit, and stretcher. They then enter the small farmhouse. A quick scene size-up reveals no immediate dangers. Tony and Lee find the patient seated at the kitchen table, obviously short of breath. They quickly perform a primary assessment. The airway is clear, the patient is moving little air, and he has a strong pulse. Tony replaces the nasal cannula placed by the first responders with a nonrebreather mask. Lee and Tony then complete a focused history and physical exam. The patient has diminished breath sounds and occasional rhonchi, and is using the accessory muscles of respiration. There is a hint of cyanosis around his mouth.

The team learns that, several years ago, doctors at the Veterans Administration (VA) hospital diagnosed the patient as having emphysema. Over the past 24 hours, he has had progressive dyspnea and didn't sleep at all the previous night. His wife reports that he paced the floor and repeatedly opened and closed windows. Vital signs reveal a blood pressure of 140/78 mmHg, a pulse of 96 beats per minute, and a respiratory rate of 28 breaths per minute. The monitor shows a sinus rhythm. Pulse oximetry reveals an oxygen saturation of 90 percent while receiving supplemental oxygen. The patient is mentally alert but slightly anxious. His current medications include an albuterol (Ventolin) metered-dose inhaler, montelukast (Singulair), and azithromycin (Zithromax). He still smokes a pack and a half of cigarettes per day and has done so for 40 years, accumulating a 60-pack/year history.

The patient wants to be transported to the VA hospital. Lee contacts medical direction and provides

a brief patient report. Medical direction approves transport to the VA hospital, as it is only 5 miles farther away than the nearest hospital. The transport time will be approximately 40 minutes. The paramedics place a saline lock. In addition, medical direction orders a nebulizer treatment with levalbuterol (Xopenex). Because of the long transport time, medical direction also orders the administration of 125 milligrams of methylprednisolone (Solu-Medrol) by IV push.

Halfway through the nebulizer treatment, the patient shows marked improvement. His respiratory rate slows to 20 breaths per minute, and his oxygen saturation reading increases to 94 percent. Transport to the VA hospital is uneventful. He remains at the VA hospital for two days and is discharged.

Introduction

The respiratory system is a vital body system responsible for providing oxygen to the tissues, while at the same time removing the metabolic waste product, carbon dioxide. Oxygen is required for the conversion of essential nutrients into energy and must be constantly available to all body tissues.

Respiratory emergencies are among the most common emergencies EMS personnel are called on to treat. You will encounter many patients in respiratory distress during your career. As a paramedic, you must promptly recognize and appropriately treat respiratory problems in order to reduce mortality and morbidity.

Several risk factors increase the likelihood of developing respiratory disease. *Intrinsic risk factors* are those that are influenced by or are from within the patient. The most important intrinsic risk factor is genetic predisposition. The likelihood of developing respiratory disease, such as bronchial asthma, **chronic obstructive pulmonary disease (COPD)**, and lung carcinoma (cancer), is increased in patients who have family members with these diseases.

Certain respiratory conditions are increased in patients who have underlying cardiac or circulatory problems. For example, patients with cardiac conditions that result in ineffective pumping of blood are prone to the development of pulmonary edema. In addition, both cardiac and circulatory disease may allow blood to pool in the large veins of the pelvis and lower extremities, leading to the development of pulmonary emboli. Both pulmonary edema and pulmonary emboli often present with a respiratory complaint as the primary complaint. Finally, the patient's level of stress may increase the severity of any respiratory complaint. Remember that stress can actually precipitate acute episodes of asthma or COPD.

CONTENT REVIEW

➤ Factors in Respiratory Disorder Development
- Most important *intrinsic factor:* genetic predisposition
- Most important *extrensic factor:* smoking

Extrinsic risk factors, those that are external to the patient, are also important in increasing the likelihood of developing respiratory disease. The most important of these is cigarette smoking. There is a strong link between cigarette smoking and the development of pulmonary diseases such as lung carcinoma and COPD. Additionally, diseases such as pneumonia and pulmonary emboli are more likely in patients who smoke. Finally, cigarette smoking has been implicated as a risk factor in the development of cardiac disease that may lead to the development of pulmonary edema. In any case, underlying lung damage caused by cigarette smoke causes virtually all lung disorders to be worse in smokers.

Another important extrinsic risk factor is environmental pollutants. Patients who live in highly industrialized areas, particularly where there is little movement of air, are at particular risk for respiratory problems. The prevalence of patients with COPD is markedly increased in areas with high environmental pollutants. The number and severity of acute attacks of both asthma and COPD are also worse under these conditions.

This chapter will help you to develop an understanding of the pathophysiology of respiratory disease, then integrate this knowledge with your assessment findings to develop a field impression and manage the patient with respiratory problems. (Before continuing with this chapter, you may want to review the chapter "Airway Management and Ventilation.")

Review of Respiratory Anatomy and Physiology

As you may recall, the airway is divided anatomically into the upper airway and the lower airway (Figure 1-1).

Upper Airway Anatomy

The upper airway (Figure 1-2) is responsible for warming and humidifying incoming air. It is also very effective in air purification. Each day, approximately 10,000 liters of air are filtered, warmed, humidified, and exchanged by the adult respiratory system.

CONTENT REVIEW

➤ The Upper Airway
- Nasal cavity
- Pharynx
- Larynx

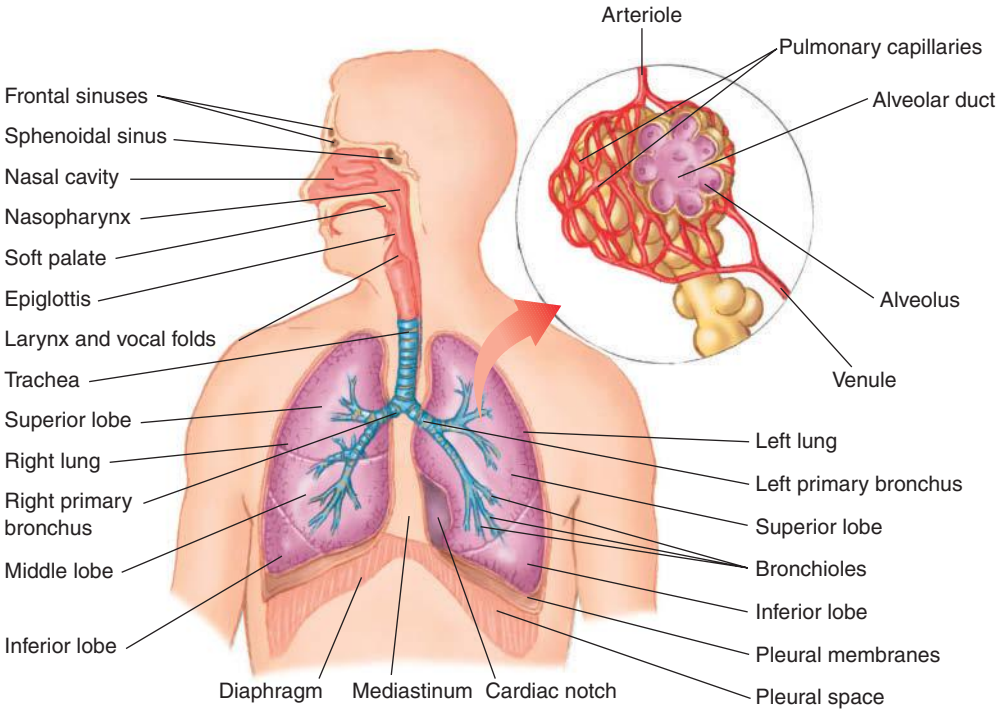

FIGURE 1-1 Overview of the upper and lower airways.

Nasal Cavity

Air enters the upper airway through the nose. It initially passes through the external nares, or nostrils, and enters the nasal cavity. The nasal cavity is divided into two chambers (right and left) by the nasal septum. In the anterior portion of the nose are many hair follicles that help trap large dust particles. The lateral wall of the nasal cavity is marked by three bony prominences called the *turbinates*. Between each set of turbinates is a passageway, or *meatus*, that leads to the *paranasal sinuses* (Figure 1-3). The turbinates cause turbulence in the incoming airflow. This facilitates the entrapment and removal of any inhaled foreign particles, such as dust.

As air passes posteriorly, the thin layer of mucus that lines the nose traps any small inhaled particles not filtered by the hair follicles. This mucus is constantly produced by goblet cells found in the mucous membrane. Some of the

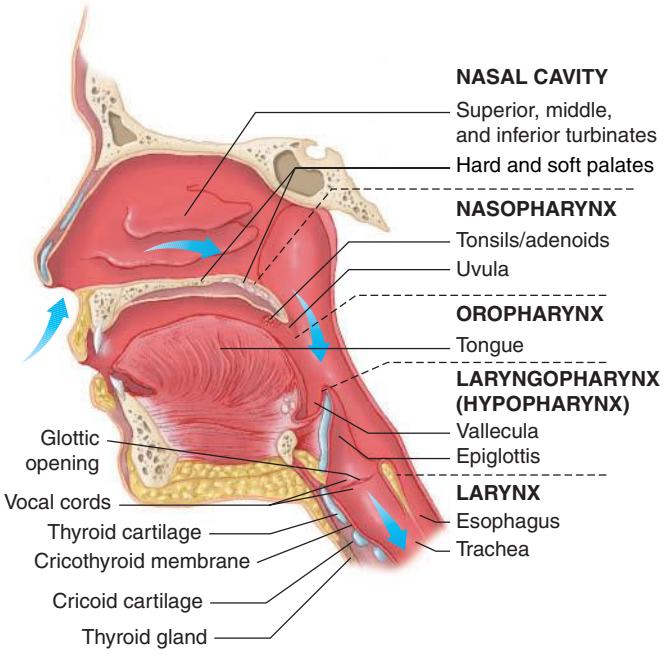

FIGURE 1-2 Anatomy of the upper airway.

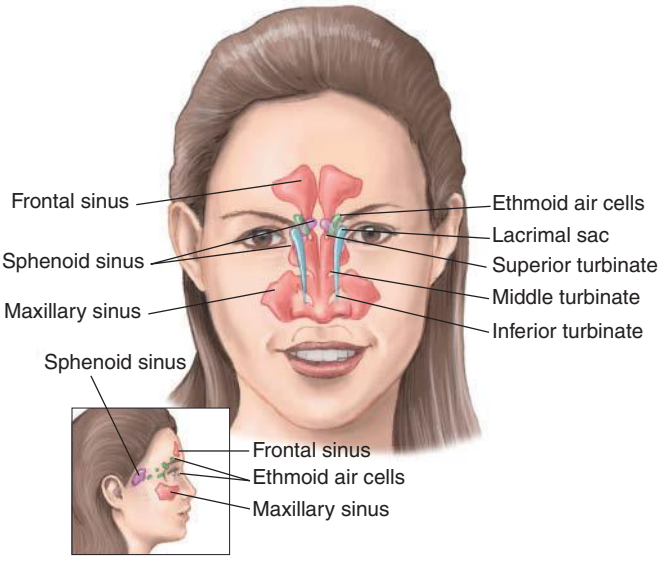

FIGURE 1-3 Paranasal sinuses.

cells lining the respiratory tract have *cilia.* Cilia are thin, fingerlike projections that have the ability to contract in a single direction. In the nose, the cilia move in a manner that produces a steady posterior flow of mucus, at the same time removing any entrapped particles. Once the mucus and any entrapped particles reach the posterior part of the nasopharynx, they are swallowed and removed from the body via the digestive tract.

There is a rich supply of blood vessels, referred to as *Kiesselbach's plexus*, in the lower nasal septum that warms the inspired air. These functions of filtering and warming are also supported by the paranasal sinuses, which are air cavities in the frontal, ethmoid, sphenoid, and maxillary portions of the skull. All are connected to the nasal cavity. The superior portion of the nose contains nerve fibers that are important to our sense of smell (olfactory sense). These fibers, derived from the first cranial nerve (CN-I, the olfactory nerve), pass through the thin cribriform plate that separates the nasal cavity from the cranial cavity.

Pharynx

The *pharynx* is a funnel-shaped structure that connects the nose and mouth to the larynx. It has three divisions: the nasopharynx, the oropharynx, and the laryngopharynx. The *nasopharynx* is the portion of the pharynx that is posterior to the nose and is the most superior aspect of the pharynx. Filtering, humidification, and warming of inspired air continue in the nasopharynx. Both food and air are conducted through the lower divisions of the pharynx, the *oropharynx* and the *laryngopharynx.* The tonsils are nodules of lymphoid tissue that are located in the posterior pharynx. There are three types of tonsils. The pharyngeal tonsils, also called the adenoids, are found in the nasopharynx. The palatine tonsils and lingual tonsils are located in the oropharynx.

Larynx

In addition to its role in speech, the larynx serves as a filtering device for the digestive and respiratory tracts. Externally, you can locate the larynx by feeling the thyroid cartilage, or Adam's apple. The larynx is composed of three pairs of cartilage (arytenoid, corniculate, and cuneiform), the thyroid cartilage, the cricoid cartilage, and the epiglottis. The larynx also possesses two pairs of folds that are derived from the internal lining of the larynx. The upper lining forms a pair of folds called the *vestibule*, or false vocal cords. The lower pair forms the true vocal cords. The vocal cords and the space in between them are referred to as the *glottic opening*. During inspiration, the three paired cartilages remain widely separated, and the epiglottis sits upright so that air can freely enter the trachea. With swallowing, the epiglottis tips backward and the cartilage pairs close, diverting food to the esophagus.

Lower Airway Anatomy

CONTENT REVIEW

- ➤ The Lower Airway
  - Trachea
  - Bronchi
  - Alveoli
  - Lungs

Trachea

During inspiration, air exits the upper airway and passes through the larynx into the *trachea* (Figure 1-4). The trachea is approximately 11 cm in length and is composed of a series of C-shaped cartilaginous rings. It is lined with the same kind of cells that line the nares. Mucus produced by these cells continues to trap air contaminants, and the cilia propel the mucus toward the pharynx. Additionally, stimulation of the trachea by food or other ingested products triggers a coughing response that helps keep the airway free of foreign material. Cigarette smoking ultimately leads to destruction of the cilia, leaving the cough reflex as the only protective mechanism.

Bronchi

At the **carina**, the trachea divides into the right and the left mainstem bronchi. The carina has many nerve endings and stimulation of this area produces violent coughing. The right mainstem bronchus is almost a straight continuation of the trachea, whereas the left mainstem bronchus angles more acutely to the left. This anatomic difference between the two mainstem bronchi helps to explain why gastric contents or other aspirated material tend to pass down the right mainstem bronchus into the lungs. It also explains why pneumonia that results from aspiration occurs more commonly in the right lung. Additionally, this is why, in most instances, an endotracheal tube advanced too far into the trachea will pass into the right mainstem bronchus.

The mainstem bronchi divide into the secondary (lobar) bronchi. These secondary bronchi divide into tertiary (segmental) bronchi, which ultimately divide into the bronchioles, or the small airways. The bronchioles are approximately 1 mm thick and contain smooth muscle that can contract, thus reducing the diameter of the airway.

The conduit system, from the trachea to the terminal bronchioles, must be intact for air to enter the lungs. Both the upper airway and lower airway must be patent so that air may pass through the bronchial system into the alveoli. The upper airway is the gateway to the body's respiratory system, and occlusion by the patient's tongue or a foreign body prevents air from reaching the alveoli. Lower airway disease such as bronchial asthma can have the same result. You can see, therefore, how important it is to maintain a patent airway as you attempt to resuscitate a patient.

After approximately 22 divisions, the bronchioles become terminal bronchioles. The terminal bronchioles divide into the respiratory bronchioles, and it is at this point that the airway shifts from being a conduit for air to

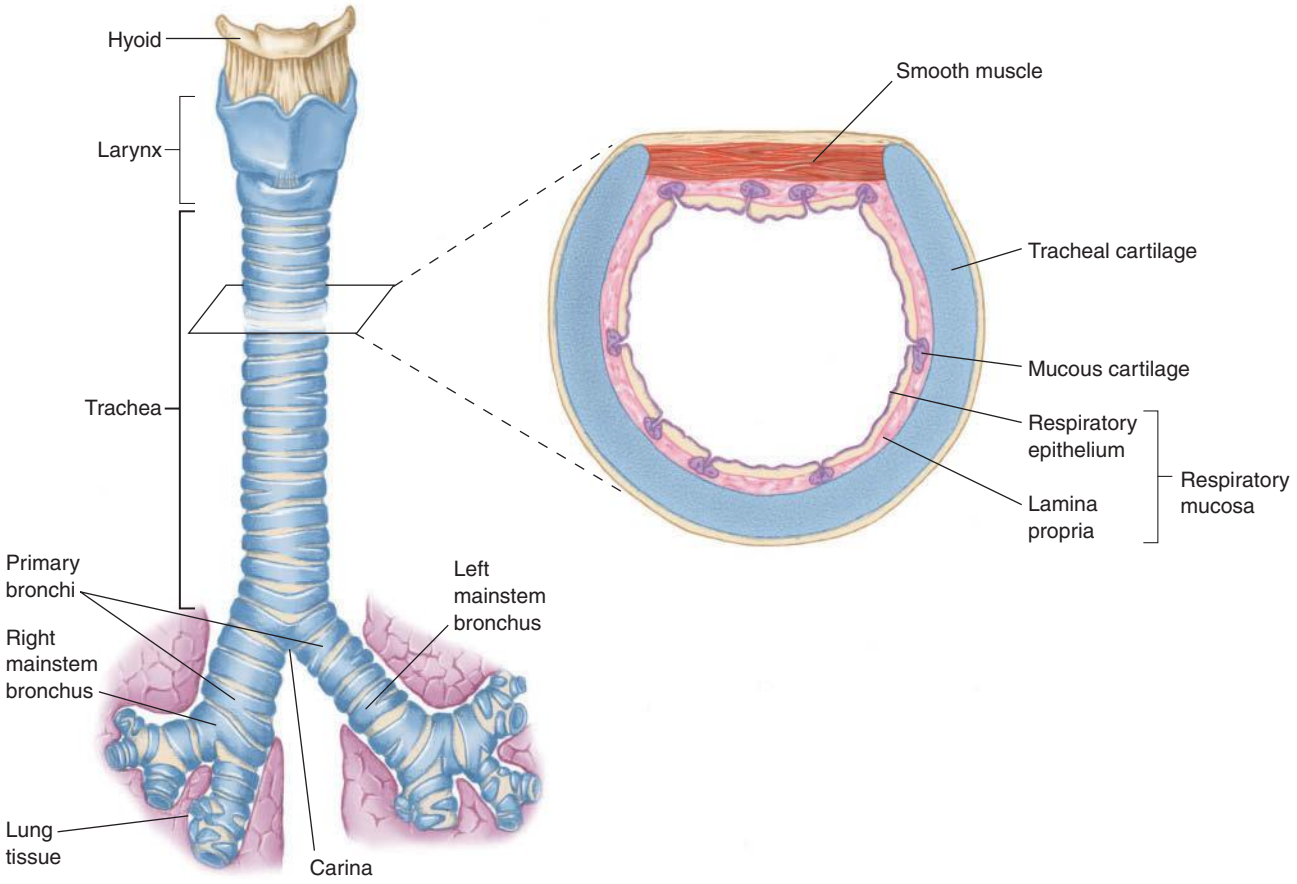

FIGURE 1-4 Anatomy of the lower airway.

an organ of gas exchange. The respiratory bronchioles contain mostly smooth muscle and have limited gas exchange ability.

Alveoli

The respiratory bronchioles divide into the alveolar ducts. These terminate in the alveolar sacs, or alveoli. It is estimated that there may be 300 million alveoli in the lungs. Most of the gas exchange (exchange of oxygen and carbon dioxide) takes place in the alveoli (Figure 1-5), although limited gas exchange may occur in the alveolar ducts and respiratory bronchioles.

The alveolar wall consists of a thin layer of cells (type I cells) that lines the surface of the lung. In close proximity to the alveoli are the pulmonary capillaries. These capillaries carry carbon dioxide-rich blood from the heart into the lungs and oxygen-rich blood away from the lungs for return to the heart. A small amount of supportive tissue contained in the interstitial space separates the capillaries from the alveolar surface (Figure 1-6).

The alveolar lining, supportive tissue, and capillaries make up the *respiratory membrane.* This gas exchange surface measures approximately 70 m^2. Diseases such as emphysema destroy the walls between the alveoli and reduce the total surface area available for gas exchange. When this surface area is reduced by more than two-thirds, oxygen diffusion will be unable to meet the needs of the resting patient.

The alveoli are moistened and kept open because of the presence of an important chemical called **surfactant**

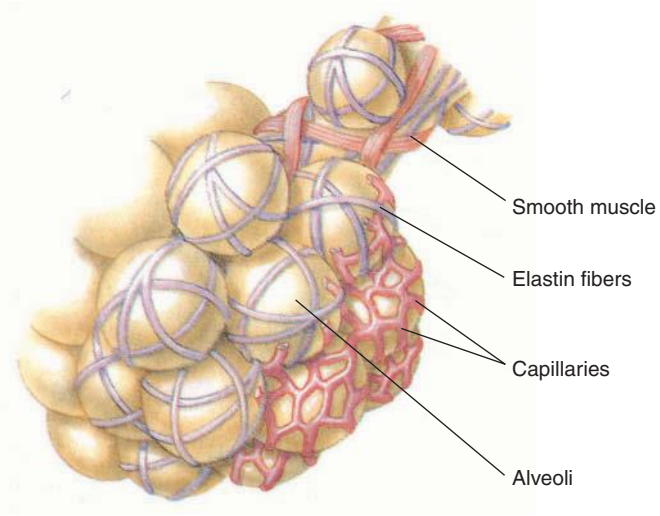

FIGURE 1-5 The alveoli and the pulmonary capillaries.

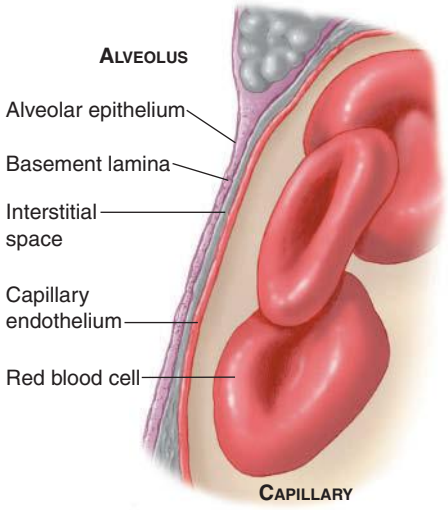

FIGURE 1-6 Supportive tissue contained in the interstitial space that separates capillaries from the alveolar surface.

that is secreted by type II cells found on the alveolar surface. Surfactant tends to decrease the surface tension of the alveoli, thus keeping them open for gas exchange. The alveolar macrophages are another type of cell found within the alveoli. These cells are part of the body's immune system and function to digest particles, bacteria, and other foreign material.

Remember that, in a normal patient, not all of the alveoli remain patent during gas exchange. This means that a small percentage of blood will pass through the alveoli without exchanging oxygen and carbon dioxide. This is referred to as *physiologic shunt* and affects approximately 2 percent of the total blood flow to the lungs.

Lungs

The lungs are the main organs of respiration. The right lung contains three main divisions, or *lobes*, whereas the left lung has only two lobes. The lungs are covered by connective tissue called *pleura.* Unattached to the lung, except at the *hilum* (the point at which the bronchi and blood vessels enter the lungs), the pleura consists of two layers, visceral and parietal. The *visceral pleura* covers the lungs and does not contain nerve fibers. In contrast, the *parietal pleura* lines the thoracic cavity and contains nerve fibers. A small amount of pleural fluid is usually found in the pleural space, a potential space between the two layers of pleura. This fluid serves as a lubricant for lung movement during respiration. The surface tension maintains the contact between the lungs and chest wall (similar to the attractive force that is generated when you place water between two glass slides).

Pulmonary and Bronchial Vessels

Blood is supplied to the lungs through two systems: the pulmonary vessels and the bronchial vessels. The pulmonary arteries transport deoxygenated, carbon dioxide-rich blood from the heart and present it to the lungs for oxygenation. The pulmonary veins then transport the oxygenated blood from the lungs back to the heart. The lung tissue itself receives little of its blood supply from the pulmonary arteries and veins. Instead, bronchial arteries that branch from the aorta provide most of the blood supply to the lungs. Bronchial veins return blood from the lungs to the superior vena cava.

Physiologic Processes

The major function of the respiratory system is to exchange gases with the environment. Oxygen is taken in while carbon dioxide is eliminated, a process known as gas exchange.

Oxygen is vital to our bodies, allowing us to generate the energy that drives our many body functions. Oxygen from the atmosphere diffuses into the bloodstream through the lungs. Oxygen is then available for use in cellular metabolism by the body's 100 trillion cells. Waste products, including carbon dioxide, produced by cellular metabolism must be eliminated from the body. In the lungs, carbon dioxide is exchanged for oxygen, and the carbon dioxide is excreted from the lungs.

Three important processes allow gas exchange to occur:

- Ventilation
- Diffusion
- Perfusion

Ventilation

Ventilation is the mechanical process of moving air in and out of the lungs. For ventilation to occur, several body structures must be intact, including the chest wall, nerve pathways, diaphragm, pleural cavity, and brainstem.

The chest wall consists of a series of ribs that are supported posteriorly by the thoracic spine and anteriorly by the sternum and costal cartilages. Each set of ribs is connected by a thick array of muscles called the *intercostal muscles*. A paired artery and vein, the intercostal vessels, nourish these muscles, which receive their nerve supply from the intercostal nerve. The nerve and blood vessels lie along the lower edge of each rib in a groove on the posterior surface (Figure 1-7). The chest wall is an important component of ventilation and also serves to protect the heart, lungs, and other organs of the thorax.

The *diaphragm*, a dome-shaped muscle, separates the thorax and abdomen. Nerve impulses from the phrenic nerve, which begins in the region of the cervical portion of the spinal cord

CONTENT REVIEW

➤ Processes of Gas Exchange
- Ventilation
- Diffusion
- Perfusion

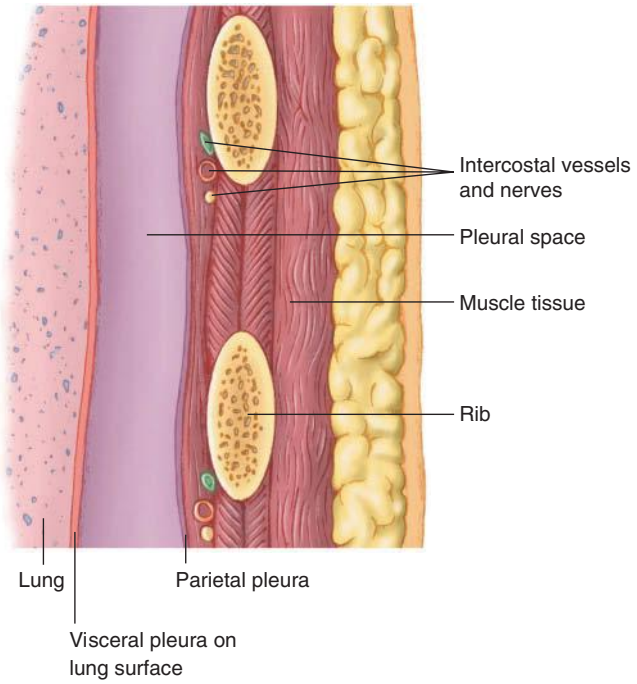

FIGURE 1-7 The intercostal vessels and nerves are located at the inferior borders of the ribs.

and travels through the chest cavity, stimulate the diaphragm to contract. Several traumatic, infectious, and even neoplastic conditions (cancer, tumors) can interrupt the nerve supply to the diaphragm.

INSPIRATION AND EXPIRATION Ventilation is divided into two phases: inspiration and expiration. During inspiration, air is drawn into the lungs. During expiration, air leaves the lungs. These phases of ventilation depend on changes in the volume of the thoracic cavity.

As inspiration begins, the diaphragm contracts and thus flattens. In addition, the intercostal muscles contract, producing an expansion in both the anteroposterior and lateral diameter of the chest cavity (Figure 1-8a). These two actions result in an expansion in the chest volume, which produces a decrease in the air pressure inside the chest cavity. This decrease to approximately 1 to 2 mmHg *below* atmospheric pressure causes air outside the body to be drawn through the trachea into the lungs. During periods of heavy respiratory demand, the accessory muscles of the neck (primarily the sternocleidomastoid and scalene muscles) and abdominal wall are recruited to assist in increasing the chest wall volume. Inspiration is always an active process, requiring energy.

Inspiration is dependent not only on an intact chest wall, but also on an intact pleural cavity. The pleural space has a pressure between 4 and 8 mmHg less than atmospheric pressure. This pressure difference between the lung and pleural space, as well as the surface tension of the pleural fluid, ensures that the lungs will move in concert with the chest wall. You can see how an opening into the pleural cavity from a knife or gunshot wound would severely disrupt the normal ventilatory mechanism. A wound opening would eliminate the negative pressure that exists in the pleural space that causes the lungs to expand with the chest wall.

During expiration, both the chest wall and diaphragm recoil to their normal resting state, which increases the pressure inside the chest to approximately 1 to 2 mmHg *above* atmospheric pressure (Figure 1-8b). This drives air out of the lungs. Expiration is generally a passive process that does not require energy. In some disease states, such as emphysema, however, the normal elasticity of the lungs is

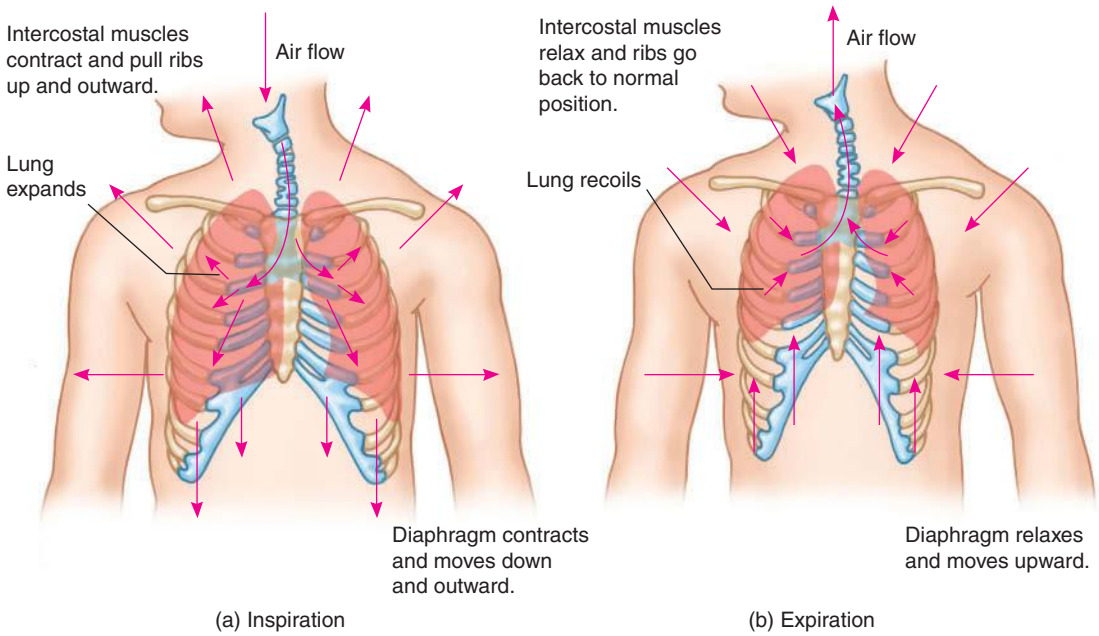

FIGURE 1-8 The phases of respiration: (a) inspiration; (b) expiration.

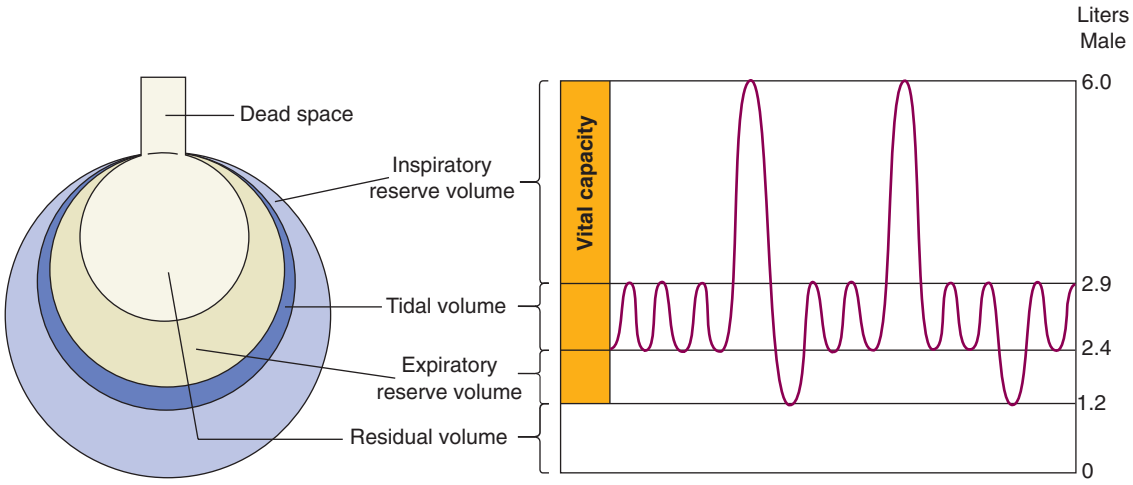

FIGURE 1-9 The lung volumes.

lost. Additionally, during heavy exercise, use of expiratory muscles (such as the rectus muscle and some of the intercostal muscles) is required to generate a larger expiratory effort. In either of these situations, expiration of air becomes an active process, requiring energy.

AIRWAY RESISTANCE AND LUNG COMPLIANCE The amount of airflow into the lungs (ventilation) is dependent not only on the difference between the pressure in the atmosphere and that inside the chest cavity, but also on two additional factors: airway resistance and lung compliance.

The more *airway resistance* (or drag to the flow of air) exists, the less air flows into the chest cavity. Of the passages that conduct air into the alveoli, the medium-sized bronchi offer the greatest resistance to airflow. In patients who have asthma, the smooth muscle within these structures is stimulated by environmental allergens, cold weather, infection, and other factors. This stimulation leads to *bronchospasm* (widespread constriction of the bronchial smooth muscle) and increased resistance to airflow. This makes breathing more strenuous for the patient. The bronchi contain smooth muscle that is also very sensitive to input from the sympathetic nervous system. This is why sympathetic stimulants such as epinephrine, or parasympathetic blocking agents such as atropine or ipratropium bromide (Atrovent), are useful in the treatment of asthma.

Another factor that influences airflow into the lungs is *lung compliance*. Simply stated, compliance refers to the ease with which the chest expands. More specifically, it is defined as the change in volume of the chest cavity that results from a specific change in pressure within the chest cavity. The more the chest wall expands as the result of a change in pressure, the greater the lung compliance. One natural change that occurs with aging is a decrease in lung compliance. This is caused by a loss of elasticity in the muscles, ribs, and cartilage that form the chest wall, which results in a shrinking of the chest wall. The reverse is true with emphysema patients. Because elastic tissue is destroyed in these patients, their lung compliance is abnormally high, so that small changes in pressure result in large expansion of lung volume.

LUNG VOLUMES The volume of air entering the lungs varies based on the metabolic needs of the patient. Several important lung volumes can be measured (Figure 1-9). Factors such as age, sex, physical conditioning, and medical illness will alter these volumes. During quiet respiration, approximately 500 mL of air move in and out of the lungs of a 70-kg adult. This is referred to as the *tidal volume*. The lungs are capable of drawing in an additional volume of air beyond the volume inspired during quiet respiration. This is referred to as the *inspiratory reserve volume*. In an adult male, this volume is approximately 3,000 mL. Similarly, the amount of air that can be forcibly expired out of the lung after a normal breath is referred to as the *expiratory reserve volume* and measures approximately 1,200 mL. An additional 1,200 mL of air remains in the lungs at all times and is important in maintaining the patency of the alveoli. This is called the *residual volume.*

Several calculated volumes can be derived from these volumes that we have already discussed. Such derived volumes are referred to as *lung capacities*. The *inspiratory capacity* is the sum of the tidal volume and inspiratory reserve volume. This is approximately 3,500 mL in adult males. The sum of the expiratory reserve volume and the residual volume is the *functional residual capacity*, which measures approximately 2,400 mL. The *vital capacity* is the amount of air that is measured from a full inspiration to a full expiration. This is the sum of the inspiratory reserve volume, tidal volume, and expiratory reserve volume and measures 4,800 mL. The total volume of air in the lungs, called the *total lung capacity*, measures approximately 6,000 mL in an adult male (Table 1-1).

Table 1-1 Lung Volumes in Healthy Resting Adult Males

Capacity/Volumes (male, in mL)	
Total lung capacity	6,000
Vital capacity	4,800
Inspiratory reserve	3,000
Tidal volume	500
Expiratory volume	1,200
Residual volume	1,200

You may come across several measures of pulmonary function when caring for patients with respiratory disorders. These measurements reflect the dynamic nature of air movement in and out of the lungs. The *minute respiratory volume* is the amount of air moved in and out of the lungs during 1 minute. It is calculated by multiplying the tidal volume and the respiratory rate. For an adult male, the typical minute respiratory volume is approximately 6,000 mL (or 500 mL × 12 breaths per minute). Similarly, the *minute alveolar volume* is the volume of air moving through the alveoli in 1 minute. It is calculated by subtracting the dead space (approximately 150 mL; explanation follows) from the tidal volume and multiplying by the respiratory rate.

The *forced expiratory volume (FEV)* is the volume of air exhaled over a measured period of time. Most commonly, the FEV_1 measures the volume of air expelled during the first second of a forced expiration. Similarly, a *peak flow* measures the maximum rate of airflow during a forced expiration. This is measured in liters of air expiration per minute. Both these measurements are commonly used in the assessment of patients with lung diseases, such as asthma or COPD, in which the expiration of gases may be impaired.

Remember that when a patient breathes in a tidal volume of 500 mL, some of that air rests in the trachea, mainstem bronchi, and bronchioles and is unavailable for gas exchange. This is called the *anatomical dead space* and is approximately 150 mL. You should also remember that under certain conditions, some alveoli might be unavailable for gas exchange (because they are collapsed or are filled with fluid). This is referred to as *alveolar dead space*. This volume varies depending on the degree of alveolar collapse.

REGULATION OF VENTILATION The lower portions of the brainstem, specifically the *medulla,* control ventilation. This area of the brain sends a constant, repetitive signal to the lungs to initiate inspiration. The medulla contains both an inspiratory and an expiratory center. However, because expiration is generally a passive process, the inspiratory center plays a more active role in the rhythm of breathing. The resting rate of respiration varies between 12 and 20 breaths per minute in an adult.

The medullary signal is transmitted through the phrenic and intercostal nerves to the primary muscles of ventilation—that is, to the diaphragm and the intercostal muscles, respectively. The medullary signal can be modified by input from voluntary centers in the cerebral cortex, from other centers in the hypothalamus and brainstem (pons), and from other areas of the medulla. Other receptors throughout the body also provide input to the respiratory center. This allows tight control of ventilation in response to the body's physiologic needs.

Stretch receptors, located on the visceral pleura and on the walls of the bronchi and bronchioles, are important body structures that provide input to the medulla's respiratory center. As the patient continues to inhale, signals from these receptors become stronger until they completely inhibit impulses transmitted from the medulla. As the lungs begin to recoil, the signals become less intense, allowing the medulla to begin another inspiratory phase. This mechanism prevents overinflation of the lungs and is called the *Hering-Breuer reflex.* The medulla also receives input to increase the ventilatory rate from receptors that are stimulated by irritants in the lung and bronchial tree and, additionally, from receptors that detect increased activity in muscles and joints.

The most important determinant of the ventilatory rate is the arterial PCO_2. An increase in the patient's arterial PCO_2 results in a decrease in the **pH** of the blood. An increase in carbon dioxide in the blood also results in an increase in carbon dioxide in *cerebrospinal fluid* (the fluid that bathes the brain and spinal cord). Carbon dioxide and water combine to produce an acid, resulting in a lowering of the pH (increasing the concentration of hydrogen ions) in the cerebrospinal fluid. Chemical receptors in the area of the medulla detect this decrease in the pH, which produces an increase in the ventilatory rate, which helps the body eliminate excess CO_2 and return the pH to a normal level. There are also chemical receptors in the carotid artery and aorta that are directly sensitive to the arterial PCO_2. Stimulation of these receptors by an increase in arterial PCO_2 will also stimulate respiration. Remember that there is instantaneous feedback through these chemical receptors to the medulla so that, once changes in cerebrospinal fluid pH and arterial PCO_2 are corrected, the stimulus to increase respiration ceases.

Unfortunately, regulation of ventilation in patients with COPD does not take place as described. In patients with this disorder, the body becomes less responsive to changes in arterial PCO_2. Instead, the major stimulus to breathing comes from the level of oxygen detected in arterial blood by receptors in the aortic arch. As a result, patients with COPD will achieve a delicate balance in the

PO_2, with the level being low enough to continually stimulate the medulla's respiratory center while having enough oxygen to maintain normal body functions. Measured PO_2 levels of between 50 and 60 mmHg are not uncommon in this patient population.

Diffusion

Diffusion is the process by which gases move between the alveoli and the pulmonary capillaries. Remember that gases tend to flow from areas in which there is a high concentration of gas into an area of low concentration. The normal concentration of oxygen in the alveoli is 104 mmHg, as opposed to a concentration of 40 mmHg in the pulmonary arterial circulation. Therefore, oxygen will move from the oxygen-rich alveoli into the oxygen-poor capillaries in response to the gradient that exists in the concentration of gases. As the red blood cells move through the pulmonary capillaries, they become enriched with oxygen. Less oxygen will pass into the bloodstream as the gradient between alveolar and capillary oxygen concentration decreases.

Similarly, carbon dioxide passes out of the blood in response to a gradient that exists between the concentration of carbon dioxide in the blood in the pulmonary capillaries (45 mmHg) and in the alveoli (40 mmHg). By the time blood leaves the pulmonary capillaries, it has a dissolved concentration of oxygen of 104 mmHg and a carbon dioxide concentration of 40 mmHg.

The respiratory membrane, which normally measures 0.5 to 1.0 micrometer in thickness, must remain intact for gas exchange to occur. Any disorder that damages the alveoli or allows them to collapse will impede oxygen from entering the body and will reduce carbon dioxide elimination. Changes in the respiratory membrane or any increase in the interstitial space will also impede the process of diffusion. For example, fluid accumulation in the interstitial space as the result of pulmonary edema or pneumonia will prevent proper diffusion of gases. Finally, the endothelial lining of the capillaries must be intact for exchange of oxygen and carbon dioxide to occur. Diseases that produce thickening of the endothelial lining will also interfere with the process of diffusion.

There are certain measures you can take to address problems with lung diffusion. Providing the patient with high concentrations of oxygen is one simple step that can be used. Remember that the concentration gradient provides the driving force in moving oxygen into the capillaries. Therefore, the larger the difference between the concentration of oxygen in the alveoli and the capillaries, the greater the diffusion of oxygen into the bloodstream. Similarly, when fluid accumulation or inflammation is the underlying cause of the thickening of the interstitial space within the alveoli, medications such as diuretic agents or anti-inflammatory drugs (corticosteroids) are given to reduce fluid and inflammation.

Perfusion

One additional process that occurs in the lungs is **perfusion**. Lung perfusion is the circulation of blood through the lungs or, more specifically, the pulmonary capillaries. Lung perfusion is dependent on three conditions:

- Adequate blood volume
- Intact pulmonary capillaries
- Efficient pumping of blood by the heart

For perfusion to proceed effectively, there must be an adequate volume of blood in the bloodstream. Equally important is the concentration of **hemoglobin**, which is the transport protein that carries oxygen in the blood. Remember that oxygen is transported in the bloodstream in one of two ways: bound to hemoglobin or dissolved in the plasma. Under normal conditions, less than 2 percent of all oxygen is transported dissolved in plasma (as measured by the PO_2), whereas more than 98 percent is carried by hemoglobin. Hemoglobin with oxygen bound is referred to as **oxyhemoglobin**. Hemoglobin without oxygen is called **deoxyhemoglobin**.

Hemoglobin has some unique properties. It is made up of four iron-containing heme molecules and a protein-containing globin portion. Oxygen molecules bind to the heme portion of the hemoglobin molecule. As oxygen binds to hemoglobin, its structure changes so that it more readily binds additional oxygen molecules. Similarly, as fully oxygen-bound hemoglobin begins to release oxygen, it more readily sheds additional oxygen. The relationship is described by the *oxygen dissociation curve* (Figure 1-10). You can see that, between 10 and 50 mmHg, there is a marked increase in the saturation of hemoglobin. However, as the PO_2 increases above 70 mmHg, there is only a small change in the saturation of hemoglobin, which is already near 100 percent.

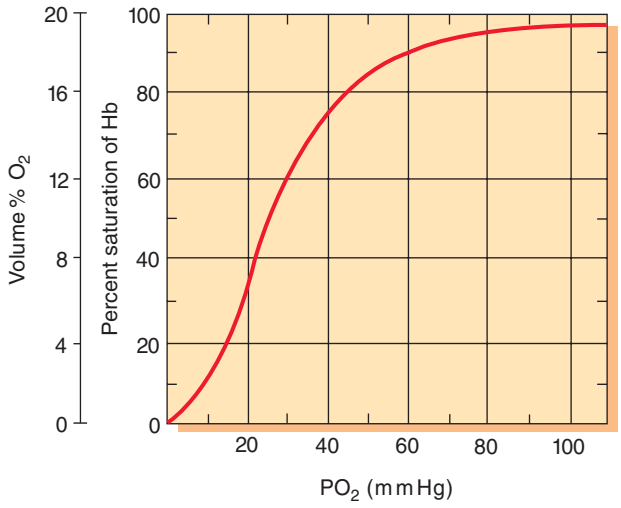

FIGURE 1-10 Oxygen dissociation curve.

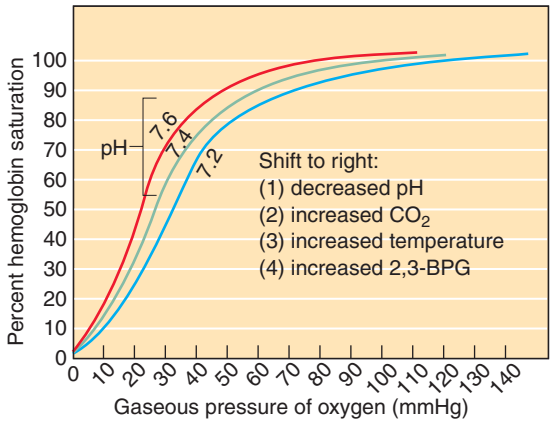

FIGURE 1-11 The Bohr effect.

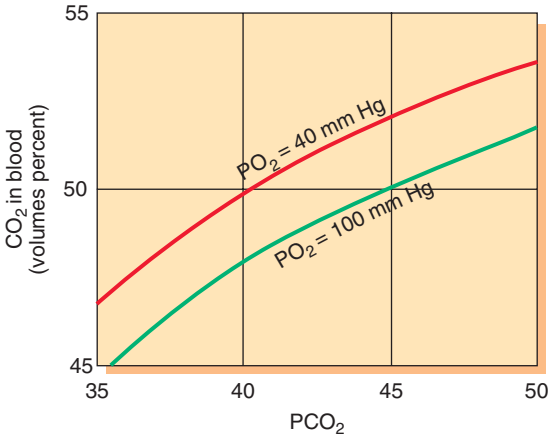

FIGURE 1-12 The Haldane effect.

Changes in the body temperature, the blood pH, and the PCO_2 can alter the oxygen dissociation curve. Within the tissues, as hemoglobin becomes bound with carbon dioxide, it loses its affinity for oxygen. As a result, more oxygen is released and is thus available to cells for metabolism (called the Bohr effect) (Figure 1-11).

Carbon dioxide is transported from the cells to the lungs in one of three ways:

- As bicarbonate ion (70 percent)
- Bound to the globin portion of the hemoglobin molecule (23 percent)
- Dissolved in plasma (measured as PCO_2) (7 percent)

As just noted, the vast majority of carbon dioxide is transported in the form of bicarbonate ion. As the CO_2 is released into the capillaries, it enters the red blood cell where an enzyme (carbonic anhydrase) combines carbon dioxide with water to form two ions, hydrogen (H^+) and bicarbonate (HCO_3^-). Bicarbonate is then released from the red blood cell and transported in plasma. In the lungs, the reverse process takes place, producing water and carbon dioxide. The carbon dioxide then diffuses into the alveoli, where it is eliminated during exhalation.

A limited amount of carbon dioxide is transported bound to hemoglobin. However, unlike oxygen, carbon dioxide does not bind to the same site as does oxygen (the heme site). Instead, it binds to an amino acid in the protein chain that makes up hemoglobin. Hemoglobin with carbon dioxide bound is called **carbaminohemoglobin**. The carbon dioxide that is bound to hemoglobin is released in the lung because of the lower concentration of this gas in the alveoli. Additionally, as the heme portion of the hemoglobin molecule becomes saturated with oxygen, it becomes acidic and more carbon dioxide is released (called the Haldane effect) (Figure 1-12).

Only a fraction of carbon dioxide is transported as a gas. It flows into the alveoli due to the gradient that exists between the concentrations of gases (PCO_2 of 45 mmHg in the pulmonary artery versus 40 mmHg in the alveoli).

For perfusion to take place, in addition to having adequate blood volume, the pulmonary capillaries must be able to transport blood through all portions of the lung tissue. These vessels must be open and not occluded, or blocked. For example, a pulmonary embolism will occlude the pulmonary artery in which it lodges, making that artery unavailable for perfusion of the portion of the lung it usually supplies with blood. Finally, the heart must pump efficiently to push blood effectively through the pulmonary capillaries to perfuse the lung tissues.

To maintain perfusion, you must ensure that the patient has an adequate circulating blood volume. In addition, take the necessary steps to improve the pumping action of the heart. For example, in patients with acute pulmonary edema, the use of diuretic agents reduces the blood return (preload) to an ineffectively pumping heart and improves cardiac efficiency.

The entire system we have just discussed provides for **respiration**, which is the exchange of gases between a living organism and its environment. Pulmonary respiration occurs in the lungs when the respiratory gases are exchanged between the alveoli and the red blood cells in the pulmonary capillaries through the respiratory membranes. Cellular respiration, on the other hand, occurs in the peripheral capillaries. It involves the exchange of the respiratory gases between the red blood cells and the various tissues. Many of the principles of gas exchange that occur in the lungs are reversed in the tissues, with oxygen being released to the cells and carbon dioxide accumulating in the plasma and red blood cells.

Pathophysiology

Remember that many disease states affect the pulmonary system and interfere with its ability to acquire the oxygen required for normal cellular metabolism. Additionally, respiratory diseases limit the body's ability to get rid of waste products such as carbon dioxide. Your understanding

of normal anatomy and physiology—ventilation, diffusion, and perfusion—will aid in understanding the mechanism of each disease process and will direct you toward the appropriate corrective actions. Ultimately, any disease process that impairs the pulmonary system will result in a derangement in ventilation, diffusion, perfusion, or a combination of these processes.

Disruption in Ventilation

Diseases that affect ventilation will result in obstruction of the normal conducting pathways of the upper or lower respiratory tract, impairment of the normal function of the chest wall, or abnormalities involving the nervous system's control of ventilation.

Upper and Lower Respiratory Tracts

Disease states that affect the upper respiratory tract will result in obstruction of airflow to the lower structures. Upper airway trauma, for example, produces both significant hemorrhage and swelling. Infections of the upper airway structures, including epiglottitis, soft tissue infections of the neck, tonsillitis, and abscess formation within the pharynx (peritonsillar abscess and retropharyngeal abscess), can obstruct airflow. A common condition, obstructive sleep apnea, occurs when the tongue (or adjoining tissues) blocks the airway during deep sleep. This results in periods of apnea that usually awaken the patient. Similarly, lower airway obstruction may be produced by trauma, foreign body aspiration, mucus accumulation (as in asthmatics), smooth muscle constriction (in asthma and COPD), and airway edema produced by infection or burns.

Chest Wall and Diaphragm

As you read earlier, the chest wall and diaphragm are mechanical components that are essential for normal ventilation. Traumatic injuries to these areas will disrupt the normal mechanics, causing loss of negative pressure within the pleural space. This occurs in patients with **pneumothorax**, including open pneumothorax, tension pneumothorax, or **hemothorax**. Infectious processes such as empyema (pus accumulation in the pleural space) or inflammatory conditions produce similar effects. Chest wall injuries, including rib fractures or **flail chest** and diaphragmatic rupture, limit the patient's ability to expand the thoracic cavity. Certain neuromuscular diseases, such as muscular dystrophy, multiple sclerosis, or amyotrophic lateral sclerosis (ALS or Lou Gehrig's disease), impair muscular function so as to limit the ability to generate a negative pressure within the chest cavity.

Nervous System

Any disease process that impairs the nervous system's regulation of breathing may also alter ventilation. Central nervous system depressants, such as alcohol, benzodiazepines, or barbiturates, alone or in combination, can alter the brain's response to important signals such as rising PCO_2. Similarly, stroke, diseases, or injuries that involve the respiratory centers within the central nervous system can change the normal ventilatory pattern. In fact, certain abnormal respiratory patterns are produced by specific brain injury (Figure 1-13):

- *Cheyne-Stokes respirations* are a ventilatory pattern with progressively increasing tidal volume, followed by a declining volume, separated by periods of **apnea** at the end of expiration. This pattern is typically seen in older patients with terminal illness or brain injury.
- *Kussmaul's respirations* are deep, rapid breaths that result as a corrective measure against conditions such as diabetic ketoacidosis that produce metabolic acidosis.

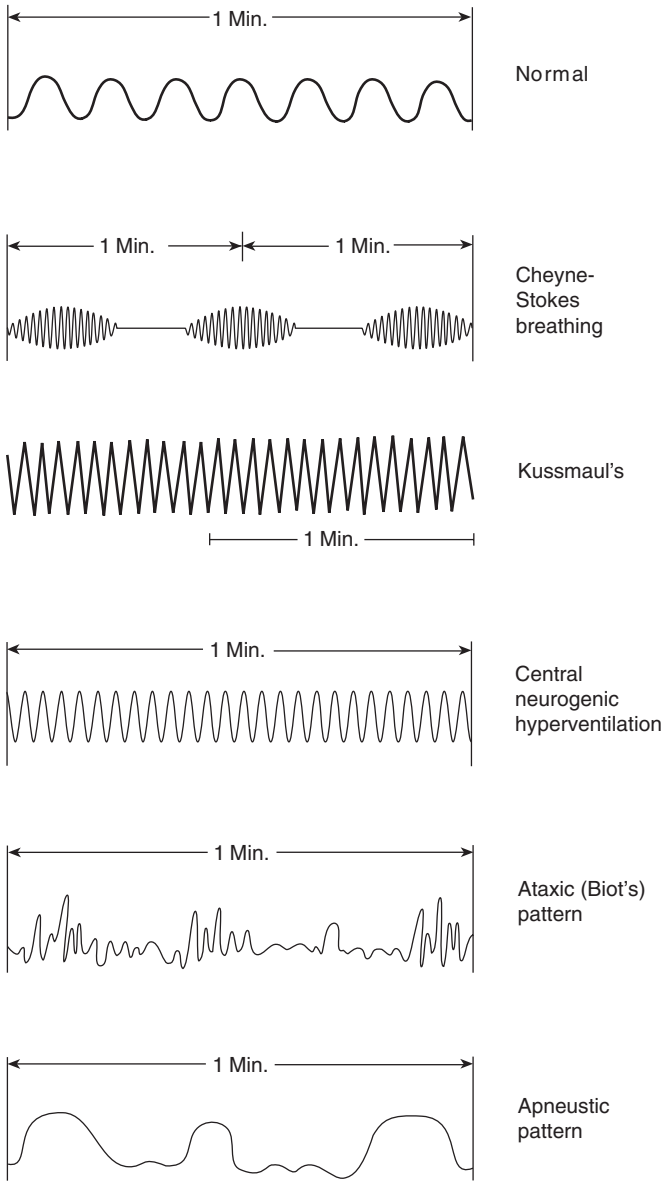

FIGURE 1-13 Abnormal respiratory patterns.

- *Central neurogenic hyperventilation* also produces deep, rapid respirations that are caused by strokes or injury to the brainstem. In this case, there is loss of normal regulation of ventilatory controls and respiratory alkalosis is often seen.
- *Ataxic (Biot's) respirations* are characterized by repeated episodes of gasping ventilations separated by periods of apnea. This pattern is seen in patients with increased intracranial pressure.
- *Apneustic respiration* is characterized by long, deep breaths that are stopped during the inspiratory phase and separated by periods of apnea. This pattern is a result of stroke or severe central nervous system disease.

Also remember that damage to the major peripheral nerves that supply the diaphragm and intercostal muscles, the phrenic nerve, and intercostal nerves will also affect normal ventilatory mechanics. Traumatic disruption of the phrenic nerve during chest surgery, with penetrating trauma, or by neoplastic (cancerous, tumorous) invasion of the nerve can paralyze the diaphragm on the side of involvement.

Disruption in Diffusion

Other disease states can disrupt the diffusion of gases. Any change in the concentration of oxygen in the alveoli, such as that which occurs when a person ascends to high altitudes, can limit the diffusion of oxygen and produce **hypoxia**, a state of insufficient oxygen. Similarly, any disease that alters the structure or patency of the alveoli will limit diffusion. Destruction of alveoli by certain environmental pathogens, such as asbestos or coal (black lung disease), in patients with COPD or in those with inhalation injury reduces the capacity of the lungs to diffuse gases.

Disease states that alter the thickness of the respiratory membrane will limit the diffusion of gases. The most common cause of this alteration is accumulation of fluid and inflammatory cells in the interstitial space. Fluid can accumulate in the interstitial space if high pressure within the pulmonary capillaries forces fluid out of the circulatory system. This is seen in patients with left-sided heart failure (cardiogenic causes) and is due to increased venous pressure as a result of poor functioning of the left ventricle. Patients with pulmonary hypertension have high resting pressures in the pulmonary circulation that ultimately lead to fluid accumulation in the interstitial space, causing right-heart failure.

Similar effects can be produced by changes in the permeability (or leakiness) of the pulmonary capillaries (noncardiogenic causes). Permeability can be affected by acute respiratory distress syndrome, asbestosis and other environmental pathogens, drowning, prolonged hypoxia, and inhalation injury. Also remember that disease states that alter the pulmonary capillary endothelial lining, such as advanced atherosclerosis or vascular inflammatory states, can affect diffusion.

Disruption in Perfusion

As detailed earlier, any alteration in appropriate blood flow through the pulmonary capillaries will limit normal gas exchange in the lungs. Any disease state that reduces the normal circulating blood volume—such as trauma, hemorrhage, dehydration, shock, or other causes of hypovolemia—will limit normal perfusion of the lungs. Remember that hemoglobin is the major transport protein for oxygen and plays a significant role in the elimination of carbon dioxide. Therefore, any reduction in the normal circulating hemoglobin will also affect perfusion. All causes of anemia, a condition in which the number of red blood cells or amount of hemoglobin in them is below normal, must be considered. Such causes include acute blood loss, iron or vitamin deficiency, malnutrition, and anemia from chronic disease states.

Remember that blood must be available to all the lung segments for maximum gas exchange to occur. When an area of lung tissue is appropriately ventilated but no capillary perfusion occurs, available oxygen is not moved into the circulatory system. This is referred to as a *pulmonary shunting*. In patients with pulmonary embolism, a blockage of a division of the pulmonary artery by a clot prevents perfusion of the lung segments supplied by that branch of the artery. As a result, there may be significant shunt with return of deoxygenated blood to the pulmonary venous circulation.

Assessment of the Respiratory System

Assessment of the respiratory system is a vital aspect of prehospital care. You must quickly assess the airway and ventilation status during the primary assessment. If the patient's complaints suggest that the respiratory system is involved in the patient's problem, the focused history and physical examination should be directed to this aspect of the assessment.

Scene Size-Up

When you approach the scene, consider two major questions: (1) Is the scene safe to approach the patient? (2) Are there visual clues that might provide information regarding the patient's medical complaint?

Remember that several hazards may result in respiratory complaints by the patient that are also potentially dangerous for emergency care providers. Certain gases and

toxic products that are causing respiratory complaints from the patient may also present a significant risk to you. Dust particles are also a risk. Some rescuers involved in the World Trade Center attacks in 2001 and the Oklahoma City bombing in 1995 developed both acute and chronic respiratory problems following dust exposure. Carbon monoxide, for example, is a colorless and odorless gas that may be present in quantities large enough to overcome unsuspecting emergency care personnel. Other toxins from incomplete combustion produced in fires or industrial processes pose a similar risk. Past incidents involving chemical agents such as sarin gas or biologic agents such as anthrax highlight the need for emergency care providers to be aware of hazards to themselves as well as to their patient.

You should also be aware that in certain rescue environments, the concentration of available oxygen is significantly reduced. This would include areas such as grain silos, enclosed storage containers, or any enclosed space in which there is an active fire. You must take the appropriate precautions before entering such environments, including the use of your own supplemental oxygen supply.

In any situation in which you believe there is a hazard to you as a care provider, make sure that the scene is appropriately secured before you enter. If specific protective items such as hazardous materials suits, self-contained breathing apparatus (SCBA), or supplemental oxygen are needed, make sure they are available before you attempt to care for your patient. Similarly, if other personnel such as fire suppression units or hazmat teams are required, contact dispatch and have them available on scene before putting yourself at risk.

Once it is safe to enter the scene, look for clues that will provide information regarding the patient's complaints. Do you see evidence of cigarette packs or ashtrays to suggest that the patient or family members are smokers? Look for any home nebulizer machines or supplemental oxygen tanks that may suggest a patient with underlying COPD or asthma. Look for possible sources of carbon monoxide exposure. If the patient is a small child, look for small items lying around the house that could suggest potential ingested foreign bodies. Using your eyes, ears, and nose can lead you to several important clues that are useful as you begin your assessment of the patient.

Primary Assessment

General Impression

Take the following considerations and steps to help form your initial impression of the patient's respiratory status:

- ***Position.*** Consider the patient's position. Patients with respiratory diseases tend to tolerate an upright posture better than lying flat. Indications of severe respiratory distress include a patient who is sitting upright with feet dangling over the side of the bed. In the most severe cases, the patient will assume the "tripod" position in which he leans forward and supports his weight with the arms extended (Figure 1-14).

CONTENT REVIEW

- General Impression of Respiratory Status
  - Position
  - Color
  - Mental status
  - Ability to speak
  - Respiratory effort

- ***Color.*** Patients with severe respiratory distress display **pallor** and **diaphoresis**. **Cyanosis** is a late finding and may be absent even with significant hypoxia. Peripheral cyanosis (bluish discoloration involving only the distal extremities) is not a specific finding and is also found in patients with poor circulation. Peripheral cyanosis reflects the slowing of blood flow and increased extraction of oxygen from red blood cells. Central cyanosis (involving the lips, tongue, and truncal skin) is a more ominous finding seen in hypoxia.
- ***Mental status.*** Briefly assess the patient's mental status. The hypoxic patient will become restless and agitated. Confusion is seen with both hypoxia (deficiency of oxygen) and hypercarbia (excess of carbon dioxide). When respiratory failure is imminent, the patient will appear severely lethargic and somnolent. The eyelids will begin to droop and the head will bob with each respiratory effort.
- ***Ability to speak.*** Assess the patient's ability to speak in full, coherent sentences. Determine the ease with

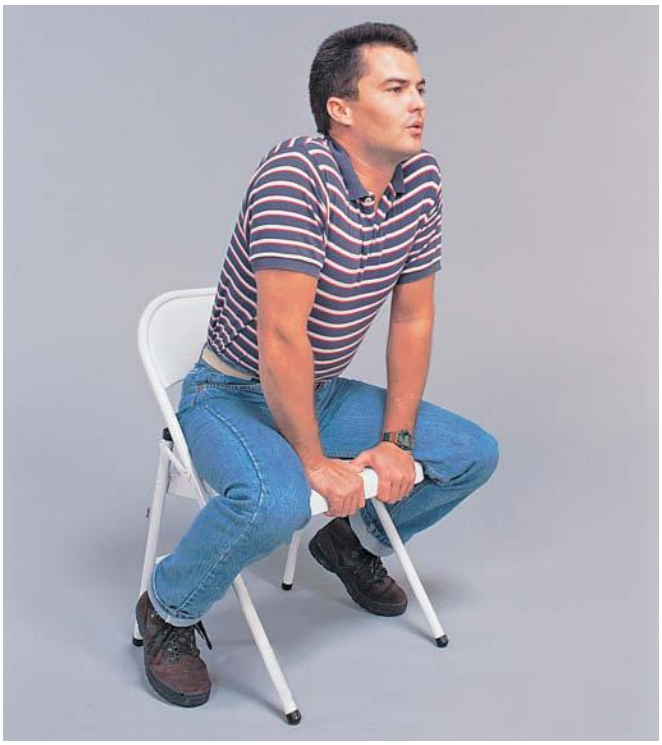

FIGURE 1-14 Tripod position.

which the patient can discuss symptoms. Patients with respiratory distress will be able to speak only one to two words before they need to pause to catch their breath. Rambling, incoherent speech indicates fear, anxiety, or hypoxia.

- ***Respiratory effort.*** As described, normal ventilation is an active process. However, the use of accessory muscles in the neck (scalenes and sternocleidomastoids) and visible contractions of the intercostal muscles indicate significant breathing effort.

As you form your general impression, also make specific note of any of the following signs of respiratory distress:

- **Nasal flaring**
- Intercostal muscle retraction
- Use of the accessory respiratory muscles
- Cyanosis
- Pursed lips
- **Tracheal tugging**

Your primary assessment of the patient is directed at identification of any life-threatening conditions resulting from compromise of airway, breathing, or circulation (the ABCs). Remember that in presumed cardiac arrest, circulation comes before airway and breathing. Because this chapter concerns the respiratory system, we focus here on assessment of airway and breathing.

Airway

Remember that oxygen is one of the most basic necessities for life, and the respiratory system is responsible for supplying it to the body tissues. As a result, any significant abnormality in the respiratory tract must be viewed as potentially life threatening.

After quickly forming your general impression, immediately focus on the patient's airway. When assessing the airway, keep these principles in mind:

- Noisy breathing nearly always means partial airway obstruction.
- Obstructed breathing is not always noisy.
- The brain can survive only a few minutes in **asphyxia**.
- Artificial respiration is useless if the airway is blocked.
- A patent airway is useless if the patient is apneic.
- If you note airway obstruction, do not waste time looking for help or equipment. Act immediately.

If the airway is compromised, quickly institute basic airway management techniques. Once you have secured a patent airway, ensure that the patient has adequate ventilation. Your primary assessment of the respiratory system should be brief and directed. A more detailed examination should be conducted once you have been able to establish that an immediate threat to life does not exist.

Breathing

The following signs should suggest a possible life-threatening respiratory problem in adults. They are listed in order from most ominous to least severe.

- Alterations in mental status
- Severe central cyanosis
- Absent breath sounds
- Audible stridor
- One-to-two-word **dyspnea** (need to breathe after every word or two)
- **Tachycardia** ≥ 130 beats per minute
- Pallor and diaphoresis
- Presence of intercostal and sternocleidomastoid retractions
- Use of accessory muscles

If any of these signs are present, direct your efforts toward immediate resuscitation and transport of the patient to a medical facility.

Secondary Assessment

History

The history and physical exam should be directed at problem areas as determined by the patient's chief complaint or primary problem. Patients with respiratory diseases will often present with a complaint of "shortness of breath" (dyspnea). Obtain a SAMPLE history. If the chief complaint suggests respiratory disease, ask the OPQRST questions, including the following questions about the current symptoms. The answers to these or similar questions will provide you with a pertinent patient history.

- How long has the dyspnea been present?
- Was the onset gradual or abrupt?
- Is the dyspnea better or worse by position? Is there associated **orthopnea** or **paroxysmal nocturnal dyspnea**?
- Has the patient been coughing?
- If so, is the cough productive?
- What is the character and color of the sputum?
- Is there any **hemoptysis** (coughing up of blood)?
- Is there any chest pain associated with the dyspnea?
- If so, what is the location of the pain?
- Was the onset of pain sudden or slow?
- What was the duration of the pain?
- Does the pain radiate to any area?

- Does the pain increase with respiration?
- Are there associated symptoms of fever or chills?
- What is the patient's past medical history?
- Does the patient have obstructive sleep apnea?
- Has the patient experienced wheezing?
- Is the patient or close family member a smoker?

It is also important to ask the patient whether he has ever experienced similar symptoms in the past. Patients with chronic medical conditions such as COPD or asthma can usually relate the severity of their current presenting complaints to other episodes that they have experienced. Question the patient or family about prior hospitalizations for respiratory disease. In particular, you should try to determine whether the patient required care in the intensive care unit (ICU) for breathing problems. Ask whether the patient has ever required endotracheal intubation and ventilatory support. Consider patients who have been previously intubated to be potentially seriously ill and approach them with great caution.

Similarly, it is important to ask the patient whether he already has a known respiratory disease. The most common reason for a call to emergency care personnel is a worsening of an already present respiratory disease. This is typical for patients with COPD, asthma, or lung cancer. If you are not familiar with the patient's diagnosis (for example, alpha-1 antitrypsin deficiency), try to determine whether the disease is affecting the process of ventilation, diffusion, or perfusion.

Continue history taking by determining:

- What current medications is the patient taking? (Pay particular attention to oxygen therapy, oral bronchodilators, corticosteroids, and antibiotics.)
- Does the patient have any allergies?

A good history of medication use is essential and may provide useful clues to the diagnosis. If time permits, gather the patient's current medications and transport them with the patient. This is a great benefit to the emergency department personnel who will be evaluating the patient. Pay particular attention to any medications that suggest pulmonary disease. These would include inhaled or oral sympathomimetics such as albuterol and related agents that are used to treat diseases such as COPD or asthma. Also ask about steroid preparations, which are used in these conditions. Other common medications used by patients with COPD or asthma include cromolyn sodium, methylxanthines (e.g., theophylline), and antibiotic agents.

Ask if the patient has a home nebulizer unit and how frequently it is used. Inquire about the use of a continuous positive airway pressure (CPAP) device (or similar device) for obstructive sleep apnea.

Also ask about drugs used for cardiac conditions, as cardiac patients often present with dyspnea. Patients with cardiac disease commonly use nitrates, calcium channel blockers, diuretic agents, digoxin, and certain antiarrhythmic agents.

Finally, inquire about medication allergies. This information is important because it helps to avoid administering agents to which the patient is allergic. It is also possible that a specific medication may be the cause of an allergic reaction that has resulted in upper airway edema and respiratory complaints.

CONTENT REVIEW

- ➤ Physical Exam of the Respiratory System
  - Head
  - Neck
  - Chest
    - Inspection
    - Palpation
    - Percussion
    - Auscultation
  - Extremities

Physical Examination

First address the patient's head and neck. Look at the lips. Pursed lips indicate significant respiratory distress. This is the patient's way of maintaining positive pressure during expiration and preventing alveolar collapse. Also examine the nose, mouth, and throat for any signs of swelling or infection that might be causing upper airway obstruction.

Occasionally, the patient may produce sputum, which can suggest an underlying cause of the patient's complaints. An increase in the amount of sputum produced suggests infection of the lungs or bronchial passages (bronchitis). Thick green or brown sputum is characteristic of these infections. On the other hand, thin yellow or pale gray sputum is more typical of inflammation or an allergic cause. Pink, frothy sputum is a sign of severe pulmonary edema. Truly bloody sputum (hemoptysis) may be seen with cancer, tuberculosis, and bronchial infection.

Assess the neck for signs of swelling or infection. Remember to look at the jugular veins for evidence of distention (Figure 1-15). This occurs when the right side of

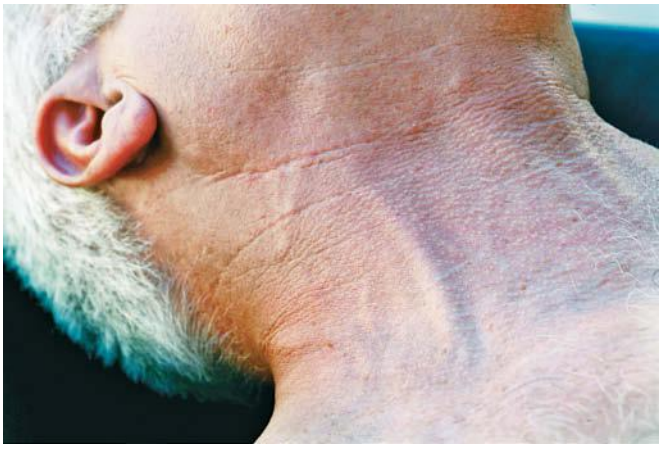

FIGURE 1-15 Jugular vein distention.

the heart is not pumping blood effectively, causing a "backup" in the venous circulation. Such findings are often accompanied by left-sided heart failure and pulmonary edema.

Physical examination of the respiratory system should follow the standard steps of patient assessment: inspection, palpation, percussion, and auscultation.

- ***Inspection.*** Inspection should include an examination of the anterior–posterior dimensions and general shape of the chest (Figure 1-16). An increased anterior–posterior diameter is suggestive of COPD. Inspect the chest for symmetrical movement. Any asymmetry may be suggestive of trauma. A paradoxical movement (moving in a fashion opposite to that expected) is suggestive of flail chest. Note any chest scars, lesions, wounds, or deformities.
- ***Palpation.*** Palpate the chest, both front and back, for abnormalities (Figure 1-17). Note any tenderness, **crepitus**, or **subcutaneous emphysema**. Palpate the anterior chest first, then the posterior. Inspect your gloved hands for blood each time you remove them from behind the patient's chest. In some instances, it may be appropriate to evaluate **tactile fremitus**, the vibration felt in the chest during speaking. When evaluating tactile fremitus, compare one side of the chest with the other. Simultaneously, palpate the trachea for **tracheal deviation**, which is suggestive of a tension pneumothorax.

FIGURE 1-17 The chest should be palpated.

- ***Percussion.*** If indicated, quickly percuss the chest (Figure 1-18). Limit percussion to suspected cases of pneumothorax and pulmonary edema. A hollow sound on percussion is often indicative of pneumothorax or

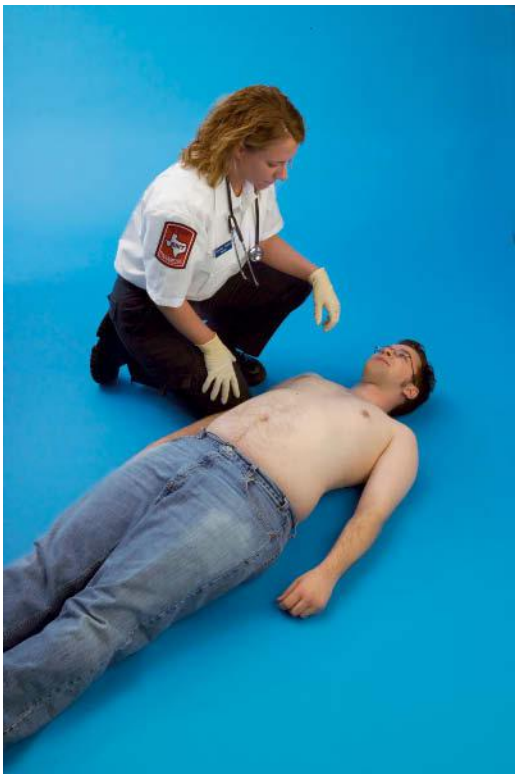

FIGURE 1-16 Inspection of the chest.

FIGURE 1-18 If indicated, the chest should be percussed.

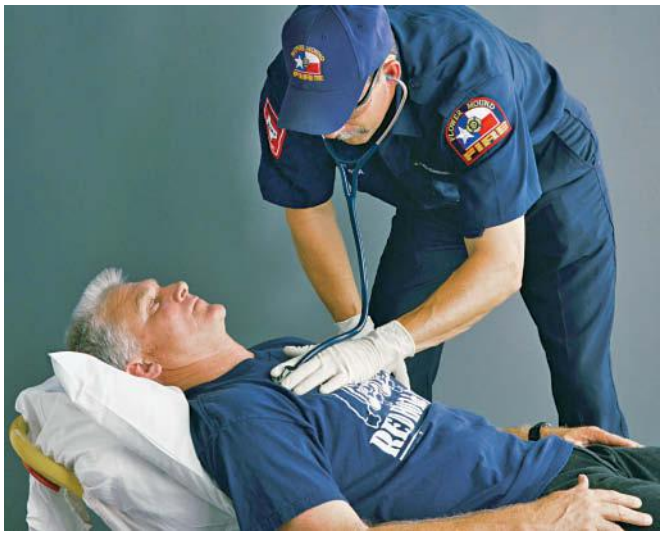

FIGURE 1-19 The chest should be auscultated.

emphysema. In contrast, a dull sound is indicative of pulmonary edema, hemothorax, or pneumonia. Remember, however, that percussion may be of little value in the noisy environment typical of most emergency scenes.

- ***Auscultation.*** Auscultate the chest. Begin by listening to the patient without a stethoscope and from a distance. Note any loud stridor, wheezing, or cough. If possible, the patient should be in the sitting position and the chest auscultated in a symmetrical pattern. When the patient cannot sit up, auscultate the anterior and lateral parts of the chest (Figure 1-19). Each area should be auscultated for one respiratory cycle.

Normal breath sounds heard during auscultation can be characterized according to the following descriptions.

Normal Breath Sounds

- Bronchial (or tubular)
  - Loud, high-pitched breath sounds heard over the trachea
  - Expiratory phase lasts longer than inspiratory phase
- Bronchovesicular
  - Softer, medium-pitched breath sounds heard over the mainstem bronchi (below clavicles or between scapulae)
  - Expiratory phase and inspiratory phase equal
- Vesicular
  - Soft, low-pitched breath sounds heard in the lung periphery

While the patient breathes in and out deeply with the mouth open, note any abnormal breath sounds and their location. Many terms are used to describe abnormal breath sounds. The following list includes some of the more common terms.

Abnormal Breath Sounds

- ***Snoring.*** Occurs when the upper airway is partially obstructed, usually by the tongue.
- ***Stridor.*** Harsh, high-pitched sound heard on inspiration and characteristic of an upper airway obstruction such as croup.
- ***Wheezing.*** Whistling sound due to narrowing of the airways by edema, bronchoconstriction, or foreign materials.
- ***Rhonchi.*** Rattling sounds in the larger airways associated with excessive mucus or other material.
- ***Crackles*** (also called rales). Fine, moist crackling sounds associated with fluid in the smaller airways.
- ***Pleural friction rub.*** Sounds like dried pieces of leather rubbing together; occurs when the pleura become inflamed, as in pleurisy.

Also examine the extremities. Look for peripheral cyanosis, which may indicate hypoxia. Examine the extremities for swelling, redness, and a hard, firm cord indicating a venous clot. This may suggest a possible cause for pulmonary embolism. Look for clubbing of the fingers (Figure 1-20), suggesting long-standing hypoxemia. This is typical of patients with COPD or cyanotic heart disease. Finally, the patient may demonstrate *carpopedal spasm*, in which the fingers and toes are contracted in flexion. This is found in patients with hyperventilation and is caused by transient shifts in the blood calcium concentration due to changes in the serum CO_2 and pH levels.

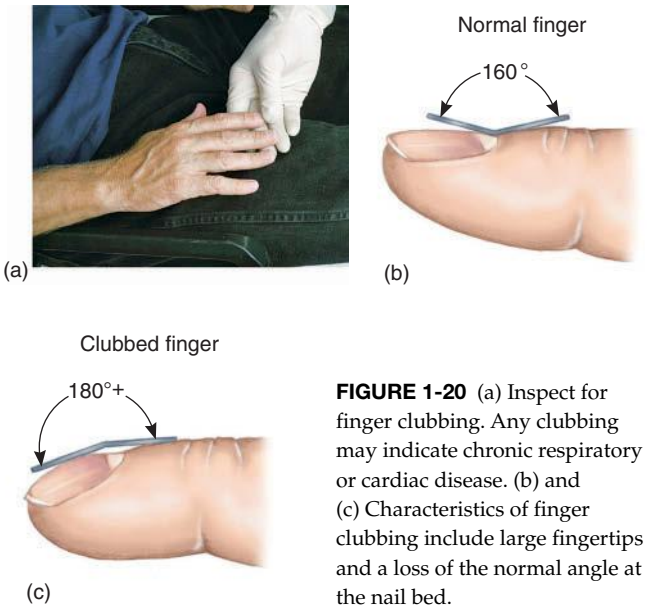

FIGURE 1-20 (a) Inspect for finger clubbing. Any clubbing may indicate chronic respiratory or cardiac disease. (b) and (c) Characteristics of finger clubbing include large fingertips and a loss of the normal angle at the nail bed.

Vital Signs

The patient's vital signs may also provide information regarding the severity of the respiratory complaints. In general, tachycardia (rapid heart rate) is a very nonspecific finding, seen with fear, anxiety, and fever. In patients with respiratory complaints, however, tachycardia may also indicate hypoxia. Remember that the patient may have recently used sympathomimetic drugs such as albuterol, which will accelerate the heart rate. These same drugs will elevate the patient's blood pressure as well. During your assessment of the blood pressure, a patient will occasionally exhibit *pulsus paradoxus,* a drop in the systolic blood pressure of 10 mmHg or more with each respiratory cycle. *Pulsus paradoxus* is associated with COPD and cardiac tamponade. As a rule, however, you should not take the time to look for *pulsus paradoxus*.

A change in a patient's respiratory rate may be one of the earliest indicators of respiratory disease. The patient's respiratory rate can be influenced by several factors, including respiratory difficulty, fear, anxiety, fever, and underlying metabolic disease. Assume that an elevated respiratory rate in a patient with dyspnea is caused by hypoxia. Although fluctuations in the respiratory rate are common, a persistently *slow* rate indicates impending respiratory arrest.

Continually reassess the patient's respiratory rate during the time that you are caring for the patient. Trends in the respiratory rate (for example, an increasing rate) can give you an overall assessment of the effectiveness of any intervention you have made. Also assess the patient's respiratory pattern. The normal respiratory pattern (eupnea) is steady, even breaths occurring 12 to 20 times per minute with an expiratory phase that lasts between 3 to 4 times as long as the inspiratory phase. **Tachypnea** describes a respiratory pattern with a rate that exceeds 20 breaths per minute. **Bradypnea** describes a respiratory pattern with a rate slower than 12 breaths per minute. Look also for any abnormal respiratory patterns (e.g., Cheyne-Stokes, Kussmaul's, or other), as discussed earlier in the chapter.

Diagnostic Testing

Three diagnostic measurements are of value in assessing the patient's respiratory status: *pulse oximetry, peak flow,* and *capnography.*

PULSE OXIMETRY Pulse oximetry offers a rapid and accurate means for assessing oxygen saturation (Figure 1-21). The pulse oximeter can be quickly applied to a finger or earlobe. The pulse rate and oxygen saturation can be continuously recorded. Pulse oximeter probes contain two light-emitting diodes (LEDs). One diode emits light in the red range (660 nm) and the other emits light in the infrared (940 nm) range. Photodetectors on the opposite side of the probe detect the two wavelengths of light that penetrate the tissues. Deoxyhemoglobin absorbs more red light than does oxyhemoglobin. Oxyhemoglobin absorbs more infrared light than red light. The ratio of the two types of absorbed light is calculated by the oximeter and compared against standardized values. The concentration of oxyhemoglobin (and thus oxygen saturation) is displayed as a percentage called the *hemoglobin oxygen saturation*. The oxygen saturation measurement obtained through pulse oximetry is abbreviated as SpO_2. Oxygen saturations obtained through blood gas analysis in hospitals are abbreviated SaO_2. Normally, the SpO_2 and SaO_2 are the same.

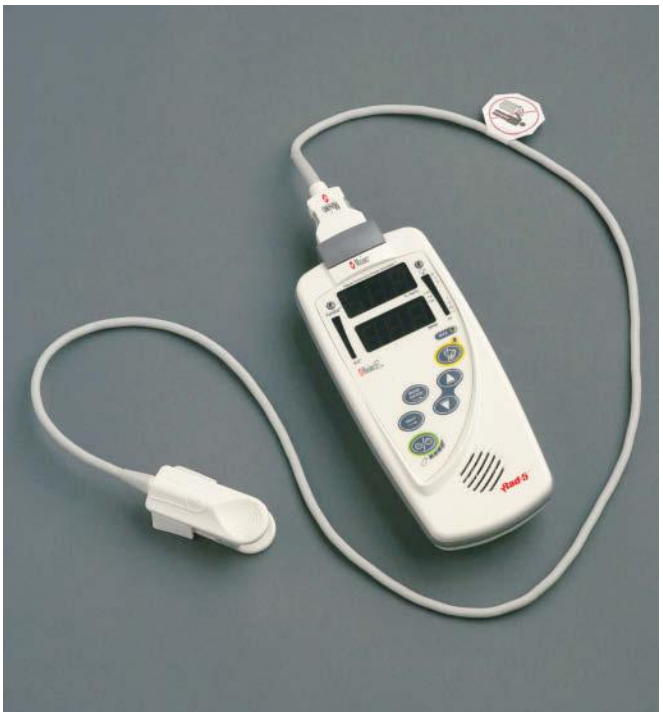

FIGURE 1-21 Sensing unit for pulse oximetry. This device transmits light through a vascular bed, such as in the finger, and can determine the oxygen saturation of red blood cells. To use the pulse oximeter, it is only necessary to turn the device on and attach the sensor to a finger. The desired graphic mode on the oximeter should be selected. The oxygen saturation and pulse rate can be continuously monitored.

Older pulse oximeters were prone to give abnormal or inconsistent readings in patients with peripheral vasoconstriction (as in sepsis or hypothermia) or low-flow states. They also tended to be inaccurate under conditions in which an abnormal substance such as carbon monoxide was bound to hemoglobin, as the instrument measures the saturation of hemoglobin without indicating which substance has saturated it. Fortunately, second-generation pulse oximeters have technology (filters, signal processors) that minimizes these effects, making them much more accurate and much less sensitive to extraneous factors.

CONTENT REVIEW

➤ Prehospital Diagnostic Tests
- Pulse oximetry
- Peak flow
- Capnometry
- CO-oximetry

FIGURE 1-22 The ability to noninvasively determine the amount of hemoglobin present (SpHb) is available on certain monitoring technologies.

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FIGURE 1-23 Combining the oxygen saturation (SpO_2) with the total hemoglobin (SpHb) allows for the calculation of the oxygen content of arterial blood (SpOC).

(© Dr. Bryan E. Bledsoe)

FIGURE 1-24 Modern prehospital patient monitors now allow for continuous monitoring of multiple physiologic parameters.

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It is important to note that the SpO_2 reflects the oxygen saturation of available hemoglobin. Most pulse oximeters cannot discern between normal and abnormal levels of hemoglobin. For example, in a patient with anemia or who has suffered significant hemorrhage, the total amount of hemoglobin available to transport oxygen is reduced. However, most pulse oximeters will reflect a normal SpO_2 despite the fact that tissue delivery of oxygen may be impaired because of inadequate hemoglobin. Some of the newer pulse oximeters have the capability of noninvasively measuring total hemoglobin (SpHb) in addition to SpO_2 and other parameters (Figure 1-22).[1] This provides more accurate information about the total amount of oxygen being transported. This value is referred to as the *oxygen content* and, when being measured noninvasively, is referred to as the SpOC (Figure 1-23).

Use of the pulse oximeter, if available, is encouraged for any patient complaining of dyspnea or respiratory problems (Figure 1-24). It is important to remember that the pulse oximeter is designed to supplement the physical examination and not replace it. However, oxygen saturation readings can help you guide care. Table 1-2 details how to interpret oxygen saturation readings and suggested interventions.

PEAK FLOW Handheld devices are available for use in determining the patient's peak expiratory flow rate (PEFR). The normal expected peak flow rate is based on the patient's sex, age, and height. Remember that the measurement of the peak expiratory flow rate is somewhat effort dependent; you must have a cooperative patient who understands the use of the device to get an accurate reading.

Table 1-2 Treatment Strategies Based on Pulse Oximetry Readings

SpO_2	Interpretation	Suggested Intervention
95–100%	Normal	Increase or decrease the FiO_2 to maintain a normal SpO_2
91–94%	Mild hypoxemia	Increase FiO_2 to increase oxygen saturation
85–90%	Moderate hypoxemia	Increase FiO_2 to increase oxygen saturation Assess and increase ventilation as indicated
<85%	Severe hypoxemia	Increase FiO_2 to increase oxygen saturation Increase ventilation

The PEFR is obtained using a Wright spirometer (Figure 1-25), which is inexpensive and easy to use. Place the disposable mouthpiece into the meter. First have the patient take in the deepest possible inspiration. Then encourage the patient to seal his lips around the device and

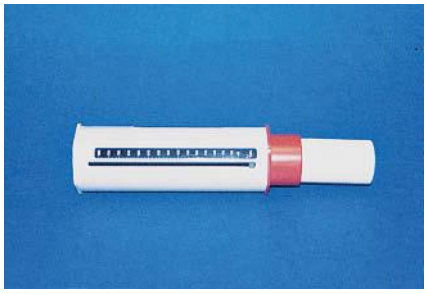


FIGURE 1-25 Wright spirometer for determining peak expiratory flow rate (PEFR).

Table 1-3 Spirometry and Peak Flow Values for Adults

Severity	FEV_1 (liters)	FVC (%)	Peak Flow (liters/min)
Normal	4.0–6.0	80–90	550–650 (Male) 400–500 (Female)
Mild	3.0	70	300–400
Moderate	1.6	50	200–300
Severe	0.6	40	100

forcibly exhale. The peak rate of exhaled gas is recorded in liters per minute. This should be repeated twice, with the highest reading recorded as the patient's PEFR (Table 1-3).

CAPNOGRAPHY As discussed in the chapter "Airway Management and Ventilation," capnography has become a commonly used diagnostic tool for prehospital care. Capnography is a noninvasive method of measuring the levels of carbon dioxide (CO_2) in the exhaled breath.

The following terms have been applied to capnography:

- ***Capnometry:*** the measurement of expired CO_2. (It typically provides a numeric display of the partial pressure of CO_2 [in torr or mmHg] or the percentage of CO_2 present.)
- ***Capnography:*** a graphic recording or display of the capnometry reading over time
- ***Capnograph:*** a device that measures expired CO_2 levels
- ***Capnogram:*** the visual representation of the expired CO_2 waveform
- ***End-tidal CO_2 ($ETCO_2$):*** the measurement of the CO_2 concentration at the end of expiration (maximum CO_2)
- ***$PETCO_2$:*** the partial pressure of end-tidal CO_2 in a mixed gas solution
- ***$PaCO_2$:*** the partial pressure of CO_2 in the arterial blood

CO_2 is a normal end product of metabolism and is transported by the venous system to the right side of the heart. It is then pumped from the right ventricle to the pulmonary artery and eventually enters the pulmonary capillaries. There, it diffuses into the alveoli and is removed from the body through exhalation. When circulation is normal, CO_2 levels change with ventilation and are a reliable estimate of the partial pressure of carbon dioxide in the arterial system ($PaCO_2$). Normal $ETCO_2$ is 1 to 2 mm less than the partial pressure of carbon dioxide ($PaCO_2$), or approximately 5 percent. A normal partial pressure of end-tidal CO_2 ($PETCO_2$) is approximately 38 mmHg (0.05 × 760 mmHg = 38 mmHg). ($ETCO_2$ is normally expressed as a percentage, whereas $PETCO_2$, a partial pressure, is expressed in mmHg.) When perfusion decreases, as in shock or cardiac arrest, $ETCO_2$ levels reflect pulmonary blood flow and cardiac output, not ventilation.

Table 1-4 Basic Rules of Capnography

Symptom	Possible Cause
Sudden drop of $ETCO_2$ to zero	• Esophageal intubation • Ventilator disconnection or defect in ventilator • Defect in CO_2 analyzer
Sudden decrease of $ETCO_2$ (not to zero)	• Leak in ventilator system; obstruction • Partial disconnect in ventilator circuit • Partial airway obstruction (secretions)
Exponential decrease of $ETCO_2$	• Pulmonary embolism • Cardiac arrest • Hypotension (sudden) • Severe hyperventilation
Change in CO_2 baseline	• Calibration error • Water droplet in analyzer • Mechanical failure (ventilator)
Sudden increase in $ETCO_2$	• Accessing an area of lung previously obstructed • Release of tourniquet • Sudden increase in blood pressure
Gradual lowering of $ETCO_2$	• Hypovolemia • Decreasing cardiac output • Decreasing body temperature; hypothermia; drop in metabolism
Gradual increase in $ETCO_2$	• Rising body temperature • Hypoventilation • CO_2 absorption • Partial airway obstruction (foreign body); reactive airway disease

Decreased CO_2 levels can be found in shock, cardiac arrest, pulmonary embolism, bronchospasm, and with incomplete airway obstruction (such as mucus plugging). Increased CO_2 levels are found with hypoventilation, respiratory depression, and hyperthermia (Table 1-4).

Capnometry provides a noninvasive measure of CO_2 levels, thus providing medical personnel with information about the status of systemic metabolism, circulation, and ventilation. It can be used to detect certain conditions by examining the waveform in the context of the physical examination. The use of capnography has become commonplace in the operating room, emergency department, and prehospital setting.

When first introduced into prehospital care, CO_2 monitoring was used exclusively to verify proper endotracheal tube placement in the trachea, and it is still used for this purpose. The presence of adequate CO_2 levels following intubation confirms that the tube is in the trachea through the presence of exhaled CO_2. CO_2 is detected by using either a colorimetric or an infrared device.

Colorimetric Devices The colorimetric device is a disposable CO_2 detector that contains pH-sensitive, chemically impregnated paper encased within a plastic chamber (Figure 1-26). It is placed in the airway circuit between the patient and the ventilation device. When the paper is

FIGURE 1-26 Colorimetric end-tidal CO_2 detector.
(© Dr. Bryan E. Bledsoe)

exposed to CO_2, hydrogen ions (H^+) are generated, causing a color change in the paper. The color change is reversible and changes from breath to breath. A color scale on the device estimates the CO_2 level. Colorimetric devices cannot detect hypercarbia or hypocarbia (increased or decreased CO_2 levels). If gastric contents or acidic drugs (e.g., endotracheal epinephrine) contact the paper in the device, subsequent readings may be unreliable.

Electronic Devices Electronic capnography detectors use an infrared technique to detect CO_2 in the exhaled breath (Figure 1-27). A heated element in the sensor generates infrared radiation. The CO_2 molecules absorb infrared light at a very specific wavelength and can thus be measured. Electronic $ETCO_2$ detectors may be either qualitative (i.e., they simply detect the presence of CO_2) or quantitative (i.e., they determine how much CO_2 is present). Quantitative devices are now routinely used in prehospital care. Most can provide a digital waveform (capnogram) that reflects the entire respiratory cycle (Figure 1-28).

Capnogram The capnogram reflects CO_2 concentrations over time. It is typically divided into four phases (Figure 1-29).

- ***Phase I*** (AB in Figure 1-29) is the respiratory baseline. It is flat when no CO_2 is present and corresponds to the late phase of inspiration and the early part of expiration (in which dead-space gases without CO_2 are released).
- ***Phase II*** (BC in Figure 1-29) is the respiratory upstroke. This reflects the appearance of CO_2 in the alveoli.

(a)

(b)

FIGURE 1-27 (a) Electronic capnography. (b) Electronic capnography sensor on a patient.

- ***Phase III*** (CD in Figure 1-29) is the respiratory plateau. It reflects the airflow through uniformly ventilated alveoli with a nearly constant CO_2 level. The highest level of the plateau (point D in Figure 1-29) is called the $ETCO_2$ and is recorded as such by the capnometer.

FIGURE 1-28 Capnography devices provide a digital waveform (capnogram) that reflects the entire respiratory cycle.

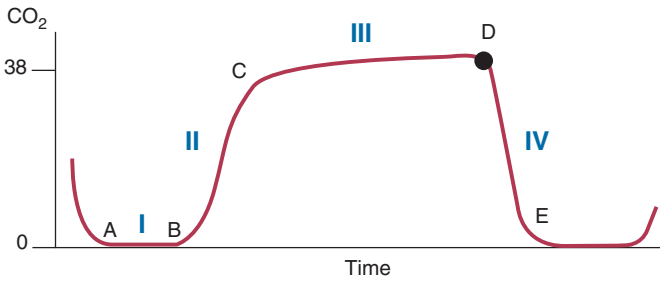


FIGURE 1-29 Normal capnogram. AB = *Phase I:* late inspiration, early expiration (no CO_2). BC 5 *Phase II:* appearance of CO_2 in exhaled gas. CD = *Phase III:* plateau (constant CO_2). D = highest point ($ETCO_2$). DE = *Phase IV:* rapid descent during inspiration. EA = respiratory pause.

- ***Phase IV*** (DE in Figure 1-29) is the inspiratory phase. It is a sudden downstroke and ultimately returns to the baseline during inspiration. The respiratory pause restarts the cycle (EA in Figure 1-29).

Clinical Applications Initially, as noted earlier, CO_2 detection was used only to determine proper endotracheal tube placement. Typically, a qualitative $ETCO_2$ device was applied to the airway circuit following intubation. If CO_2 levels were detected, then proper tube placement was verified. However, it is difficult to continuously monitor the airway with a quantitative device. Now, continuous waveform capnography is available and allows continuous monitoring of airway placement and ventilation for intubated patients. Continuous waveform capnography also has utility in monitoring nonintubated patients. By following trends in the capnogram, prehospital personnel can continuously monitor the patient's condition, detect trends, and document the response to medications.

CO_2 detection is also useful in CPR. During cardiac arrest, CO_2 levels fall abruptly following the onset of cardiac arrest. They begin to rise with the onset of effective CPR and return to near-normal levels with a return of spontaneous circulation. During effective CPR, CO_2 levels have been found to correlate well with cardiac output, coronary perfusion pressure, and even with the effectiveness of CPR compressions.

Continuous waveform capnography is rapidly becoming a standard of care in EMS (Figure 1-30). Misplaced endotracheal tubes represent a significant area of liability in EMS, and the documentation provided by this technology can provide irrefutable evidence of proper endotracheal tube placement.

Management of Respiratory Disorders

The following sections address the pathophysiology, assessment, and management of the more common respiratory disorders encountered in prehospital care. The discussion begins with a look at general principles that can and should be applied to all respiratory emergencies.

Management Principles

In cases of acute respiratory insufficiency, the following principles should guide your actions in the prehospital setting:

- The airway always has first priority. In trauma victims who may have associated cervical spine injuries,

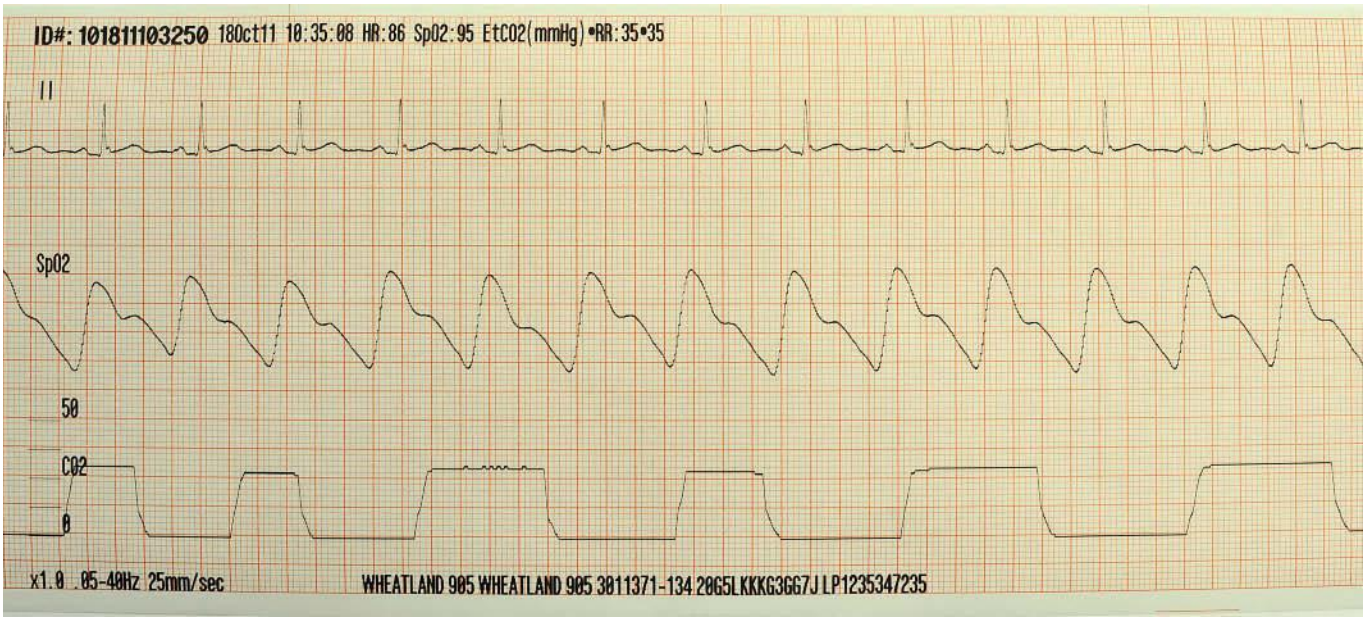


FIGURE 1-30 Continuous waveform (bottom tracing).

protect and maintain the airway without extending the neck.

- Any patient with hypoxia should receive oxygen.
- Any patient whose illness or injury suggests the possibility of hypoxia should receive oxygen until pulse oximetry is available.
- If there is a question whether oxygen should be given, as in COPD, administer enough oxygen to maintain an adequate SpO_2 level (typically ≥96 percent). This state of normal, adequate oxygen is known as **normoxia**. You should strive for normoxia in your patient and avoid both hypoxia and **hyperoxia** (too little or too much oxygen), if possible.

Oxygenation

Supplemental oxygen administration has always been the mainstay of respiratory emergency management. Although supplemental oxygen is an essential and important therapy in prehospital care, several recent studies have demonstrated that administering too much oxygen can actually worsen patient outcomes.[2,3,4] **Hypoxia**, a state of insufficient oxygen, can result in the formation of toxic chemicals called **free radicals**. These chemicals, also called **reactive oxygen species (ROS)**, can damage body cells and tissues—a process called **oxidative stress**. Because of this, the goal of oxygen therapy is to provide just enough oxygen to treat hypoxia (as guided by pulse oximetry) without causing hyperoxia. Oxygen should be treated like any other drug in the formulary. It should be used when needed, but judiciously. As already emphasized, the goal is normoxia (Figure 1-31).

Keep these precautions in mind as you read through the descriptions of pathophysiology, assessment, and management of respiratory disorders frequently encountered in the field.

FIGURE 1-31 Excess amounts of oxygen (hyperoxia) have been associated with worsened outcomes in critically ill patients. Always provide enough oxygen to treat hypoxia but avoid hyperoxia.

Specific Respiratory Diseases

Upper Airway Obstruction

The most common cause of upper airway obstruction is the relaxed tongue. In an unconscious patient in the supine position, the tongue can fall into the back of the throat and obstruct the upper airway. Additionally, such common materials as food, dentures, or other foreign bodies can obstruct the upper airway. A typical example of upper airway obstruction is the "café coronary," which tends to occur in middle-aged or elderly patients who wear dentures. These people often are unable to sense how well they have chewed their food. Thus, they accidentally inhale a large piece of food (often meat) that obstructs their airway. Concurrent alcohol consumption is often implicated in the café coronary. Furthermore, obstruction of the upper airway can be the result of facial or neck trauma, upper airway burns, or allergic reactions. In addition, the upper airway can become blocked by an infection that causes swelling of the epiglottis (epiglottitis) or subglottic area (croup).

Foreign bodies may cause either a mild or a severe airway obstruction. Intervention is usually required only if there is a severe airway obstruction. Signs of a severe airway obstruction include a silent cough, cyanosis, and the inability to speak or breathe. If the patient is conscious, ask, "Are you choking?" If the victim indicates yes (by nodding the head) without speaking, you should assume that the obstruction is severe.

Assessment

Assessment of the patient with an upper airway obstruction varies, depending on the cause of the obstruction and the history of the event. The unresponsive patient should be evaluated for snoring respirations, possibly indicating tongue or denture obstruction. If confronted by a patient suffering a café coronary, determine whether the victim can speak. Speech indicates that, at present, the obstruction is incomplete. If the victim is unresponsive and has been eating, strongly suspect a food bolus lodged in the trachea. If a burn is present or suspected, assume laryngeal edema until proven otherwise.

Patients who may be having an allergic reaction to food or medications will often report an itching sensation in the palate followed by a "lump" in the throat. The situation may progress to hoarseness, inspiratory stridor, and

CONTENT REVIEW

➤ Common Causes of Airway Obstruction
- Tongue
- Foreign matter
- Trauma
- Burns
- Allergic reaction
- Infection

complete obstruction. Pay particular attention to the presence of urticaria (hives). Intercostal muscle retraction and use of the strap muscles of the neck for breathing suggest attempts to ventilate against a partially closed airway.

Capnography can be useful in identifying upper airway obstruction. Generally, depending on the degree of upper airway obstruction, you will see a steady increase in CO_2 levels.

Management

Management of the obstructed airway is based on the nature of the obstruction. Blockage by the tongue can be corrected by opening the airway, using either the head-tilt, chin-lift, jaw-thrust, or jaw-thrust without head extension maneuver. The airway can be maintained by employing either a nasopharyngeal or oropharyngeal airway. If possible, remove obstructing foreign bodies using the following basic airway maneuvers for an adult.[5] (For management of a foreign body airway obstruction in an infant or child, see the chapter "Pediatrics.")

CONSCIOUS ADULT In an adult patient (or child >1 year of age) who is conscious:

1. Determine whether there is a complete obstruction or poor air exchange. Ask the patient: "Are you choking?" "Can you speak?" If the patient can speak, he should be asked to produce a forceful cough to expel the foreign body.
2. If the patient has a severe obstruction or poor air exchange, provide rapid abdominal thrusts in rapid sequence until the obstruction is relieved. If abdominal thrusts are ineffective (or if the patient is obese), attempt chest thrusts instead. If the patient is pregnant, omit the abdominal thrusts and proceed straight to chest thrusts. Often, more than one technique is required. These should be applied in rapid sequence until the obstruction is relieved.

UNCONSCIOUS ADULT If the patient is unconscious or loses consciousness:

1. Use the head-tilt, chin-lift, jaw-thrust, or jaw-thrust without head extension maneuver in an attempt to open the airway.
2. Begin CPR.
3. Each time you open the airway during CPR, look for an object in the victim's mouth and remove it.
4. If the obstruction persists and ventilation cannot be provided, visualize the airway with the laryngoscope. If you can see the foreign body, grasp it with the Magill forceps and remove. Once the obstruction has been removed, begin ventilation and administer supplemental oxygen (if hypoxia is detected or suspected).

In cases of airway obstruction caused by laryngeal edema (e.g., anaphylactic reactions, angioedema), establish the airway by the head-tilt, chin-lift, jaw-thrust, or jaw-thrust without head extension maneuver. Then administer supplemental oxygen if hypoxia has been determined by pulse oximetry. Attempt bag-valve-mask ventilation. Often, air can be forced past the obstruction and the patient adequately ventilated using this technique. Next, start an IV with a crystalloid solution and administer intravenous or intramuscular epinephrine. Then administer diphenhydramine (Benadryl). Transtracheal ventilation may be required if the patient does not respond to the treatments described. (See the "Pediatrics" chapter for pediatric techniques.)

Noncardiogenic Pulmonary Edema/ Acute Respiratory Distress Syndrome

Acute respiratory distress syndrome (ARDS) is a life-threatening condition that adversely affects gas exchange in the lungs. It is a form of pulmonary edema that is caused by fluid accumulation in the interstitial space within the lungs. Patients with *cardiogenic* pulmonary edema have a poorly functioning left ventricle. This leads to increases in hydrostatic pressure and fluid accumulation in the interstitial space. In patients with ARDS, however, fluid accumulation occurs as the result of increased vascular permeability and decreased fluid removal from the lung tissue. This occurs in response to a wide variety of lung insults including:

- Sepsis, particularly with Gram-negative organisms
- Aspiration
- Pneumonia or other respiratory infections
- Pulmonary injury
- Burns
- Inhalation injury
- Oxygen toxicity
- Drugs such as aspirin or opiates
- High altitude
- Hypothermia
- Near-drowning
- Head injury
- Emboli from blood clot, fat, or amniotic fluid
- Tumor destruction
- Pancreatitis
- Procedures such as cardiopulmonary bypass or hemodialysis
- Other insults such as hypoxia, hypotension, or cardiac arrest

The mortality in patients who develop ARDS is quite high, approaching 70 percent. Although many patients die as the result of respiratory failure, many succumb to failure of several organ systems, including the liver and kidneys.

Pathophysiology

ARDS is a disorder of lung diffusion that results from increased fluid in the interstitial space. Each of the underlying conditions cited previously results in the inability to maintain a proper fluid balance in the interstitial space. Severe hypotension, significant hypoxemia as the result of cardiac arrest, drowning, seizure activity or hypoventilation, high-altitude exposure, environmental toxins, and endotoxins released in septic shock all can cause disruption of the alveolar–capillary membrane. Increases in pulmonary capillary permeability, destruction of the capillary lining, and increases in osmotic forces act to draw fluid into the interstitial space and contribute to interstitial edema. This increases the thickness of the respiratory membrane and limits diffusion of oxygen. In advanced cases, fluid also accumulates in the alveoli, causing loss of surfactant, collapse of the alveolar sacs, and impaired gas exchange. This results in a significant amount of pulmonary shunting with deoxygenated blood returning to the circulation. The result is significant hypoxia (Figure 1-32).[6]

Assessment

Specific clinical symptoms are related to the underlying cause of ARDS. For example, patients who develop ARDS as the result of sepsis will have symptoms related to their underlying infection. Determine whether there is a history of prolonged hypoxia, head or chest trauma, inhalation of gases, or ascent to a high altitude without prior acclimation, all of which can suggest an underlying cause for the respiratory complaints.

Patients with ARDS experience a gradual decline in their respiratory status. In rare cases, a seemingly healthy patient has a sudden onset of respiratory failure and hypoxia. Such a presentation is characteristic of patients with high-altitude pulmonary edema (HAPE).

Dyspnea, confusion, and agitation are often found in patients with noncardiogenic pulmonary edema. Patients may also report fatigue and reduced exercise ability. Symptoms such as orthopnea, paroxysmal nocturnal dyspnea, or sputum production are not commonly reported but may be seen.

The prominent physical findings are generally those associated with the underlying lung insult. Tachypnea and tachycardia are often found in association with ARDS. Crackles (rales) are audible in both lungs. Wheezing may also be heard if there is any element of bronchospasm. Severe tachypnea, central cyanosis, and signs of imminent respiratory failure are seen in severe cases. Pulse oximetry will demonstrate low oxygen saturations in patients with advanced disease. In patients requiring ventilatory support, decreased lung compliance will be noted. (It will require more operator force to deliver an adequate lung volume.)

Management

Specific management of the patient's underlying medical condition is the hallmark of treatment for this disorder. Treatment of Gram-negative sepsis with appropriate antibiotics, removal of the patient from any inciting toxin, or rapid descent to a lower altitude in patients with HAPE are the most important therapies for this condition. The patient will usually tolerate an upright position with the legs dangling off the cart.

Because the hypoxia seen in ARDS is the result of diffusion defects, supplementation is often essential for all patients with this condition. Establish intravenous access, but provide fluids only if hypovolemia exists. Establish cardiac monitoring. Suctioning of lung secretions is often required to maintain airway patency.

Use positive pressure ventilation to support any ARDS patient who demonstrates signs of respiratory failure. Use bag-valve-mask ventilation for initial respiratory support while preparing a continuous positive airway pressure (CPAP) device. Use of CPAP can often avoid the need for endotracheal intubation and mechanical ventilation. **Positive end-expiratory pressure (PEEP)**, via CPAP, will help to maintain patency of the alveoli and adequate oxygenation. Diuretics and nitrates, which are used in patients with cardiogenic pulmonary edema, are usually not helpful in patients with ARDS. Your medical director may occasionally order corticosteroids for patients with ARDS/noncardiogenic pulmonary edema. Corticosteroids are thought to stabilize the alveolar–capillary membrane, although clinical studies have not demonstrated any benefit to their use.

Maintain cardiac monitoring and pulse oximetry throughout transport of the patient. Transport patients to a facility capable of advanced hemodynamic monitoring (including via Swan-Ganz catheter) and mechanical ventilation support.

Obstructive Lung Disease

Obstructive lung disease is widespread in our society. The most common obstructive lung diseases encountered in prehospital care are asthma, emphysema, and chronic bronchitis (the last two are often discussed together as chronic obstructive pulmonary disease, or COPD). Asthma afflicts 4 to 5 percent of the U.S. population and COPD is found in 25 percent of all adults. Chronic bronchitis alone

CONTENT REVIEW

➤ Obstructive Lung Diseases
- Emphysema
- Chronic bronchitis
- Asthma

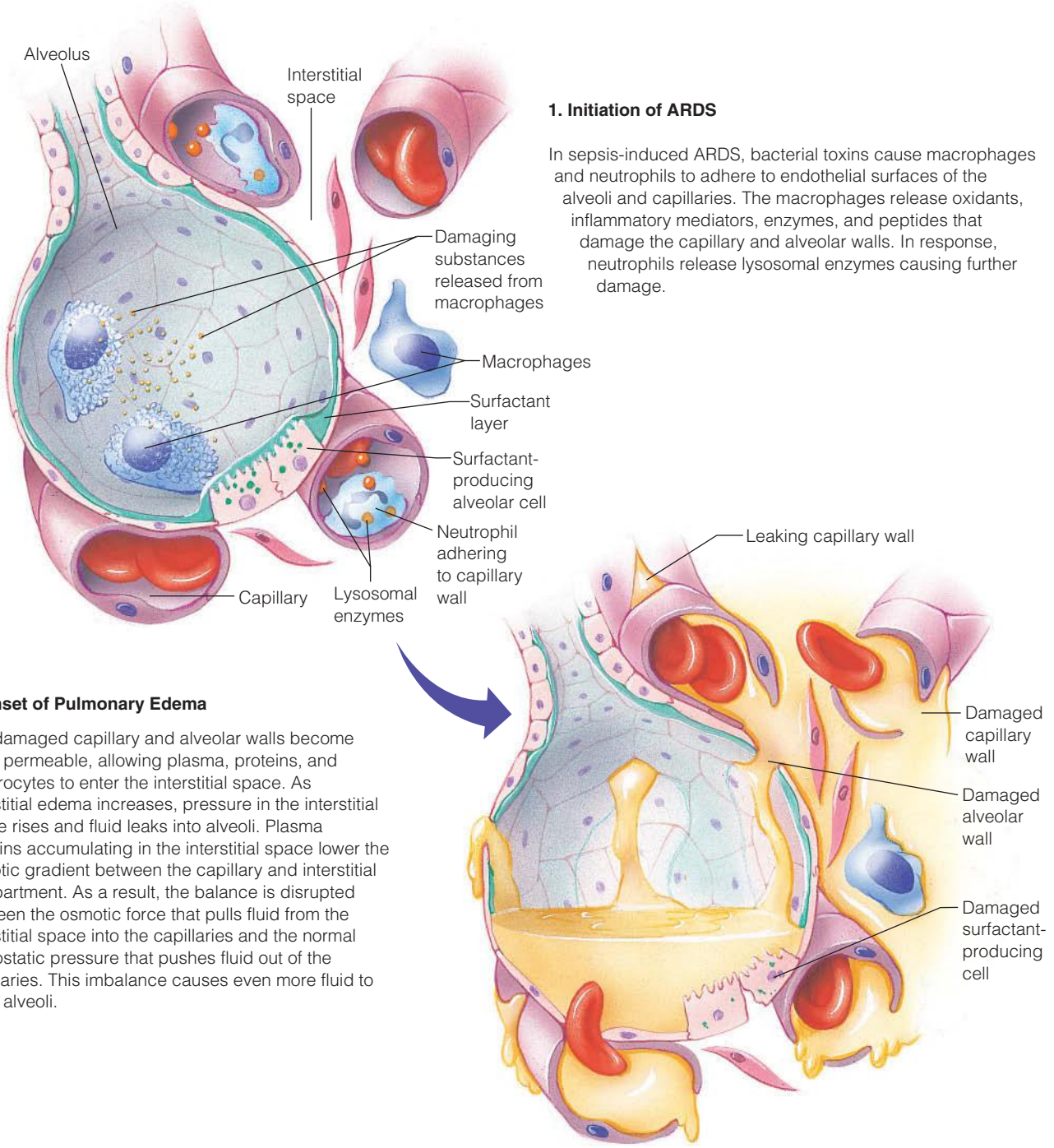


FIGURE 1-32

1. ***Initiation of ARDS*** In septic ARDS, bacterial toxins cause inflammation that damages the alveolar and capillary walls.
2. ***Onset of Pulmonary Edema*** The damaged alveolar and capillary walls become more permeable, allowing plasma and other substances to enter the interstitial space, thus causing fluid entry into the alveoli.
3. ***End-Stage ARDS*** Fibrin and cell debris form hyaline membranes that reduce alveolar compliance and adversely affect diffusion.
4. ***Alveolar Collapse*** Protein-rich fluid accumulates in the alveoli and damages the cells that manufacture surfactant, causing stiffening of the alveoli and atelectasis.

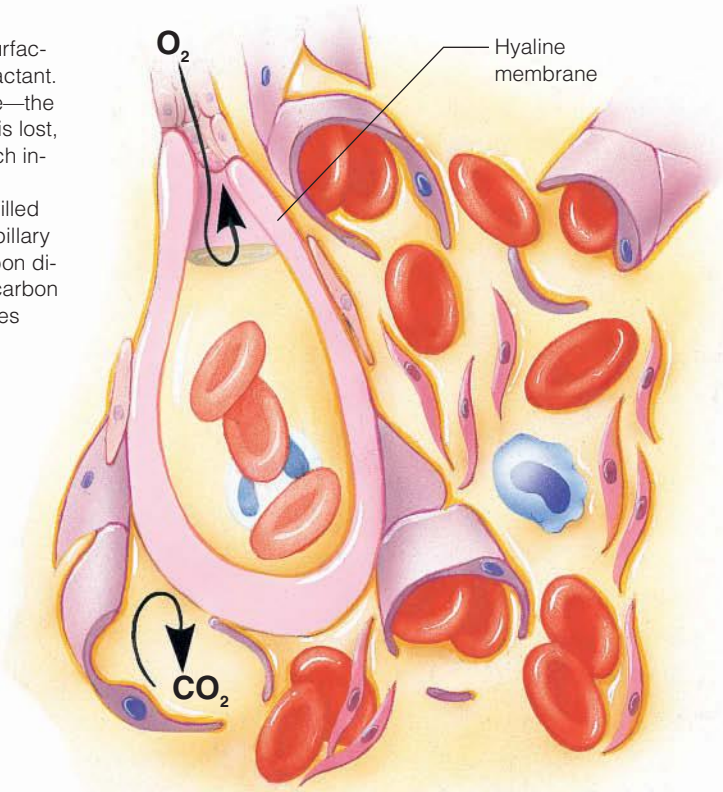
affects one in five adult males. Patients with COPD have a 50 percent mortality within 10 years of the diagnosis.

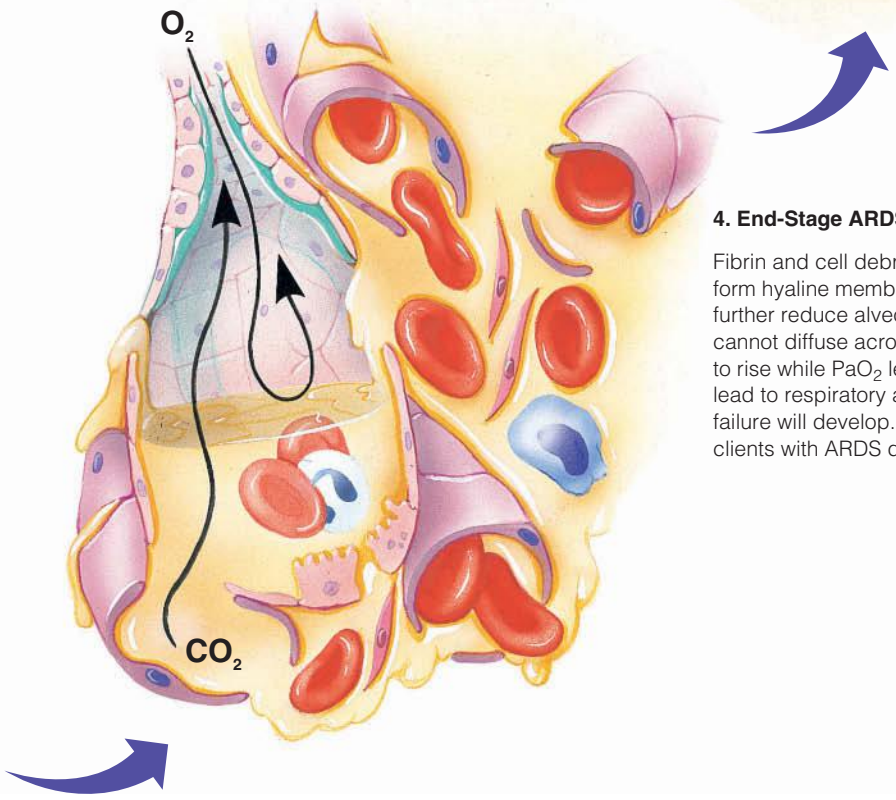
Although asthma may have a genetic predisposition, COPD is known to be directly caused by cigarette smoking and environmental toxins. Other factors have been shown to precipitate symptoms in patients who already have obstructive airway disease. Intrinsic factors include stress, upper respiratory infections, and

3. Alveolar Collapse

Protein-rich fluid accumulates in the alveoli, inactivating surfactant and damaging type II alveolar cells that produce surfactant. (Surfactant is important in maintaining alveolar compliance—the ability of tissue to stretch or distend.) As active surfactant is lost, the alveoli stiffen and collapse, leading to atelectasis, which increases breathing effort.

Decreased alveolar compliance, atelectasis, and fluid-filled alveoli interfere with gas exchange across the alveolar-capillary membrane. Blood oxygen (PaO_2) levels fall. Because carbon dioxide diffuses more readily than oxygen, however, blood carbon dioxide ($PaCO_2$) levels also fall initially as tachypnea causes more CO_2 to be expired.





4. End-Stage ARDS

Fibrin and cell debris from necrotic cells combine to form hyaline membranes, which line the interior of the alveoli and further reduce alveolar compliance and gas exchange. Because CO_2 cannot diffuse across hyaline membranes, $PaCO_2$ levels now begin to rise while PaO_2 levels continue to fall. Rising $PaCO_2$ levels can lead to respiratory acidosis. Without respiratory support, respiratory failure will develop. Even with aggressive treatment, almost 50% of clients with ARDS die.

FIGURE 1-32 (*Continued*)

exercise. Extrinsic factors include tobacco smoke, drugs, occupational hazards (chemical fumes, dust, and others), and allergens such as foods, animal danders, dusts, and molds.

Obstructive lung diseases all have abnormal ventilation as a common feature. This abnormal ventilation is a result of obstruction that occurs primarily in the bronchioles. Several changes occur within these air conduits.

Cultural Considerations

Cultural Acceptance of Smoking. Cigarette smoking has clearly been demonstrated to be a contributing factor in the development of respiratory disease—especially chronic obstructive pulmonary disease and bronchogenic cancers. Although the use of tobacco products in the United States has declined overall, it is still more acceptable in some areas of the country than in others. For example, in some southern and eastern states where tobacco is the cash crop, there is a higher incidence of tobacco use. Smoking also is more acceptable and better tolerated in some cultures than in others, and tobacco products are more readily available where these cultures prevail. Thus, over time, one would expect the incidence of chronic obstructive pulmonary diseases and bronchogenic cancers to be higher in these groups.

Bronchospasm (sustained smooth muscle contraction) occurs, which may be reversed by beta-adrenergic receptor stimulation. Agents such as albuterol (Ventolin, Proventil), levalbuterol (Xopenex), and epinephrine are used to accomplish this stimulation. Increased mucus production by goblet cells that line the respiratory tree also contributes to obstruction. This effect may be worsened by the fact that in many patients, the cilia are destroyed, resulting in poor clearance of excess mucus. Finally, inflammation of the bronchial passages results in the accumulation of fluid and inflammatory cells. Depending on the underlying cause, some elements of bronchial obstruction are reversible, whereas others are not. Ipratropium (Atrovent), a parasympathetic blocker, may aid in the drying of bronchial secretions and in reversing bronchospasm.

During inspiration, the bronchioles will naturally dilate, allowing air to be drawn into the alveoli. As the patient begins to exhale, the bronchioles constrict. When this natural constriction occurs—in addition to the underlying bronchospasm, increased production of mucus, and inflammation that exist in patients with obstructive airway disease—the result is significant air trapping distal to the obstruction. This is one of the hallmarks of obstructive lung disease. The next sections discuss each of these disease processes—emphysema, chronic bronchitis, and asthma—detailing the pathophysiology, assessment, and treatment of each.

Emphysema

Emphysema results from destruction of the alveolar walls distal to the terminal bronchioles. It is more common in men than in women. The major factor contributing to emphysema in our society is cigarette smoking. Significant exposure to environmental toxins is another contributing factor.

Pathophysiology

Continued exposure to noxious substances, such as cigarette smoke, results in the gradual destruction of the walls of the alveoli. This process decreases the alveolar membrane surface area, thus lessening the area available for gas exchange. The progressive loss of the respiratory membrane results in an increased ratio of air to lung tissue. The result is diffusion defects. Additionally, the number of pulmonary capillaries in the lung is decreased, thus increasing resistance to pulmonary blood flow. This condition ultimately causes pulmonary hypertension, which in turn may lead to right-heart failure, **cor pulmonale**, and death (Figure 1-33).

Emphysema also causes weakening of the walls of the small bronchioles. When the walls of the alveoli and small bronchioles are destroyed, the lungs lose their capacity to recoil and air becomes trapped in the lungs. Thus, residual volume increases while vital capacity remains relatively normal. The destroyed lung tissue (called *blebs*) results in alveolar collapse. To counteract this effect, patients tend to breathe through pursed lips. This creates continued positive pressure similar to PEEP (positive end-expiratory pressure) and prevents alveolar collapse.

As the disease progresses, the PaO_2 further decreases, which may lead to increased red blood cell production and **polycythemia** (an excess of red blood cells, resulting in an abnormally high hematocrit). The $PaCO_2$ also increases and becomes chronically elevated, forcing the body to depend on hypoxic drive to control respirations. Finally, remember that emphysema is characterized by irreversible airway obstruction.

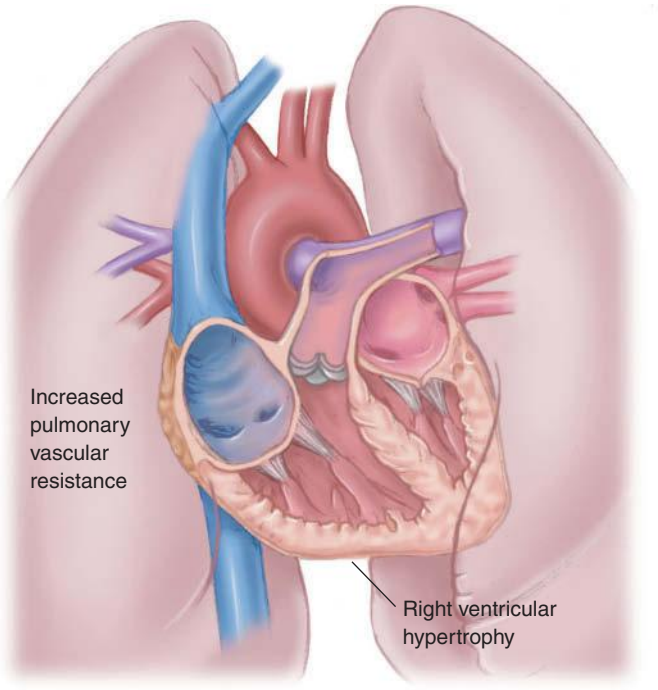


FIGURE 1-33 Chronic obstructive pulmonary disease of long duration can cause pulmonary hypertension, which in turn may lead to cor pulmonale.