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Dental Management of Patients with Obstructive Pulmonary Diseases

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Dental health care providers must be aware that patients with compromised airway function have diminished overall health and ability to tolerate dental treatment. Delivery of routine dental care may need to be altered because of existing pulmonary disease or the medications that are prescribed to treat them.

Many diseases and conditions can cause obstruction of the human airway. In children, the most common cause is bronchial asthma. In adults, the differential diagnosis is much longer, and may include compromise of other systems of the body as well (Box 1) [1–4]. The consequences of airway obstruction and the subsequent hypoxic state can be severe. Chronic obstructive pulmonary disease (COPD), the fourth most common cause of death in the United States [5,6], prevents air exchange due to pathology of the respiratory passages. This article focuses on the three most common obstructive pulmonary diseases: chronic bronchitis, emphysema, and bronchial asthma.

Incidence and prevalence

Estimates of prevalence of the obstructive pulmonary diseases and other lung diseases are made annually by the American Lung Association (Table 1) [6–8]. Over the past 20 years the morbidity and mortality of COPD has steadily decreased as Americans have reduced their use of tobacco and received the benefits of more sophisticated pharmacotherapeutics for managing airway obstruction. The burden of asthma in the United States has increased. However, within the last few years, mortality and hospitalizations due to

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Box 1. Differential diagnosis of obstructive pulmonary disease in adults

- Chronic obstructive pulmonary disease (includes emphysema and chronic bronchitis)
- Bronchial asthma
- Congestive heart failure
- Sinusitis
- Acute bronchitis
- Bronchiectasis
- Pneumonia
- Neoplasm
- Laryngeal dysfunction
- Pulmonary infarction or embolism
- Mechanical airway obstruction
- Vocal cord dysfunction

asthma have decreased, and asthma prevalence has stabilized, possibly indicating a higher level of disease management [8–10].

Anatomy and pathophysiology

A review of respiration and pulmonary function makes for better understanding of obstructive pulmonary diseases. Loss of function of any part of the respiratory system can lead to hypoxia. The lower respiratory system is typically affected the most by these diseases. The lungs, bronchi, and trachea are specialized tissues and will each be described below. The obstructive pulmonary diseases are often multifaceted, affecting more than one aspect of

Table 1
Incidence, prevalence, and mortality of obstructive pulmonary diseases in the United States in 2003

Disease	Number affected	Prevalence (%)	Number of deaths
Chronic bronchitis	8.6 million	40.2 per 1000 (4.0)	120,555 (combined deaths for chronic bronchitis and emphysema)
Emphysema	3.1 million	14.6 per 1000 (1.5)	
Bronchial asthma	20 million (including 6.2 million children (<18 years))	Adults: 7.7 Children: 8.8	4,261[a]

[a] Data for 2002.
Data from Refs. [6–8].

the respiratory system. In many patients, the diseases coincide and symptoms overlap. Typically, the patient's symptoms change over time.

The lungs contain the pulmonary alveoli. These terminal air spaces, or recesses, are located at the end of the respiratory tree. The pulmonary alveoli are lined with specialized epithelium and are supported by a rich vascular supply to the endothelium. The exchange of oxygen and carbon dioxide occurs in these recesses by rapid diffusion, a passive process. This process of the exchange of oxygen and carbon dioxide between the atmosphere and the cells of the body is called respiration.

The epithelial cells lining the alveoli are called pneumocytes. Type-I cells allow the gas exchange, while the type-II cells produce surfactant to decrease alveolar surface tension, support other metabolic activities, and may also proliferate to replace aging or damaged type-I cells [11]. The pneumocytes are supported by connective tissue that contains the capillaries of both the oxygen-poor bronchiolar vessels and the oxygen-rich pulmonary vessels [11].

Air reaches the alveoli when inhalation occurs. Normal ventilation is an automatic, unconscious process, although it can be altered intentionally. Negative pressure develops when the diaphragm contracts and the elasticity of the lungs and pleura expand to fill the thoracic cavity. In some situations, the expansion of the space to create the negative pressure is assisted by the external intercostal muscles and other accessory muscles of respiration. The sternocleidomastoid and scalene muscles can elevate the ribcage to assist with this expansion [11].

The second part of the process of ventilation is called exhalation, which generally occurs because of the relaxation of the thoracic cavity and diaphragm. The internal intercostal muscles may assist the expiratory function, as do the abdominal muscles when needed to elevate the diaphragm. The lungs are delicate and would collapse if the air pressure inside the chest exceeded that inside the lungs. For this reason, the air pressure inside the thoracic cavity is slightly lower than the pressure within the lungs.

The alveoli are connected to the bronchioles by alveolar ducts, and then to the larger-diameter bronchi that branch off of the trachea. These passageways are lined with pseudostratified, ciliated, columnar epithelium and mucous glands. The effect of the cilia is to remove small particles of dust and other inhaled foreign bodies, while the mucus protects the lining of the airway by capturing foreign materials and pathogens. The trachea and bronchi are held patent by rings of cartilage. The lungs are covered with the pleural membrane, which is contiguous with the wall of the thoracic cavity [12].

Other nonrespiratory functions of the lungs include synthesis of arachidonic acid into products of the cyclo-oxygenase and lipoxygenase pathways; the metabolism of bradykinin, serotonin, and some prostaglandins; and the conversion of angiotensin I to angiotensin II in the regulation of blood pressure [11,12]. Patients being treated for hypertension or other conditions may be prescribed medications that block angiotensin converting enzyme (ACE).

These medications are ACE inhibitors (eg, lisinopril). These patients may develop a nonproductive cough in response to the buildup of angiotensin I in the lungs.

Clinical findings

Patients with pulmonary diseases typically present with one or more of the following symptoms: cough, dyspnea, sputum, hemoptysis, wheeze, or chest pain [12]. Coughing is the most frequent and earliest symptom, and is a rapid exhalation with high velocity airflow intended to clear the airway of secretions or foreign bodies. Breathlessness is characteristic of dyspnea wherein the patient has become aware of difficulty in getting enough air exchange. Dyspnea does not always signify disease as it may also occur during over-exertion or exercise.

Excretions are normal, but if they become glue-like (sputum) or blood-tinged (hemoptysis) they signify the progression of respiratory diseases. Wheezing is a high-pitched noise on inhalation that comes from air rushing through airways with reduced diameters. Chest pain is most commonly from inflamed pleura, but can also result from infection, bony involvement, neural involvement, or lung neoplastic disease [12].

COPD has been described for many years as a combination of chronic bronchitis and emphysema. Each disease has its own characteristics leading to the obstruction of the airway, but both may represent the spectrum of an irreversible process, and occur together. Overlapping symptoms make the diagnosis difficult, and for the majority of patients, cigarette smoking is the most significant etiologic factor. The airway obstruction in bronchial asthma is essentially reversible, but many years of recurrent exacerbations may cause permanent airway remodeling.

Chronic bronchitis

In chronic bronchitis, the airway is obstructed because of excessive mucus production and inflammation of the smaller airways. Surfactant production is lost and alveoli collapse. Gas exchange becomes inefficient. Without the surfactant to dilute it, thickened mucus plugs the bronchioles. The walls of the bronchioles become infiltrated with inflammatory cells, hyperplastic goblet cells, and enlarged mucous glands. The cross-section of the airway thus shrinks.

By definition, chronic bronchitis is diagnosed when a patient has a chronic, productive cough on most days for at least 3 months of a year for at least 2 consecutive years. [12–14] Along with the cough comes wheezing, shortness of breath, exertional dyspnea, and frequent respiratory infections. The term ''blue-bloater'' is often used to signify the cyanotic appearance of the patient, who is likely to be sedentary and overweight

[12,13,15]. These patients are typically over 50 years of age, but some younger patients exhibit the same symptoms as a result of alpha-one antitrypsin deficiency [4,16].

Emphysema

In emphysema, obstruction to gas exchange results from damage to the alveoli. Long-term exposure to cigarette smoke has recently been noted to modify type-IV collagen [17]. This contributes to increased macrophage adhesion and activation, which causes the destruction of the alveolar septa. The terminal air spaces are enlarged and, conversely, the gas exchange surface area shrinks. The bronchioles and bronchi lose supporting tissue, and the elasticity of the lung tissue is reduced. Exhalation is difficult.

Emphysema patients are termed ''pink-puffers'' as the effort of exhaling builds the muscles of the chest to a barrel shape and air becomes trapped in the lungs. The rate of ventilation increases and the patient may purse his lips in an attempt to exhale more completely. The accessory muscles cause the chest to rise more vertically than normal and the diaphragm rests in a more horizontal position. Fatigue and weight-loss progress with the disease. Coughing is not likely to be a significant feature in these patients. The diagnosis is confirmed with a chest radiograph that shows a relatively small heart and overinflated lungs [12,14].

Bronchial asthma

Bronchial asthma may be described as reactive airway disease. The patient has recurring episodes of wheezing, coughing, and dyspnea. An attack may last for several minutes, then resolve spontaneously with rest or in response to drug therapy. Patients often have quiescent periods, then experience a series of exacerbations. Some children may have complete resolution of their asthma at the onset of puberty, while other patients continue with the disease. There may be a genetic component to the susceptibility of certain patients [18,19].

In patients with bronchial asthma, the airway is hyperactive, and while different mechanisms can lead to the obstruction in an asthma attack, it appears to be mostly reversible. These mechanisms include bronchial smooth muscle spasm and constriction, inflammation and edema of the mucosa, and excessive mucus secretion. Rapid resolution of the attack after use of inhaled bronchodilator medication is now being used to help differentiate asthma from other diseases [10,16].

Different stimuli cause reactions that lead to the airway obstruction of asthma. Extrinsic or allergic asthma involves the activation of mast cells by antigen–antibody complexes. The allergens may be dust, pollen, or animal dander, and the attack ensues when the mast cells release histamine

and many other vasoactive and chemotactic factors. Bronchioles become obstructed by the stimulated mucus production, edema of the tissue, and leakage of plasma into the airways. The mucosa shows an increased inflammatory infiltrate and smooth-muscle spasm. Extrinsic asthma is the most common etiology, estimated at about 35% of all adult cases, and it is the likely cause in children [2,3,9,12,13].

Intrinsic asthma is not as clearly understood as extrinsic. There is a narrowing of the airway because of bronchospasm mediated by the vagus nerve. Some triggers are emotional or physical stress, anxiety, or nervousness. Intrinsic asthma is also called nonallergic, and usually affects patients over 30 years of age. There are often comorbidities, such as gastro-esophageal reflux disease or pregnancy [15].

Some asthmatics suffer attacks associated with exercise, inhalation of cold air, or after taking certain medications. Still others suffer airway constriction after infections caused by viruses, bacteria, or mycoplasma organisms [12]. Inflammation and accumulation of fluid in the tissues, bronchial smooth-muscle spasm and thick mucus secretion are typical findings.

Most patients have characteristics of several etiologies. Rather than focus on the specific stimuli of an asthma attack, physicians have developed a classification system that is based on clinical severity. Medical therapy is designed according to these stages of severity as well as the history of triggers. The term chronic is applied when the condition continues for more than 3 months [12].

Mild intermittent asthma is described as intermittent wheezing fewer than 2 days per week, with brief exacerbations and periods of no symptoms. These patients have good exercise tolerance and nocturnal symptoms fewer that two times per month.

Mild persistent asthma patients have exacerbations that affect activity and sleep on 2 to 5 days per week, with nocturnal attacks more than two times per month. The history may include a recent emergency room visit.

Moderate persistent asthma affects patients with wheezing attacks daily. These patients have the need to use bronchodilators daily and have nocturnal exacerbations at least one time per week. There is limited exercise tolerance and a history of emergency room visits.

Severe persistent asthma patients have daily and frequent exacerbations, with more than four nocturnal attacks per month. Exercise is not tolerable, and patients require periodic hospitalization.

Status asthmaticus is a persistent condition that continues for more that 24 hours despite therapy (See emergency treatment below).

Current concepts of diagnosis by primary care physicians include the use of spirometry, a measure of the amount of air that can be inhaled or exhaled [20]. The patient uses a simple volumetric device to measure the volume of each ventilation, often measured over time (eg, forced expiratory volume for 1 second). Tests include the use of short-acting bronchodilators to evaluate

the patient's airway response, as well as the bronchoprovocation test using methacholine [2,16,21]. The complete blood cell count with a differential assessment may show a mild eosinophilia, which supports the connection between asthma and allergies [16].

Medical management

Correct diagnosis of obstructive pulmonary diseases is essential before treatment is implemented. Proper use of spirometry to achieve this is advocated by the National Heart, Lung, and Blood Institute and the World Health Organization [1,22]. History is important, including pulmonary diseases in family members, triggers, seasonal variability, and age at inception or change of symptoms. In special populations, such as the elderly, some patients have both asthma and COPD [21]. Patients should be advised to consult their physician if symptoms become more intense or frequent. Respiratory-tract inflammation that goes untreated can lead to further pulmonary fibrosis.

There is no cure for chronic bronchitis or emphysema, but symptoms can be alleviated. Bronchodilators such as short- and long-acting beta adrenergic agonists, are the first line of medications. Inhaled corticosteroids improve lung function, reduce the severity of acute exacerbations, and reduce the number of COPD-related mortalities [21]. The use of beta adrenergic agonists with steroids actually shows a synergistic effect because as smooth muscle contracts, it generates inflammatory cytokines [23]. New evidence supports the use of anticholinergic medication to fight the bronchoconstriction caused by vagal mediation [24]. See the following section on asthma for more information about different inhaled medications.

Pulmonary rehabilitation includes a multidisciplinary program of incremental exercises and activities over a 6- to 10-week period [25]. Quality of life also improves with tobacco cessation [21,26]. A new procedure called noninvasive ventilation (NIV) has been introduced to help COPD patients who are hospitalized with severe exacerbations. NIV employs a close-fitting face mask to provide ventilatory support, eliminating the need for intubation and sedation. Mortality dropped from 10% to 20% with this change alone [27].

For patients with emphysema, a surgical procedure called lung volume reduction surgery is available in some centers. With the use of a minimal-access laparoscopic approach, the damaged emphysematous lung tissue is removed from the upper lobes. Patients have shown improvements in objective lung function, symptoms, and quality of life [26].

Appropriate referral for evaluation of depression is important for any patient with a chronic disease, especially those patients with diseases requiring long-term oxygen therapy. Depression and anxiety are also linked to acute exacerbations of COPD [21,28]. Other conditions and diseases may also cause exacerbations of obstructive pulmonary diseases and should be treated or prevented quickly. Gastroesophageal reflux disease may cause irritation of the

vocal cords, contributing to airway irritation [16]. Patients with obstructive pulmonary diseases should receive influenza and pneumococcal vaccines.

Proper management of asthma requires a step-wise approach with a specific written action plan developed for the individual patient. Studies show a direct reduction in hospitalizations and asthma mortality when patients have more knowledge and confidence [29,30]. Preventive "controller" medications (eg, inhaled corticosteroids, leukotriene receptor antagonists, and mast cell stabilizers) [10] and "rescue" [1,16] medications (eg, short-acting bronchodilators) are organized so that the patient has more participation in his or her treatment. The goal of asthma therapy is to keep the number and severity of attacks as low as possible while allowing the patient to have normal activity levels.

The recommended pharmacologic therapy for mild intermittent asthma consists of a short-acting beta-2 adrenergic agonist (eg, bronchodilator) as needed. These patients do not appear to benefit from the use of inhaled corticosteroids [10]. Commonly prescribed medications in the United States include albuterol and pirbuterol. These inhalers are considered rescue medications, and action begins within 5 minutes of administration. Duration may be as long as 4 hours. Mild intermittent asthmatics are not likely to need a controller medication [16].

Mild persistent asthma patients should show improvement with the early use of an inhaled corticosteroid as the initial controller medication [30]. Anti-inflammatory activity is delivered to the lungs with little systemic effect. Mounting evidence supports the efficacy of the rapid-onset, long-acting (up to 12 hours) beta adrenergic agonist salmeterol for mild persistent asthma [10]. Inhaled corticosteroids, such as triamcinolone, fluticasone, budesonide, and beclomethasone, are common. A combination of salmeterol and fluticasone is available in an inhaler used twice a day. Patient compliance is improved as only one inhaler is needed. Some patients find better control with inhaled anticholinergic medications, such as ipratropium bromide, which promotes smooth-muscle relaxation and acts as a bronchodilator. This medication is available in combination with albuterol.

Several other medications have been classified as controller drugs. Mast cell stabilizers (eg, cromolyn, nedocromil) control the release of inflammatory mediators, including histamine, leukotrienes, and prostaglandins. The methyxanthine medication theophylline works to inhibit phosphodiesterase, resulting in smooth-muscle relaxation. Side effects and drug interactions are common with theophylline and its use has been less frequent recently. Other methylxanthines are being developed to reduce these harmful effects [16]. Leukotriene modifiers focus at the substances and the pathways that lead to inflammation and bronchoconstriction. Montelukast is a leukotriene receptor antagonist, and zileuton is a lipoxygenase inhibitor that interrupts the formations of leukotrienes [16].

In very severe and persistent asthma, patients are treated with the addition of systemic steroids, such as prednisone and methylprednisolone,

preferably for short periods. Adverse side effects include cataracts, osteoporosis, diabetes mellitus, and adrenal insufficiency.

New trends and future areas of interest in treating obstructive pulmonary diseases are developing. Longer-acting medications, such as bronchodilators and anticholinergics, will help by reducing the amount of medications needed. Novel drugs that interact with inflammatory chemokines and cytokines may help to reduce the destruction of lung tissue. A monoclonal antibody against interleukin-8 in COPD has been developed [31]. Other targets are neutrophil proteases and other proteolytic enzymes [18,32]. Still other drugs under consideration would target mucus hypersecretion and molecular components, increase the hydration of mucus, and enhance mucociliary clearance. Lastly, an investigation of the human genome may reveal a specific marker that makes individuals more susceptible to COPD and asthma and lead to gene-based therapy to reverse or intercept these diseases [16,18].

Oral manifestations

Proper oxygenation of orofacial tissues is essential for maintaining good health, fighting infection, and ensuring proper healing after surgery or injury. Obstructive pulmonary diseases may lead to a hypoxic state that affects the entire body. The exposure to cigarettes is a major risk factor for oral cancer and a thorough screening examination is important to investigate for early signs.

Patients who have difficulty breathing may resort to using their mouths to increase the amount of air that can pass. In these patients, dentists are likely to see some decrease in the amount of saliva and, consequently, effects of oral dryness (xerostomia). In the anterior teeth, the gingival appears more edematous, and there may be an increase in the amount of calculus on the facial of the maxillary incisors [33]. This tendency to mouth breathing secondary to asthma in young children may alter the development of the facial structures, resulting in increased upper anterior and total anterior facial height, higher palatal vaults, greater overjets, and a higher prevalence of crossbites [34]. The levels of dental erosion in children with asthma are higher than in children without asthma [35].

There do not appear to be any direct effects of COPD on oral health [36]. The medications prescribed for patients with obstructive pulmonary diseases can lower the pH of dental plaque [37]. Xerostomia is a side effect of beta adrenergic agonist inhalers, antihistamines, and other medications and can lead to an increased caries risk, gingivitis, and oropharyngeal candidosis [3,15,38–40]. Inhaled and systemic corticosteroids may alter the ability to heal after surgery or trauma, and can lead to gingivitis and candidosis [15]. Dentists may also be consulted for the diagnosis and treatment of dysgeusia or mucosal erosions that may be secondary to medications.

Dental management

Dental health care providers should be aware of the patient's level of control of his disease. A prudent dentist will review and discuss the medications, frequency of exacerbations, and triggers of attacks before dental treatment. Dental materials that have powder as a component (eg, alginate and allergens used in latex gloves) may be implicated in the worsening of the patient's airway obstruction if the powder is inhaled.

Some patients with advanced emphysema may be using a portable oxygen delivery system and a nasal cannula. Furthermore, these patients may not be able to tolerate a horizontal position in the dental chair. Rubber dams should be used to avoid possible aspiration of instruments or aerosols that may contain bacteria as upper and lower respiratory infections contribute to airway obstruction [33].

Anxiety can trigger an asthma attack and may exacerbate chronic bronchitis by increasing smooth-muscle contraction [3]. Nitrous oxide sedation is possible for patients with mild to moderate asthma, but should be avoided in those with severe asthma or emphysema. This is because the primary drive to breathe is hypercarbia and in patients with COPD, this is replaced by hypoxia, a secondary drive to breathe. Nitrous oxide and oxygen delivers twice the oxygen as room air, which may suppress the patient's drive to inhale. Oral premedication with diazepam can be used in small doses [14].

Certain medications should be avoided in susceptible patients. There are some patients who experience asthma attacks after using aspirin or other nonsteroidal anti-inflammatory drugs. Sulfites used as an antioxidant and preservative for vasoconstrictors, such as epinephrine or levonordefrin in anesthetic cartridges, may also trigger an attack, but this appears to be a rare occurrence [13,14,41]. The analgesics of choice are acetaminophen and propoxyphene. Codeine-related drugs (eg, oxycodone and hydrocodone) and the compounds containing these may aggravate bronchospasm [41]. Narcotics, sedatives, and tranquilizers should be avoided because of the side effect of respiratory depression [15].

Medical emergency during dental care

Acute airway obstruction (eg, a dental crown or a rubber dam clamp that is swallowed) can be a life-threatening situation if not managed properly. Prevention is the best management, but immediate recognition and treatment are essential. Malamed's *Medical Emergencies in the Dental Office* [42] provides a comprehensive approach to dealing with many different medical emergencies and to preventing problems if possible. Cardiopulmonary resuscitation (CPR) certification is now required by most states to renew one's dental license. Within the CPR training are methods to assist in clearing a mechanical airway obstruction.

A patient with a chronic obstructive respiratory disease or asthma may have an acute exacerbation in the dental office. For a patient who is known to have these diseases, it is important to understand the patient's respiratory function status. What are the medications that have been prescribed? Is the patient taking them correctly, or at all? When was the last acute exacerbation, and what caused it? Having a clear picture of the patient's medical history allows the dentist to avoid a medical emergency. It is therefore necessary to frequently update the patient history.

For an asthmatic patient, the rescue inhaler should be readily available during the dental appointment. Status asthmaticus can occur in any patient with asthma, although it is more likely to occur in those with moderate to severe persistent asthma. If the patient does not respond to inhaled bronchodilators, hospitalization is necessary to provide life support. Parenteral delivery of bronchodilators is indicated when the airway is nearly or totally obstructed. Signs include extreme fatigue, dehydration, severe hypoxia, cyanosis, peripheral vascular shock, tachycardia, and low blood pressure [42].

Chronic use of systemic corticosteroids for treatment of any obstructive pulmonary disease can lead to iatrogenic adrenal insufficiency. These patients may not be able to respond to surgical stress, infection, or pain without supplementation, especially for major dental procedures [3,15,36]. Inhaled corticosteroids rarely cause adrenal suppression [13].

Tobacco cessation

The rate of smoking in the United States has dropped over the past several decades. The American Lung Association reports that cigarette consumption has declined steadily over the past 40 years to the 2004 level of 20.9% of adults (estimated) or about 44.5 million [43]. The amount of tobacco consumed by individuals appears to have decreased as well [43], although there was an increase in the number of cigars that were consumed by about 9% from the previous year [43].

Tobacco use, especially cigarettes, is responsible for more than 438,000 deaths each year [5]. Cigarette smoking is the major risk factor for chronic bronchitis, emphysema, and some cases of chronic asthma [44], and has been attributed to 70% of chronic lung diseases [45]. A patient may have a history of smoking 10 to 20 cigarettes per day for 20 or more years before the onset of symptoms [46]. When a patient with chronic obstructive pulmonary disease stops smoking, the patient's rate of decline slows. However, the damage caused by the disease can never be repaired [44,47].

The reasons dentists and other dental health care providers give to encourage patients stop using tobacco have focused on diseases and conditions in the mouth. Current campaigns from the American Dental Association (ADA), the American Dental Hygiene Association, and Oral Health America (Table 2) all concentrate on reducing oral cancer, slowing periodontal

Table 2
Public information programs of dental organizations

Organization	Campaign title	More information
American Dental Association	Dentist Saves Patient's Life! Early Oral Cancer Detection And Tobacco Use Cessation	www.ada.org
American Dental Hygiene Association	Smoking Cessation Initiative: Ask, Advise, Refer. Quitlines (state and local information)	1-8000-243-ADHA x220 www.askadviserefer.org
Oral Health America National Spit Tobacco Education Program (NSTEP)	Smoking Does NOT Mean Harmless	www.NOSPIT.com www.chewfree.com www.nstep.org

disease progression and gingival recession, avoiding other soft tissue defects and abrasion, improving the rate of healing, and reducing stains and halitosis [48,49]. However, as health care providers, dentists must also take into consideration a patient's overall health. Tobacco cessation is important for not only improving orofacial conditions, but also for reducing morbidity and mortality associated with heart and lung diseases [50].

In 2003, the ADA included the US Public Health Service Clinical Practice Guideline [51] into the third edition of the ADA Guide to Dental Therapeutics [49,52]. A study of dentists in east Texas found that most were unaware of this guideline, and that a lack of training is the greatest barrier to conducting tobacco interventions [49]. A simple plan tailored for a busy office is designated the "Five A's [51,53,54]:

- Ask every patient about tobacco use.
- Advise tobacco users to stop.
- Assess patient's willingness to make an attempt to quit.
- Assist patients interested in quitting.
- Arrange for follow-up contact.

Another recently published study shows that current smokers perceive that they need more dental treatment than nonsmokers do [55]. These patients should receive a brief tobacco cessation message from each member of the dental health care team. Those dental patients who also have obstructive pulmonary diseases should find a benefit for their lungs and heart as well.

Former US Surgeon General C. Everett Koop said, "Cigarette smoking is the chief, single most avoidable cause of death in our society and the most important public health issue of our time." Dental health care providers can and should take the opportunity to spend a few minutes with patients to stress tobacco cessation that will lead to improvements in both their oral and overall health.

Summary

Many dental patients have obstructive pulmonary diseases, such as chronic bronchitis, emphysema, and bronchial asthma. These diseases have different etiologies but may have overlapping signs and symptoms. Diagnosis and treatment are becoming more sophisticated so that morbidity and mortality are improving. Most dental patients with these diseases can be treated safely with only minor adjustment to procedures. Cigarette smoking is a common risk factor that dental health care providers can address with a simple cessation program called the five A's.

References

[1] GOLD guidelines, National Heart, Lung, and Blood Institute/World Health Organization. The GOLD workshop panel. Global strategy for the diagnosis, management and prevention of chronic obstructive pulmonary disease: NHLBI/WHO workshop report. Bethesda (MD): National Heart and Blood Institute; 2001. NIH publication #2701.
[2] Sollecito TP, Tino G. Asthma. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;92: 485–90.
[3] Steinbacher DM, Glick M. The dental patient with asthma: an update and oral health considerations. J Am Dent Assoc 2001;132:1229–39.
[4] Tilles SA. Differential diagnosis of adult asthma. Med Clin N Am 2006;90:61–76.
[5] Average number of deaths, 1997–2001. Centers for Disease Control, Morbidity and Mortality Weekly Report 2005;54(25):625–8.
[6] Trends in chronic bronchitis and emphysema: morbidity and mortality. American Lung Association Epidemiology and Statistic Unit, Research and Program Services; May 2005. Tables 1, 4, 7. Available at: www.lungusa.org. Accessed February 13, 2006.
[7] Estimated prevalence and incidence of lung disease by lung association territory. American Lung Association Epidemiology and Statistic Unit, Research and Program Services; July 2005. Executive summary and table 1. Available at: www.lungusa.org. Accessed February 13, 2006.
[8] Trends in asthma: morbidity and mortality. American Lung Association Epidemiology and Statistic Unit, Research and Program Services; May 2005. Executive summary and table 1. Available at: www.lungusa.org. Accessed February 13, 2006.
[9] Waldman HB, Swerdloff M, Perlman SP. An increasing number of your pediatric patients may have asthma: the demographics of asthma. J Dent Child 2000;67:98–101.
[10] Lim KG. Mild persistent asthma. Postgrad Med 2004;115:40–6.
[11] Andreoli TE, Carpenter CCJ, Griggs RC, et al, editors. Cecil essentials of medicine. 6th edition. Philadelphia: WB Saunders; 2004. p. 126–46.
[12] Bricker SL, Langlais RP, Miller CS. Oral diagnosis, oral medicine, and treatment planning. 2nd edition. Hamilton (Canada): BC Decker; 2002. p. 165–91.
[13] Little JW, Falace DA, Miller CS, et al. Dental management of the medically compromised patient. 6th edition. St. Louis (MO): Mosby; 2002. p. 125–36.
[14] Hatch CL, Canaan T, Anderson G. Pharmacology of the pulmonary diseases. Dent Clin N Am 1996;40:521–41.
[15] Tyler MT, Lozada-Nur F, Glick M, editors. Clinician's guide to medically complex dental patients. 2nd edition. Baltimore (MD): The American Academy of Oral Medicine; 2001. p. 57–62.
[16] Mathur AK, Busse WW. Asthma: diagnosis and management. Med Clin N Am 2006;90: 39–60.
[17] Kamholz SL. Pulmonary and cardiovascular consequences of smoking. Med Clin N Am 2004;88:1415–30.

[18] Buhl R, Farmer SG. Current and future pharmacologic therapy of exacerbations in chronic obstructive pulmonary disease and asthma. Proc Am Thorac Soc 2004;1:136–42.
[19] Jarjour NN, Kelly EAB. Pathogenesis of asthma. Med Clin N Am 2002;86:925–36.
[20] Martinez FJ, Standiford C, Gay SE. Is it asthma or COPD? Postgrad Med 2005;117:19–26.
[21] Decramer M, Selroos O. Asthma and COPD: differences and similarities. J Clin Pract 2005; 59:385–98.
[22] National Institute for Clinical Excellence (NICE). Chronic obstructive pulmonary disease: national clinical guideline for management of chronic obstructive pulmonary disease in adults in primary and secondary care. Thorax 2004;59(Suppl I):1–232.
[23] Schleimer RP. Drug pathways and their interactions in COPD therapy. Proc Am Thorac Soc 2005;2:340–1.
[24] Doherty DE. The pathophysiology of airway dysfunction. Am J Med 2004;117(12A): 11S–23S.
[25] British Thoracic Society. Pulmonary rehabilitation (standards of care). Thorax 2001;56: 827–34.
[26] Ware JH. The national emphysema treatment trial—how strong is the evidence? N Engl J Med 2003;348:2055–6.
[27] Plant PK, Owen JL, Elliott MW. Early use of non-invasive ventilation for acute exacerbations of chronic obstructive pulmonary disease on general respiratory wards: a multicentre randomised controlled trial. Lancet 2000;355:1931–5.
[28] Sprenkle MD, Niewoehner DE, Nelson DB, et al. The veterans short form 36 questionnaire is predictive of mortality and health-care utilization in a population of veterans with a self-reported diagnosis of asthma or COPD. Chest 2004;126:81–9.
[29] Bryant E. Chronic disease management for patients with respiratory disease. Nurs Times 2005;101:46–8.
[30] British Thoracic Society. Scottish Intercollegiate Guidelines Network (SIGN). British guideline on the management of asthma. Thorax 2003;58(Suppl I):11–94.
[31] Mahler DA, Huang S, Tabrizi M, et al. Efficacy and safety of a monoclonal antibody recognizing interleukin-8 in COPD: a pilot study. Chest 2004;126:926–34.
[32] MacNee W. Pathogenesis of chronic obstructive pulmonary disease. Proc Am Thorac Soc 2005;2:258–66.
[33] Scannapieco FA, Ho AW. Potential associations between chronic obstructive respiratory disease and periodontal disease: analysis of national health and nutrition examination survey III. J Periodontol 2001;72:50–6.
[34] Bresolin D, Shapiro PA, Shapiro GG, et al. Mouth breathing in allergic children: its relationship to dentofacial development. Am J Orthod 1983;83:334–40.
[35] Shaw L, Al-Dlaigan YH, Smith A. Childhood asthma and dental erosion. J Dent Child 2000; 67:102–6.
[36] Ghezzi EM, Ship JA. Systemic diseases and their treatments in the elderly: impact on oral health. J Public Health Dent 2000;60:289–96.
[37] Kargul B, Tanboga I, Ergeneli S, et al. Inhaler medicament effects on saliva and plaque pH in asthmatic children. J Clin Pediatr Dent 1998;22:137–40.
[38] Ryberg M, Moller C, Ericson T. Effect of beta 2-adrenoceptor agonists on saliva proteins and dental caries in asthmatic children. J Dent Res 1987;66:1404–6.
[39] Ryberg M, Moller C, Ericson T. Saliva composition and caries development in asthmatic patients treated with beta 2-adrenoceptor agonists: a 4-year follow-up study. Scand J Dent Res 1991;99:212–8.
[40] McDerra E, Pollard MA, Curzon MEJ. The dental status of asthmatic British school children. Pediatr Dent 1998;20:281–7.
[41] Eversole LR. Respiratory diseases. In: Silverman S, Eversole LR, Truelove EL, editors. Essentials of oral medicine. Hamilton (Canada): BC Decker; 2001. p. 42–53.
[42] Malamed SF. Medical emergencies in the dental office. 5th edition. St. Louis (MO): Mosby; 2000. p. 168–99, 209–23.

[43] Trends in tobacco use. American Lung Association Epidemiology and Statistic Unit, Research and Program Services; July 2005. Executive summary and tables 1–4. Available at: www.lungusa.org. Accessed February 13, 2006.
[44] Kanner R. Early intervention in chronic obstructive pulmonary disease: a review of the lung health study results. Med Clin N Am 1996;80:523–47.
[45] Johnson NW, Bain CA. Tobacco and oral disease. Br Dent J 2000;189:200–6.
[46] Anonymous. What you need to know about…chronic obstructive pulmonary disease. Nurs Times 2003;99(47):37.
[47] British Thoracic Society. BTS guidelines for the management of chronic obstructive pulmonary disease. Thorax 1997;52:S1–28.
[48] Benson W, Christen AG, Crews KM, et al. Tobacco-use prevention and cessation: dentistry's role in promoting freedom from tobacco. J Am Dent Assoc 2000;131:1137–43.
[49] Hu S, Pallonen U, McAlister AL, et al. Knowing how to help tobacco users: dentists' familiarity and compliance with the clinical practice guideline. J Am Dent Assoc 2006;137:170–9.
[50] Wagena EJ, van der Meer RM, Ostelo RJ, et al. The efficacy of smoking cessation strategies in people with chronic obstructive pulmonary disease: results from a systematic review. Respir Med 2004;98:805–15.
[51] Fiore MC, Bailey WD, Cohen SF, et al. Treating tobacco use and dependence: clinical practice guideline. Rockville (MD): US Department of Health and Human Services, Public Health Service; 2000.
[52] Mecklenburg RE. Cessation of tobacco use. In: Ciancio SG, editor. ADA guide to dental therapeutics. 3rd edition. Chicago: American Dental Association; 2003. p. 587–99.
[53] Tomar SL. Dentistry's role in tobacco control. J Am Dent Assoc 2001;132:30S–5S.
[54] Crews KM, Gordy FM, Penton-Eklund N, et al. Tobacco cessation: a practical dental service. Gen Dent 1999;47:476–83.
[55] Dye BA, Morin NM, Robison V. The relationship between cigarette smoking and perceived dental treatment needs in the United States: 1988–1994. J Am Dent Assoc 2006;137:224–34.