Respiratory infections are commonly encountered among dental patients. Commonalities between chemotherapeutic options and the anatomic proximity with the oral cavity lead to much interplay between oral and respiratory infections. There is a growing body of literature pointing to a direct association between oral pathogens and respiratory diseases. Recent studies have reported on oral bacteria as causative pathogens in respiratory diseases and conditions associated with significant morbidity and mortality. Furthermore, some respiratory illnesses (such as asthma) may have an effect on orofacial morphology or even on the dentition. This chapter discusses the more common respiratory illnesses and explores the relationship between these conditions and oral health.

**UPPER-AIRWAY INFECTIONS**

- Viral Upper Respiratory Infections
- Allergic Rhinitis and Conjunctivitis
- Otitis Media
- Sinusitis
- Laryngitis and Laryngotracheobronchitis
- Pharyngitis and Tonsillitis

**LOWER-AIRWAY INFECTIONS**

- Acute Bronchitis
- Pneumonia
- Bronchiolitis
- Asthma
- Chronic Obstructive Pulmonary Disease
- Cystic Fibrosis
- Pulmonary Embolism
- Pulmonary Neoplasm

There are several major oral health concerns for patients with upper respiratory infections. These concerns are about infectious matters, such as the possible transmission of pathogens from patients to health care workers and such as re-infection with causative pathogens through fomites such as toothbrushes and removable oral acrylic appliances. Furthermore, antibiotic resistance may develop because of the use of similar types of medications for upper respiratory infections and odontogenic infections. Lastly, oral mucosal changes (such as dryness due to decongestants and mouth breathing) and increased susceptibility to oral candidiasis in patients using long-term glucocorticosteroid inhalers may be noticed.
**Viral Upper Respiratory Infections**

The most common cause of acute respiratory illness is viral infection, which occurs more commonly in children than in adults. Rhinoviruses account for the majority of upper-respiratory infections in adults. These are ribonucleic acid (RNA) viruses, which preferentially infect the respiratory tree. At least 100 antigenically distinct subtypes have been isolated. Rhinoviruses are most commonly transmitted by close person-to-person contact and by respiratory droplets. Shedding can occur from nasopharyngeal secretions for up to 3 weeks, but 7 days or less is more typical. In addition to rhinoviruses, several other viruses, including Coronavirus, influenza virus, parainfluenza virus, adenovirus, Enterovirus, coxsackievirus, and respiratory syncytial virus, have also been implicated as causative agents. Infection by these viruses occurs more commonly during the winter months in temperate climates.

**PATHOPHYSIOLOGY**

Viral particles can lodge in either the upper or lower respiratory tract. The particles invade the respiratory epithelium, and viral replication ensues shortly thereafter. The typical incubation period for Rhinovirus is 2 to 5 days. During this time, active and specific immune responses are triggered, and mechanisms for viral clearance are enhanced. The period of communicability tends to correlate with the duration of clinical symptoms.

**CLINICAL AND LABORATORY FINDINGS**

Signs and symptoms of upper-respiratory-tract infections are somewhat variable and are dependent on the sites of inoculation. Common symptoms include rhinorrhea, nasal congestion, and oropharyngeal irritation. Nasal secretions can be serous or purulent. Other symptoms that may be present include cough, fever, malaise, fatigue, headache, and myalgia. A complete blood count (CBC) with differential shows an increase in mononuclear cells, lymphocytes, and monocytes (“right shift”).

Laboratory tests are typically not required in the diagnosis of upper respiratory infections. Viruses can be isolated by culture or determined by rapid diagnostic assays. However, these tests are rarely clinically warranted.

**DIAGNOSIS**

The diagnosis is made on the basis of medical history as well as confirmatory physical findings. Diagnoses that should be excluded include acute bacterial rhinosinusitis, allergic rhinitis, and group A streptococcal pharyngitis.

**MANAGEMENT**

The treatment of upper respiratory infections is symptomatic as most are self-limited. Analgesics can be used for sore throat and myalgias. Antipyretics can be used in febrile patients, and anticholinergic agents may be helpful in reducing rhinorrhea. Oral or topical decongestants, such as the sympathomimetic amines, are an effective means of decreasing nasal congestion. Adequate hydration is also important in homeostasis, especially during febrile illnesses.

Antimicrobial agents have no role in the treatment of acute viral upper respiratory infections. Presumptive treatment with antibiotics to prevent bacterial superinfection is not recommended. The excessive use of antibiotics can result in the development of drug-resistant bacteria.

**PROGNOSIS**

As most patients recover in 5 to 10 days, the prognosis is excellent. However, upper respiratory infections can put patients at risk for exacerbations of asthma, acute bacterial sinusitis, and otitis media; that is especially so in predisposed patients such as children and such as patients with an incompetent immune system.

**ORAL HEALTH CONSIDERATIONS**

The most common oral manifestation of upper respiratory viral infections is the presence of small round erythematous macular lesions on the soft palate. These lesions may be caused directly by the viral infection, or they may represent a response of lymphoid tissue. Individuals with excessive lingual tonsillar tissue also experience enlargement of these foci of lymphoid tissue, particularly at the lateral borders at the base of the tongue.

Treatment of upper respiratory infections with decongestants may cause decreased salivary flow, and patients may experience oral dryness (see Chapter 9 for a discussion of the treatment of oral dryness).

Although there has been some discussion in the dental literature in regard to a relationship between dentofacial morphology and mouth breathing, this association has not been verified in prospective longitudinal studies.

**Allergic Rhinitis and Conjunctivitis**

Allergic rhinitis is a chronic recurrent inflammatory disorder of the nasal mucosa. Similarly, allergic conjunctivitis is an inflammatory disorder involving the conjunctiva. When both conditions occur, the term “allergic rhinoconjunctivitis” is used. The basis of the inflammation is an allergic hypersensitivity (type I hypersensitivity) to environmental triggers. Allergic rhinoconjunctivitis can be seasonal or perennial. Typical seasonal triggers include grass, tree, and weed pollens. Common perennial triggers include dust mites, animal dander, and mold spores.

Allergic rhinitis is the most prevalent chronic medical disorder. More than 35 million Americans are affected. Allergic rhinitis is associated with a significant health care cost burden, and more than $2 billion (US) are spent annually in the United States on medication for this condition alone. In addition, allergic rhinitis accounts for more than 2 million lost school days per year and more than 3 million lost workdays per year.

**PATHOPHYSIOLOGY**

Patients with allergic rhinoconjunctivitis have a genetically predetermined susceptibility to allergic hypersensitivity reactions (atopy). Prior to the allergic response, an initial phase of sensitization is required. This sensitization phase is dependent on exposure to a specific allergen and on recognition of the
allergen by the immune system. The end result of the sensitization phase is the production of specific immunoglobulin E (IgE) antibody and the binding of this specific IgE to the surface of tissue mast cells and blood basophils. Surface IgE can bind to the same allergens upon re-exposure. Once bound by surface IgE, subsequent IgE cross-linking will occur, which triggers degranulation of the mast cell and the release of mast cell mediators. This is the early-phase allergic reaction. Histamine is the primary preformed mediator released by mast cells, and it contributes to the clinical symptoms of sneezing, pruritus, and rhinorrhea. Mast cells also release cytokines that permit amplification and feedback of the allergic response. These cytokines cause an influx of other inflammatory cells, including eosinophils, resulting in the late-phase allergic reaction. Eosinophils produce many proinflammatory mediators that contribute to chronic allergic inflammation and to the symptom of nasal congestion.

**CLINICAL AND LABORATORY FINDINGS**

The symptoms of allergic rhinoconjunctivitis can vary from patient to patient and depend on the specific allergens to which the patient is sensitized. Conjunctival symptoms may include pruritus, lacrimation, crusting, and burning. Nasal symptoms may include sneezing, pruritus, clear rhinorrhea, and nasal congestion. Other symptoms can occur, such as postnasal drainage with throat irritation, pruritus of the palate and ear canals, and fatigue.

The clinical signs of allergic rhinoconjunctivitis include injection of the conjunctiva with or without “cobblestoning”; prominent infraorbital creases (Dennie-Morgan folds/pleats), swelling, and darkening (“allergic shiners”), a transverse nasal crease; and frequent upwards rubbing of the tip of the nose (the allergic “salute”). Direct examination of the nasal mucosa reveals significant edema and a pale blue coloration of the turbinates. A copious clear rhinorrhea is often present. Nasal polyps may also be visible. Postnasal drainage or oropharyngeal cobblestoning might be identified upon examination of the oropharynx. A high-arched palate, protrusion of the tongue, and overbite may be seen.

Laboratory investigations are usually kept to a minimum. Patients with allergic rhinitis might have elevated levels of serum IgE and an elevated total eosinophil count. These findings are not, however, sensitive nor specific indicators of atopy. Microscopic examination of nasal secretions often demonstrates significant numbers of eosinophils. The radioallergosorbent test (RAST) is a method of testing for specific allergic sensitivities that is based on circulating levels of specific IgE. Specific IgE levels are determined by using serum samples and are quantified by using radioactive markers. Although the RAST is somewhat less reliable than skin testing (see below), it is a useful test in certain situations (such as pregnancy or severe chronic skin disorders, including atopic dermatitis).

**CLASSIFICATION**

There is no universal classification system for allergic rhinoconjunctivitis. Many authors make the distinction between perennial and seasonal allergy, with the former being caused mainly by indoor allergens and the latter being triggered primarily by outdoor allergens. Perennial allergic rhinitis sufferers might benefit more from specific environmental control measures than would seasonal allergic rhinitis sufferers.

**DIAGNOSIS**

The diagnosis of allergic rhinoconjunctivitis is usually apparent, based on history and physical examination. Patients present with a history suggestive of allergic sensitivity, recurrent symptoms with specific exposures, or predictable exacerbations during certain times of the year. Symptoms that have recurred for 2 or more years during the same season are very suggestive of seasonal allergic disease. Alternatively, the history might indicate a pattern of worsening symptoms while the patient is at work or on vacation; this pattern is highly suggestive of perennial allergic disease with indoor triggers. The presence of the characteristic physical findings described above would confirm the presence of allergic rhinoconjunctivitis.

The preferred method of testing for allergic sensitivities is skin testing, which is performed with epicutaneous (prick/scratch) tests, often followed by intradermal testing. Prick skin testing is the type most widely used. With prick testing, a small amount of purified allergen is inoculated through the epidermis only (ie, epicutaneously) with a pricking device. Positive (histamine) and negative (albumin-saline) controls are used for comparison. Reactions are measured at 15 minutes, and positive reactions indicate prior allergen sensitization. Tests that yield negative results may be repeated intradermally to increase the sensitivity of the testing. All tests with positive results need to be interpreted carefully, with attention to each patient’s history and physical findings.

**MANAGEMENT**

Three general treatment modalities are used in the treatment of allergic rhinoconjunctivitis: allergen avoidance, pharmacotherapy (medication), and immunotherapy (allergy injections). The best treatment is avoidance of the offending allergen. This requires the accurate identification of the allergens implicated and a thorough knowledge of effective interventions that can minimize or eliminate the exposure. Complete avoidance is rarely possible.

Pharmacotherapy is often recommended for patients with incomplete responses to allergen avoidance and for patients who are unable to avoid exposures. Many treatment options are available. For patients with prominent sneezing, pruritus, or rhinorrhea, antihistamines are an excellent treatment option. Second-generation nonsedating antihistamines such as loratadine and fexofenadine are now available. These medications deliver excellent antihistaminic activity with few side effects. Oral decongestants (sympathomimetic amines) can be added to oral antihistamines to relieve nasal congestion and obstruction. Combination medications are available in once-daily and twice-daily dosage forms for ease of administration. For patients with daily nasal symptoms or severe symptoms that are
not relieved with antihistamine-decongestants, topical anti-inflammatory agents for the nasal mucosa are available. These medications include cromolyn sodium and topical corticosteroid sprays. The benefits of topical corticosteroids include once-daily dosing, superior efficacy (when compared to cromolyn sodium), and relief of the total symptom complex.

Immunotherapy is an effective means of treatment for patients with allergic rhinoconjunctivitis. Numerous studies have shown the efficacy of long-term allergen immunotherapy in inducing prolonged clinical and immunologic tolerance. Immunotherapy is available for a variety of airborne allergens, including grass, tree, and weed pollens; dust mites; animal dander; and mold spores. Excellent candidates for immunotherapy include those patients who are unable to avoid exposures, patients with suboptimal responses to pharmacotherapy, patients who prefer to avoid the long-term use of medications, and women who are contemplating pregnancy.

PROGNOSIS

Although allergic rhinoconjunctivitis is not a life-threatening disorder, it does have a significant impact on the patient’s quality of life. With proper allergy care, most patients can lead normal lives with an excellent quality of life.

ORAL HEALTH CONSIDERATIONS

The use of decongestants and first-generation antihistamines may be associated with oral dryness. There may also be an increased incidence of oral candidiasis in long-term users of topical corticosteroid-containing sprays.

Otitis Media

Otitis media is inflammation of the middle-ear space and tissues. It is the most common illness that occurs in children who are 8 years of age or younger. Approximately 70% of children experience at least one episode of otitis media by age 3 years; of these, approximately one-third experience three or more episodes in this same time interval.

Otitis media can be subdivided into acute otitis media, recurrent otitis media, otitis media with effusion, and chronic suppurative otitis media. The underlying problem in all types of otitis media is dysfunction of the eustachian tube. A poorly functioning eustachian tube does not ventilate the middle-ear space sufficiently. This lack of proper ventilation results in pressure changes in the middle ear and subsequent fluid accumulation. The fluid frequently becomes infected, resulting in acute otitis media. The most common infectious causes are viruses, *Streptococcus pneumoniae, Haemophilus influenzae*, and *Moraxella catarrhalis*. In infants younger than 6 weeks of age, other bacteria, including *Staphylococcus aureus*, *Escherichia coli*, *Klebsiella*, and *Enterobacter*, have also been implicated.

PATHOPHYSIOLOGY

There are several factors that influence the pathogenesis of otitis media. Nasopharyngeal colonization with large numbers of pathogenic viruses and bacteria such as *Streptococcus pneumoniae, Haemophilus influenzae*, or *Moraxella catarrhalis* can increase the risk of otitis media. The likelihood of aspiration of these nasopharyngeal pathogens can be increased by nasal congestion or obstruction, negative pressure in the middle-ear space, acute viral upper respiratory infections, and exposure to tobacco smoke. For infants, breast-feeding can decrease the risk of otitis media whereas impaired immune responsiveness can increase this risk.

Under normal circumstances, the eustachian tube acts to ventilate the tympanomastoid air cell system during the act of swallowing. Any process that impairs normal eustachian tube function can lead to negative pressure in the middle-ear space. Transient impairments of eustachian tube function are seen in conditions that cause nasopharyngeal mucosal edema and obstruction of the eustachian tube orifice, such as allergic rhinitis and viral upper respiratory infections. Chronic eustachian tube obstruction can be seen with several conditions, including cleft palate and nasopharyngeal masses. Aspiration of nasopharyngeal pathogens can then occur due to negative pressure in the middle-ear space, with subsequent infection by these pathogens. This leads to the clinical manifestations of otitis media.

CLINICAL AND LABORATORY FINDINGS

The most common symptoms in acute otitis media are fever and otalgia. Other symptoms include irritability, anorexia, and vomiting. Parents may note their child pulling or tugging at one or both ears. Symptoms of a viral upper respiratory infection might also be present, preceding the development of otitis media. On physical examination, the tympanic membrane may appear erythematous and bulging, suggesting inflammation of the middle ear. Other otoscopic findings include a loss of landmarks and decreased mobility of the tympanic membrane as seen by pneumatic otoscopy.

In otitis media with effusion, patients often complain of “clogged” ears and “popping.” Otoscopic examination reveals serous middle-ear fluid, and air-fluid levels may be present. The mobility of the tympanic membrane is usually diminished, and mild to moderate conductive hearing loss may be demonstrated. In chronic suppurative otitis media, otosrhea is present and can be visualized either from a tympanic membrane perforation or from surgically placed tympanostomy tubes.

Investigations that can aid the diagnosis or management of otitis media include tympanometry and myringotomy with aspiration. Tympanometry is a technique that measures the compliance of the tympanic membrane by using an electroacoustic impedance bridge. Decreased compliance of the tympanic membrane indicates a middle-ear effusion. Myringotomy with aspiration can be useful in situations when culture of the middle ear fluid is needed, such as with immunocompromised hosts or with patients who have persistent effusions despite medical management.

CLASSIFICATION

Acute otitis media is defined as middle-ear inflammation with an infectious etiology and a rapid onset of signs and symp-
Diagnosis

The diagnosis of otitis media is made on the basis of history and physical examination. The most useful tool for diagnosing otitis media is pneumatic otoscopy, which allows the clinician not only to visualize the tympanic membrane but also to assess its mobility. As stated above, an immobile tympanic membrane probably represents the presence of middle-ear fluid, and (in the context of a confirmatory medical history) the diagnosis of otitis media is made in such a case.

Management

Antibiotics are the treatment of choice for acute otitis media. Initial antibiotic therapy is directed towards the most common middle-ear pathogens. Common choices include amoxicillin, trimethoprim plus sulfisoxazole, and erythromycin plus sulfisoxazole. In recalcitrant cases, treatment is directed towards β-lactamase–producing organisms and antibiotic-resistant strains of Streptococcus pneumoniae. Common choices include second- and third-generation cephalosporins, clarithromycin, and amoxicillin plus clavulanate (amoxicillin/clavulanate). The duration of therapy varies from 5 to 14 days.

Insertion of tympanostomy tubes is indicated when a patient experiences more than six acute otitis media episodes during a 6-month period or has recurrent otitis media superimposed on otitis media with persistent effusion. Persistent bilateral effusions for longer than 4 months are also an indication for tympanostomy tubes. Adenoidectomy as an adjunctive therapy can be considered in children older than 3 years of age. A trial of antibiotic prophylaxis is commonly carried out prior to surgical consultation.

The management of chronic suppurative otitis media often includes parenteral antibiotics to cover infection by Pseudomonas spp and anaerobic bacteria. However, medical therapy is ineffective when a cholesteatoma (a mass filled with cellular debris and cholesterol crystals) is present.

Prognosis

Although treatment with antibiotics is the norm, up to 81% of patients achieve resolution of acute otitis media without antibiotic treatment. Therefore, the prognosis for acute otitis media is excellent. However, complications can occur, more commonly in patients younger than 1 year of age. The most common complication is conductive hearing loss related to persistent effusions. Serious complications, including mastoiditis, cholesteatoma, labyrinthitis, extradural or subdural abscesses, meningitis, brain abscess, and lateral sinus thrombosis, are uncommon.

Oral Health Considerations

Many children with recurrent otitis media are treated frequently (and sometimes for extensive periods) with various antibiotics. Included in the antibiotic armamentarium are medications that are also used for odontogenic infections. Oral health care providers need to be aware of what type of antibiotics the patient has taken within the previous 4 to 6 months, to avoid giving the patient an antibiotic to which resistance has already developed. Furthermore, the extended use of antibiotics may result in the development of oral candidiasis.

Sinusitis

Sinusitis is defined as an inflammation of the epithelial lining of the paranasal sinuses. The inflammation of these tissues causes mucosal edema and an increase in mucosal secretions. The most common trigger is an acute upper respiratory infection although other causes (such as exacerbations of allergic rhinitis, dental infections or manipulations, and direct trauma) can be implicated. If blockage of sinus drainage occurs, retained secretions can promote bacterial growth and subsequent acute bacterial sinusitis.

Acute sinusitis is a very common disorder, affecting more than 31 million Americans per year. This accounts for more than 18 million office visits to primary care physicians per year and for 124 million lost days from work each year. Chronic sinusitis is also very common.

Pathophysiology

The paranasal sinuses are air-filled cavities that are lined with pseudostratified columnar respiratory epithelium. The epithelium is ciliated, which facilitates the clearance of mucosal secretions. The frontal, maxillary, and ethmoid sinuses drain into an area known as the ostiomeatal complex. Rhythmic ciliary movement and the clearance of secretions can be impaired by several factors, including viral upper respiratory infections, allergic inflammation, and exposure to tobacco smoke and other irritants. In addition, foreign bodies (accidental or surgical) or a severely deviated nasal septum can cause obstruction. If blockage of the sinus ostia or obstruction of the ostiomeatal complex occurs, stasis of sinus secretions will allow pooling in the sinus cavities, which facilitates bacterial growth.

The most common organisms found in acute sinusitis are Streptococcus pneumoniae, Haemophilus influenzae, and Moraxella catarrhalis. In approximately 8 to 10% of cases of acute sinusitis, Bacteroides spp and Staphylococcus aureus are causative. Organisms that are commonly associated with chronic sinusitis are anaerobic bacteria such as Bacteroides spp, Fusobacterium spp, Streptococcus, Veillonella, and Corynebacterium spp. Sinusitis due to a fungal infection can rarely occur, usually in immunocompromised patients and in patients who are unresponsive to antibiotics.

Clinical and Laboratory Findings

The symptoms of acute sinusitis include facial pain, tenderness, and headache localized to the affected region. Sinusitis affecting the sphenoid sinuses or posterior ethmoid sinuses...
can cause headache or pain in the occipital region. Other symptoms that are commonly described include purulent nasal discharge, fever, malaise, and postnasal drainage with fetid breath. Occasionally, there may be toothache or pain with mastication. Patients with chronic sinusitis often present with other symptoms that are often vague and poorly localized. Chronic rhinorrhea, postnasal drainage, nasal congestion, sore throat, facial “fullness,” and anosmia are common complaints.

Physical examination reveals sinus tenderness and purulent nasal drainage. On occasion, erythema and swelling of the overlying skin may be evident. The nasal mucosa will appear edematous and erythematous, and nasal polyps might be visible.

Although not often needed, plain-film sinus radiography can be helpful in the diagnosis of acute maxillary or frontal sinusitis. Poor visualization of the ethmoid sinuses and limited visualization of the sphenoid sinuses affect the usefulness of this type of radiography. Plain-film radiography is not helpful for establishing osteomeatal complex disease. Computed tomography (CT) is the study of choice for documenting chronic sinusitis with underlying disease of the osteomeatal complex and is superior to magnetic resonance imaging (MRI) for the identification of bony abnormalities. CT can also accurately assess polyps, reactive osteitis, mucosal thickening, and fungal sinusitis.

**CLASSIFICATION**

Sinusitis is classified as either acute or chronic, based on the duration of the inflammation and underlying infection. Patients with persistent symptoms for 3 to 8 weeks or longer are considered to have chronic disease.

**DIAGNOSIS**

The diagnosis of acute sinusitis is made on the basis of history and physical examination. As previously noted, radiologic evaluations might be helpful in certain situations. Patients with recurrent disease need to be evaluated for underlying factors that can predispose to sinusitis. Allergy evaluation for allergic rhinitis is often helpful. Other predisposing factors such as tobacco smoke exposure, immunodeficiency, and septal deviation should be considered.

CT usually aids the diagnosis of chronic sinusitis. Evaluation of the osteomeatal complex is crucial in the management of these patients. In addition, rhinoscopy may be helpful for direct visualization of sinus ostia.

**MANAGEMENT**

Initial medical treatment consists of antibiotics to cover the suspected pathogens, along with topical or oral decongestants to facilitate sinus drainage. First-line antibiotics such as amoxicillin are often effective although second-generation cephalosporins, clarithromycin, and amoxicillin plus clavulanate can be helpful in resistant cases. Many patients who also have underlying allergic rhinitis may benefit from the addition of a topical nasal corticosteroid (many are available). Treatment courses often last 2 to 3 weeks. Acute frontal or sphenoid sinusitis is very serious because of the potential for intracranial complications. Intravenous antibiotics are indicated, and surgical intervention is considered, based on the condition’s response to medical management.

The management of chronic sinusitis involves antibiotics of a broader spectrum, and a prolonged treatment course may be required. Topical corticosteroids or short courses of oral corticosteroids may help reduce the swelling and/or obstruction of the osteomeatal complex. Avoidance of exacerbating factors such as allergens or tobacco smoke should be emphasized. Patients with histories suggestive of allergy should undergo a thorough allergy evaluation.

Patients who have chronic sinusitis with evidence of disease of the osteomeatal complex who fail medical management often require surgical intervention. Functional endoscopic sinus surgery involves the removal of the osteomeatal obstruction through an intranasal approach. This procedure can be performed with either local or general anesthesia and without an external incision. The recovery time from this procedure is short, and morbidity is generally low.

**PROGNOSIS**

Patients treated for acute sinusitis usually recover without sequelae. Children with sinusitis, particularly ethmoid and maxillary sinusitis, are at risk for periorbital or orbital cellulitis. Periorbital cellulitis is most often treated with intravenous antibiotics. Orbital cellulitis, on the other hand, requires prompt surgical intervention to prevent involvement of the globe or intracranial structures.

Frontal sinusitis can extend through the anterior wall and present as Pott’s puffy tumor. Sinusitis can also spread intracranially and result in abscess or meningitis. These complications, although uncommon, are more likely to occur in male adolescent patients.

Patients with chronic sinusitis are more likely to require a prolonged recovery period, with a resultant decrease in quality of life. Chronic medication use can lead to side effects or other complications, such as rhinitis medicamentosa from prolonged use of topical decongestants. Surgical intervention and underlying-factor assessment will often reverse the chronic process, leading to an improvement in quality of life.

**ORAL HEALTH CONSIDERATIONS**

Patients with sinus infections who present with a complaint of a toothache are commonly encountered in a dental office. The oral health care professional evaluating the patient must be able to differentiate between an odontogenic infection and sinus pain. On history, sinus infections usually present with pain involving more than one tooth in the same maxillary quadrant whereas a toothache usually involves only a single tooth. Ruling out odontogenic infections by a dental examination and appropriate periapical radiography strengthens a diagnosis.

Chronic sinus infections are often accompanied by mouth breathing. This condition is associated with oral dryness and (in longtime sufferers) increased susceptibility to oral conditions such as gingivitis.
As with other conditions for which the prolonged use of antibiotics is prescribed, the potential development of bacterial resistance needs to be considered. Switching to a different class of antibiotics to treat an odontogenic infection is preferable to increasing the dosage of an antibiotic that the patient has recently taken for another condition.

The use of decongestants may be associated with oral dryness, which may need to be addressed.

**Laryngitis and Laryngotracheobronchitis**

The upper airway is the site of infection and inflammation during the course of a common cold, but respiratory viruses can attack any portion of the respiratory tree. Laryngitis is defined as an inflammation of the larynx, usually because of a viral infection. Laryngotracheobronchitis (also termed viral croup) is an inflammation (also due to a viral illness) involving the larynx, trachea, and large bronchi. Although these illnesses have distinct presenting features, both result from a similar infectious process and the reactive inflammation that follows. Laryngitis can present at any age although it is more common among the adult population. In contrast, laryngotracheobronchitis is an illness seen primarily in young children and has a peak incidence in the second and third years of life. These infections are most common during the fall and winter months, when respiratory viruses are more prevalent.

The viruses most commonly implicated in laryngitis are the coxsackieviruses, adenoviruses, and herpes simplex virus. The viruses most commonly associated with laryngotracheobronchitis are parainfluenza virus, respiratory syncytial virus, influenza virus, and adenovirus.

Acute laryngitis can also result from excessive or unusual use of the vocal cords or from irritation due to tobacco smoking.

**PATHOPHYSIOLOGY**

The underlying infectious process is quite similar to that seen in viral infections of the upper respiratory tract (see above). After infection of the respiratory epithelium occurs, an inflammatory response consisting of mononuclear cells and polymorphonuclear leukocytes is mounted. As a result, vascular congestion and edema develop. Denudation of areas of respiratory epithelium can result. In addition to edema, spasm of laryngeal muscles can occur. Because the inflammatory process is triggered by viral infection, the disease processes are usually self-limited.

**CLINICAL AND LABORATORY FINDINGS**

Patients with laryngitis usually have an antecedent viral upper respiratory infection. Complaints of fever and sore throat are common. The most common manifestation of laryngitis is hoarseness, with weak or faint speech. Cough is somewhat variable in presentation and is more likely when the lower respiratory tract is involved.

Children presenting with viral croup commonly have an antecedent upper respiratory infection, which may include fever. Shortly thereafter, a barking cough and intermittent stridor develop. Stridor at rest, retractions, and cyanosis can occur in children with severer inflammation. Neck radiography will demonstrate subglottic narrowing (a finding termed “steeple sign”) on an anteroposterior view.

**CLASSIFICATION**

There is no universal classification system for these illnesses. The anatomic site most affected describes these diseases.

**DIAGNOSIS**

The diagnosis of laryngitis is based on the suggestive history. There are no specific findings on physical examination or laboratory tests although the presence of hoarseness is suggestive. The differential diagnosis includes other causes of laryngeal edema, including obstruction of venous or lymphatic drainage from masses or other lesions, decreased plasma oncotic pressure from protein loss or malnutrition, increased capillary permeability, myxedema of hypothyroidism, and hereditary angioedema. Carcinoma of the larynx can also present with hoarseness.

The diagnosis of laryngotracheobronchitis is usually apparent and is based on a suggestive history, with radiography confirming the clinical impression. With children, it is important to rule out other causes of stridor, including foreign body aspiration, acute bacterial epiglottitis, and retrophyaryngeal abscess.

**MANAGEMENT**

Most cases of laryngitis are mild and self-limited, so only supportive care need be prescribed. The use of oral corticosteroids in severe or prolonged cases can be considered although their routine use is controversial.

Treatment of laryngotracheobronchitis is also supportive. Cool-mist therapy and hydration are usually sufficient treatment. Hospitalization is usually indicated for patients with stridor at rest. Although somewhat controversial, a short course of oral or parenteral corticosteroids can reduce inflammation and help hasten recovery. Nebulized racemic epinephrine has been shown to temporarily relieve airway obstruction although rebound airway edema is common. The uncommon patient with impending respiratory failure requires endotracheal intubation or tracheotomy if intubation fails.

**PROGNOSIS**

As with viral upper respiratory infections, most cases of laryngitis and laryngotracheobronchitis are self-limited and require minimal medical intervention. Recovery within a few days to a week is the rule. In some cases, laryngotracheobronchitis can recur although the factors influencing this are not well understood.

**Pharyngitis and Tonsillitis**

Inflammation of the tonsils and pharynx is almost always associated with infection, either viral or bacterial. More than 90% of cases of sore throat are related to viral infections. These infections can be associated with fever, rhinorrhea, and cough.
The major viral etiologies are Epstein-Barr virus, coxsackievirus A, adenovirus, Rhinovirus, and measles virus.36

The most common bacterial cause of acute tonsillopharyngitis is group A beta-hemolytic Streptococcus (GABHS) infection, specifically Streptococcus pyogenes infection. Proper diagnosis and treatment of this infection is extremely important in order to prevent disease sequelae, namely, acute rheumatic fever and glomerulonephritis. Less common bacterial causes include Corynebacterium diphtheriae, Neisseria gonorrhoeae, Chlamydia, and Mycoplasma pneumoniae.

Chronic mouth breathing, chronic postnasal drainage, and inflammation due to irritant exposure can also cause pharyngitis and tonsillitis.

**PATHOPHYSIOLOGY**

Streptococcal infections are spread through direct contact with respiratory secretions. Transmission is often facilitated in areas where close contact occurs, such as schools and day care centers. The incubation period is 2 to 5 days.

**CLINICAL AND LABORATORY FINDINGS**

Sore throat is the predominant symptom. Associated clinical findings are based on the infectious etiology. Patients with Epstein-Barr virus infections develop infectious mononucleosis, a disease characterized by exudative tonsillitis, lymphadenopathy, fever, and fatigue. Physical examination can reveal hepatosplenomegaly. Common laboratory findings include leukocytosis with more than 20% atypical lymphocytes on blood smear. Blood chemistries may reveal elevated liver enzymes.

Infection with coxsackievirus can cause several distinct illnesses, each associated with tonsillitis. Herpangina is a disease that is characterized by ulcers that are 2 to 3 mm in size and located on the anterior tonsillar pillars and possibly the uvula and soft palate. Hand-foot-and-mouth disease is characterized by ulcers on the tongue and oral mucosa, in association with vesicles found on the palms and/or soles. Small yellow-white nodules on the anterior tonsillar pillars characterize lymphonodular pharyngitis; these nodules do not ulcerate.

Pharyngitis is characterized by exudative tonsillitis, conjunctivitis, and fever. Infection is due to an adenovirus.

Measles is a disease with a prodromal phase that is characterized by symptoms of upper respiratory infection, tonsillopharyngitis, and small white lesions with erythematous bases on the buccal mucosa and inner aspect of the lower lip (Koplik’s spots). These lesions are pathognomonic of early measles infection.

Streptococcal pharyngitis is characterized by exudative tonsillitis and fever. Physical examination often reveals a beefy red uvula, cervical adenitis, and oral petechiae. Laboratory evaluation should include a throat culture for group A Streptococcus.37

**CLASSIFICATION**

Pharyngotonsillitis is classified on the basis of etiology and clinical presentation (see above).

**DIAGNOSIS**

Diagnosis is based on a history of sore throat and is established by appropriate physical findings and results of a throat culture (see above). A rapid antigen detection test is available for diagnosing streptococcal pharyngitis. The test has a high specificity (95%+) but a low sensitivity (60 to 95%). Therefore, negative results should be confirmed by throat culture.

Antistreptolysin O titers rise about 150 U within 2 weeks of acute infection. These titers are useful for documenting recent streptococcal infections, especially in the course of acute rheumatic fever.

**MANAGEMENT**

The viral causes of tonsillopharyngitis are treated symptomatically. Gargle solutions, analgesics, and antipyretics are often helpful. The course is always self-limited.3

Acute streptococcal pharyngitis is treated with a 10-day course of oral penicillin V or erythromycin (for penicillin-sensitive individuals). Alternatives include an intramuscular injection of benzathine penicillin G or oral cephalosporins. Failure rates for penicillin vary from 6 to 23%, so an additional antibiotic course may be necessary.37

**PROGNOSIS**

The prognosis for viral tonsillopharyngitis is very good as the infections are self-limited. Late sequelae from group A streptococcal tonsillitis can be avoided by prompt diagnosis and treatment.38 Other complications due to streptococcal tonsillitis are uncommon but include cervical adenitis, peritonsillar abscesses, otitis media, cellulitis, and septicemia.

**ORAL HEALTH CONSIDERATIONS**

The association between GABHS infection and the development of severe complications, such as rheumatic fever and its associated heart condition, is well known. Although failure to successfully treat GABHS infections was more common in the pre-penicillin era, there are some concerns today regarding re-infection in cases in which penicillin is unable to eradicate the organism. One study found a significant association between the persistence of GABHS on toothbrushes and removable orthodontic appliances and the recovery of GABHS in the oropharynx of symptomatic patients after 10 days of treatment with penicillin.39 Interestingly, when toothbrushes were rinsed with sterile water, organisms could not be cultured beyond 3 days whereas nonrinsed toothbrushes harbored GABHS for up to 15 days. Thus, patients with GABHS infections should be instructed to thoroughly clean their toothbrushes and removable acrylic appliances daily. It is also advisable to change to a new toothbrush after the acute stage of any oropharyngeal infections.

**LOWER-AIRWAY INFECTIONS**

The association between oral health and respiratory diseases has recently received renewed attention. Several articles have suggested that dental plaque may be a reservoir for respiratory
Acute Bronchitis

Acute bronchitis is an inflammatory process of the large airways (trachea and bronchi) or what is commonly termed the lower respiratory tract. In patients who are otherwise healthy and without underlying pulmonary disease, bronchitis is most commonly caused by a viral infection. The viruses most commonly implicated are Rhinovirus, Coronavirus, influenza virus, parainfluenza virus, and adenovirus. Acute bronchitis due to bacterial infection is less common and is seen more commonly in patients who have chronic lung disease. The most common cause in this group is Streptococcus pneumoniae. Infection with Haemophilus influenzae is common in patients with chronic obstructive pulmonary disease (COPD). Other causes of acute bacterial bronchitis include Mycoplasma pneumoniae, Chlamydia pneumoniae, Bordetella pertussis, and Legionella spp. Staphylococcus and gram-negative bacteria are common causes of bronchitis among hospitalized individuals.

PATHOPHYSIOLOGY

The pathophysiology of acute bronchitis is similar to that of other respiratory tract infections. Following infection of the mucosal cells, congestion of the respiratory mucosa develops. Inflammation causes an increase in secretory activity, resulting in increased sputum production. Polymorphonuclear leukocytes infiltrate the bronchial walls and lumen. Desquamation of the ciliated epithelium may occur, and spasm of bronchial smooth muscle is common.

CLINICAL AND LABORATORY FINDINGS

Acute viral bronchitis usually presents with a viral prodrome consisting of fever, malaise, myalgias, headache, and weakness. Upper-respiratory-tract symptoms that may include sore throat and rhinorrhea usually follow. As the illness progresses, lower tract symptoms develop, with a prominent nonproductive cough. Chest discomfort may occur; this usually worsens with persistent coughing bouts. Other symptoms, such as dyspnea and respiratory distress, are variably present. Physical examination may reveal wheezing. The presentation may closely resemble an acute asthma exacerbation. Symptoms gradually resolve over a period of 1 to 2 weeks. Patients with underlying chronic lung disease might also experience respiratory compromise, with a significant impairment in pulmonary function.

The presentation of acute bacterial bronchitis is very similar to that of bacterial pneumonia (see below). Symptoms may include fever, dyspnea, productive cough with purulent sputum, and chest pain. Bacterial bronchitis can be differentiated from pneumonia by the lack of significant findings on chest radiography.

CLASSIFICATION

Although there is no universal classification scheme, acute bronchitis can be differentiated on the basis of etiology. Viral bronchitis presents differently than bacterial bronchitis, as described above.

DIAGNOSIS

Diagnosis of acute bronchitis is based on a suggestive history and a physical examination. Neither blood cell counts nor sputum analyses are particularly diagnostic in otherwise healthy patients. Chest radiography may be helpful in distinguishing bacterial bronchitis from pneumonia. Patients with recurrent bouts of acute bronchitis should be evaluated for possible asthma. This evaluation would include pulmonary function testing.

Patients with persistent symptoms in the course of presumed viral bronchitis should be evaluated to determine possible underlying etiologies. Sputum culture might prove useful in these circumstances.

MANAGEMENT

Viral bronchitis can be managed with supportive care only as most individuals who are otherwise healthy recover without specific treatment. If significant airway obstruction or hyperreactivity is present, inhaled bronchodilators such as albuterol can be useful. Cough suppressants such as codeine can also be used for patients whose coughing interferes with sleep.

The treatment of bacterial bronchitis includes antibiotics. Amoxicillin is an excellent first-line agent although macrolide antibiotics can be used for patients with penicillin allergy. Second-generation cephalosporins and amoxicillin/clavulanate are good second-line agents for patients with suspected infection due to β-lactamase–producing organisms. Inhaled bronchodilators are also helpful in cases with a bronchospastic component.

PROGNOSIS

Acute bronchitis carries an excellent prognosis for patients who are without underlying pulmonary disease, and recovery without sequelae is the norm. However, for patients with chronic lung disease and respiratory compromise, bronchitis can be quite serious and may often lead to hospitalization and respiratory failure. In other high-risk individuals, such as those with human immunodeficiency virus (HIV) infection or other immunodeiciencies, acute bronchitis may lead to the development of bronchiectasis.

ORAL HEALTH CONSIDERATIONS

Resistance to antibiotics may develop rapidly and last for 10 to 14 days. Thus, patients who are taking amoxicillin for acute bronchitis should be prescribed another type of antibiotic, (such as clindamycin or a cephalosporin) when an antibiotic is needed for an odontogenic infection.

Pneumonia

Pneumonia is defined pathologically as an infection and a subsequent inflammation involving the lung parenchyma. Both
viruses and bacteria are causes, and the presentation is dependent on the causative organism. There are an estimated 2.5 million cases of pneumonia each year. These cases can be broadly classified as either community acquired or nosocomial. Nosocomial infections are infections that are acquired in a hospital or health care facility and often affect debilitated or chronically ill individuals. Community-acquired infections can affect all persons but are more commonly seen in otherwise healthy individuals.

The most common bacterial cause of community-acquired pneumonia is *Streptococcus pneumoniae*, followed by *Haemophilus influenzae*. *Staphylococcus aureus* and gram-negative bacteria are common causes of nosocomial pneumonia. Pneumonia due to *Klebsiella pneumoniae* is seen in predominantly older patients and in those with a history of alcoholism. Atypical organisms commonly associated with pneumonia include *Mycoplasma pneumoniae*, *Legionella*, and *Chlamydia*.48 The atypical organisms cause a pneumonia that differs in clinical presentation from that caused by the aforementioned bacteria (see below). Pneumonia can also be caused by viruses; by fungi such as *Candida*, *Histoplasma*, *Cryptococcus*, and *Aspergillus*; and by protozoa such as *Pneumocystis carinii* (seen in immunocompromised hosts), *Nocardia*, and *Mycobacterium tuberculosis*. Infection with these organisms can often be differentiated by chest radiography.

**PATHOPHYSIOLOGY**

The pathophysiology of pneumonia is dependent on the causative infectious organism. In bacterial pneumonia caused by *Streptococcus pneumoniae*, for example, the bacteria first enter the alveolar spaces after inhalation. Once inside the alveoli, the bacteria rapidly multiply, and extensive edema develops. The bacteria cause a vigorous inflammatory response, which includes an influx of polymorphonuclear leukocytes. In addition, capillary leakage is pronounced. As the inflammatory process continues, the polymorphonuclear leukocytes are replaced by macrophages. Subsequent deposition of fibrin ensues as the infection is controlled, and the inflammatory response resolves.52

Atypical infections of the lung (ie, viral, mycoplasmal, etc.) are interstitial processes. The organisms are first inhaled into the alveolar spaces. The organisms then infect the type I pneumocytes directly. As these pneumocytes lose their structural integrity and necrosis ensues, alveolar edema begins. Type II pneumocytes proliferate and line the alveoli, and an exudative cellular debris accumulates. An interstitial inflammatory response is mounted, primarily by mononuclear leukocytes. This process can occasionally progress to interstitial fibrosis although resolution is the norm.

**CLINICAL AND LABORATORY FINDINGS**

Pneumonia due to community-acquired bacterial infection (*Pneumococcus*) typically presents acutely, with a rapid onset of symptoms. A prodrome similar to that seen with acute infections of the upper respiratory tract is unusual. Common symptoms include fever, pleuritic chest pain, and coughing that produces purulent sputum.49 Chills and rigors are also common. Pneumonia due to *Haemophilus influenzae*, which is seen more commonly in patients with COPD or alcoholism, presents with fever, cough, and malaise. Chest pain and rigors are less common.

Nosocomial pneumonia due to *Staphylococcus* or gram-negative bacteria is usually associated with a prodrome due to an antecedent viral upper respiratory infection. Symptoms of pneumonia, including cough and fever, develop several days after the onset of the upper respiratory symptoms.

Physical examination demonstrates crackles (rales) in the affected lung fields. Decreased breath sounds and dullness to percussion might also be noted. Signs of respiratory distress may be present in severely affected individuals.

As many as 25% of all cases of community-acquired pneumonia are considered atypical. Symptoms usually develop over 3 to 4 days and initially consist of low-grade fever, malaise, a nonproductive cough, and headache. Sputum production, if present, is usually minimal. Findings on physical examination of the chest are usually unremarkable, with only scattered rhonchi. Infection due to *Mycoplasma* is common among younger patients. This organism is commonly spread to other family members. The onset of symptoms is gradual, and symptoms often include pharyngitis. Evidence of bullous disease of the tympanic membranes (bullous myringitis) is highly suggestive of mycoplasmal infection. Pneumonias due to viral causes have a similar presentation but can have a more rapid onset. Influenza virus is the most common viral etiology.

Infection with *Legionella* (legionnaires’ disease) begins with a prodrome consisting of fever and malaise and progresses rapidly to an acute phase of high fever, rigors, pleuritic chest pain, gastrointestinal complaints, and confusion. The cough is typically nonproductive and is only variably present. Elevated liver enzymes and proteinuria indicate renal and hepatic involvement. Hypoxia can also develop and can rapidly progress. Legionnaires’ disease was first described at an American Legion convention in Philadelphia in 1976. The causative organisms have a predilection for moist areas such as air-conditioning ducts and cooling towers. The infection tends to occur more commonly among middle-aged men with a history of tobacco smoking.

**CLASSIFICATION**

Pneumonia is initially classified on clinical presentation as either bacterial or atypical. Different classifications based on radiologic or pathologic manifestations are less commonly used.

**DIAGNOSIS**

When a patient with probable pneumonia is being evaluated, the possible causative organism will be suggested by (1) the clinical presentation and course of the illness, (2) the degree of immunocompetency of the patient, (3) the presence or absence of underlying lung disease, and (4) the place of acquisition (hospital or community). Ultimately, the goal is rapid diagnosis to establish an etiology so that appropriate antimicrobial therapy can be initiated. Sputum analysis is the most
important tool for diagnosis and management. Spontaneously coughed or induced sputum should be immediately analyzed by Gram’s stain. This will allow identification of the likely etiology and thus a more directed antibiotic therapy. Gram-positive cocci in pairs (diplococci) are suggestive of pneumococcal infection. Gram-positive cocci in clusters suggest infection with Staphylococcus aureus. Gram-negative pleomorphic rods are typical of Haemophilus influenzae, whereas Klebsiella is identified by its short plump gram-negative-rod appearance. Numerous polymorphonuclear leukocytes are also often seen.

Culture of sputum samples is used to help identify the causative organism. Routine culture can identify Streptococcus pneumoniae, Haemophilus influenzae, Staphylococcus aureus, and gram-negative rods. Specialized culturing techniques are needed to identify Legionella, Mycobacterium, Nocardia, Mycoplasma, and fungi. Tissue cultures are used to identify viruses and Chlamydia. Other cultures, such as blood and pleural fluid cultures, can be analyzed. Blood cultures are done routinely because many organisms are identified in this manner. For example, blood cultures are positive in 35% of patients with pneumococcal disease and in 15% of those with Klebsiella infection.

Chest radiography can be a valuable tool in the evaluation of the patient with pneumonia. The radiologic presentation is dependent on the infectious etiology and the underlying medical condition of the patient. A pattern of lobar consolidation and air bronchograms is seen most commonly in cases of pneumococcal pneumonia. The lower lobes and right middle lobe are most commonly involved. A pattern of patchy non-homogenous infiltrates, pleural effusion, and cavitary lesions are common with staphylococcal pneumonia. Klebsiella pneumonia typically involves multiple lobes and can also be associated with effusion and cavitation. Viral or atypical organisms usually present with an interstitial infiltrative pattern or patchy segmental infiltrates. Organisms such as Nocardia, Mycobacterium, and fungi often cause nodular or cavitary lesions, which are demonstrable on chest radiography. Rapid accumulation of pleural fluid or empyema is seen most often with bacterial infection. Pneumococcal pneumonia is associated with pleural effusion in 10 to 15% of cases.

The presence of cold agglutinins is suggestive of Mycoplasma infection. Cold agglutinins are antibodies (produced in response to Mycoplasma infection) that agglutinate red blood cells upon cold exposure. Titters reach maximal levels in 3 to 4 weeks but can be detected 1 week after the onset of disease. These antibodies can be found in 60 to 70% of patients with Mycoplasma pneumonia but are not specific to this disease.

Legionella pneumonia is diagnosed either by culture of the organisms, using specialized media, or by direct fluorescent antibody staining of sputum.

MANAGEMENT

Empiric treatment is started immediately upon diagnosis of pneumonia. When a pneumococcal infection is suspected, treatment with penicillin is effective although penicillin resistance has emerged in several areas of the world. Alternatives include the cephalosporins and macrolide antibiotics. Symptoms begin to improve within 1 to 2 days although chest radiograph abnormalities may persist for months. Treatment for Haemophilus influenzae pneumonia includes second-generation cephalosporins or ampicillin/clavulanate. Clarithromycin or quinolone antibiotics are alternatives. Erythromycin is the antibiotic of choice for pneumonia caused by Legionella or Mycoplasma. Alternatives include the quinolone antibiotics or clarithromycin.

Non-specific treatment for patients with pneumonia includes aggressive hydration to aid in sputum clearance. Chest physiotherapy is advocated by many clinicians although evidence of efficacy is lacking. If hypoxia is present, supplemental oxygen is given. A pneumococcal vaccine is available for active immunization against pneumococcal disease. The vaccine is effective for preventing disease from 85% of pneumococcal serotypes. It is effective for adults and for children older than 2 years of age and is recommended for high-risk individuals, such as those with asplenia and all individuals over the age of 65 years.

PROGNOSIS

Mortality due to community-acquired pneumonia is low. The risk of mortality is higher for older patients, patients with underlying pulmonary disease, patients with immunodeficiency (ie, asplenia), and patients with positive blood cultures. Most deaths occur within 5 days of the onset of disease.

Mortality due to staphylococcal pneumonia is high, and patients who do recover often have residual pulmonary abnormalities. Mortality due to atypical pneumonia is low, with the exception of Legionella pneumonia, which has a 15% mortality rate if left untreated.

ORAL HEALTH CONSIDERATIONS

The aspiration of salivary secretions containing oral bacteria into the lower respiratory tract can cause pneumonia. Numerous periodontally associated oral anaerobes and facultative species have been isolated from infected pulmonary fluids. Although most reports suggest increased susceptibility to the development of nosocomial pneumonia from periodontal pathogens, other oral bacteria (such as Streptococcus viridans) have been implicated in community-acquired pneumonia. Colonization of dental plaque and oral mucosa with respiratory pathogens is more prevalent among patients in medical intensive care units (ICUs). Furthermore, the amount of dental plaque among ICU patients has been shown to increase over time, resulting in the occurrence of nosocomial pneumonia with pathogens isolated from the dental plaque. However, prerinsing with a 0.12% chlorhexidine gluconate mouth rinse may significantly reduce the mortality of nosocomial pneumonia in ICU patients.

Elderly individuals residing in nursing homes have an increased prevalence of poor oral health, including increased plaque retention. Studies have evaluated the occurrence of
pneumonia in cohorts of elderly individuals who were receiving and not receiving oral care. In one such study, the relative risk of developing pneumonia increased 67% in the group without access to oral health interventions, compared with individuals who had access to oral care. This data support the benefit of increased awareness and increased oral health interventions in hospitalized and institutionalized individuals. More intervention studies are needed to assess the impact of oral pathogens on the incidence of pneumonia, but at present, there is ample evidence that poor oral health status is a risk indicator for the development of pneumonia.

**Bronchiolitis**

Bronchiolitis is a disease that affects children under the age of 2 years; it is most common among infants aged 2 to 12 months. It is characterized by inflammation of the lower respiratory tract, with the bronchioles being most affected. The inflammatory response is secondary to an infectious trigger, usually respiratory syncytial virus (RSV). Other organisms associated with bronchiolitis are parainfluenza virus, influenza virus, adenovirus, and *Mycoplasma pneumoniae*.63

**PATHOPHYSIOLOGY**

Infection of the bronchioles leads to a marked inflammatory response with a prominent mononuclear cell infiltrate. This inflammatory response results in mucosal edema with cellular debris, mucosal thickening, and mucous hypersecretion and plugging. Bronchiolar spasm is an occasional feature. Due to these changes, the lumina of the bronchioles are critically narrowed, leading to areas of microatelectasis and emphysema. Respiratory compromise is common, with decreased blood oxygen saturation, hypercarbia, respiratory acidosis, and in severe cases, respiratory failure.

**CLINICAL AND LABORATORY FINDINGS**

Infants first develop signs and symptoms of an infection of the upper respiratory tract, with low-grade fever, profuse clear rhinorrhea, and cough. Signs of infection in the lower respiratory tract soon follow, including tachypnea, retractions, wheezing, and (on occasion) cyanosis. Crackles can be audible, and thoracic hyper-resonance can be noted on percussion. Associated findings can include conjunctivitis, otitis media, and pharyngitis.

Chest radiography shows peribronchial cuffing, flattening of the diaphragms, hyperinflation, and increased lung markings.

Laboratory studies reveal a mild leukocytosis with a prominence of polymorphonuclear leukocytes ("left shift").

**CLASSIFICATION**

Bronchiolitis can be classified by the causative agent, as is the case with acute bronchitis.

**DIAGNOSIS**

The diagnosis is clinical, based on history and physical examination. The etiology can be determined (and the diagnosis confirmed) by performing a nasopharyngeal culture for RSV and other respiratory viruses. Rapid viral diagnostic assays are also available.

The differential diagnosis includes other causes of wheezing and respiratory distress in this age group, such as asthma, heart failure due to congenital heart disease, and cystic fibrosis.

**MANAGEMENT**

Infants are usually managed in cool-mist oxygen tents, where continuous oxygen administration can be given. Due to an increase in insensible water losses, hydration must be ensured. Aerosolized bronchodilators are often used although their routine use is not recommended. Oral or parenteral corticosteroids are often used although validation of their efficacy through clinical trials is lacking.

Antiviral therapy with ribavirin is recommended for infants with severe disease, congenital heart disease, or underlying pulmonary disease. Ribavirin is delivered by aerosol on a semicontinuous basis for up to 1 week.64

Mechanical ventilation is required in the infant with respiratory failure. Very young infants (less than 1 month of age) are at risk for apnea due to RSV infection, so close observation is required.

Anti-RSV immunoglobulin preparations are available for passive immunization against RSV. These preparations are currently recommended for high-risk patient populations. A vaccine is under development for the prevention of RSV-related disease.65

**PROGNOSIS**

Although mortality due to bronchiolitis is not uncommon, most patients recover without sequelae. A subset of patients develop recurrent bronchospasm following RSV bronchiolitis. It is not known whether or not this represents a risk factor for persistent asthma in later childhood.66

**Asthma**

Asthma is a chronic disease that affects the lower airways. It is characterized by recurrent and reversible airflow limitation due to an underlying inflammatory process. The etiology of asthma is unknown, but allergic sensitivity is seen in most patients with asthma. Genetic factors play a role, but no single gene or combination of genes has yet been identified as causative.

In the United States, asthma affects more than 14 million people. Its onset is most commonly during childhood, and almost 5 million children are affected. Asthma mortality numbers 5,000 people per year. In addition, the care of asthmatic persons represents a significant economic and social burden, accounting for numerous hospitalization days and days missed from school and work. These trends do not appear to be declining despite advances in our understanding of asthma and despite new pharmacologic modalities.

**PATHOPHYSIOLOGY**

The clinical features of asthma are due to the underlying chronic inflammatory process. Although the etiology is not
known, certain histopathologic features provide insights into the chronic process. Inflammatory infiltrates are rich in activated eosinophils and T helper lymphocytes, suggesting an allergic process. Degranulated mast cells are in close proximity to the affected airway, most likely representing allergen-mediated activation. The inflammatory cascade results in disruption of the integrity of the normal airway. There is destruction of the airway epithelium, bronchial smooth-muscle hypertrophy, sub-basement membrane collagen deposition, edema, and mucous plugging, all of which are demonstrable in vivo.

Airway inflammation has been characterized as acute, subacute, or chronic. The acute inflammatory state is due to the release of mediators from activated inflammatory cells that are resident in the airways (such as histamine release from degranulated mast cells). Subacute inflammation is characterized by early cellular infiltrates, most notably eosinophils, and the release of mediators with direct toxicity to airway epithelium. Chronic inflammation is characterized by persistent ongoing inflammation mediated by lymphocytes and eosinophils, with resultant damage and repair processes. This may lead to irreversible airway obstruction in a subset of patients with asthma.

Airflow limitation develops as a result of the airway inflammation. Several inter-related factors play a role, including mucous plugging, airway edema, bronchospasm and constriction, and airway remodeling due to basement membrane thickening and sub-basement membrane fibrosis. The signs and symptoms of asthma are the direct results of these processes.

Atopy is the strongest risk factor associated with the development of asthma. Persistent exposure to relevant allergens in a sensitized individual can lead to chronic allergic inflammation of the airways. Although atopy is seen more commonly in childhood-onset asthma, it can also play an important role in asthma in adults.

**CLINICAL AND LABORATORY FINDINGS**

The hallmark clinical features of asthma are recurrent reversible airflow limitation and airway hyper-responsiveness. These factors lead to the development of the signs and symptoms of asthma, which include intermittent wheezing, coughing, dyspnea, and chest tightness. Symptoms of asthma tend to worsen at night and in the early morning hours. In addition, well-defined triggers may precipitate asthma symptoms. These triggers include allergens, exercise, cold air, respiratory irritants, emotional extremes, and infections (especially viral infections). Symptoms can progress slowly over time, or they may develop abruptly.2,67,68

Historical points that suggest asthma are chronic coughing with nocturnal awakenings, dyspnea or chest tightness with exertion, recurrent “bronchitis” associated with infections of the upper respiratory tract, and wheezing that occurs on a seasonal basis. Physical examination of patients with mild disease often shows no abnormalities. However, common findings in patients with more severe disease include an increased anteroposterior chest diameter, a prolonged expiratory phase, wheezing, and diminished breath sounds. Digital clubbing is rarely seen. Concurrent allergic disease such as allergic rhinitis may be present. During acute exacerbations, patients may show signs of respiratory distress, with tachypnea, intercostal retractions, nasal flaring, and cyanosis.

Spirometry is the best tool available to aid in the diagnosis and management of asthma. Spirometry allows the measurement of lung capacity and airflows and can be performed during a routine office visit. The technique involves a maximal forced expiration following a maximal inspiration. The key measurements are the forced vital capacity (FVC), which is the amount of air expired during the forced expiration, and the forced expiratory volume in 1 second (FEV₁), which is the amount of air expired during the first second of expiration; FEV₁ is a measure of the rate at which air can be exhaled. Given the FEV₁ and the FEV₁/FVC ratio, an objective determination of airflow limitation is possible. Reversibility can be demonstrated after administration of a short-acting bronchodilator (such as albuterol) and a repeat spirometric measurement. In patients with normal baseline spirometry values, a demonstration of bronchial hyperresponsiveness is useful. This is performed by bronchoprovocation, using nonspecific triggers such as histamine or methacholine. When delivered by aerosol, these agents allow the determination of bronchial hyperreactivity by triggering a decrease in the FEV₁ immediately following inhalation.

Measurement of the peak expiratory flow rate (PEFR) can be a useful adjunct for diagnosis and management of asthma. Patients with asthma often demonstrate a diurnal variation of 20% or more, when early-morning values are compared to evening values. The PEFR is easy to determine, and durable metering devices are available at little cost. In addition, measurement of PEFRs can predict when asthma is worsening and is often used as an indicator of asthma severity in patients who require daily anti-inflammatory therapy.

Allergen skin testing is another valuable tool. This testing allows the accurate identification of allergic triggers, which can translate into more specific therapies such as allergen avoidance and immunotherapy (see “Allergic Rhinitis and Conjunctivitis,” above). Chest radiography may be useful, especially as a means of excluding other diseases from the diagnosis.

**CLASSIFICATION**

Asthma is classified according to its severity. Although there is no universal classification scheme, the guidelines set forth by the National Asthma Education and Prevention Program (NAEPP) are the most widely used in the United States.69 Asthma patients are classified as having mild-intermittent, mild-persistent, moderate-persistent, or severe-persistent disease. The categories are defined by both subjective (historical) and objective (spirometric) points. Treatment guidelines are based on the level of severity of the patient’s disease, and the classification can therefore change over time.70 Asthma may also be classified by the underlying trigger (eg, exercise-induced asthma and occupational asthma).
DIAGNOSIS

The diagnosis of asthma is made on the basis of a suggestive history, confirmatory physical findings, and the demonstration of reversible airflow limitation. This can be documented during hospitalization, by outpatient use of spirometry or PEFR determinations, or by clinical assessment after therapeutic trials.

The differential diagnosis of asthma includes other causes of chronic coughing and wheezing. The diseases that are usually considered are chronic rhinitis or sinusitis, cystic fibrosis, gastroesophageal reflux disease, airway narrowing due to compression (ie, masses), and COPD (chronic bronchitis). Factors favoring the diagnosis of asthma include intermittent symptoms with asymptomatic periods, complete or nearly complete reversibility with bronchodilators, the absence of digital clubbing, and a history of atopy.

MANAGEMENT

The goals of asthma management include the patient’s having little or no chronic symptoms, few or no exacerbations, no hospitalizations, and minimal or no activity limitation. Ideal control would include no need for short-acting bronchodilators, normal PEFRs, no PEFR variability, and no adverse effects from controller medications. All patients with asthma, regardless of its severity, should have an asthma control plan to aid in understanding the underlying process and treatment options and to effectively treat asthma exacerbations. Regular monitoring of asthma is important; spirometry, PEFR measurement, and questionnaires are useful for this purpose. Avoidance control measures are regularly emphasized, focusing on allergen and irritant triggers. Treatment for concomitant diseases that may exacerbate asthma (such as allergic rhinitis, gastroesophageal reflux disease, and chronic sinusitis) should be instituted.

Pharmacotherapy of asthma is based on the severity of disease. NAEP guidelines provide written algorithms to aid in treatment plan development. Patients with mild-intermittent disease usually require short-acting bronchodilators on an as-needed basis. These medications (such as albuterol) are preferably administered by inhalation. Preparations are available in metered-dose inhalers, in dry-powder inhalers, and as solutions for a nebulizer. Patients with mild-persistent asthma require routine therapy for control of underlying airway inflammation. Inhaled corticosteroids are the most widely used and most effective asthma anti-inflammatory agents.

They have an excellent safety profile at conventional doses although high-dose therapy can put patients at risk for corticosteroid side effects. These medications have been used for decades in both children and adults without significant long-term side effects in most patients. However, a concern about growth suppression in children has mandated warning labels on all inhaled corticosteroids. The long-term consequences of this short-term growth suppression are yet to be determined. Alternative medications include the non-steroidal anti-inflammatory agents nedocromil and cromolyn. These medications have less potent anti-inflammatory effects and are therefore most helpful for patients with mild disease. Leukotriene receptor antagonists such as montelukast and zafirlukast have been used successfully as monotherapy in patients with mild-persistent asthma. Although the role of leukotrienes in allergic inflammation is well known, the long-term benefit of these agents when used alone is yet to be determined.

Patients with moderate and severe persistent disease require more intensive therapy. Long-acting bronchodilators such as salmeterol have been shown to have an additive effect when used with inhaled corticosteroids and are useful additions to inhaled-corticosteroid therapy. Leukotriene receptor antagonists also are often a helpful addition to inhaled corticosteroids. A minority of patients might require long-term corticosteroids; these patients are difficult to manage, but adequate symptom control while minimizing the dose is of paramount importance.

Patients with allergic triggers may benefit from allergen immunotherapy. Many studies have now documented improvement from following a 3- to 5-year course of specific immunotherapy. This is an excellent means of minimizing medications while maintaining control for many patients.

PROGNOSIS

Although asthma is not a curable disease, it is a controllable disease. Asthma education programs are extremely important in making early diagnosis and interventions possible. Despite an increase in our knowledge of the underlying pathophysiology, asthma mortality rates have not declined. With early diagnosis and a comprehensive management plan, patients with asthma can experience a normal life expectancy with good quality of life.

ORAL HEALTH CONSIDERATIONS

The main concern when treating any medically complex patient is to avoid exacerbation of the underlying condition. Several protocols suggesting appropriate procedures for dental treatment of asthmatic patients have been put forth. However, few studies assessing the respiratory response of patients to dental care have been performed. One recent study indicated that although 15% of asthmatic pediatric patients will have a clinically significant decrease in lung function, no clinical parameter or historical data pertaining to asthma can predict this phenomenon.

However, numerous dental products and materials, including toothpaste, fissure seals, tooth enamel dust, and methyl methacrylate, have been associated with the exacerbation of asthma whereas other items (such as fluoride trays and cotton rolls) have been suggested as being so associated.

There is still no consensus regarding the association between asthma and dentofacial morphology. Although nasal respiratory obstruction resulting in mouth breathing has been implicated in the development of a long and tapered facial form, an increased lower facial height, and a narrow maxillary arch, this relationship has never been substantiated with unequivocal evidence.

Oral manifestations include candidiasis, decreased salivary flow, increased calculus, increased gingivitis, increased peri-
odontal disease, increased incidence of caries, and adverse effects of orthodontic therapy. It is possible that prolonged use of β₂-agonists may cause reduced salivary flow, with a resulting increase in cariogenic bacteria and caries and an increased incidence of candidiasis. The increased incidence of caries is further accelerated by the use of cariogenic carbohydrates and sugar-containing anti-asthmatic medications.

Dental treatment for asthmatic patients needs to address the oral manifestations of this condition, as well as its potential underlying systemic complications. Elective dental procedures should be avoided in all but those whose asthma is well controlled. The type and frequency of asthmatic attacks, as well as the type of medications used by the patient, indicate the severity of the disease.

The following are considerations and recommendations for administering dental care to patients who have asthma:

1. Fluoride supplements should be instituted for all asthmatic patients, particularly those taking β₂-agonists.
2. The patient should be instructed to rinse his or her mouth with water after using inhalers.
3. Oral hygiene should be reinforced to reduce the incidence of gingivitis and periodontitis.
4. Antifungal medications should be administered as needed, particularly in patients who are taking inhaled corticosteroids.
5. Steroid prophylaxis need to be used with patients who are taking long-term systemic corticosteroids (see Chapter X).
6. Use stress-reducing techniques. Conscious sedation should be performed with agents that are not associated with bronchoconstriction, such as hydroxyzine. Barbiturates and narcotics should be avoided due to their potential to cause bronchospasm and reduce respiratory functions. Nitrous oxide can be used for all but patients with severe asthma as it may irritate the airways.
7. Avoid dental materials that may precipitate an attack. Acrylic appliances should be cured prior to insertion. Dental materials without methyl methacrylate should be considered.
8. Schedule these patients’ appointments for late morning or later in the day, to minimize the risk of an asthmatic attack.
9. Have oxygen and bronchodilators available in case of an exacerbation of asthma.
10. There are no contraindications to the use of local anesthetics containing epinephrine, but preservatives such as sodium metabisulfite may contribute to asthma exacerbation in susceptible patients. Nevertheless, interactions between epinephrine and β₂-agonists may result in a synergistic effect, producing increased blood pressure and arrhythmias.
11. Judicious use of rubber dams will prevent reduced breathing capability.
12. Care should be used in the positioning of suction tips as they may elicit a cough reflex.
13. Up to 10% of adult asthmatic patients have an allergy to aspirin and other nonsteroidal anti-inflammatory agents. A careful history concerning the use of these type of drugs need to be elicited. Although the use of acetaminophen has been proposed as an alternative to the use of aspirin, recent data suggest caution because these type of drugs have also been associated with more severe asthma.
14. Drug interactions with theophylline are common. Macrolide antibiotics may increase the level of theophylline whereas phenobarbitals may reduce the level. Furthermore, drugs such as tetracycline have been associated with more accentuated side effects when given together with theophylline.
15. During an acute asthmatic attack, discontinue the dental procedure, remove all intraoral devices, place the patient in a comfortable position, make sure the airway is opened, and administer a β₂-agonist and oxygen. If no improvement is noted, administer epinephrine subcutaneously (1:1,000 concentration, 0.01 mg/kg of body weight, up to a maximum of 0.3 mg) and alert emergency medical assistance.

Chronic Obstructive Pulmonary Disease

“Chronic obstructive pulmonary disease” is a term used to describe chronic and largely irreversible airway obstruction due to inflammation of the lower airways. Chronic bronchitis is COPD due to chronic bronchial inflammation. Chronic bronchitis is diagnosed on clinical criteria and is defined as coughing and sputum production for 3 or more months per year for at least 2 consecutive years. Emphysema is diagnosed by histopathology and is defined by enlarged air spaces and the loss of alveolar tissue. The hallmark features of COPD are dyspnea and hypoxemia. Alveolar hypoventilation and diffusion impairment causes hypercarbia, which may result in pulmonary hypertension and cor pulmonale. COPD is almost always due to the smoking of tobacco although air pollution has also been implicated. A deficiency in the enzyme α₁-antitrypsin causes a syndrome of emphysema only. This enzyme is responsible for inhibiting the activity of trypsin and other proteases in the serum and tissues. The characteristic panlobular emphysematous changes that are seen in α₁-antitrypsin deficiency are related to the loss of alveolar walls.

Tobacco smoking accelerates this process.

The clinical course of patients with COPD is quite varied. Most patients display some degree of progressive dyspnea, exercise intolerance, and fatigue. In addition, patients are susceptible to frequent exacerbations, usually caused by infections of the upper or lower respiratory tract. Most patients with COPD have little respiratory reserve. Therefore, any process that causes airway inflammation can lead to clinical deterioration.

PATHOPHYSIOLOGY

Many toxins in tobacco smoke can cause a vigorous inflammatory response. Acrolein, for example, causes impairment of both ciliary and macrophage activities. Nitrogen dioxide
causes direct toxic damage to the respiratory epithelium. Hydrogen cyanide is responsible for the functional impairment of enzymes that are required for respiratory metabolism. Carbon monoxide causes a decrease in the oxygen-carrying capacity of red blood cells by associating with hemoglobin to form carboxyhemoglobin. Lastly, polycyclic hydrocarbons have been implicated as carcinogens.

Episodes of infection can precipitate exacerbations of chronic bronchitis. Patients with chronic bronchitis develop bacterial colonization of the tracheobronchial tree. It is not known, however, whether these bacteria are responsible for clinical deterioration. Infection due to *Mycoplasma* pneumonia or viruses is associated with exacerbation in up to one-third of patients with chronic bronchitis.

Histopathologically, patients with end-stage chronic bronchitis display an increased number of airway goblet cells, hypertrophy of mucus glands, squamous metaplasia of the airway epithelium, and mucosal edema. The airways are obstructed by mucous plugging and edema due to the ongoing inflammatory infiltrate. Mucous plugging and narrowing due to fibrosis is also seen in the smaller (2 to 3 mm) airways. Destruction of the alveolar walls leads to emphysema due to fibrosis is also seen in the smaller (2 to 3 mm) airways. Extensive fibrosis leads to the characteristic clinical patient presentation termed the “blue bloater.”

Hypoxemia is the result of the ventilation-perfusion mismatch that accompanies airway obstruction and emphysema. Portions of the lung that are not aerated due to obstruction cannot oxygenate the blood. This causes a decrease in overall oxygen concentrations. In addition, emphysema causes a decreased diffusion capacity because of a loss of air-space capillary units. Hypercarbia also develops and is often progressive and asymptomatic. Pulmonary hypertension can result from chronic hypoxia due to vas constriction of pulmonary vessels.

Patients with emphysema alone have less ventilation-perfusion mismatching early in the course of the disease; this is due to the loss of both air space and supplying blood vessels. Severe hypoxia, pulmonary hypertension, and cor pulmonale are not seen until late in the disease process. Emphysema manifests as loss of the elastic recoil of the lungs, making the lungs more compliant. The work of breathing is therefore not significantly increased. However, the decrease in recoil allows the easy collapse of the peripheral airways, leading to airway obstruction and airflow limitation.

**CLINICAL AND LABORATORY FINDINGS**

Patients with chronic bronchitis present with dyspnea, cough, and sputum production. An increase in the production of often purulent sputum is a sign of exacerbation due to respiratory infection. Physical findings include diffuse wheezing, possibly associated with signs of respiratory distress including the use of accessory muscles of respiration (retractions) and tachypnea. Liver enlargement due to congestion, ascites, and peripheral edema can develop as the disease progresses to pulmonary hypertension and cor pulmonale. This leads to the characteristic clinical patient presentation termed the “blue bloater.”

Patients with emphysema present primarily with dyspnea. Patients can be adequately oxygenated in the early stages of the disease and thus can have fewer signs of hypoxia; the term “pink puffer” has been used to describe these patients. Physical findings include an increase in chest wall size. Wheezing is present to varying degrees.

Chest radiography may show evidence of an increase in lung compliance, with flattened diaphragms, hyperexpansion, and an increase in anteroposterior diameter. Spirometry will show evidence of airflow limitation, with decreases in the FEV1 and the FEV1/FVC ratio. Complete pulmonary function studies will also indicate an increase in residual volume (RV) and total lung capacity. Pulmonary diffusion capacity will be decreased due to a loss of gas-exchanging units.

**CLASSIFICATION**

COPD is traditionally divided into two major categories: chronic bronchitis and emphysema. Airway obstruction in chronic bronchitis is due to bronchospasm, edema, and mucous plugging of the airways as a result of chronic inflammation. In emphysema, airway obstruction occurs because of the loss of lung elasticity and the resultant collapse of the airways. It is uncommon for patients to fit neatly into one category. Most patients have features of both emphysema and chronic bronchitis.

**DIAGNOSIS**

The diagnosis is suggested by the history and physical findings. Alternative diagnoses such as asthma, cystic fibrosis, and congestive heart failure should be considered. Complete pulmonary function tests are a valuable means of assessing airflow limitation and the reversibility of airflow obstruction. For patients with more severe disease, assessment of oxygen status with pulse oximetry is a valuable office procedure. A determination of arterial blood gases is important for patients who are clinically deteriorating and for the management of hospitalized patients. Chest radiography can be helpful, but it is often used only as an adjunct to the above diagnostic investigations.

**MANAGEMENT**

There are no curative treatments for chronic bronchitis and emphysema. Management focuses on maintaining quality of life and preventing exacerbations. Maintenance therapy includes trials of inhaled bronchodilators such as albuterol and ipratropium bromide. Theophylline products have also been used with some efficacy. Inhaled corticosteroids do not benefit all patients with COPD although some patients might experience some improvement. A recent large multicenter study indicated that patients treated with inhaled corticosteroids did not show any significant slowing of the decline in lung function but did have fewer symptoms and improved sensitivity of the lungs to external stimuli.

Chest physiotherapy has not been proven to be of value in the management of COPD. During exacerbations, oxygen therapy is often required. Caution must be used when administering oxygen to patients
with COPD as their ventilatory drive will often be diminished. This is the result of chronic retention of carbon dioxide and subsequent insensitivity to hypercarbia. As a result, patients with COPD are sensitive to increases in oxygen tension, which provides the major stimulus for respiratory drive. A partial pressure of arterial oxygen (PaO₂) of 55 to 60 mm Hg is often a reasonable goal to help reduce hypoxemia while maintaining respiratory drive. Oxygen therapy during sleep can also be a useful means of limiting hypoxemia and subsequent pulmonary hypertension.

Antibiotics are used during exacerbations of chronic bronchitis. Typical treatment includes 7 to 10 days of an oral broad-spectrum antibiotic, such as a second-generation cephalosporin. Although an underlying infectious etiology should be sought, clinical responses to antibiotic treatment do not always correlate with organisms isolated from sputum cultures.

Early inflammatory changes are visible in the bronchi of young smokers. Education about smoking risks is therefore imperative. Patients should be instructed that smoking cessation does lead to the resolution of symptoms and early-stage disease.

**PROGNOSIS**

The prognosis is poor for patients who are frequently symptomatic due to COPD. The 5-year mortality rate is approximately 50%, with most patients dying from respiratory failure. Two-thirds of patients who survive one bout of respiratory failure will die within 2 years.106

**ORAL HEALTH CONSIDERATIONS**

Several epidemiologic studies have suggested an association between oral infections and COPD.104,105 Apart from the periodontal pathogens mentioned above, *Streptococcus viridans* has been shown to be the causative pathogen of exacerbation in 4% of individuals with COPD.107 One prospective study suggested that oral colonization with respiratory pathogens in patients residing in a chronic care facility was significantly associated with COPD.40 The relationship between oral pathogens and exacerbations of COPD clearly deserves serious consideration. It is essential that elderly individuals (particularly, institutionalized patients) receive adequate oral hygiene in order to minimize respiratory complications.

Drug interactions with theophylline may arise (see above), and a change of medications by the oral health care provider may be appropriate.

As mentioned above, increased oxygen tension may diminish respiratory function in patients with COPD. Extreme caution must be exercised when administering supplemental oxygen in emergencies.

**Cystic Fibrosis**

Cystic fibrosis (CF) is a genetic disorder characterized by hyperviscous secretions in the respiratory and gastrointestinal tracts. The sweat glands, hepatobiliary system, and reproductive organs are also affected. Thickened secretions affect the pancreas and intestinal tract, causing malabsorption and intestinal obstruction. In the lungs, viscid mucus causes airway obstruction, infection, and bronchiectasis. Pulmonary complications are the major factors affecting life expectancy in patients with CF. This section focuses on the pulmonary manifestations of CF.

Cystic fibrosis is an autosomal recessive inherited disease. The responsible gene, which codes for the cystic fibrosis transmembrane conductance regulator (CFTR), is located on chromosome 7. The incidence of CF among white people is approximately 1 in 2,000 to 3,000 births; the incidence is lower among those of other races.108

**PATHOPHYSIOLOGY**

The primary defect in the CFTR gene results in a defective chloride transport system in exocrine glands. As a result, mucous production occurs without sufficient water transport into the lumen. The resultant mucus is dry, thick, and tenacious and leads to inspissation in the affected glands and organs. In the airways, the viscous secretions impair mucociliary clearance and promote airway obstruction and bacterial colonization. Bacterial superinfection is common and can lead to respiratory compromise.

**CLINICAL AND LABORATORY FINDINGS**

Patients with CF may present in infancy with extrapulmonary manifestations such as meconium ileus or failure to thrive. Pulmonary manifestations include coughing, recurrent infections of the lower respiratory tract, and bronchospasm. Tachypnea and crackles can be found on physical examination. As the disease progresses, digital clubbing and bronchiectasis may become apparent.

Spirometry is a useful tool for documenting and monitoring airflow limitation. Airway obstruction tends to worsen with disease progression although some patients with CF have mild pulmonary disease.

A sweat test can be performed to confirm the diagnosis. The procedure involves the collection of sweat after stimulation with pilocarpine. Samples containing > 60 mEq/L chloride are considered positive. Patients with indeterminate values (40 to 60 mEq/L) can be further assessed by using tissue genotyping.

**CLASSIFICATION**

There is no universally accepted classification system for CF.

**DIAGNOSIS**

The diagnosis of CF is based on the presence of pulmonary or extrapulmonary symptoms, as described above. A sweat chloride test result of > 60 mEq/L confirms the diagnosis.

**MANAGEMENT**

Conventional treatment of CF has included antibiotics, bronchodilators, anti-inflammatory agents, chest physiotherapy with postural drainage, and mucolytic agents. In addition to oral and parenteral antibiotics, inhaled antibiotics are used to help minimize systemic effects.109 The use of anti-inflammatory agents is controversial but may help minimize airway inflammation. Recombinant deoxyri-
Pulmonary Embolism

Pulmonary embolism (PE) is defined as a blockage of a pulmonary arterial vessel due to a thromboembolic event. The embolus may originate anywhere, but it is usually due to a thrombosis in the lower extremities. Risk factors for PE include prolonged immobilization (such as in a postoperative state), lower-extremity trauma, a history of deep-vein thromboses, and the use of estrogen-containing oral contraceptives (especially in association with tobacco smoking).113

PATHOPHYSIOLOGY

PE causes occlusion of pulmonary arterial vessels, which results in a ventilation-perfusion mismatch. Massive PE causes right-sided heart failure and is rapidly progressive. Local bronchoconstriction may occur due to factors released by platelets and mast cells at the sites of occlusion. Pulmonary hypertension due to vessel occlusion and arterial vasospasm is a common finding.

CLINICAL AND LABORATORY FINDINGS

Patients usually present with dyspnea. Other features that are variably present include chest pain, fever, diaphoresis, cough, hemoptysis, and syncope. Physical findings can include evidence of a lower-extremity deep-vein thrombosis, tachypnea, crackles or rub on lung auscultation, and heart murmur.

Measurements of arterial blood gases are helpful as patients may demonstrate a decrease in PaO₂ and partial pressure of arterial carbon dioxide (PaCO₂), with an increase in hydrogen ion concentration (pH). However, normal arterial blood gases do not rule out the possibility of PE. Chest radiography is often unhelpful but may reveal suggestive signs such as elevated diaphragms, pleural effusions, and pulmonary artery dilatation.

Ventilation-perfusion (V-Q) scanning is a noninvasive study that can exclude the diagnosis of PE; however, it is inadequate for establishing the diagnosis of PE. V-Q scanning uses radioactive tracers to measure ventilation and perfusion in different parts of the lung. An area that shows no perfusion but normal ventilation is suggestive of PE. Pulmonary arteriography is the "gold standard" study and is usually performed when the results of V-Q scanning are inconclusive.114

CLASSIFICATION

Four separate PE syndromes have been described: (1) massive PE, (2) PE with pulmonary infarction, (3) PE without infarction or cor pulmonale, and (4) organized emboli in central arteries. There is significant overlap among these syndromes.115

DIAGNOSIS

The diagnosis is made on the basis of history and physical findings. V-Q scanning should be performed in suspected cases, with pulmonary arteriography reserved for suspected cases with inconclusive V-Q scanning results.

MANAGEMENT

Fibrinolytic agents, such as streptokinase and urokinase, are effective for rapid lysis of emboli. Newer fibrinolytic agents include tissue-type plasminogen activator, single-chain urokinase-like plasminogen activator, and anistreplase. A heparin infusion is started, followed by long-term management with warfarin. Surgical embolectomy can be performed in unstable patients for whom anticoagulants are contraindicated. Patients with recurrent disease may be candidates for vena caval interruption by placement of a Greenfield vena caval filter.

PROGNOSIS

Although many patients with PE die before medical attention is received, the rate of mortality due to PE once adequate anticoagulation therapy is initiated is less than 5%.

ORAL HEALTH CONSIDERATIONS

The main concern in the provision of dental care for individuals with PE is the patient who is being managed with oral anticoagulants. As a general rule, dental care (including simple extractions) can safely be provided for patients with prothrombin times of up to 20 seconds or an international normalized ratio of 2.5. However, it is recommended that any dental care for these patients be coordinated with their primary medical care provider.

Pulmonary Neoplasm

Lung cancer is the leading cause of cancer deaths in both men and women. More than 100,000 people in the United States die each year due to lung cancer. Men and women who are over the age of 45 years and who have a long history of tobacco smoking are at highest risk.116

Squamous cell carcinomas account for one-third of all lung cancers. The neoplasm derives from bronchial epithelial cells that have undergone squamous metaplasia. This is a slow-growing neoplasm that invades the bronchi and leads to airway obstruction.

Small cell carcinomas account for approximately one-fourth of all lung cancers. These derive from neuroendocrine cells in the airways and metastasize rapidly. Most small cell tumors have metastasized prior to diagnosis.
Adenocarcinomas account for approximately one-third of all lung cancers. These neoplasms are of glandular origin and develop in a peripheral distribution. They grow more rapidly than squamous cell carcinomas and tend to invade the pleura. The bronchoalveolar tumor (a type of adenocarcinoma) is derived from bronchiolar or alveolar epithelium. This cancer is not associated with exposure to tobacco smoke.117

Large cell carcinomas, which account for most of the remaining lung cancers, include anaplastic and giant cell tumors. They are poorly differentiated tumors that resemble neither squamous cell carcinomas nor adenocarcinomas.

**PATHOPHYSIOLOGY**

Metaplasia of the respiratory epithelium occurs in response to injury, such as that induced by tobacco smoking. With continued injury, the cells become dysplastic, with the loss of differentiating features. Neoplastic change first occurs locally; invasive carcinoma usually follows shortly thereafter.118

**CLINICAL AND LABORATORY FINDINGS**

A chronic nonproductive cough is the most common symptom. Sputum production may occur, usually associated with obstructive lesions. Hemoptysis is present in up to 30% of patients.119 Dyspnea is variably present. Facial edema, cyanosis, and orthopnea indicate the possibility of superior vena cava syndrome, caused by compression of the superior vena cava by tumor. The acute onset of hoarseness may signal tumor compression of the recurrent laryngeal nerve. Shoulder and forearm pain might suggest the presence of Pancoast’s tumor, which is found in the apical region of the lungs below the pleura.

Metastatic and paraneoplastic effects are also common. The symptoms of metastasis depend on the sites involved and on the size of the tumor. The bones, the brain, and the liver are common sites of metastasis. Paraneoplastic effects include endocrine abnormalities that are due to tumors that secrete hormones such as antidiuretic hormone, adrenocorticotropic hormone, and parathyroid hormone–related peptides.120

**CLASSIFICATION**

The World Health Organization has differentiated pulmonary neoplasms into 12 distinct histologic types. The major clinical distinction is between small cell types and non-small-cell types; each type has different therapeutic implications. The four major pathologic categories are squamous cell carcinoma, small cell carcinoma, adenocarcinoma, and large cell carcinoma.

**DIAGNOSIS**

Diagnosis is suggested by history and physical examination. Chest radiography should be performed in suspected patients. Radiographs of symptomatic patients are normal 90% of the time. Computerized tomography is useful for patients who show no visible lesions on plain-film radiography. Sputum cytology may establish the diagnosis, even in the absence of an abnormal chest radiograph. Bronchoscopy is often performed to obtain a tissue diagnosis and to help localize the tumor.

**MANAGEMENT**

Surgical excision is the treatment of choice. Nonresectable tumors are difficult to treat because most are only minimally responsive to chemotherapy; the exception is small cell carcinoma, which may respond dramatically, especially if the disease is limited to one hemithorax. Radiation therapy is an important palliative measure, especially for patients with superior vena cava syndrome, brain metastases, or bone lesions.

**PROGNOSIS**

The 5-year survival rate for all patients with lung cancer is 8%. However, the 5-year survival rate for patients treated by resection is greater than 50%. For patients with small cell carcinoma confined to one hemithorax, a 1-year survival rate of 70% can be achieved with chemotherapy.

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