The respiratory system includes the nasal and oral cavities: the sinuses and larynx as the upper airway, and the trachea, bronchi, bronchioles, and alveoli as the lower airway. Many of the diseases that occur in the oral cavity are also found in the upper airway regions. Inflammatory lesions, both infectious and allergic, are found in the nose, sinuses, nasopharynx, oropharynx, and larynx. Tumors of the upper air passages are similar to oral neoplasms, with a few lesions being unique to the sinonasal region. The respiratory mucosal lining overlies mucinous glands, and these glands can give rise to salivary type tumors. Since these tissues are in close proximity to the oral cavity, the dentist should be familiar with the basic disease processes that are diagnosed and treated by the otolaryngologist.

Diseases of the lower tract are typically referred to as pulmonary diseases. Acute and chronic pulmonary infections are of concern to the dental team since some of these infections are transmissible in the dental office. Pulmonary infections can compromise respiratory function and pose a risk during inhalation anesthesia. Allergic reactions of the airway, particularly anaphylactic shock, can be induced by a variety of drugs and may constitute a medical emergency. Asthma is also an immunopathologic condition that is characterized by airway constriction and can become an emergency when a severe attack develops. A group of chronic pulmonary diseases progressively lead to a loss of pulmonary function, a condition known as chronic obstructive pulmonary disease (COPD). Patients with COPD have difficulty breathing, particularly when reclined, and they also pose a risk during inhalation anesthesia. Chronic obstructive pulmonary disease is one of the major causes of death each year in the United States.

Pathophysiology

The upper air passages are lined by either stratified squamous or respiratory pseudostratified columnar epithelia. Respiratory epithelium is found throughout the sinonasal tract, whereas stratified squamous epithelium is found throughout the oral cavity, in certain regions of the oropharynx, and covering the vocal apparatus in the larynx. In all regions, minor mucous-secreting glands are found, and these glands transmit their secretions through ducts that empty onto the surface of the airway epithelium to form the moist mucosal surface.

Anatomically, the paranasal sinuses are located around the nasal cavity, and all of these sinuses have exit sites, or ostia, that allow mucous secretions to drain into the nasal cavity. The sinuses, being hollow cavities within the skull, reduce the bony mass and weight of the head while serving to moderate the temperature of inspired air. The connective tissues of the lateral nasal walls are traversed by a rich supply of blood vessels. Inflammatory diseases of the sinonasal region are common and are usually of an allergic nature, although bacterial and viral mucosal infections are also common in these regions.

Most of the sinonasal allergies are IgE-mediated immediate hypersensitivity reactions. Inspired allergens, such as pollens, dander, and various other particles, can stimulate a specific IgE response (Figure 5–1). These IgE antibodies (reagins) are bound to mast cell membranes by a specific receptor that binds to a ligand on the Fc region. When allergen passes into the mucous membrane and diffuses into the submucosa, binding to the IgE-mast cell complex causes bridging of two contiguous immunoglobulins, a conformational event that triggers internal signaling pathways that initiate degranulation and release of...
Neoplastic processes of the nose and sinuses are of both the benign and malignant varieties. There are three histologic variants of papillomas that occur in the nose, usually arising on the lateral wall and also extending into the antrum. Fungiform papillomas are innocuous, whereas inverting type papillomas are aggressive, causing osseous destruction. Inverting papillomas are associated with human papillomavirus (HPV), usually types 6, 11, and 16, although Epstein-Barr virus DNA has also been identified in the papilloma tissues. It is more probable that the HPVs are causative. About 5% of inverting papillomas undergo carcinomatous transformation. The cylindric cell papilloma is histologically unique and is rare.

Histamine induces vasodilation and increased capillary permeability with leakage of proteins from serum, including kinins and other mediators that cause pruritus and sneezing. Protein leaks increase tissue osmotic pressure, culminating in edema and swelling of the mucous membranes. Prolonged exposure to allergens over many years may stimulate proliferation of the soft tissues, the result of which is the formation of nasal and antral polyps. Mucosal edema that develops in either infectious or allergic inflammations can lead to swelling and occlusion of the sinus ostia, with resulting pain and fluid retention. These changes are readily visualized on computed tomography (CT) scans and magnetic resonance imaging (MRI) of the sinus regions.

Figure 5-1 IgE-mediated reactions in allergic pulmonary inflammatory diseases. Allergens react with reagin (IgE) antibodies that are bound to receptors on mast cells. Histamine and leukotrienes are released and exert pharmacologic effects on endothelial cells, mucus-secreting epithelial cells, and smooth muscle. In anaphylactic shock, acute and severe vasodilation with bronchospasms occur following systemic introduction of allergen. In asthma, hypotensive reaction is lacking, since the allergen is introduced via the airway and only affects bronchiolar smooth muscle.
Squamous cell carcinoma is the most common cancer to arise in the sinuses. Adenocarcinomas are seen in the nasal cavity; hardwood saw dust is considered a risk factor for these adenocarcinomas, many of which microscopically have the appearance of intestinal epithelium. As stated previously, the presence of numerous salivary type glands accounts for the occurrence of salivary gland tumors in the airway. Certain neoplasms are unique to the sinonasal region. The olfactory neuroblastoma derives from neuroblasts in the olfactory bulb. Craniopharyngiomas arise in the pituitary gland and may invade the upper nasopharynx. A unique vascular tumor that arises in young boys is located in the posterior nasal cavity and apparently arises from the vascular tissues that are so plentiful in the lateral nasal wall. Carcinomas that are poorly differentiated or undifferentiated are also found in this region. The sinonasal undifferentiated carcinoma is a neoplasm with a poor prognosis that is encountered in the adult. Nonkeratinizing squamous cell carcinoma is a nasopharyngeal malignancy of teenage males that is often first detected as a neck metastasis.

The larynx is also a site of inflammatory disease. Both allergies and infections can cause laryngitis. Severe infections with marked edema can compromise the airway and even lead to death. Laryngospasm or bronchospasm secondary to anaphylactic shock can also be a fatal event. The same mechanisms described earlier are operational in anaphylactic shock. Allergen IgE responses with histamine release can be systemic or local with profound hypotension and loss of consciousness. The effect of histamine release on bronchiolar smooth muscle is constriction, with airway stricture. A similar reaction is encountered in asthma. Leukoplakia of the larynx is relatively common, and squamous cell carcinomas of the larynx account for more than 1200 malignancies each year in the United States.

Inflammatory diseases of the lungs include allergies, as well as bacterial, viral, and fungal infections. These infections involve the bronchioles and the alveolar air sacs (Figure 5–2). Pneumonia is a widespread infection of the lung parenchyma in which a lobe or an entire lung becomes infected with either bacteria or virus, resulting in purulent exudate accumulation in the alveolar air sacs (Figure 5–3). Gaseous exchange can become severely compromised, leading to death. When the infection is multifocal throughout the lungs, the condition is referred to as bronchopneumonia. When the infection localizes to an entire lobe, the term lobar pneumonia is used.

Foreign bodies may be aspirated into the airway from the oral cavity. Endodontic files and reamers coated with pathogenic microorganisms can cause lung abscess. There are instances where crown castings and even partial dentures have been aspirated. Because the right main stem bronchus courses vertically, whereas the left bronchus is angled to the left, most foreign bodies lodge in the right lung. Many can be visualized radiographically; the majority can be retrieved during bronchoscopy.

There are a group of microorganisms that cause chronic granulomatous infections of the lung; tuberculosis is the most common. Certain fungi, including histoplasmosis, blastomycosis, and coccidioidomycosis cause chronic lung infections with granuloma formation. As these granulomas enlarge over time, they may erode vessels, causing hemoptysis, coalesce, and compromise pulmonary function. Organisms are subsequently seeded into other alveolar air sacs, thus disseminating the infection throughout both lungs. The granulomas are readily visualized radiographically if sufficiently large.

Tuberculosis (TB) is contracted by aerosol spread in close contact with an infected subject. The primary infection occurs at the periphery of the lung tissue, the Ghon focus, and via lymphatics, a hilar lymph node becomes infected and subsequently enlarged with granulomas, the Ghon complex (see Figure 5–3). This primary infection usually becomes quiescent. Reinfection or reactivation of the tubercle bacillus results in secondary TB. The secondary infection occurs in the face of an intact T-cell response and a positive tuberculin
When the terminal alveoli along the pleural margin are dilated as blister-like sacules, the condition is referred to as bullous emphysema. In bronchitis, irritants cause mucinous secretions to accumulate in the bronchioles, with resulting chronic productive cough. Long-standing COPD shows classic clinical features, and affected patients are at risk for serious pulmonary infections and cor pulmonale (pulmonary artery hypertension).

Bronchiectasis is a lesion that occurs in the bronchioles after repeated bouts of influenza or other pulmonary infections. The bronchiolar walls become thin and aneurysmal. These focal dilatations accumulate mucins, leading to chronic productive cough and COPD. Bronchiectasis is particularly problematic in cystic fibrosis. This childhood illness is an autosomal recessive disease that results from a mutation on chromosome 7, involving a gene that encodes a transmembrane chloride channel. In sweat ducts, excessive chloride is secreted, whereas in lung tissues chloride does not pass
from the epithelial lining cells into the lumen, and sodium is retained as well. The result is a dry airway with compensatory hyperplasia of the epithelial lining. This airway loses its mucinous wet layer, and ciliary action is impaired, leading to repeated pulmonary infections that progress to bronchiectasis.

Asthma is also considered a form of COPD. Unlike bronchitis, emphysema, and bronchiectasis, asthma begins at a young age and is an allergic disorder with a psychosomatic element and a genetic predisposition. The condition is equivalent to anaphylactic shock in that allergen binding in conjunction with emotional stress leads to airway constriction. Severe attacks are known as status asthmaticus, an emergency situation in which the airway shuts down.

Restrictive pulmonary disease is a condition that evolves as a consequence of environmental exposure to toxic chemicals or to certain infections. Common to all of these diseases is interstitial fibrosis. The tissues between the alveoli and bronchioles are loose, areolar, and fibrovascular. In the restrictive lung diseases, this interstitial tissue becomes progressively scarified, and as it does, the alveolar air spaces become compressed. In pneumoconiosis, a group of fibrosing lesions evolve from prolonged exposure to inspired chemicals, such as coal dust (anthracosis), beryllium, asbestos, and silica. Certain infections with viruses and bacteria lead to interstitial pneumonitis, whereby the interstitial spaces are edematous and inflamed and heal by fibrosis.

Cancer of the lungs can arise from the bronchiolar epithelium, mucous glands, or the pleural cells. The most common form of lung cancer is bronchogenic carcinoma; 80% of cases are related to smoking tobacco. The bronchiolar epithelium undergoes squamous metaplasia and dysplasia and, ultimately, transforms into squamous cell carcinoma. Interestingly, in laboratory animals, forced smoking does not cause lung tumors. Nevertheless, a variety of potential carcinogens exist in tobacco smoke. Prolonged heavy exposure to asbestos also increases the risk for bronchogenic carcinoma, and when tobacco and asbestos exposure are combined, the relative risk for lung cancer is 90-fold. Adenocarcinomas are rare, as is mesothelioma, a cancer of the lung pleura that is typically encountered in asbestos mine workers.

The diseases that compromise pulmonary function, particularly COPD and restrictive lung disease, affect respiratory physiology. Pulmonary function tests can be used to assess various parameters that are diagnostically useful. The spirometer is an instrument that measures various inspiratory and expiratory volumes. In a normal individual without lung disease, the total lung capacity is over 5500 mL of air (Figure 5–5). Regular breathing involves a volume of about 500 mL of air with each inspiration and expiration, a measure referred to as the tidal volume. The amount of air that is able to be inspired maximally, in excess of inspired tidal volume level is called the inspiratory reserve volume. This represents the maximum amount of air that can possibly enter the lungs, about 3000 mL. After a normal exhalation during the tidal cycle, more air can be forcefully exhaled; this is the excretory reserve volume. Even after this maximal exhalation there is still residual air in the alveoli (the residual volume) that is not available for respiratory function. In pulmonary diseases, in which alveolar surface area is decreased, or in lesions that restrict gaseous exchange, perturbations in these volumes can be identified when testing with the spirometer (Figure 5–6).
In expiratory reserve volume, and a delay in expiration after maximal inhalation, owing to air trapped in dilated alveoli. Chronic obstructive pulmonary disease (COPD) = chronic obstructive pulmonary disease. Easement attributable to pneumoconiosis, the inspiratory reserve volume and vital capacity are decreased because of loss of airway space. COPD = chronic obstructive pulmonary disease.

Clinical features

Upper airway diseases present with sinonasal symptoms. The inflammatory diseases typically manifest nasal stuffiness, sneezing, rhinorrhea, dysosmia, mucosal itchiness, nasal obstruction, or pain. The temperature should be taken in these situations; if elevated, infection is favored over allergy. Allergic rhinitis and sinusitis are typically seasonal. Some patients have had allergy testing and, therefore, are aware of the environmental allergens that trigger an immediate hypersensitivity response. Physical examination discloses a nasal discharge along with erythema of the mucous membranes. Long-standing allergic disease may result in polyp formation. Nasal polyps are a common cause of loss of smell. With a nasal speculum, the polyps appear as pink fleshy masses high in the nasal cavity, usually above the middle turbinate.

When the ostia become obstructed, sinus pain may develop and can mimic toothache. The sinuses may be tender to palpation over the malar eminence and the entire maxillary quadrant may manifest a chronic dull aching pressure sensation. All teeth in the quadrant are often percussion sensitive. The pain and pressure are exacerbated if the patient places his or her head below the knees while seated. Transillumination of the sinuses in a dark room shows clouding. Radiography may disclose soft-tissue thickening of the sinus membranes or an air-fluid level may be seen on an anterior posterior skull radiogram, Waters’ sinus projection, or computed tomography (CT) scan. When the patient is febrile, an infectious process is probable and may be of viral or bacterial origin. Acute pain symptoms in the sinuses are most indicative of bacterial sinusitis, and culture of the nasal discharge is in order.

Sinonasal symptoms indicative of neoplasias include persistant nasal obstruction, nasal speech, dysosmia, and swelling in the lateral nasal wall or the palate. Infraorbital paresthesia or hyposthesia are suspicious signs for malignancy within the maxillary sinus, and epistaxis may occur with sinonasal malignancies. Imaging studies discloses soft-tissue, space-occupying lesions, bony wall expansion and osseous perforation or destruction (Figure 5–7). Biopsy is then required to derive a definitive diagnosis.

Nasopharyngeal carcinoma is characterized by pain, stuffiness, unilateral hearing loss, epistaxis, and cervical lymph node enlargement. It is not uncommon for an enlarged indurated node to be the first sign of nasopharyngeal carcinoma. Benign vascular tumors (juvenile nasopharyngeal angiofibroma) also cause nasal stuffiness and obstruction.

Pharyngeal symptoms most commonly center around irritation and pain. Laryngitis is characterized by hoarseness, throat pain, and dysphonia. Examination of the larynx shows edematous swelling of the both true and false vocal cords along with erythema. Laryngitis may be caused by vocal cord trauma (eg, yelling, loud forced singing), allergy, or infection with either virus or bacteria. Neoplastic disease of the vocal structures is similar to that seen in the oral cavity. Laryngeal papillomatosis is encountered primarily in children and teenagers. On laryngoscopy polypoid masses are seen on the true and false cords. These HPV-induced lesions are difficult to eradicate; recurrence after excision or laser ablation may occur.
Carcinoma of the laryngeal mucosa is typically associated with smoking. These tumors are squamous cell carcinomas. The signs are hoarseness and dysphonia; symptoms include a scratchy feeling and pain. Occasionally, blood-tinged saliva is found from tumor bleeding. Clinically, early lesions are white, red, or mixed red and white lesions with focal ulcerations. Tumefaction is seen, and when the tumor invades the adjacent cartilages, auscultation discloses a loss of normal crepitus, since the cartilagenous structures become invaded and fixed. Invasion of contiguous structures may also cause true cord paralysis.

The primary clinical manifestations of pulmonary disease include dyspnea, cough (productive or nonproductive), hemoptysis, and respiratory distress. The accumulation of viscous mucus within bronchioles and bronchi, as well as constriction of the airway because of smooth muscle contraction act as impediments to air flow. These obstructive changes may be detectable with the stethoscope placed over the lung fields on the patient’s back. A constricted airway causes wheezing on both inspiration and expiration. Mucus accumulation produces crackling and gurgling sounds termed rales and rhonchi. Orthopnea, shortness of breath while supine, is another sign of pulmonary disease. The signs and symptoms may be associated with a variety of pulmonary diseases, and alone are not diagnostic of any one disorder.

The most common infectious diseases to affect the lungs are the common cold and respiratory flu, both of which are viral in origin. These common infections are characterized by productive cough, fever, and malaise, with a 7- to 10-day course. Serious infections of the lungs are the pneumonias. Pneumonia is a widely disseminated lung disease that may be caused by either viruses or bacteria. The more common organisms to cause pneumonia are the bacteria *Streptococcus pneumoniae*, *staphylococci*, *Haemophilus influenzae*, *Pseudomonas aeruginosa*, and coliform rods. When the infection is widely disseminated within the parenchyma as multiple foci, the disease is termed bronchopneumonia. When the infection diffusely involves an entire lobe it is referred to as lobar pneumonia. In response to infection, the air passages secrete excessive mucins and the fibrovascular septae show vessel engorgement and congestion with exudative fluid accumulation in the airway. This leads to dyspnea along with deep cough that is productive in the beginning, yet as the infection consolidates, a dry cough follows. The patient is febrile. Percussion and auscultation of the chest disclose evidence of fluid accumulation, and radiographs show patchy opacification in bronchopneumonia and diffuse opacification in lobar pneumonia. Patients with pneumonia require hospitalization and selection of appropriate antibiotics when bacteria are causative. In debilitated patients, death is a common outcome. Pneumonia and flu are leading causes of death in the United States. Disseminated malignant neoplasms and immunodeficiencies of various types are often complicated by pneumonia.

Infections of the lung parenchyma may extend to the pleural lining where inflammatory foci develop and fibrin may be deposited, a lesion known as pleuritis. As the lungs expand and contract, these inflammatory foci may scrape across adjacent regions of the parietal pleura lining the internal chest wall. These movements can be painful and upon chest auscultation the scraping sounds often are detectable as a friction rub.

Influenza virus, respiratory syncytial virus, and adenovirus among others cause interstitial pneumonia. *Mycoplasma pneumoniae*, a bacterium, also causes infection, with inflammatory lesions involving the interstitial fibrovascular tissues that constitute the lung septae. Patients complain of headache, extremity muscle pain, fever, and cough. Elevated serum cold agglutinin titers are encountered in *M. pneumoniae* infection, yet are absent in most other viral forms of interstitial pneumonia. Most of these infections run their course without complication; however, fatal outbreaks have been encountered.

Pulmonary infections are commonly seen in cystic fibrosis along with other exocrine lesions, such as bowel obstruction, malabsorption, and xerostomia. Only homozygotes are affected. Children develop bronchiectasis with dyspnea and chronic cough due to a lack of airway wetting. The mucins are thick and ropey (muco-
viscidosis) and obstruct the airway. These problems lead to pneumonia or cor pulmonale and eventuate in death for many of the affected children.

Chronic granulomatous infections of the lungs, TB being the most prevalent, begin with coughing in the absence of fever, or there may be a low-grade elevation in temperature. Weight loss and fatigue are frequently associated. As more and more lung parenchyma becomes involved and as granulomas enlarge, they may cavitate in the zones of caseous necrosis. A productive cough ensues and hemoptysis is commonly encountered. These individuals are infectious and can transmit TB organisms. Sputum specimens can be cultured or smeared to identify tubercle bacillus organisms. Recall that deep invasive fungi, such as histoplasmosis, blastomycosis, and coccidioidomycosis, can cause similar granulomatous infections. Chest auscultation and percussion are abnormal in late disease. Radiographs disclose the presence of granulomas and hilar lymph node enlargement. Long-term antibiotic treatment is required for active tuberculosis, and most patients are hospitalized until the infection is controlled. In contrast to the global epidemic, TB in the United States is under control. The main TB problem in industrialized countries resides in poverty areas of large cities, in immunocompromised patients, and with antibiotic-resistant mycobacteria.

Dyspnea is a common sign in congestive heart failure (see Chapter 3). Recall that left-sided failure results in passive congestion of the lungs with accumulation of edema fluid and inflammation in the alveolar spaces. Physical examination, cardiac auscultation, echocardiography, and radiographic studies identify the nature of the cardiopathy.

Patients suffering from the various forms of COPD manifest dyspnea as the chief complaint. In emphysema and bronchitis, the signs and symptoms begin in midlife and become severe in the elderly. Cigarette smoking is the primary causative factor for both of these conditions. Bronchitis is characterized by chronic productive cough as tobacco irritants stimulate inflammation and mucus secretion in the bronchi and bronchioles (bronchiolitis). Emphysema is characterized by dyspnea. As the terminal airway loses septal surface area for gaseous exchange, expirations must be forced. Affected patients are slender and barrel chested. In most cases, bronchitis, bronchiolitis, and emphysema occur together. Spirometer readings disclose normal tidal volume and inspiration reserve volume with a significantly decreased expiratory reserve and an increased residual volume (see Figure 5–6). After forced inhalation, there is a delay in the duration of exhalation as air is forced by contraction of the intercostal muscles. Chronic obstructive pulmonary disease in this setting is irreversible. Cessation of smoking is essential to prevent the complications and death that may ultimately occur.

Two common appellations applied to COPD patients define their clinical appearance. The euphoniously so-called Red Puffer suffers from emphysema. The facial skin is flushed from over-inspiration and oxygenation, and the lips are puckered as the affected person forces air from the lungs on expiration. These patients have normal blood gas values. The second descriptive term is applied to the patient with chronic severe bronchitis: the Blue Bloater is cyanotic with facial palor and a bluish cast to the skin as a result of poor oxygenation with hypercapnia. The neck is full from being distended as a consequence of constant coughing.

Asthma is a complex disease that is divided into two major subtypes: extrinsic and intrinsic (Table 5–1). In both forms, the condition usually evolves in early childhood. Extrinsic asthma secondary to airway-introduced allergens is frequently worsened during episodes of stress and anxiety. As the airway becomes constricted and accumulates mucous plugs, the chief clinical sign is wheezing and coughing, without fever. When the asthmatic attack is severe and prolonged over many days, bronchospasm may occur and can be fatal, a condition referred to as status asthmaticus. Asthmatics are treated with orally administered bronchodilator drugs, corticosteroids, and inhalation bronchodilators. In some forms of childhood allergic asthma, the condition improves with ensuing age. Occupational asthma has similar signs and symptoms and represents an idiosyncratic reaction among select individuals upon exposure to certain chemicals found in the workplace. Epoxy resins, formaldehyde, toluene, cotton fibers, and wood dust are common precipitating elements that can initiate an IgG or IgE response. Intrinsic asthma is caused by hyper-reactivity of the airway to infectious agents, aspirin, or vigorous physical activity.

The restrictive lung diseases are uncommon. As the interstitium becomes scarified, the total lung volume is decreased. The spirometer patterns show decreased inspiratory reserve and vital capacity volumes (see Figure

<table>
<thead>
<tr>
<th>Extrinsic</th>
<th>Intrinsic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allergen-induced (IgE response)</td>
<td>Nonreaginic respiratory infections</td>
</tr>
<tr>
<td>Occupational chemical exposure (IgE and IgG response)</td>
<td>Sports asthma (high physical activity)</td>
</tr>
<tr>
<td>Aspergillosis allergy</td>
<td>Aspirin sensitivity</td>
</tr>
</tbody>
</table>
5–6). Patients may also have a chronic cough secondary to bronchiolitis from toxic irritants. Dyspnea is also a major sign. Pneumoconiosis is a group of lung diseases caused by inhalation of irritant chemicals. Anthracosis (coal miner’s black lung disease) results in severe interstitial fibrosis with marked restriction in airway volume. Asbestosis is usually only seen in asbestos miners who have been breathing asbestos dust for many years. As mentioned earlier, these individuals are at increased risk for development of bronchogenic carcinoma and mesothelioma. Other forms of restrictive lung disease include the following: sarcoidosis, a granulomatous inflammatory disease of unknown etiology (see Chapter 24); hypersensitivity to allergens that find their way into the terminal bronchioles; hemorrhagic lung syndromes such as Goodpasture syndrome (a condition that also affects the kidneys; Wegener granulomatosis (see Chapter 24); and lupus erythematosus (see Chapter 21).

Bronchogenic carcinoma is a leading cancer among males and is increasing in females. Morbidity and mortality are high. Most cases are associated with smoking cigarettes. Chronic cough is a sign of pulmonary irritation from inhaled smoke, with COPD developing after many years of use. When these signs are accompanied by hemoptysis, carcinoma should be suspected, and chest films must be secured. If a cough is productive, sputum cytology may show malignant cells. In fact, lung cancer can be a silent killer and is often discovered after routine chest radiographs disclose a mass. In some cases after metastasis has occurred at another site, a biopsy indicates malignant cells primary in the lung. Small-cell cancer of the lung is not smoking-related. Some of these small “oat” cell carcinomas secrete hormones, and the first manifestation of disease is clinical endocrinopathy, with hyperparathyroidism being common, owing to secretion of a parathormone-like peptide by the malignant cells (see Chapter 10).

Oral manifestations

Patients with sinonasal diseases often show oral signs and symptoms. Halitosis can be a common sign of chronic allergic sinusitis and rhinitis whereby postnasal secretions fall upon the base of the tongue and may cause malodor (see Chapter 27 on special senses). Cases of antral cancer are seen in which the tumor erodes the palatal bone and presents as an ulceration or fistula (see Figure 5–7; Figure 5–8). Invasive fungal infections of the sinonasal region can also erode into the palate as can nasal midline necrotizing diseases such as Wegener granulomatosis (Figure 5–9) and malignant reticulosis, a form of angiocentric T-cell lymphoma (Figure 5–10). Infraorbital paresthesia is a sign of antral carcinoma, and when present, appropriate imaging studies must be ordered. Cranial base malignancies may cause neurologic deficits of the cranial nerves including paresthesias and motor deficits of cranial nerves III, IV, V, and VI.

Oral signs are rare in pulmonary diseases. Patients with lung cancer often develop metastases to bone, and the mandible may become a focus of distant spread. Typically, the patient complains of an insidious onset of paresthesia of the lower lip on the affected side as tumor cells invade the inferior alveolar nerve. A dental radiograph discloses a poorly margined radiolucency, which usually is confined to the posterior body and ramus of the mandible yet may also occur as a soft-tissue mass (Figure 5–11). Biopsy discloses the presence of metastatic malignant cells with morphology consistent with lung carcinoma. As mentioned previously, uncovering a metastatic focus may be the first indication that the patient has a primary cancer in the lung; but it also indicates advanced disease with essentially no chance for cure.
Tuberculosis and invasive fungi may also spread to the oral cavity from the primary infection in the lung. The organisms gain access to the oral regions by hematogenous spread, and when they adhere to oral tissues, a site of granulomatous inflammation develops. These granulomas may be red and granular or ulcerated with rolled margins, thus resembling a primary carcinoma (Figure 5–12). Tuberculous lymphadenitis occurs in individuals who have ingested nonpasteurized milk contaminated with mycobacteria. The nodes are firm to palpation, movable, and often bilateral (Figure 5–13). Tuberculosis and histoplasmosis are the more common granulomatous infections seen in the oral mucosa. Biopsy is required to make the diagnosis, and the pathologist usually needs to apply special stains to identify the microorganisms. Acid-fast Ziehl-Neelsen staining is used to identify mycobacteria; periodic acid-Schiff (PAS) or gram methenamine silver identifies histoplasma and other fungal organisms, all of which have distinct morphologic features. Histoplasmosis in the oral cavity can be an opportunistic infection among human immunodeficiency virus (HIV)-infected subjects and may or may not be associated with pulmonary disease (see Chapter 14).

In cystic fibrosis, all secretions of exocrine origin are affected, including those of the major and minor salivary glands. Affected children complain of xerostomia, and may require artificial saliva preparations to keep the mucosa moist and comfortable.

**Dental management**

Patients with pulmonary diseases usually do not pose a significant problem for dental care unless general anesthesia is to be performed. It must be realized that the common respiratory infections are transmissible in the dental operatory, necessitating adherence to infection control procedures (see Chapter 19). Currently, in most dental practices, patients are placed in a supine position, and in the patient with a compromised airway, orthop-
unaware if they were exposed once before and were never given another prescription after the initial dosing. Food allergens can also induce anaphylactic reactions. At the onset of an anaphylactic event, the patient notices difficulty breathing, and wheezing may be detected. Severe respiratory distress ensues rapidly as histamine and leukotrienes are released in the airway mucosa and submucosa. The blood pressure also drops, and the patient may lose consciousness, owing to hypoperfusion of cerebral vessels. Death may ensue if this medical emergency is not treated. A call to 911 should be undertaken, oxygen should be administered and 1:1000 epinephrine should be injected sublingually, because vascular collapse makes intravenous injection difficult if not impossible. To be effective, diphenhydramine as an intravenous emergency drug can be administered only in the early stages, prior to airway closure and hypotensive shock. Because this antihistamine is a histamine-receptor blocker, its use in late-stage anaphylaxis is of no benefit, since the receptors are already occupied by the IgE-mediated release of histamine; alternatively, epinephrine exerts a direct dilating effect on bronchiolar smooth muscle, opening up the airway within seconds to minutes.

Patients with asthma are at increased risk for an asthmatic attack while undergoing any dental care that may elevate stress levels. Pain and anxiety are common precipitating factors for both allergic and nonallergic asthma may become a significant problem. When patients have a full stomach and are placed in supine position for prolonged periods, there is always the risk for regurgitation with subsequent aspiration of acidic contents into the lungs. Aspiration of dental instruments, materials, tooth fragments, and prostheses that may be contaminated with oral microorganisms may initiate a lung abscess. The use of the rubber dam helps to eliminate the chance for introduction of such foreign bodies.

Recognition of the patient’s pulmonary disease status with regard to severity of dyspnea and orthopnea is an important consideration when long periods of dental therapy are contemplated and the patient is placed in a reclined position. It is advisable to seat the patient upright or to stop all procedures on a regular basis and return them to an upright position periodically. Oxygen should be readily available and equipment should be in place in case assisted breathing is required. It should be noted, however, that forcing air into the lungs of a patient with emphysema, particularly the bullous variant, can burst the pleural lining resulting in pneumothorax, a condition that leads to respiratory arrest, owing to collapse of the lungs.

It is axiomatic that patients with upper respiratory infections with nasal congestion or blockage are unable to receive routine dental care when a rubber dam is to be used, because the oral airway will be blocked. If the nasal mucus can be expelled by blowing the nose, treatment can be implemented; if not, the patient should be rescheduled.

The most severe respiratory emergency is anaphylactic shock. Such an event in the dental office setting is extremely rare, yet can occur if the patient is introduced to a provoking antigen just prior to or during an office visit. Recall that a variety of pharmacologic agents can cause anaphylaxis, particularly penicillins. Most patients are aware of an allergy to penicillin, yet others may be unaware if they were exposed once before and were never given another prescription after the initial dosing. Food allergens can also induce anaphylactic reactions. At the onset of an anaphylactic event, the patient notices difficulty breathing, and wheezing may be detected. Severe respiratory distress ensues rapidly as histamine and leukotrienes are released in the airway mucosa and submucosa. The blood pressure also drops, and the patient may lose consciousness, owing to hypoperfusion of cerebral vessels. Death may ensue if this medical emergency is not treated. A call to 911 should be undertaken, oxygen should be administered and 1:1000 epinephrine should be injected sublingually, because vascular collapse makes intravenous injection difficult if not impossible. To be effective, diphenhydramine as an intravenous emergency drug can be administered only in the early stages, prior to airway closure and hypotensive shock. Because this antihistamine is a histamine-receptor blocker, its use in late-stage anaphylaxis is of no benefit, since the receptors are already occupied by the IgE-mediated release of histamine; alternatively, epinephrine exerts a direct dilating effect on bronchiolar smooth muscle, opening up the airway within seconds to minutes.

Patients with asthma are at increased risk for an asthmatic attack while undergoing any dental care that may elevate stress levels. Pain and anxiety are common precipitating factors for both allergic and nonallergic

Figure 5–12 Tuberculous ulcer in a patient with concomitant pulmonary tuberculosis.

Figure 5–13 Scrofuloderma. Enlarged cervical nodes are evident.
forms of asthma. Sedative drugs with anticholinergic effects are to be avoided. Narcotics and barbiturates are known to trigger an attack. Local anesthetics used in dentistry contain sodium metabisulfite, an antioxidant that prevents oxidation of epinephrine. Twenty percent of asthmatics are allergic to sulfur compounds, and therefore, administration of local anesthetics should be undertaken with caution, always questioning patients as to whether they have experienced any adverse reactions to local anesthetics. The severity and periodicity of asthmatic attacks is highly variable from one affected patient to another. Before engaging in extensive dental procedures, particularly surgical interventions, the history should be explored in detail. If attacks are mild and infrequent, treatment can be initiated with caution. It is always prudent to be certain that patients maintain their bronchodilator daily drug regimen prior to dental treatment and any inhaler devices should be placed within easy reach. Severe asthmatics may also be taking corticosteroids. In these individuals there may be an increased risk for infection. Patients with a history of severe and frequent bronchospasms should be hospitalized for extensive and invasive dental care. Consultation with the physician must be undertaken, and an anesthesiologist should be included on the treatment team.

In rare instances, patients develop asthma after administration of aspirin. Any patient who gives a history of asthma should be questioned about the role of aspirin in the causation of their disease, and obviously, aspirin-containing drugs should be eliminated as analgesic medications. Acetaminophen or propoxyphene should be considered as substitute analgesics for attenuation of dental pain. Oxycodone and hydrocodone should be administered with care since codeine-related drugs and opiates may aggravate asthmatic attacks. Some asthmatic patients have coincidental complaints of xerostomia. Sialagogues (parasympathomimetics) are contraindicated, because they cause airway congestion.

Children with cystic fibrosis have dry airways, and administration of inhalation sedation can be dangerous when the gases are not humidified. General anesthesia is also problematic because concurrent administration of anticholinergic drugs further aggravates airway dryness. If anesthesia is required for dental care, the patient should be hospitalized and managed by an anesthesiologist.

General anesthetics for adult patients with pulmonary disease must also be used with caution. Mild COPD or restrictive lung disease is generally not problematic. Moderate to severe pulmonary disease can be aggravated and degenerate to severe respiratory distress when inhalation anesthetics are used. This is particularly so in conjunction with intravenous drugs that depress the respiratory center of the central nervous system, and with anticholinergic drugs that may be administered during intubation. When general anesthesia is necessary for patients in this category, they should be hospitalized and managed by an anesthesiologist. In addition, the dentist should consult with the patient's physician prior to rendering treatment.

Suggested reading


