SIXTH EDITION

Fishman's

PULMONARY DISEASES AND DISORDERS

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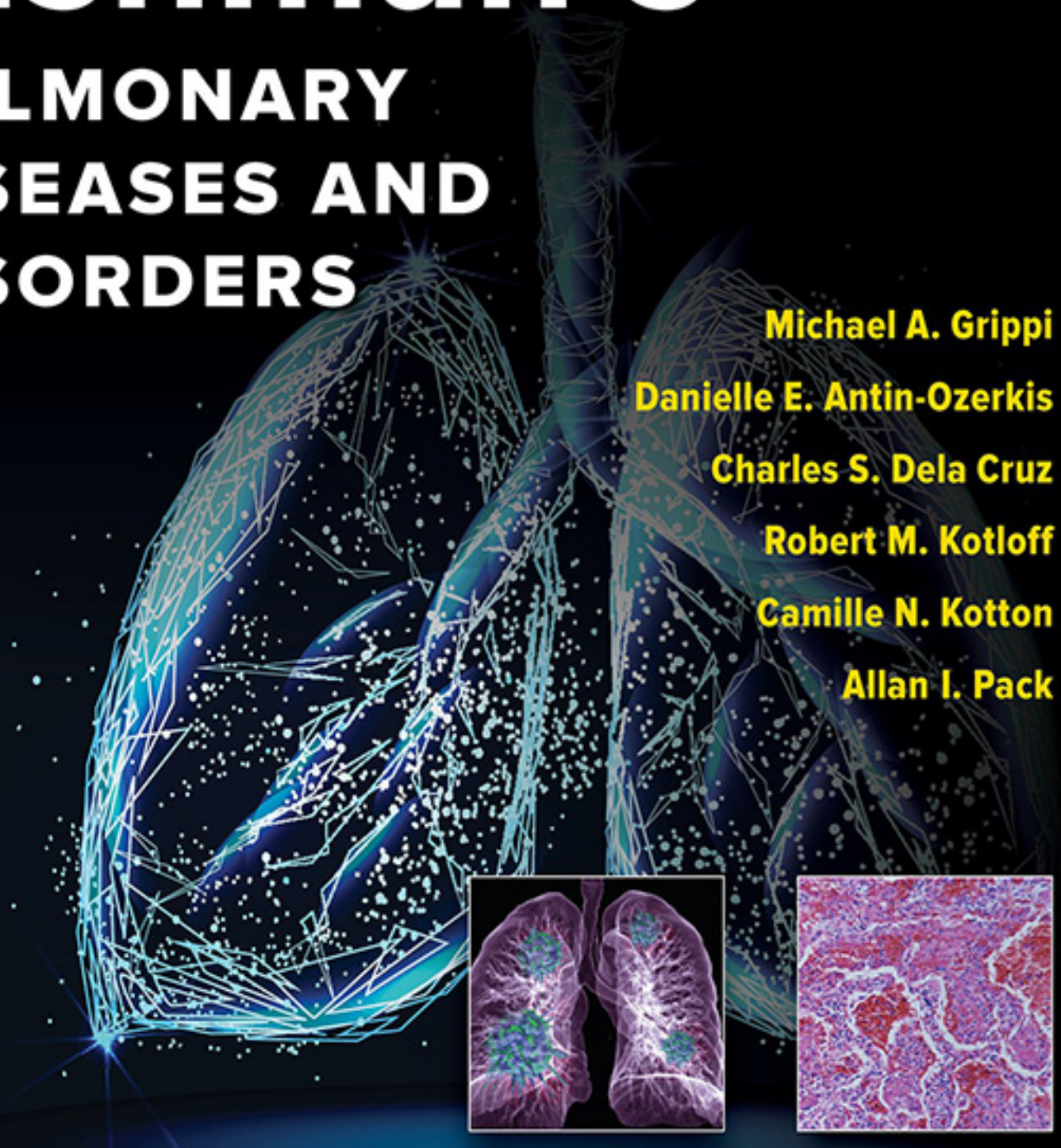

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Fishman's Pulmonary Diseases and Disorders

Sixth Edition

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CHAPTER 47

Upper Airway Obstruction in Adults

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Christine Won
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INTRODUCTION

The upper airway is conventionally described as being made up of all the structures that conduct air between the carina and the nares and includes the trachea, larynx, pharynx, nasal airway, and oral airway. Upper airway structures may change their physiologic function in response to pressures around them and anatomic structures near them. Thus, physiologically, the segments of the upper airway behave differently when they are subject to pleural pressures (anatomically intrathoracic) or ambient pressures (anatomically extrathoracic). Furthermore, extrathoracic airway function may change with posture, sleep/wake state, and the function and anatomy of tissues surrounding the airway.

The upper airway evolved anatomically in humans to subserve several important functions including swallowing, breathing, and vocalization.[1] The multifunctionality of the upper airway increases the risk of certain diseases (e.g., aspiration and sleep apnea).[2] These functions require that different segments of the airway have differing properties. The trachea receives some support by the tracheal rings, the nasal airway is surrounded by rigid structures, and the oral airway has a rigid bony roof, the hard palate. On the other hand, the pharyngeal airway does not have rigid structures supporting it. It is a collapsible tube whose patency is maintained by muscles whose function is affected by arousal state (sleep/wake, and more specifically during sleep, the stage of sleep), the structures around it, and posture. Thus, the pharyngeal airway is divided anatomically and physiologically into the nasopharynx, retropalatal oropharynx, retroglossal oropharynx, and hypopharynx.

Clinically significant obstruction in adults may occur anywhere within the upper airway. Common etiologies of upper airway obstruction (UAO) include neoplasia, scar formation, skeletal facial malformations, infection, inflammatory disorders, trauma, extrinsic compression related to pathology of adjacent structures, and functional changes related to posture and sleep/wake state. Airway obstruction may be classified as extrinsic, intrinsic, or mixed (Fig. 47-1).

UAO can be acute and life-threatening, or chronic and resulting in significant symptoms, some of which may not even relate to the respiratory system. Initial management of acute UAO focuses on securing the airway and stabilizing the patient. Often the intervention is surgical. Some diseases require bypassing the obstruction using translaryngeal intubation or tracheostomy. Definitive long-term management depends on the underlying etiology and physiology and may include both medical and surgical interventions. The still-evolving fields of imaging and interventional pulmonology offer new diagnostic and management modalities. This chapter provides an overview of acute and chronic UAO in adults and focuses on clinical presentation, assessment, etiology, and management. Obstructive sleep apnea is covered in Chapter 98 of this volume.

CLINICAL FEATURES

Upper and lower airway obstruction may present with similar symptoms (e.g., shortness of breath, noisy breathing) and physical findings (e.g., wheezing, diminished breath sounds). Asthma and chronic obstructive pulmonary disease, common causes of lower airway obstruction, are often incorrectly assumed to be causing the patient's symptoms.

Significant UAO may be asymptomatic for a prolonged period of time, resulting in delayed presentation and diagnosis, and possibly a catastrophic outcome. When UAO develops or worsens acutely, asphyxia and death may result within minutes to hours. When UAO develops slowly, diagnoses may be delayed or incorrect and, in the case of malignancy in the upper airway, may lead to incurable disease.

Dyspnea and noisy breathing, the most common symptoms of UAO, are often prominent during exercise and may also be intensified or relieved by a change in body position. The patient may indicate that breathing is labored while supine and may experience sleep disordered breathing in the forms of obstructive sleep apnea or upper airway resistance syndrome (Chapter 98). Therefore, daytime somnolence may be a prominent feature of UAO. In severely affected patients, peripheral edema as a result of cor pulmonale may be present as a consequence of chronic hypoxemia and hypercarbia during sleep and wakefulness.

In most cases, significant anatomic obstruction precedes the development of symptoms. By the time dyspnea on exertion occurs, the airway diameter at the site of obstruction is likely to be reduced to about 8 mm. Shortness of breath at rest often develops when

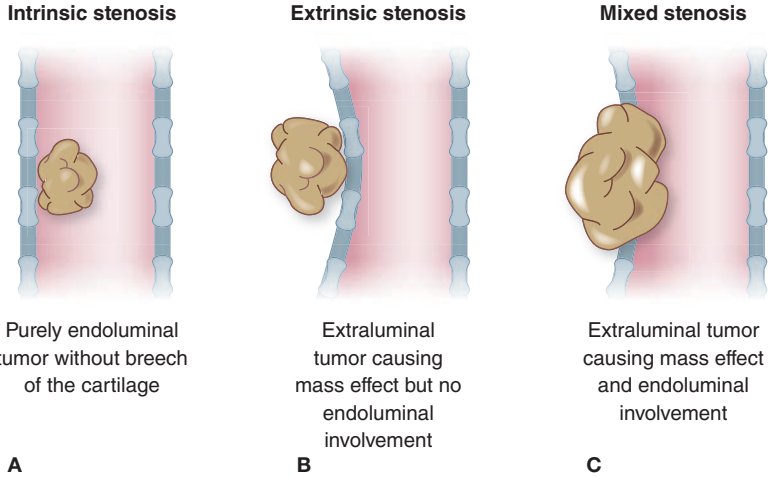


Figure 47-1 Classification of airway tumor involvement. **A.** Intrinsic stenosis. Purely endoluminal tumor without breech of the cartilage. **B.** Extrinsic stenosis. Extraluminal tumor causing mass effect but no endoluminal involvement. **C.** Mixed stenosis. Extraluminal tumor causing mass effect and endoluminal involvement.

the airway diameter is about 5 mm, and stridor is now often present. Although airway radius does play a significant role in symptom development, it is important to note that other factors such as length of stenosis and pressure differential across the stenosis also influence perceived symptoms. Stridor is a loud, musical sound of constant pitch that usually indicates the presence of extrathoracic airway obstruction most often affecting the larynx or upper trachea.

Unlike wheezing, which is a feature of diffuse peripheral airway obstruction and occurs primarily during expiration, stridor usually occurs during inspiration and is loudest in the neck. The sound can usually be appreciated without using a stethoscope. Although one would expect to be able to distinguish stridor from wheezing coming from lower airways, sound recordings from the neck and chest have shown that the sounds from the asthmatic wheeze and stridor have similar frequencies.

Breathing maneuvers that increase inspiratory airflow, such as forced inspiration or voluntary hyperventilation, accentuate the intensity of stridor. Neck flexion may change the loudness of stridor. When the obstructing lesion is fixed, both inspiratory and expiratory stridorous sounds may be heard. At times, the character of a patient's voice may be a clue to the presence of UAO. Hoarseness may be a sign of a laryngeal abnormality. Muffling of the voice may be present when vocal cords are paralyzed.

LABORATORY ASSESSMENT

Just as UAO must be quite advanced before development of symptoms, physiologic abnormalities do not become apparent on lung function testing until severe obstruction (<8 mm or 80% of normal medial tracheal diameter) occurs. The forced expiratory volume in 1 s (FEV_1) remains above 90% of control until a 6-mm orifice is created. Therefore, spirometry may not be an effective way to detect upper airway abnormalities. The peak expiratory flow rate (PEFR) and maximal voluntary ventilation (MVV) are more sensitive than the FEV_1 in detecting UAO. Of note, a noncritical airway may become so with a lesser degree of obstruction in the context of airway secretions, edema, or bleeding.[9]

The flow–volume loop, which is a recording of maximal inspiratory and expiratory flows at various lung volumes, is an important tool for the diagnosis of UAO. The configuration of the normal flow–volume loop is shown in Fig. 47-2. During a forced expiratory maneuver from total lung capacity (TLC), the maximal flow achieved during the first 25% of the forced vital capacity is dependent on effort—that is, an increase in driving pressure (effort) may result in increased flow. During the remaining 75% of the forced vital capacity maneuver, flow is determined by the mechanical properties of the lungs and is not effort dependent. During this portion of forced exhalation, a linear deceleration of flow is caused by dynamic compression of the intrathoracic airways (Fig. 47-3A). An increase in effort and therefore pleural pressure causes further compression of the intrathoracic airways and a further limitation of airflow. At higher lung volumes, flow may be limited by a UAO. At low lung volumes, flow may not be affected by a UAO, since measurement of flow in this effort-independent portion of the curve represents the function of the peripheral airways. Since the FEV_1 reflects a large portion of flow at these lower lung volumes, it is not a sensitive test for UAO. Because the PEFR reflects flow at higher lung volumes, it may be abnormal when the FEV_1 is not.

In generating the flow–volume loop, forced inspiratory flow is limited by effort during the entire inspiratory maneuver. Flow increases from residual volume to near the midportion of the curve, where it becomes maximal at the peak inspiratory flow rate. Flow then declines until TLC is reached. The pressure surrounding the extrathoracic portion of the upper airway is atmospheric. The turbulent nonlaminar airflow, which occurs during forced inspiration and causes airway pressure to fall in this portion of the airway,

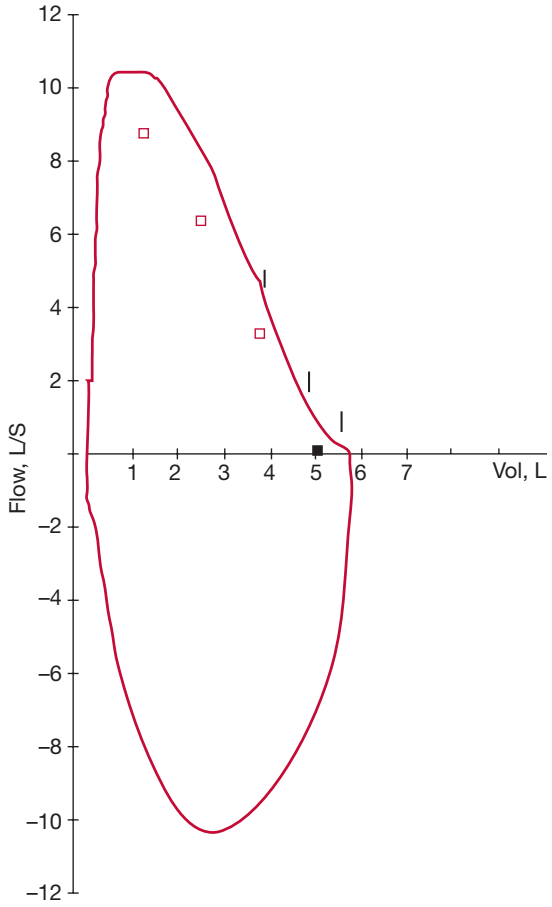


Figure 47-2 Normal flow–volume loop following maximal expiratory *(above)* and inspiratory *(below)* effort. Small vertical lines denote seconds.

favors slight narrowing of the extrathoracic airway (Fig. 47-3A, B). Peak inspiratory flow, therefore, is less than peak expiratory flow in normal subjects. Because of the dynamic compression of the intrathoracic airways that occurs during exhalation, flow during the middle of inspiration, that is, the forced inspiratory flow at 50% of the forced vital capacity ($FIF_{50\%}$), is usually greater than flow during the middle of forced expiration, that is, the forced expiratory flow at 50% of the forced vital capacity ($FEF_{50\%}$). Typical patterns of the flow–volume loop may be seen, depending on whether the obstruction to flow is "fixed" or "variable," and whether the site of the obstruction is above or below the thoracic outlet or suprasternal notch.

Fixed obstructions of the upper airway are those in which the cross-sectional area does not change in response to transmural pressure differences during inspiration or expiration. A fixed obstruction may occur in either the intrathoracic or extrathoracic airways. Irrespective of the site of the obstruction, a fixed lesion results in the flattening of the inspiratory and expiratory phases of the flow–volume loop. A *variable obstruction* is one that responds to transmural pressure changes, eliciting varying degrees of obstruction during the respiratory cycle. Since the stresses on the intrathoracic and extrathoracic airways are different, changes seen in the flow–volume loop vary according to the site of the obstruction.

A number of conditions have been associated with nondistensible narrowing of the upper airway and fixed airway obstruction. Benign strictures and malignancy are common examples. Maximal inspiratory and expiratory flow–volume loops with fixed obstruction show constant flow, represented by a plateau during both inspiration and

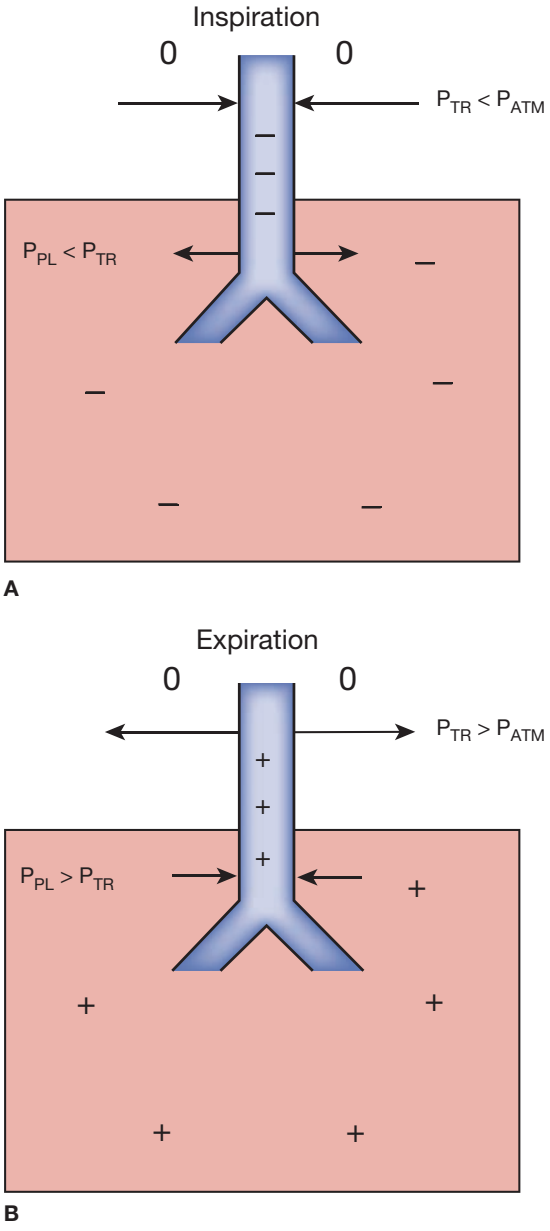


Figure 47-3 Forces acting on intra- and extrathoracic airway walls during inspiration and expiration. 0, atmospheric pressure; +, positive pressure; —, negative pressure. **A.** During inspiration, extrathoracic tracheal pressure (P_{TR}) falls below atmospheric pressure (P_{ATM}), favoring narrowing of the lumen *(arrows)*. Intrapleural pressure (P_{PL}) becomes negative, favoring airway enlargement *(arrows)*. **B.** During expiration, the extrathoracic tracheal pressure (P_{TR}) becomes positive and, therefore, greater than P_{ATM}, favoring enlargement of the lumen *(arrows)*. Intrapleural pressure (P_{PL}) is positive, causing dynamic compression of the intrathoracic trachea *(arrows)*.

expiration (Fig. 47-4A, B). On the expiratory curve, the plateau effect is seen in the effort-dependent portion of the curve near TLC; very little change is noted in the effort-dependent portion near residual volume. Since the inspiratory curve is similar in appearance, the ratio of $FEF_{50\%}$ to $FIF_{50\%}$ is normal (close to 1). The forced inspiratory volume in 1 second (FIV_1) and FEV_1 are nearly the same in fixed UAO.

Unilateral vocal cord paralysis is a common cause of variable extrathoracic obstruction. A variable extrathoracic airway obstruction increases the turbulence of inspiratory flow, and intraluminal pressure falls markedly below atmospheric pressure. This leads to partial collapse of an already narrowed airway and a plateau in the inspiratory flow loop (Fig. 47-5A, B). Expiratory flow is not significantly affected, since the markedly positive pressure in the airway tends to decrease the obstruction. The ratio of $FEF_{50\%}$ to $FIF_{50\%}$ is high (usually >2). Similarly, the FEV_1 is greater than the FIV_1.

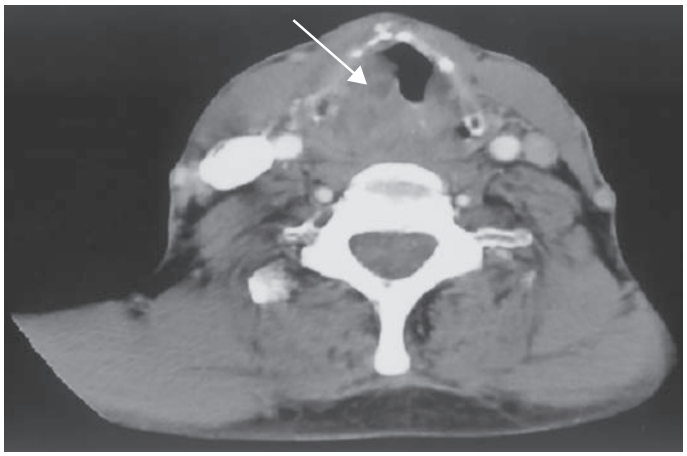
A variable obstruction in the intrathoracic airways reverses the situation. A predominant reduction in maximal expiratory flow is associated with a relative preservation of maximal inspiratory flow. This association occurs because intrapleural pressure becomes decidedly positive during forced expiration and causes dynamic compression of the intrathoracic airways. The obstruction caused by an intrathoracic lesion is accentuated, and a plateau in expiratory flow occurs on the flow–volume loop (Fig. 47-6A, B). A plateau of flow suggests that the lesion has caused the airway lumen to reach its minimal size. A flow peak may precede the plateau, suggesting that the obstruction may not affect flow until a certain lung volume is reached. During inspiration, intrapleural pressure is markedly negative; therefore, the obstruction is decreased. The ratio of $FEF_{50\%}$ to $FIF_{50\%}$ is very low and may approach 0.3. Similarly, the FEV_1 is considerably lower than the FIV_1. Although the flow ratios are similar to those seen in patients with COPD and chronic asthma, these disorders are distinguished from UAO by the appearance of the flow–volume loop. Thus, the expiratory curve in patients with COPD and asthma is primarily altered in the effort-independent portion of the curve, leading to a characteristic shape unlike the plateau configuration of a UAO (Fig. 47-7).

When a hospital laboratory or physician's office is not equipped to perform flow–volume loops, results of other tests, such as routine spirometry, may be helpful. If the forced spirogram shows that the PEFR is reduced disproportionately to the reduction in FEV_1, a UAO should be suspected. Other findings that suggest the diagnosis include a ratio of less than 1.0 for the inspiratory flow between 25% and 75% of the inspired vital capacity ($FIF_{25–75\%}$) and a value of less than 1.0 for the expiratory flow between 25% and 75% of the expired vital capacity ($FEF_{25–75\%}$). Another indication is an FEV_1 that is decreased to the same degree as the $FEF_{25–75\%}$. The MVV also may be a useful test, since it measures both inspiratory and expiratory flows. A ratio of MVV to FEV_1 of less than 25% is often found with UAO. Whenever the MVV is reduced in association with a normal FEV_1, a diagnosis of UAO should be considered.

In contrast to the situation in patients with diffuse obstructive disease of the lower airways (e.g., COPD, asthma), the distribution of ventilation in the lungs is normal, and ventilation–perfusion mismatch does not occur. Hypercarbia is not seen unless the degree of obstruction is very severe, although nocturnal hypercarbia may occur while daytime levels of P_{CO_2} are normal. Hypoxemia is also not present except during exercise and with severe airflow limitation, when it may accompany increases in the level of P_{CO_2}. In contrast to asthma and many instances of COPD, the airflow obstruction caused by an upper airway lesion does not resolve following the inhalation of a bronchodilator unless there is also a component of small airway disease that is often the case.

IMAGING

When acute airflow obstruction occurs as a result of an abnormality of the extrathoracic airway, roentgenographic studies of the soft tissues of the upper chest and neck in the emergency setting may be helpful (Fig. 47-8). However, computed tomography (CT) has afforded the most important approach to imaging of the extrathoracic airways (Fig. 47-9). The standard chest roentgenogram is often not helpful in detecting UAO. The trachea is usually well visualized on the posteroanterior (PA) and lateral views in chest



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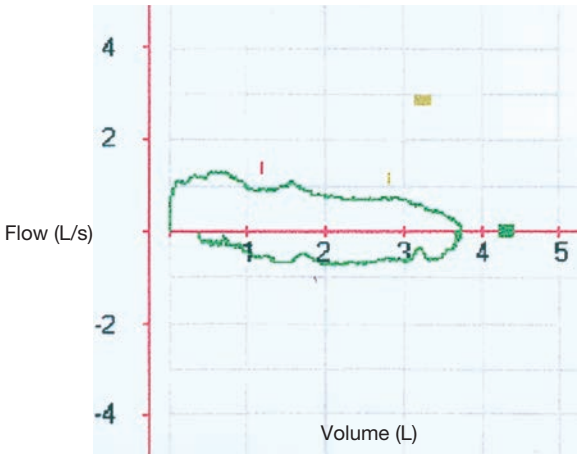
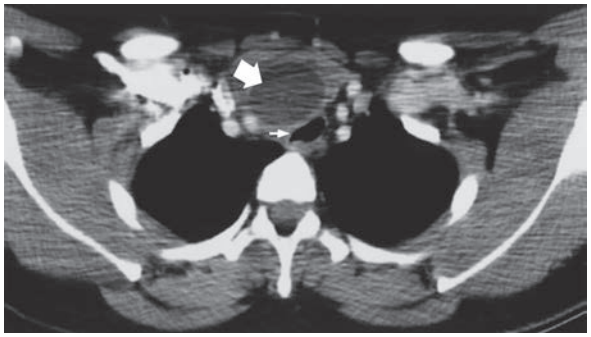


B

Figure 47-4 **A, B.** Flow–volume loop in fixed upper airway obstruction due to laryngeal abscess in a 56-year-old man who developed persistent wheezing, hoarseness of voice, and intermittent stridor for 3 months after a brief intubation for asthma exacerbation. Computed tomography scan of the neck (**A**) shows a laryngeal abscess with significant impingement on the laryngeal inlet. The flow–volume loop (**B**) demonstrates a plateau of flow during inspiration and expiration; the $FEF_{50\%}/FIF_{50\%}$ ratio is near 1.



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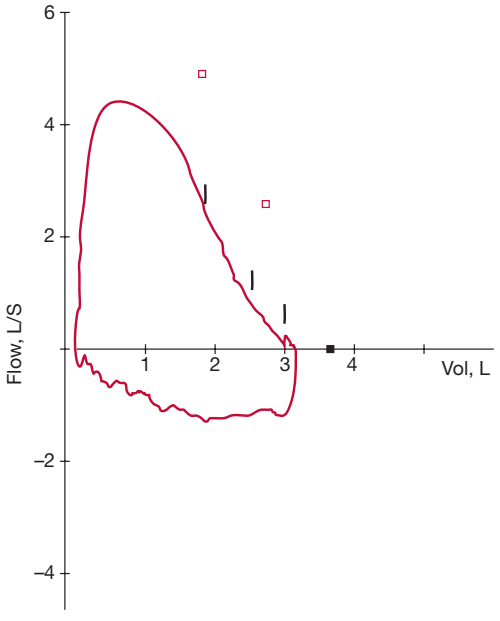
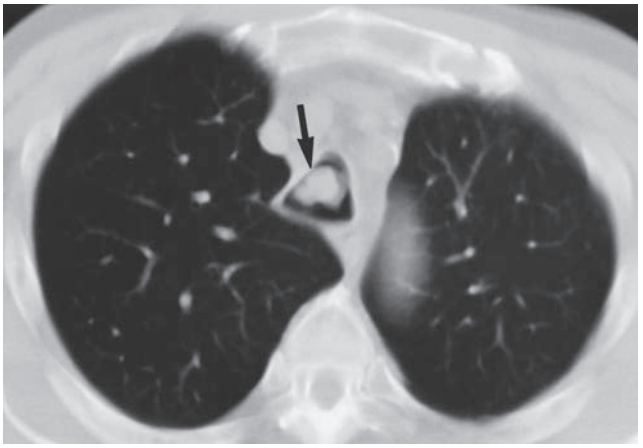


B

Figure 47-5 Variable extrathoracic obstruction due to thyroid cyst in a 32-year-old woman with dyspnea on exertion. **A.** Computed tomography of the neck shows a 10- × 4-cm cystic mass *(large arrow)* in the thyroid gland compressing the trachea *(small arrow)*. **B.** Flow–volume loop shows inspiratory obstruction. $FEF_{50\%}/FIF_{50\%}$ is very high, and the inspiratory curve is flattened.



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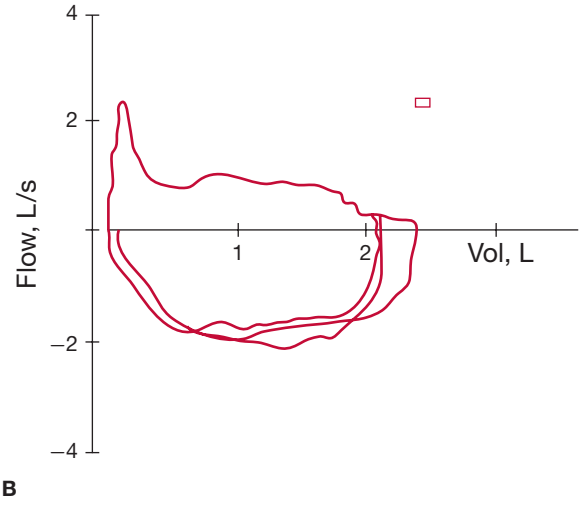


B

Figure 47-6 Variable intrathoracic obstruction due to squamous cell carcinoma of the trachea. **A.** Computed tomography of the chest shows a tracheal lesion *(arrow)*, which was not readily apparent on plain chest radiograph. **B.** Superimposed flow–volume loops show a plateau of expiratory flow preceded by a peak of flow at higher lung volumes. The forced inspiratory flow is preserved in comparison to expiratory flow, but it is also reduced. $FEF_{50\%}/FIF_{50\%}$ is 0.4.

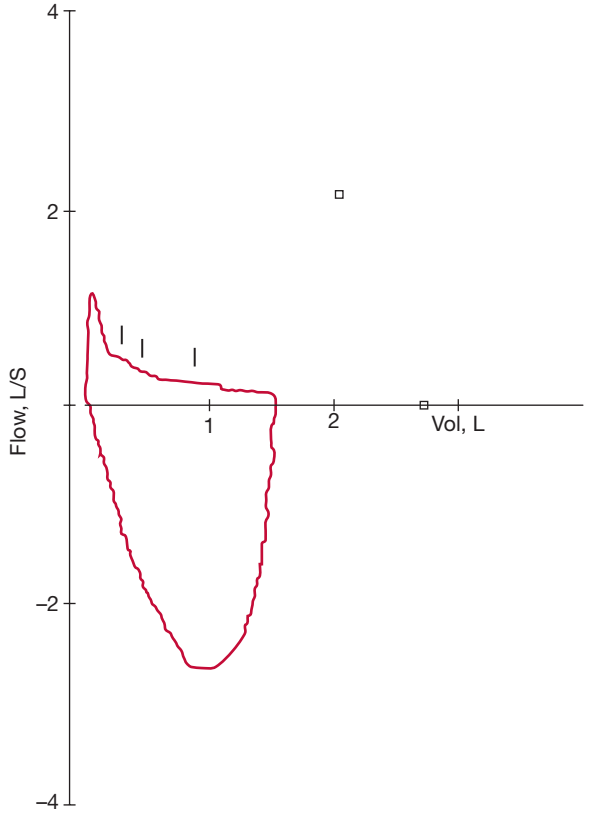


Figure 47-7 Flow–volume loop typical of chronic obstructive lung disease. Very low $FEF_{50\%}/FIF_{50\%}$ and typical curvilinear shape are noted.

Figure 47-8 Acute epiglottitis. Lateral soft tissue radiograph of the neck of a patient with stridor shows swelling of the epiglottis *(large arrow)* and loss of normal convexity of the edematous aryepiglottic folds *(small arrows)*.

roentgenograms of good quality. It is located in the midline and is moderately deviated at the level of the aortic arch. However, many standard roentgenograms are underpenetrated so that the trachea may become a "blind spot." In one study, only 13 of 53 tracheal tumors were evident to the radiologist on the standard PA roentgenogram. A lateral roentgenogram can provide a less obscured image of a tracheal abnormality, especially when the posterior wall is involved. Thoracic CT studies have become the procedure of choice for imaging the upper airway.

Helical CT (HCT) scanning minimizes artifacts due to respiratory motion and provides imaging of the whole thoracic volume during a single breathhold.[10] The technique represents an improvement over conventional CT scanning in that it allows detection of intraluminal, submucosal, and extraluminal lesions (Figs. 47-10A, B and 47-11). Since the early 1990s, HCT has become the preferred noninvasive modality for evaluation of the central airways. The use of HCT using multidetector technology and thin collimation provides high-resolution images of the entire thorax, improved special resolution, greater speed of image acquisition, and excellent contrast enhancement. HCT techniques using multiplanar and three-dimensional reconstruction can provide virtual images of the thorax that enhance the perception of local and diffuse anatomic lesions of the upper airways (Fig. 47-12). The images may demonstrate the degree of tracheal widening or narrowing, show the location and longitudinal extent of abnormalities, assess tracheal wall thickness, and demonstrate associated extratracheal diseases.

The use of paired inspiratory dynamic and expiratory multislice HCT has proved helpful for the diagnosis of tracheomalacia. Because the maximal degree of collapse in tracheomalacia usually occurs during exhalation rather than at end expiration, dynamic expiratory imaging is preferable to end-expiratory imaging. If complete collapse is not demonstrated during expiration, then

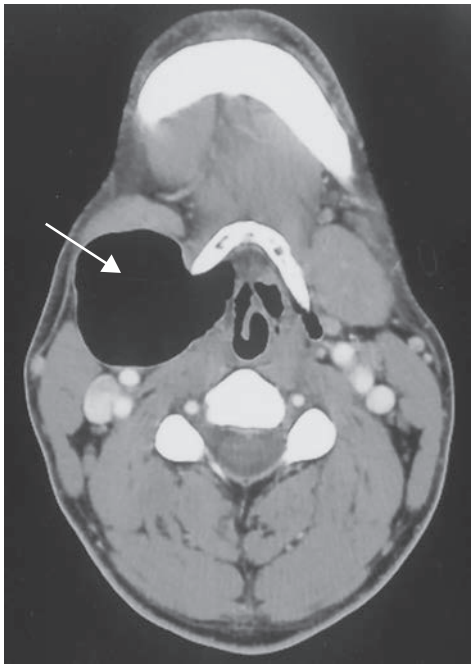
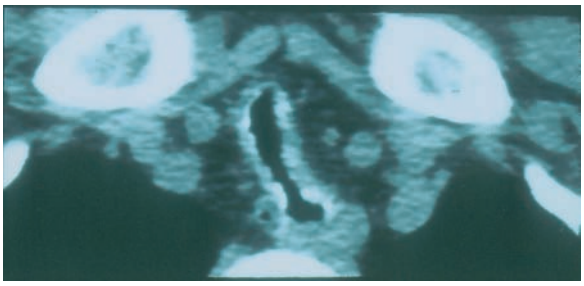


Figure 47-9 Computed tomography scan of the neck demonstrating a large laryngocele compressing the lateral wall of the larynx *(arrow)* causing positional air flow obstruction.



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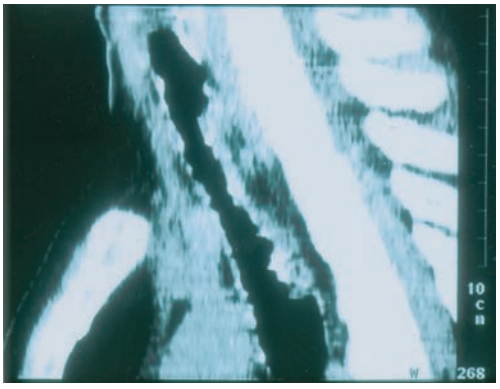


B

Figure 47-10 **A.** Computed tomography scan of the chest demonstrating marked narrowing of the trachea with intraluminal calcified nodular projections in a patient with tracheopathia osteoplastica. **B.** Computed tomography scan of the chest demonstrating multiplanar reformation of the trachea in the sagittal plane of the same patient.

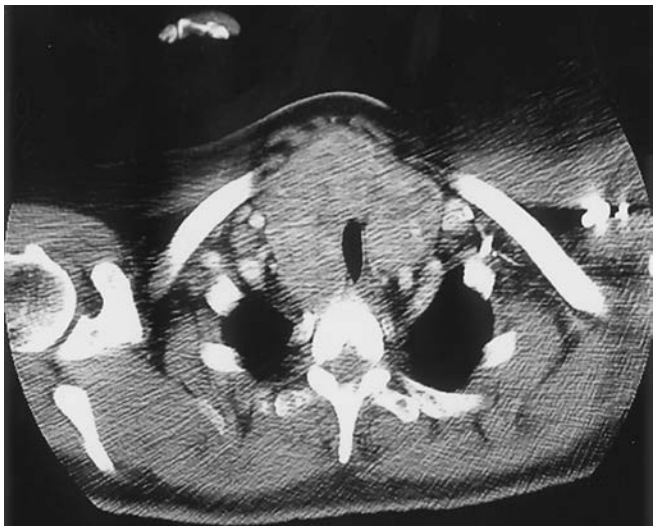
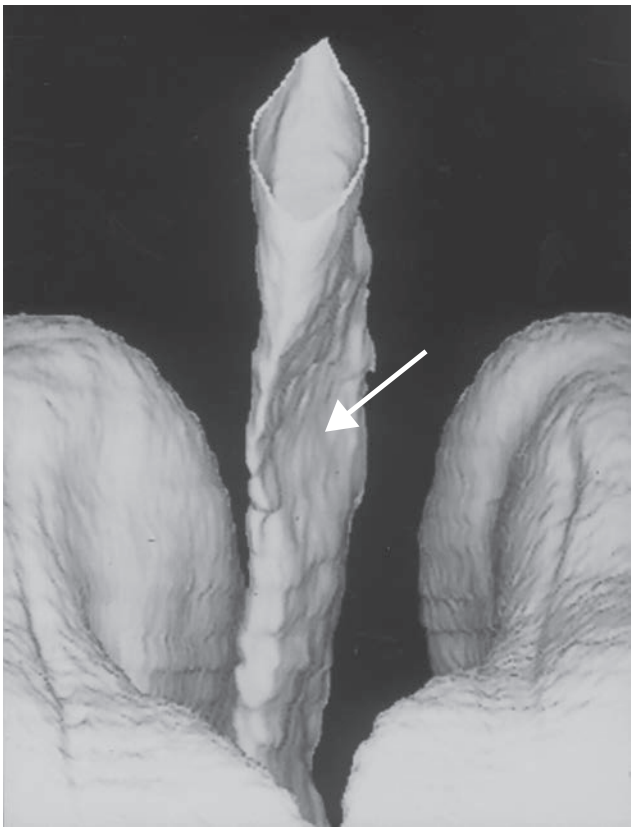


Figure 47-11 Computed tomography scan of the chest demonstrating marked extraluminal compression of the trachea caused by intrathoracic goiter.



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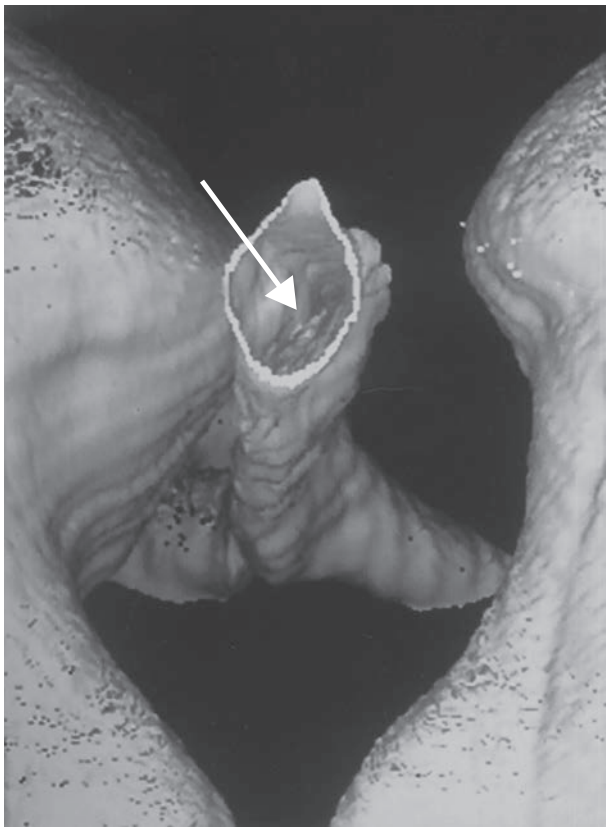


B

Figure 47-12 Helical computed tomography scan of the chest with three-dimensional reconstruction of the upper airway showing focal tracheal compression *(arrows)* (**A, B**).

one should confirm the diagnosis by quantitatively measuring the degree of airway luminal narrowing during expiration.

Another novel CT-based imaging technique is virtual bronchoscopy. The use of volumetric imaging allows for an intraluminal three-dimensional reconstruction of the airways and surrounding tissues. The technique has been used with a high degree of accuracy in assessing the width, length, and contour of fixed airway lesions.

Magnetic resonance imaging (MRI) provides a multiplane image of the chest without the need for contrast material. It is best used to investigate vascular structures surrounding central airways rather than the airways themselves, which are better visualized using CT scanning.

ENDOSCOPY

Bronchoscopy and direct laryngoscopy provide a real-time morphologic and functional evaluation of the upper airway. Imaging techniques are often limited by their inability to document the dynamic nature of many airway lesions and may underestimate the extent of an obstruction. With respect to laryngeal disease, endoscopy allows the operator to determine whether the vocal cords are mobile or fixed in abduction or adduction or alternatively moving paradoxically. This valuable information can help guide management. In cases of traumatic or thermal injury to the airways, edema, laryngeal fracture, or carbonaceous materials can be readily identified.

CLASSIFICATION

Stenosis classification systems are an objective tool that can be used between clinicians to provide an accurate characterization of the stenosis. While several classification systems exist for upper airway obstruction and none is universally accepted, broadly they include severity of airway narrowing, vertical length, location in the airway, cause, morphology/shape of stenosis, voice characteristics, swallowing, and functional impairment.[11]

Severity of the airway narrowing refers to the degree of reduction of cross-sectional area, with mild obstruction being less than 50% reduction in area and severe greater than 71%. Vertical length is important when considering treatment plans, as lesions >4 cm are generally considered inoperable. Location in the airway is described as relative to both the vocal cords and main carina (glottis, subglottic, tracheal, tracheobronchial). Morphology describes the shape of the airway lumen and can help determine etiology. Terms used to describe morphology include simple (short segment, concentric, <1 cm in length, and without malacia), complex (long segments >1 cm, with tracheal wall injury and/or malacia), pseudoglottic (triangular), eccentric, and circumferential. Classification of voice characteristics is on a spectrum ranging from no problems, to slight to significant impairments in audibility, to no voice. Finally, functional class is a subjective measurement of symptomatology with activities of daily living.

CAUSES OF UPPER AIRWAY OBSTRUCTION

UAO may be observed in a wide variety of settings, including infections, trauma (including iatrogenic), vascular disorders, and a wide variety of benign and malignant tumors. Each is discussed below.

Infection

A broad variety of infections may eventuate in UAO.

Deep Cervical Space Infections

Deep cervical space infections occur in potential spaces bounded by the deep cervical fascia. Patients with deep cervical space infections may present with sore throat, odynophagia, neck swelling, pain, fever, and dyspnea. Stridor and profound respiratory difficulty are signs of significant UAO. Parapharyngeal, peritonsillar, submandibular, and retropharyngeal abscesses appear to be common locations in adults. The bacteriology and initiating event of deep cervical infections appear to have changed over time.

Mixed infections caused by aerobic and anaerobic infections are common and have been reported in up to two-thirds of cases.[12] *Streptococcus viridans* and *Klebsiella pneumonia* are common pathogens. *Staphylococcus aureus*, *Staphylococcus epidermidis*, *Streptococcus pneumoniae*, *Escherichia coli*, and *Haemophilus influenzae* are other agents that are commonly reported. Alpha- and beta-hemolytic *streptococci* appear to have significantly declined in frequency. Overall, an odontogenic origin is probably most common. Intravenous drug abuse, mandibular fractures, iatrogenic and noniatrogenic traumatic injury to the upper airway, underlying malignancy, and poor underlying immune status are associated conditions. Ludwig's angina—an infection of the submandibular space and the floor of the mouth—is potentially lethal and is commonly associated with significant UAO. This entity is usually a cellulitic process and can affect the submandibular spaces bilaterally. Conservative management with antibiotics leads to significantly higher incidence of airway compromise compared with surgical drainage.[13] Many cases with true Ludwig's angina require tracheostomy.

Treatment of deep cervical infections involves maintenance of oxygenation and ventilation by securing an adequate airway, administration of appropriate antibiotics, and when indicated, use of surgical drainage. Complications of deep cervical infections include UAO, Lemierre's syndrome (see next paragraph), distant infection, septic embolization, carotid artery rupture, pulmonary embolism, direct extension of infection resulting in mediastinitis and empyema, and rupture of the abscess during intubation or other interventions.[14]

One particularly virulent cervical infection, known as Lemierre's syndrome, arises from a nasopharyngitis or peritonsillar abscess commonly due to *Fusobacterium necrophorum*. This lateral pharyngeal space infection results in suppurative thrombophlebitis of the internal jugular vein, septicemia, and metastatic abscess formation, particularly in the lungs and joints. Symptoms begin with a sore throat, fever, and painful swelling in the neck, followed by tender lymphadenopathy and tenderness along the sternocleidomastoid muscle due to thrombophlebitis of the internal jugular vein. Dysphagia, trismus, and UAO may occur as a result of swelling of the lateral pharyngeal space. Contrast-enhanced CT scan of the neck demonstrates thrombosis of the internal jugular vein, soft tissue abscesses, fasciitis, and myositis. Treatment with prompt antibiotics, such as high-dose penicillin with metronidazole, or monotherapy with clindamycin is key has led to the recent mortality rate of 5%.[15]

Epiglottitis

Epiglottitis is an infectious process that causes variable degrees of inflammation and edema of the epiglottis and supraglottic structures. Supraglottic structures usually are involved with variable involvement of the epiglottis in adults. Clinical presentation includes odynophagia, with inability to swallow secretions, sore throat, dyspnea, hoarseness, fever, tachycardia, and stridor. In one review, 44% of the patients had a normal routine oropharyngeal examination.[16]

Fiberoptic laryngoscopy is necessary to make the diagnosis and should be done without delay. Radiographic studies can be helpful in ruling out other etiologies with similar presentations and in evaluating potential complications but should not delay diagnosis or management.

Supraglottitis may involve the base of the tongue, uvula, pharynx, and false vocal cords. The disease may be increasing in prevalence among adults and declining in children, perhaps reflecting

introduction of *Haemophilus b* conjugate vaccines. Young adult males are commonly affected. The disorder appears to be more prevalent in colder winter months and in smokers. Blood cultures are positive in less than one-third of cases. Adult supraglottitis may be caused by a variety of organisms, including *Haemophilus influenzae*, pneumococci, group A streptococci, *S. aureus*, *Streptococcus viridans*, a variety of anaerobic organisms, mycobacteria, fungi, and viruses. Throat cultures can be helpful in diagnosis and management; however, treatment should not be delayed while awaiting culture results.

Illicit drug use may be associated with epiglottitis, with inhalation of heated objects (e.g., metal pieces from a crack cocaine pipe or the tip of a marijuana cigarette) causing thermal injury to supraglottic structures. Signs, symptoms, and roentgenographic and laryngoscopic findings are similar to those of infectious epiglottitis.

Initial antibiotic therapy using a third-generation cephalosporin or extended-spectrum penicillin is reasonable. The prevalence of resistant organisms should be considered when choosing empiric antibiotic coverage. Corticosteroids often are used in management of acute epiglottitis despite lack of evidence to support their use. Based on anecdotal case reports, epinephrine also is used.

Patients should be observed closely, and experienced staff should be available immediately to secure the airway by intubation or surgical approach, if needed. In a recent study, up to 10% of patients required ICU admission, and 4.4% required airway intervention.[17] Elevated blood glucose levels and CRP as well as a relative neutropenia predicted the need for higher level of care. Males were more likely to have recurrent disease, which was associated with an increased need for airway intervention. Securing the airway is extremely important in patients who develop stridor and other signs of significant airway obstruction. Mortality is around 2% to 3% but can be significantly higher, up to 17.6%, in those who require airway intervention.[18]

Laryngotracheobronchitis and Bacterial Tracheitis

Laryngotracheobronchitis, often called croup, is commonly seen in children who present with hoarseness, barking cough, shortness of breath, and stridor. This is an acute viral illness characterized by narrowing of the subglottic area. Adult croup is a rare condition. Rare instances of diphtheritic croup have been described in adults. Noninfectious membranous tracheitis related to trauma also has been reported.

Acute bacterial tracheitis refers to involvement of the subglottic trachea by bacterial infection and usually follows an episode of viral laryngotracheobronchitis. Thick, purulent exudates and mucosal edema may cause symptoms of UAO. *S. aureus* appears to be the predominant organism. Prompt antibiotic therapy, close observation with attention to airway compromise, and frequent suctioning are important. Data to suggest effectiveness of steroids or epinephrine in adults are lacking.

Rhinoscleroma is a chronic, progressive granulomatous infection of the upper airway that may cause airflow obstruction. This disorder affects primarily the nose and paranasal sinuses, but also may involve the nasopharynx, larynx, trachea, and bronchi. The causative organism is *Klebsiella rhinoscleromatis*. Rhinoscleroma is endemic in Africa, Asia, and South America and is rare in North America. About 5% of patients have diffuse narrowing of the trachea.[19] Prolonged antibiotic therapy with trimethoprim/sulfamethoxazole is effective.

Tuberculosis

Involvement of the upper airway can be observed both with and without pulmonary disease. Primary disease can develop due to direct inoculation of the airway with acid-fast bacilli, whereas secondary disease is due to either direct spread from advanced pulmonary disease or hematogenous or lymphatic seeding. Laryngeal tuberculosis is relatively uncommon, accounting for less than 1% of tuberculosis cases. The true vocal cords and epiglottis are the areas most affected. Laryngeal tuberculosis may present as progressive hoarseness and ulceration or a laryngeal mass. Endobronchial tuberculosis can be seen throughout the tracheobronchial tree and may result in significant airflow limitation that is related to the initial lesion or subsequent stricture formation. A barking cough and sputum production are common findings. In the appropriate clinical context, a positive purified protein derivative (PPD) skin test and acid-fast bacilli in sputum may suggest the diagnosis. However, a biopsy from the laryngeal or endobronchial abnormality usually is required. Biopsy features include caseating granulomas and acid-fast bacilli. Treatment with antituberculous medications is usually adequate and should be instituted promptly, since the disease is highly contagious. Additionally, prompt treatment with antituberculous medications should decrease the development of endobronchial fibrostenosis and resultant airflow limitation. Surgical interventions, including tracheostomy, are reserved for airway obstruction and long-term complications and, in one report, were required in 12% of patients with laryngeal involvement.[20]

Upper Airway Tumors

Both head and neck cancers and tracheal tumors may cause UAO.

Head and Neck Cancer

The great majority of cancers that impact the upper airway are squamous cell carcinomas, and the incidence of oropharyngeal cancer appears to be increasing in some countries (Fig. 47-13).[21] Other less common cancers occurring in the oropharynx include salivary gland carcinomas, lymphomas, and lymphoepitheliomas. Tobacco, alcohol abuse, and infection with human papillomavirus (HPV), especially HPV type 16, are the most common risk factors for the development of head and neck cancers.[22–24] The incidence of head and neck cancer is declining in the Unites States, concurrent with a reduction in tobacco use, accounting for about 3% of all malignancies.[25]

The clinical manifestations of head and neck cancer depend on the location and stage. Cancers at the base of the tongue and pharyngeal walls are insidious. These cancers grow in an infiltrative or exophytic pattern. Because of the lack of pain fibers at the base of the tongue, these tumors are often asymptomatic until they are far advanced. The most common location of a primary tumor of the

Figure 47-13 Laryngeal squamous cell carcinoma.

Figure 47-14 Squamous cell carcinoma of the lung.

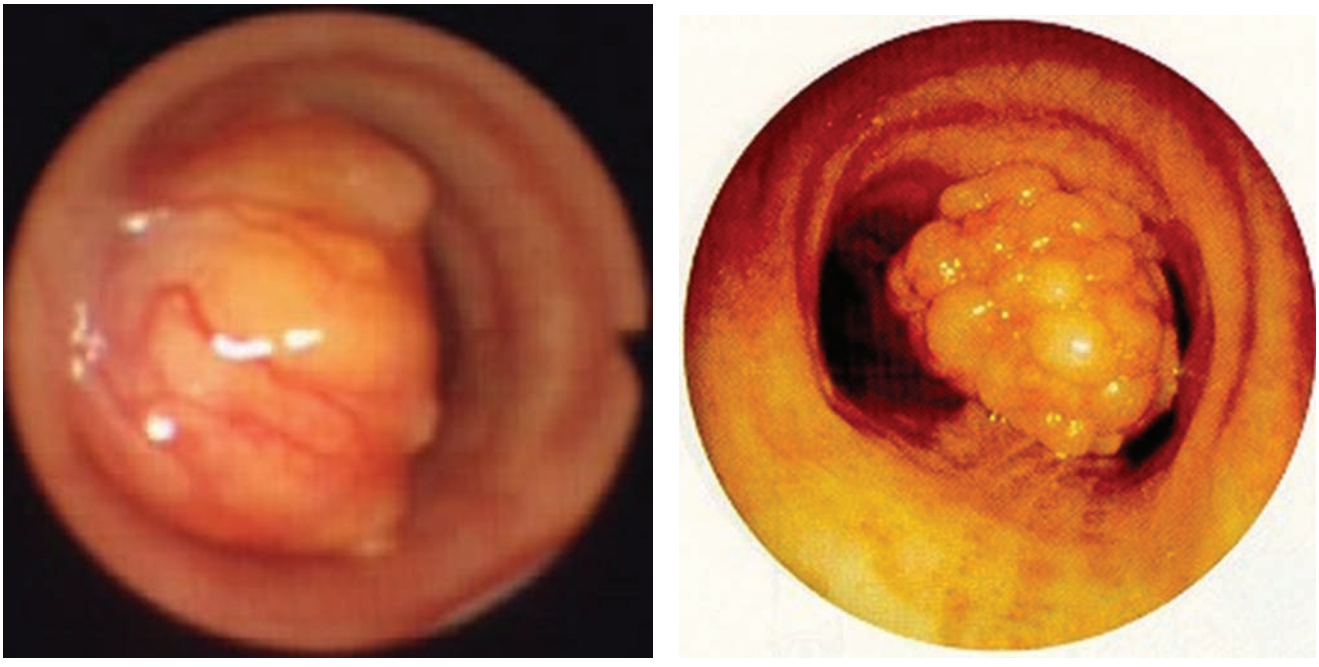
oropharynx is the anterior tonsillar pillar or tonsil. Symptoms of these cancers include hoarseness, hemoptysis, sore throat, dysphagia, referred otalgia due to cranial nerve involvement, and trismus due to pterygoid muscle involvement. Life-threatening UAO may be seen. Patients who have had head and neck surgery for cancer have a very high prevalence of obstructive sleep apnea.

Tracheal Cancer

Tumors that originate in the trachea are uncommon and make up only about 2% of all tumors that form in the upper airway. Although rare, when they do occur, approximately 80% of tracheal tumors are malignant. Primary tracheal cancers are rare, with a reported incidence of 0.1 per 100,000 per year.[26] Dyspnea, cough, wheeze, and stridor are frequent presenting symptoms. Squamous cell carcinoma (SCC) is the predominant histology (45%), followed by adenoid cystic carcinoma (ACC) (16%).[27] SCC is almost always associated with cigarette smoking. These tumors grow quickly, and in nearly half of cases they are too large to be removed by the time they are discovered. Hemoptysis is often another presenting symptom. ACC grows much more slowly and has not been found to be related to smoking. Men and women have the same risk for this type of cancer, and it is commonly diagnosed around the age of 40 years. Hemoptysis is an uncommon presentation for ACC. Surgery remains the most effective management. Emergency treatment with procedures to recanalize the airway, including airway stenting, may be necessary pending definitive surgery. Postoperative radiation therapy appears useful for primary tracheal malignancies, particularly when surgical margins are positive. Palliative radiation is used for local control when surgery is contraindicated. Five-year survival is highly dependent on resectability. For resectable adenoid cystic and squamous cell carcinomas, survival rates are reported at 52% and 39%, respectively, whereas in unresectable disease these rates are 39% and 7%, respectively.[28] Late recurrence of ACC is possible due to early perineural infiltration. Favorable prognostic factors include negative airway margins at the time of resection and adenoid cystic histology.

More commonly, malignancies found in the trachea result from direct extension of primary lung cancer or regional extension to the lymph nodes (Fig. 47-14). Metastases to central airways from nonpulmonary malignancies are uncommon, although endobronchial metastases from breast, colorectal, renal, ovarian, thyroid, uterine, testicular, nasopharyngeal, and adrenal carcinomas, as well as sarcomas, melanomas, and plasmacytomas have been described. In an autopsy series of more than 1300 patients with solid tumors, metastatic disease to central airways occurred in 2%.[29] The mediastinal lymph nodes are also a common site for disease involvement from hematologic malignancies such as lymphoma.

Nonmalignant tracheal tumors include papillomas, chondromas, and hemangiomas (Fig. 47-15). Chondromas are the most common



A **B**

Figure 47-15 Nonmalignant tracheal tumors. **A.** Hamartoma. **B.** Papillomatosis.

C

Figure 47-15 *(Continued)* **C.** Chondromas.

type of benign tracheal tumor. They are formed from the cartilage rings of the trachea and have the potential to become malignant over time. Recurrent respiratory papillomatosis in adults, caused by human papillomavirus types 6 or 11 (or, much less commonly, types 16 or 18) may result in UAO and death. Although the larynx is most commonly affected (Fig. 47-16), the tracheobronchial tree may be involved, with a predilection toward areas with prior mucosal injury, including tracheostomy sites and tracheal injuries. Lesions tend to progress down through the tracheobronchial tree. Pulmonary parenchymal involvement is rare, but it may be severe, and bronchiectasis, pulmonary nodules, and abscess formation may occur. Malignant transformation also is possible. The course

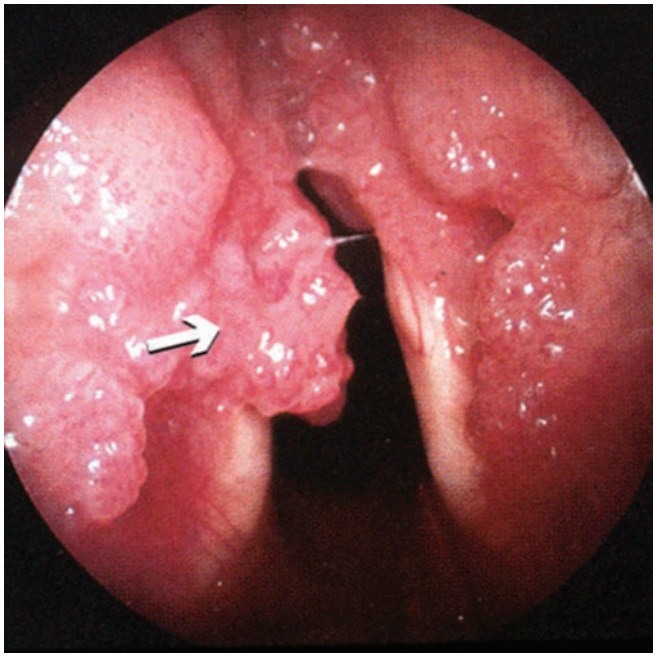


Figure 47-16 Recurrent laryngeal papillomatosis *(arrow)*.

of the disease is difficult to predict. Recurrent endoscopic interventions (debulking), with attendant risk of airway stenosis, are often required. It is important to minimize intervention unless clinically relevant obstruction exists due to concerns regarding extension secondary to airway injury. No controlled trials on the role of antiviral therapy have been conducted. Available data suggest beneficial effects of intralesional cidofovir, and it is recommended to treat with cidofovir when debulking is required at least every 2 to 3 months.[30] Bevacizumab also has been shown to have benefit when used in combination with debulking.[31] Favorable effects also have been reported with the use of interferon alpha, although the agent is not often used due to systemic side effects. Chemotherapy, radiation therapy, and targeted surgical resection are utilized for confirmed malignant transformation. The HPV vaccine Gardasil has shown promising results for secondary prevention of RPP as well as disease control.[32]

Laryngeal and Tracheal Stenosis

Common causes of laryngeal and tracheal stenosis include prior intubation and tracheotomy. However, many other causes have been reported.

Postintubation and Posttracheotomy Stenosis (PITS and PTTS)

Endotracheal intubation, tracheostomy, and prior laryngotracheal instrumentation account for most cases of laryngotracheal stenosis. The reported frequencies of tracheal stenosis following tracheostomy or laryngotracheal intubation vary widely, but recent large retrospective studies suggest that the incidence of clinically significant stenosis is low (0%–1.8%) (Figs. 47-17 and 47-18).[33,34] Patient demographics associated with an increased incidence of PITS/PTTS include female sex (75%), obesity (66%), diabetes mellitus (35.4%), hypertension (51.6%), cardiovascular disease (45.1%), and current smoker status (38.7%).[35] The incidence of symptomatic tracheal stenosis following percutaneous tracheostomy is comparable to the incidence that occurs after open techniques.[36]

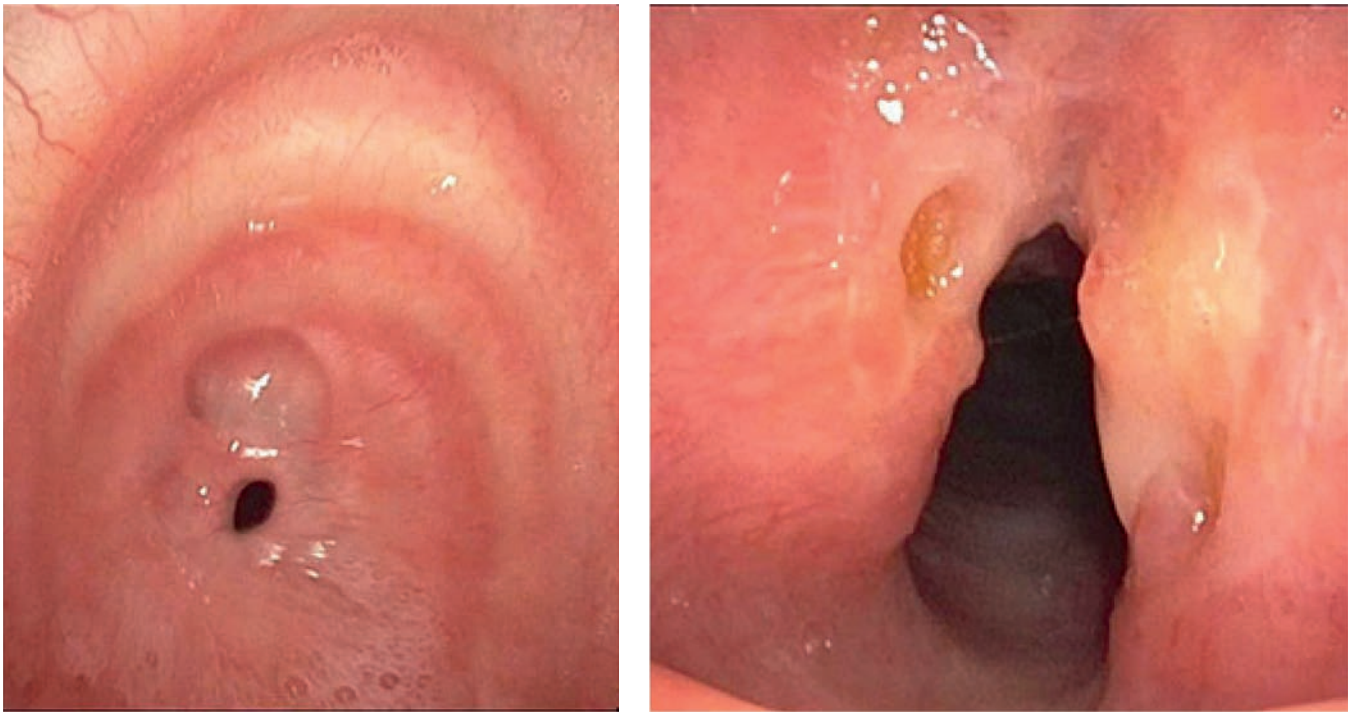
Tracheal stenosis in the region of the tube cuff is related to pressure-induced ischemic injury of the mucosa and cartilage, and its risk can be minimized by use of large-volume, low-pressure cuffs. The duration of translaryngeal intubation also affects the frequency and severity of laryngotracheal stenosis.[37]

Stenosis following tracheostomy may be above the stoma, at the level of the stoma, at the cuff site, or at the tip of the cannula. Damage to the cartilage above the stoma is a common cause of tracheal stenosis after tracheostomy. In addition to ischemic mucosal injury and ischemic chondritis, anterior and lateral tracheal wall damage, with "buckling in" fractures of the cartilage, is an important factor. The fractures can be minimized by avoiding excessive pressure on the cartilage during the procedure, selecting the appropriate size and length of the tracheostomy tube, avoiding infection, and using the lowest possible cuff pressure. Obesity (BMI >30) is a significant independent risk factor for development of posttracheostomy stenosis.[38]

Treatment of PITS/PTTS consists of bronchoscopic dilation, stent placement, and/or surgery for appropriate candidates. Electrocautery needle knife incisions coupled with balloon dilation has been shown to be effective.[39] Silicone stent placement is preferred over metallic stent. Stent placement is most effective when done within 6 months of stenosis and can be successfully removed without restenosis in 40% of patients.[40]

Other Causes of Tracheal Stenosis

Granulomatosis with polyangiitis may present with significant subglottic stenosis, a complication reported in 16% to 23% of patients.[41] Subglottic stenosis may be the only manifestation of granulomatosis



A B

Figure 47-17 Posttracheostomy stenosis. **A.** Weblike simple stenosis. **B.** A-framed stenosis.

with polyangiitis and may have a clinical course distinct from other manifestations of the disease. Endoscopic biopsy of suspected sites of involvement is positive in only 5% to 15% of cases.

Sarcoidosis may be associated with granulomatous infiltration and obstruction of the upper airways (Fig. 47-19). Laryngeal involvement is more common, but tracheal stenosis has been described. Radiographs may show diffuse tracheal stenosis, which progresses despite corticosteroid therapy. Bronchoscopy may reveal extensive tracheal narrowing.

Pulmonary amyloidosis includes tracheobronchial manifestations. The chest roentgenogram may show diffuse narrowing and wall thickening involving a long tracheal segment. Involvement is diffuse and circumferential, often with ossification of the amyloid deposits (Fig. 47-20). Bronchoscopy demonstrates multiple plaques on tracheal walls or localized tumor-like masses.

Relapsing polychondritis is a rare systemic disease characterized by recurrent episodes of inflammation of cartilaginous structures. Respiratory manifestations are often severe and may be life-threatening. Inflammation occurs in all cartilage types, including the elastic cartilage of the ears and nose, hyaline cartilage of all peripheral joints, and axial fibrocartilage. The most common presenting symptom is pain in the external ear due to auricular chondritis. Respiratory tract involvement may develop years after the first occurrence of auricular chondritis. Symptoms include hoarseness,

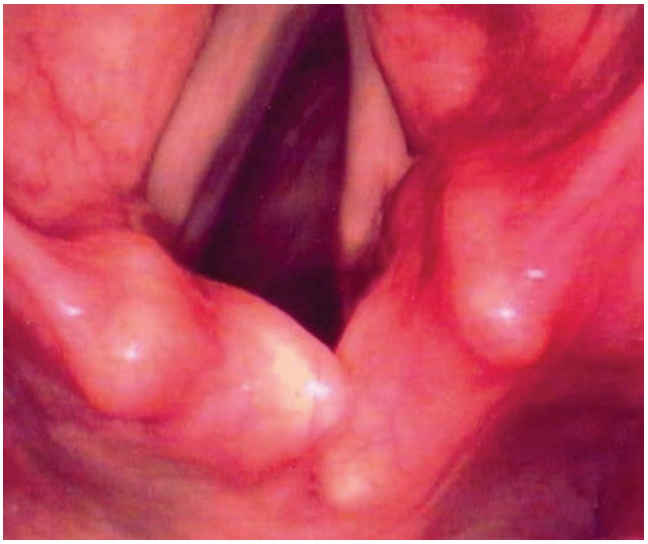


Figure 47-18 Postintubation trauma.

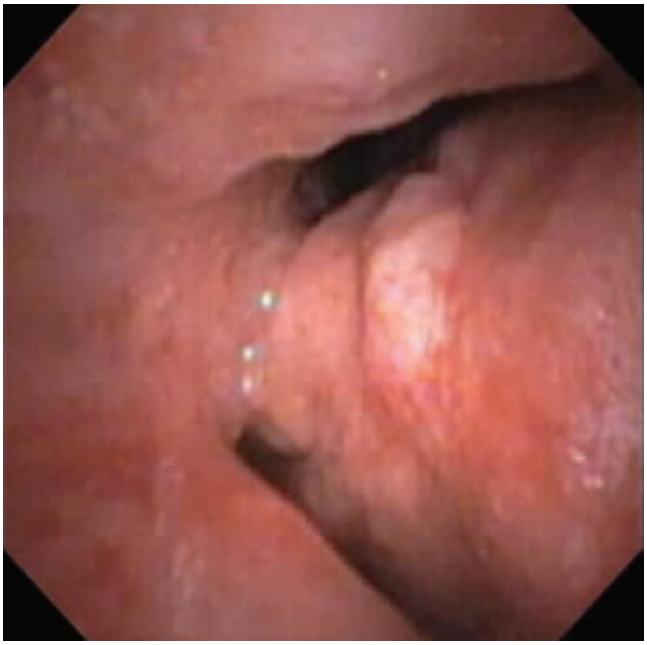


Figure 47-19 Airway sarcoid.

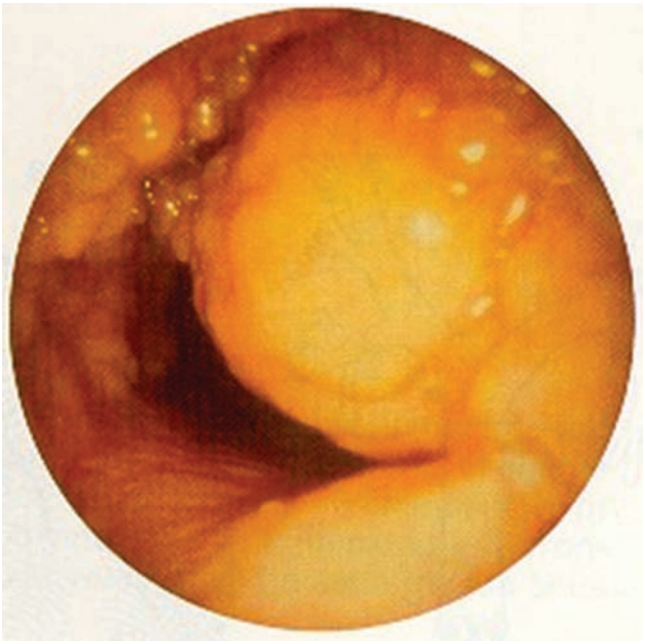


Figure 47-20 Amyloid pseudotumor.

aphonia, and choking. Tenderness over the thyroid and laryngeal cartilages may be present. When the trachea is involved, endoscopic examination shows inflammation and stenosis. CT demonstrates major airway collapse caused by destruction of cartilaginous rings or airway narrowing due to inflammatory edema and fibrosis. CT findings also include diffuse, smooth thickening of the trachea and proximal bronchi; thickened, densely calcified cartilaginous rings; tracheal wall nodularity; and diffuse narrowing of the tracheobronchial lumen. The posterior tracheal membrane is spared.[42]

Tracheopathia osteoplastica is a rare, benign disease of the trachea and major bronchi in which cartilaginous or osseous nodules project into the airway lumen, often causing considerable airway deformity. The posterior membranous portion of the tracheal wall is spared. The disorder may begin just below the larynx, but most often it affects the lower two-thirds of the trachea. Extension into the proximal portions of the major bronchi may be noted. The condition usually occurs over the age of 50 years and may cause severe airflow obstruction. Its etiology is unknown.

On rare occasions, inflammatory bowel disease produces tracheobronchial stenosis and severe airflow obstruction. The associated airway mucosal inflammation may be steroid responsive early in the course of illness. If fibrosis ensues, medical management has limited success.

Idiopathic laryngotracheal stenosis is a diagnosis of exclusion in the absence of a clear, underlying etiology. It predominantly affects women in the fifth decade of life.[43] GERD is commonly concurrently diagnosed. Since most affected patients are females, a hormonal etiology has been proposed. An imbalance in estrogen and progesterone receptor expression has been demonstrated in surgical specimens of resected stenosis.[44] Treatment includes bronchoscopic dilation. Steroid injection and aggressive medical management with proton pump inhibitors and inhaled corticosteroids may prolong the interval between required procedures.[45,46]

In addition to medical management, repeated rigid and flexible bronchoscopy-based interventions aimed at reestablishing airway patency may be necessary, particularly in those who are not considered to be surgical candidates or in those with simple weblike stenosis. In cases of weblike stenosis, radial incisions sparing the posterior membrane are made and then the airway is dilated, using either a balloon or the rigid bronchoscope barrel. With respect to more complex stenosis, endoscopic intervention is generally considered a bridge in patients considered good operative candidates with potentially resectable disease. Unfortunately resection and re-anastomosis is limited by the length of trachea to be resected.[47,48] A multidisciplinary approach incorporating medical and surgical specialists as described by Brichet is utilized in many centers.[49]

Tracheomalacia

Tracheomalacia refers to loss of tracheal rigidity and resulting susceptibility to collapse. Tracheomalacia may be diffuse or localized to a tracheal segment. The affected portion may be intrathoracic, in which airway obstruction is accentuated during expiration. Less common is extrathoracic obstruction resulting from cervical malacia, in which airway obstruction is most marked during inspiration. Tracheobronchomalacia is the term used to describe the condition when the mainstem bronchi are involved.

Tracheomalacia in adults may be classified as congenital or acquired. The congenital form, described more extensively in children, is related to a variety of congenital disorders and associated syndromes. The disorder may persist into adult life and is referred to as "idiopathic giant trachea," "tracheomegaly," or the "Mounier-Kuhn syndrome" (Fig. 47-21). Bronchiectasis and recurrent respiratory infections are common. Tracheal diverticuli have been reported in more advanced disease. Although atrophy of the longitudinal elastic fibers and muscularis layer has been described, the etiology of these changes is unclear. The diagnosis is made when the diameters of the trachea or right or left mainstem bronchi exceed the upper limits of normal by 3 or more standard deviations. Patients with Mounier-Kuhn syndrome may benefit symptomatically from airway stenting and tracheobronchoplasty.[50]

Acquired or secondary tracheomalacia in adults may be related to a variety of conditions.[51] Tracheostomy and endotracheal intubation are probably the most common etiologies. Usually, limited, focal weakness of the trachea and excessive dynamic airway collapse are present. Tracheomalacia may be caused by conditions that are associated with chronic pressure on the tracheal wall, inflammation of the cartilaginous support or mucosa, interference with tracheal

Figure 47-21 Bronchoscopic view of tracheomegaly or Mounier-Kuhn syndrome.

blood flow, or chronic infection. Traumatic injury to the central airways or surgical interventions also may lead to tracheomalacia.

Symptoms of tracheomalacia include dyspnea, a seal-barking paroxysmal cough, recurrent pulmonary infections, and respiratory failure in a small subset.[52] Many of these patients will also have decreased exercise tolerance and, in severe cases, impaired quality of life.

Tracheomalacia is defined as a reduction in cross-sectional area of greater than 50% during expiration on CT imaging. However, this cut point may not be discriminatory enough to detect clinically significant tracheomalacia.[53] Bronchoscopic visualization can confirm significant narrowing of the tracheal lumen during regular, forced expiration.

Application of continuous positive airway pressure (CPAP) has been reported to be as beneficial as pneumatic splint for the airways. Tracheobronchoplasty may be useful in selected patients and has been shown to improve quality of life and exercise tolerance.[52] Airway stents can be used to identify patients that may benefit from surgical airway stabilization.[54,55] In select patients who are not surgical candidates, but who derive both symptomatic and objective benefit from airway stents, silicone stents may be used for long-term management with appropriate monitoring for complications. Optimal medical management includes treatment of associated infections and of distal airway disease.

Extrinsic Compression of the Central Airway

The upper airway is subject to extrinsic compression by a variety of pathologies that involve adjacent structures. The compression may affect the intrathoracic trachea or extrathoracic trachea and upper airway.

Mediastinal Masses and Lymphadenopathy

Rarely, mediastinal masses present with serious limitation to airflow that develop either acutely or indolently. Common symptoms include chest pain, fever, dyspnea, and cough. The anterosuperior compartment is the most common site of mediastinal malignancies. Thymic neoplasms and lymphoma are the most common malignancies, followed by neurogenic tumors and teratomas. Both Hodgkin and non-Hodgkin lymphomas may be manifested by severe respiratory compromise due to airway compression. A similar syndrome may be due to a metastatic tumor in the mediastinal lymph nodes arising from bronchogenic or other carcinomas.

Patients with large mediastinal masses present a challenge during the perioperative period because of the potential for development of acute UAO and other respiratory complications. In adults, complete airway obstruction during induction of anesthesia is rare. Serious pulmonary complications develop intra- and postoperatively in about 4% and 7% of patients, respectively. Complications may occur while the patient is placed in the supine position, during induction, or following extubation. Patients with severe symptoms, including stridor, and those with greater than 50% airway obstruction, appear to be at high risk for respiratory complications; asymptomatic patients are at significantly less risk. Patients with reduced peak expiratory flow and mixed obstructive–restrictive patterns on pulmonary function testing also appear to be at increased risk for postoperative complications.

Middle mediastinal masses include benign cysts that are bronchogenic, enterogenous (duplication), pericardial, pleural, and thymic in origin. Most bronchogenic cysts are asymptomatic. However, some evoke cough, chest pain, and dyspnea. Severe respiratory distress and compressive symptoms can occur. Usually, cyst contents appear to have the density of water on CT or MRI. Mucoid contents may give the impression of solid appearance on CT. Surgical resection and transthoracic or transbronchial drainage are options for management. Surgical intervention appears to be the preferred treatment in patients who are symptomatic. The role of interventions, including surgery, in asymptomatic patients is controversial. Enterogenous cysts are usually removed surgically.

Enlarged mediastinal lymph nodes that compress the airway may arise from infectious and noninfectious benign etiologies. One notable example is fibrosing mediastinitis, defined as the presence of excessive mediastinal fibrous tissue that tends to invade and destroy normal structures. The entity is thought to represent a reaction to an infectious granulomatous disease, especially histoplasmosis. The incidence in populations exposed to histoplasmosis remains low. Constriction of the central airways and vessels and the resulting cardiopulmonary limitations may develop several years after the initial infection. Hemoptysis is common, as are cough, dyspnea, and chest pain. CT imaging shows mediastinal fibrosis, calcification, and compression of mediastinal structures. Bronchoscopic findings include concentric airway narrowing and mucosal edema with hyperemia. Unfortunately, hemoptysis tends to be recurrent, and the disease does not respond to corticosteroids or antifungal agents. Surgical intervention is generally ineffective and may be hazardous.

Neck- and Thyroid-Related Causes

Retrosternal extension of a diffuse goiter may cause extrathoracic or intrathoracic airway obstruction. Patients with substernal goiter may have obstructive respiratory and gastrointestinal symptoms.[56] Orthopnea is prevalent when the goiter is intrathoracic and may be enhanced by obesity. Flow–volume loops show evidence of UAO in one-third of patients. Lack of correlation has been reported between symptomatic obstruction and CT findings.[57]

Laryngoceles and saccular cysts, which are abnormal dilatations of the laryngeal saccule (ventricle), are uncommon. Saccular cysts usually are filled with mucus. Laryngoceles communicate with the laryngeal lumen, resulting in air-filled structures noted on radiographic studies. Laryngoceles may be internal (i.e., confined to the larynx), external (i.e., extending into the thyrohyoid membrane superiorly), or combined. Most are asymptomatic. Hoarseness, dysphagia, pain, or signs of airway obstruction or infection may occur. A neck mass during the Valsalva maneuver may be detectable. Pyocele formation (i.e., infection in the laryngocele) may result in airway obstruction, aspiration pneumonia, or infection of the lateral pharyngeal space. The incidence of laryngeal carcinoma in association with laryngoceles makes close evaluation necessary. Endoscopic and surgical approaches may be employed in management.

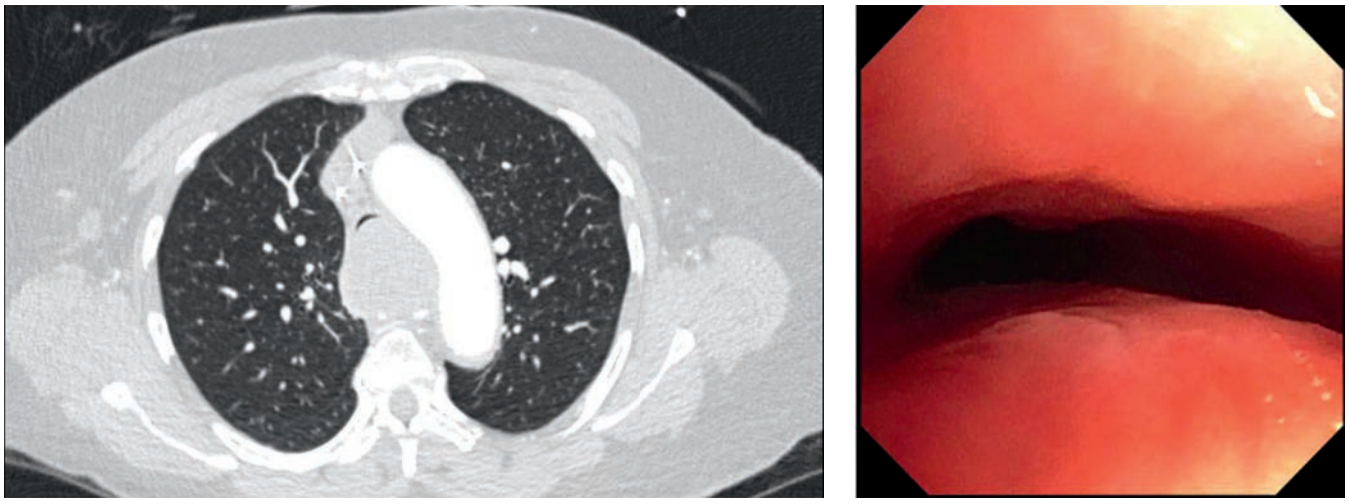
Parathyroid cysts may be located in the neck or mediastinum. Fifty percent are accompanied by clinical hyperparathyroidism. Paroxysmal symptoms of airway obstruction can develop. Surgical excision is the treatment of choice; results are generally good.

Cervical osteophytes, common in the elderly, are related to either degenerative spinal arthritis or more generalized idiopathic skeletal hyperostosis; the osteophytes may be associated with dysphagia. In addition, airway narrowing and ulcerations due to osteophytes have been reported. The airway compression may make even elective endotracheal intubation difficult, despite adequate preoperative evaluation.

Finally, significant upper airway compression may arise from cervical lymph node involvement with infectious or malignant disorders, hematomas or pseudoaneurysms (related to trauma, surgical interventions, central line placement, or coagulation abnormalities), abscess formation, or other expanding lesions in the soft tissue of the neck.

Diseases of the Esophagus

Involvement of the trachea, glottis, or vocal cords by advanced esophageal cancer is common and associated with a poor prognosis; estimated 1-year survival is less than 10%. Airway obstruction



A B

Figure 47-22 Distal tracheal compression due to esophageal tumor. **A.** CT view. **B.** Bronchoscopic view.

requiring stent placement is associated with a median survival of 1 to 4 months after the placement (Fig. 47-22A, B).[58] Tracheal obstruction may develop if an esophageal stent is placed in the setting of significant tracheal compromise. Development of tracheoesophageal fistula represents a devastating complication.

Placement of stents simultaneously in the trachea and esophagus is effective palliation for a tracheoesophageal fistula.[59] If such double stenting is anticipated for a fistula or for simultaneous esophageal and tracheal obstructions, the tracheal stent is placed first to ensure patency of the airway, followed by the esophageal stent. Palliative external or local radiation therapy, chemotherapy, or other treatment modalities (e.g., PDT) may be effective with or without accompanying airway interventions. The risk of esophageal disruption and rupture should be considered if stenting is performed after these local measures are employed.

Achalasia may cause a variety of pulmonary complications, including cough, aspiration with pneumonia or abscess formation, and rarely UAO. Tracheal compression by a dilated megaesophagus is the usual etiology. Ensuring patency of the airway and decompressing the esophagus are necessary in urgent management.

Vascular Abnormalities

Vascular rings, defined as anomalies of the aortic arch or its branches that compress the trachea or esophagus, are rare in adults (incidence <0.2%). Respiratory symptoms are common. Right-sided aortic arch occurs in less than 0.1% in adults and may be associated with complete vascular rings, while double aortic arch and right-sided aortic arch with aberrant left subclavian artery appear to be the most common etiologies of vascular rings in adults.

The right-sided aortic arch usually crosses over the right main-stem bronchus and descends on either the right or the left side. The vascular ring is usually completed by the ligamentum arteriosum arising from the descending aorta, an aberrant left subclavian artery, or an aortic diverticulum. With a double aortic arch, the left arch crosses over the left main-stem bronchus and joins the descending aorta to complete the ring; the ligamentum arteriosum does not contribute to the vascular ring. Symptoms, resulting from malacia of the compressed airway and resultant dynamic airway obstruction, may be misdiagnosed as exercise-induced asthma. An increase in aortic diameter due to rising blood pressure during exercise, intravenous fluid administration, or anatomic changes with aging may contribute to symptoms. Surgical intervention is indicated in symptomatic patients.

Pulmonary artery sling with anomalous origin of the left pulmonary artery from the right pulmonary artery is very rare in adults. In neonates, the condition is symptomatic and can be fatal without surgical intervention. However, in adults the condition is usually diagnosed incidentally on imaging a patient who has no significant symptoms. This disorder may be associated with a complete tracheal ring, forming the "sling-ring" complex. This condition may present with a right paratracheal mass noted on the chest radiograph.

Compression of the trachea by large aortic or innominate artery aneurysms or pseudoaneurysms may occur and complicate management in the perioperative period. Surgical repair is indicated to relieve symptoms.

■ Foreign Body Aspiration

Foreign body aspiration, more common in children than adults (in whom the peak incidence is in the sixth decade), is usually recognized from the patient's history. Foreign bodies commonly lodge in the bronchi after migrating through the trachea. In adults, food products are the most commonly aspirated material. The penetration syndrome, defined as the sudden onset of choking and intractable cough after aspirating a foreign body, with or without vomiting, is often followed by persistent cough, fever, chest pain, dyspnea, and wheezing. Impairment of the normal protective airway mechanisms is common; among the frequent associations are neurologic disorders, trauma with loss of consciousness, sedative or alcohol use, poor dentition, and advanced age. Emergency measures, entailing a food extractor or the Heimlich maneuver, can be life-saving. Flexible bronchoscopy is usually successful in removing foreign bodies, although backup rigid bronchoscopy should be available and is preferred as the primary procedure at some centers. A complicating chemical bronchitis from aspiration of vegetables or nuts may affect visualization and management of the foreign body. Certain nut oils also cause a significant foreign body mucosal reaction, resulting in the development of granulation tissue that may be misdiagnosed as endobronchial tumor.

■ Trauma

A variety of traumatic injuries may eventuate in UAO.

Facial Trauma

Emergency access to the airway is necessary in up to 6% of cases of facial trauma complicating motor vehicle accidents and other causes

of crush injuries. If intubation is difficult or impossible due to the injury or related airway obstruction, emergency cricothyroidotomy or tracheostomy must be considered.

Laryngotracheal Injuries

Blunt and penetrating injuries to the laryngotracheal airway are rare. Without a high index of suspicion, clinicians may miss the diagnosis. The incidence of penetrating injuries appears to be increasing.

Stridor, wheezing, dysphonia, hemoptysis, and general neurologic deficits are common. Cervical crepitus and subcutaneous emphysema also may be present. Cervical ecchymoses and hematomas, pneumomediastinum, and pneumothorax should prompt consideration of a laryngotracheal injury.

Management includes prompt securing of the airway, but blind endotracheal intubation should be avoided, since it carries the risk of complete airway obstruction. Some experts recommend tracheostomy as the primary airway management strategy. Awake fiberoptic intubation can be useful. Flexible fiberoptic laryngoscopy, rigid or flexible bronchoscopy, and CT imaging may be helpful in assessing the degree of injury. Unfortunately, the mortality of laryngotracheal injuries remains high.[60] Thoracic injuries and closed head injuries are commonly associated pathologies that can influence management and prognosis.

Inhalation Injuries

Thermal and chemical injuries to the upper respiratory tract may lead to serious consequences, including airway obstruction. The mortality rate increases significantly when burns are accompanied by inhalational injury.[61] Symptoms can be delayed in becoming manifest, making early recognition and intervention vital in the management of patients with inhalational injuries. The presence of cough, dyspnea, hoarseness, or loss of consciousness, or the findings of singed nasal hairs, carbonaceous sputum, or burns involving the face indicate a high likelihood of inhalation injury.

Early flexible bronchoscopy remains important in evaluation and management of patients with inhalation injuries, enabling the assessment of the extent and severity of the injury, procurement of samples for bacteriologic studies, and bronchoscopic intubation, as necessary.[62] Translaryngeal intubation is the standard method of securing the airway in inhalation injury; early tracheostomy is used in some centers. A role for prophylactic corticosteroids or antibiotics is currently not supported by published reports. Significant tracheal stenosis may develop in patients who survive the initial insult especially in those with larger total body surface area involved, higher grade of inhalational injury, and longer duration of intubation.[63]

Endotracheal Tube–Related Trauma

Postextubation stridor due to glottic edema, laryngospasm, or laryngotracheal stenosis is a serious event. Risk factors include duration of intubation, emergency intubation, and height/endotracheal tube size ratio.[37] Reintubation rates for UAO due to endotracheal tube–related trauma in critically ill patients have been reported to range from 4% to 33%. An "acceptable" rate is considered to be 5% to 15%. Clinical practice guidelines support the use of preextubation glucocorticoids in patients deemed high risk for post extubation stridor who have failed a cuff leak test.[64]

Translaryngeal intubation may also produce either unilateral or bilateral vocal cord paralysis. Affected patients may present with hoarseness or airway obstruction. Findings may occur immediately after extubation or be delayed. Prolonged intubation, use of a large endotracheal tube, placement of the tube cuff close to the vocal cords, or use of excessive cuff pressure are risk factors. The condition usually resolves spontaneously within 10 weeks.

Vocal cord (contact) granuloma may develop 4 to 6 weeks after intubation. Symptoms include prolonged hoarseness, exertional dyspnea, and stridor. Management, using antireflux medications, inhaled and systemic corticosteroids, antibiotics, botulinum toxin injection, speech therapy, smoking cessation, and rest of the voice are usually successful. Surgical intervention is reserved for cases that fail conservative management.

On occasion, dislocation of the arytenoid cartilages occurs during intubation. Rheumatoid arthritis that affects the cricoarytenoid cartilage is a risk factor for this condition. Rigid bronchoscopy or surgical interventions may be needed to reduce the dislocation. Other disorders that may cause complications during intubation include hyperostosis of the cervical spine due to ankylosing spondylitis and cricoarytenoid joint disease due to systemic lupus erythematosus.

Neuromuscular Disorders

Neuromuscular disorders may affect the bulbar muscles, many of which surround the upper airway (Fig. 47-23). When this occurs, resistance to airflow is increased, and the flow–volume loop often shows an inspiratory flow plateau typical of variable extrathoracic UAO. In addition, a pattern of flow oscillations during inspiration ("sawtooth pattern") may be seen.[65] The abnormal flow pattern, first noted in patients with sleep apnea,[66] is commonly seen in extrapyramidal disorders, myasthenia gravis, and motor neuron disease; it may also be seen in patients who have functional stridor and wheezing (see "Vocal Cord Dysfunction," below). In extrapyramidal disorders, the flow oscillations correspond to vocal cord tremor. In motor neuron diseases, muscle denervation causes irregular muscle fasciculation, resulting in tremor of upper airway muscles.

Upper airway symptoms may be seen in Shy–Drager syndrome with extrapyramidal involvement and in Parkinson disease.[67,68] Patients may present with symptoms of chronic dyspnea or with stridor and respiratory failure relieved by endotracheal intubation or tracheostomy. Bilateral vocal cord paralysis may also occur with familial bulbar spinal muscle atrophy, postpoliomyelitis syndrome, Parkinson disease, multiple sclerosis, acute poliomyelitis, amyotrophic lateral sclerosis, Guillain–Barré syndrome, brain stem stroke, and large cerebral hemisphere stroke. Dystonic extrapyramidal reactions due to neuroleptic medications (e.g., haloperidol) may

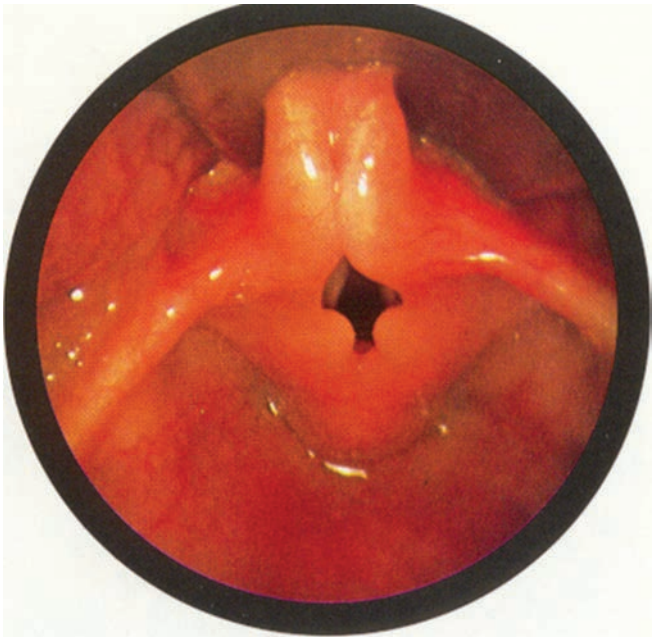


Figure 47-23 Laryngomalacia.

Figure 47-24 Bilateral vocal cord paralysis.

cause significant UAO. The usual reactions to these medications are akathisia, dyskinesia, dysarthria, and dystonic reactions such as torticollis. Laryngeal–pharyngeal dystonia may cause severe upper airway dysfunction, and if not reversed, symptoms can last for days or lead to respiratory arrest.

Bilateral Vocal Cord Paralysis

Bilateral vocal cord paralysis is a serious condition resulting in nocturnal stridor, oxygen desaturation, sleep disruption, or, in extreme cases, acute respiratory failure (Fig. 47-24). Thyroidectomy is the most common cause of bilateral recurrent laryngeal nerve injury, with its consequent bilateral vocal cord paralysis.[69] As mentioned earlier, bilateral vocal cord paralysis may occur with numerous neurologic conditions. Nonneurologic causes include endotracheal intubation injury, laryngeal trauma, infection, and thoracic aortic aneurysm. Bilateral vocal cord paralysis results in abnormalities of inspiratory flow and a distinctive flow–volume loop.

Spontaneous recovery of vocal cord motility is rare after surgical injury of the recurrent laryngeal nerve. For those patients showing no spontaneous functional recovery, several surgical techniques to achieve a glottic space enlargement are available. CO_2 laser endoscopic surgery has led to shorter hospitalization and higher patient compliance.[70,71] Endoscopic surgeries include arytenoidectomy with or without posterior true and false cord cordectomy.[72] Surgeries aim to improve breathing with minimal deglutition impairment and improve quality of voice. Restenosis of the glottis space is the main problem that leads patients to reoperations.

Vocal Cord Dysfunction

The glottis plays an active role in adjusting airflow, both voluntarily and through reflex control from laryngeal and lung receptors. During a normal respiratory cycle, the vocal folds widely abduct during inspiration and slightly adduct during expiration. Occasionally, the glottis can become dysfunctional in the absence of organic disease. The disorder, most commonly called vocal cord dysfunction or paradoxical vocal fold motion, is characterized by paradoxical closure of the vocal cords intermittently during inspiration. The glottis angle formed by the true vocal folds can become acute and cause airflow obstruction during inspiration and/or expiration. The mechanism is unknown but likely multifactorial. Laryngeal hypersensitivity has been associated with reflux, upper respiratory tract infections, postnasal drip, irritants, and exercise, as well as psychogenic factors.[73]

Signs and symptoms of vocal cord dysfunction resemble those of laryngeal edema, laryngospasm, vocal cord paralysis, or asthma.[74] Wheezing or stridor and shortness of breath are typical and are often so dramatic that they suggest acute asphyxia and respiratory failure. Intubation and other emergency measures are used frequently. However, symptoms are often brief and may remit without intervention. Distinguishing physical examination findings include tracheal localization of wheezing or stridor with absent peripheral airway expiratory wheezing; however, it should be understood that this might also occur during severe asthma attacks.

On pulmonary function testing, patients with vocal cord dysfunction typically have normal spirometry without evidence of obstructive ventilatory defect.[75] Some may demonstrate a pattern of variable extrathoracic airway obstruction, resulting in an increase in the ratio of $FEF_{50\%}$ to $FIF_{50\%}$. Some patients show a pattern of "sawtoothing," or fluttering of the inspiratory limb of the flow–volume loop, representing fluctuations in the abnormal cord motion (Fig. 47-25). Often, attempts to perform the flow–volume loop maneuver generate variable results from test to test. A normal alveolar–arterial oxygen gradient and absence of bronchial hyperresponsiveness are other clues to the diagnosis.

The diagnosis of vocal cord dysfunction is confirmed during direct visualization of the vocal cords during an attack. The anterior two-thirds of the vocal folds adduct and give the presence of a posterior diamond-shaped chink in the folds during inspiration.[76]

The true prevalence of the disorder is unknown because it is often mistakenly diagnosed as asthma, and many patients in fact have both vocal cord dysfunction and asthma.[77] Patients without asthma are predominantly women who have been misdiagnosed as having asthma for an average of 5 years. Typically, patients have been treated with large doses of oral corticosteroids and have experienced frequent emergency room visits, hospitalizations, and endotracheal intubations.

Psychiatric disorders are common in these patients.[78,79] Major psychiatric disorders, personality disorders, and sexual and physical

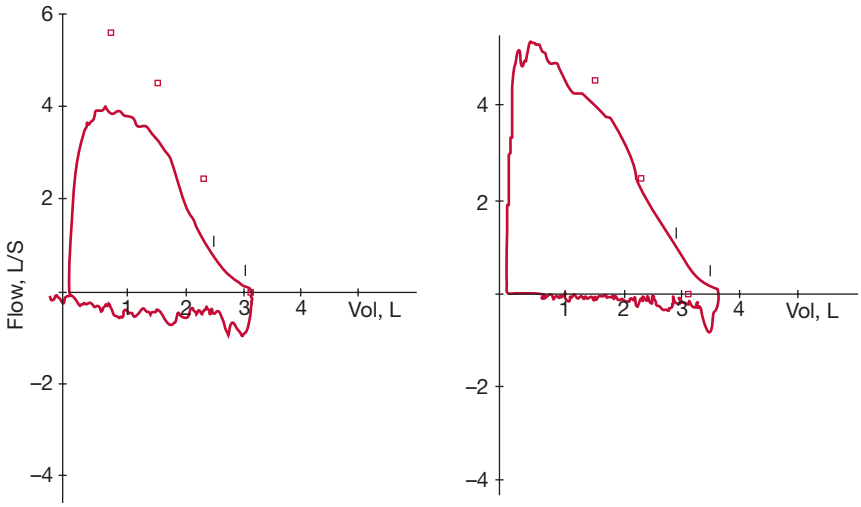


Figure 47-25 Variable extrathoracic obstruction due to vocal cord dysfunction. Two consecutive flow–volume loops from a young woman with inspiratory stridor. Variable effort accounts for the differences in configuration. $FEF_{50\%}/FIF_{50\%}$ in each is very high. The inspiratory loop is flat and demonstrates a sawtooth pattern. This pattern has also been associated with sleep apnea syndrome and various neuromuscular disorders.

abuse are commonly uncovered. Whereas many patients are unaware of their self-induced wheeze or stridor, others appear to derive secondary gain from their symptoms and manifest factitious illness. A high index of suspicion is warranted when the adventitious sounds are loudest over the neck in a patient who presents with wheezing, stridor, or both. Despite their respiratory distress, patients often have little difficulty completing full sentences and can hold their breath; the laryngeal-induced sounds disappear during a panting maneuver.

Treatment includes discussion of the diagnosis with the patient, discontinuation of unnecessary medications, and referral to a speech therapist or psychotherapist. The response to bronchodilator therapy is usually poor. Use of continuous positive airway pressure or administration of an inhaled helium–oxygen mixture may alleviate symptoms during an acute attack.[80] Botulinum toxin injection may be used in refractory cases.[81]

Angioedema

Angioedema is characterized by well-demarcated swelling of the face, lips, tongue, and mucus membranes of the nose, mouth, and throat. When the larynx is involved, UAO may occur and can be fatal. In most instances, the cause of angioedema is unclear; prior exposure to common allergens, such as drugs, chemical additives, and insect bites, should be suspected.

Counterintuitively, the most common causes of angioedema are not IgE initiated. They include reactions to histamine-releasing drugs, such as narcotics and radiocontrast materials, aspirin, other nonsteroidal anti-inflammatory drugs, and angiotensin-converting enzyme inhibitors. Hereditary angioedema, a rare cause of UAO, is an autosomal-dominant trait that causes a deficiency in production or function of C1 esterase inhibitor, a serum protease inhibitor that regulates the complement, fibrinolytic, and kinin pathways. Hereditary angioedema (HAE) is characterized by painless nonpitting edema of the face and upper airway. The disorder usually begins in childhood and becomes more prominent in adolescence. Swelling progresses over many hours and then resolves spontaneously over 1 to 3 days. Despite the slow progression, death may occur from laryngeal obstruction. Physical stimuli (cold, heat, stress) and circulating immune complex diseases (e.g., due to serum sickness or systemic lupus erythematosus) are also known to cause angioedema.

Emergency management includes foremost securing the airway. Epinephrine is used for angioedema with anaphylaxis. Administration of corticosteroids and antihistamines are often used, although current evidence suggests that such a therapy is rather ineffective in kinin-induced angioedema.[82] Thus, symptom-related medical care should be provided until the swelling has disappeared. Treatment of HAE includes C1-inhibitor concentrate, recombinant C1 inhibitor, kallikrein inhibitors (ecallantide) and bradykinin type 2 receptor antagonists (icatibant).[83–86]

MANAGEMENT OF UPPER AIRWAY OBSTRUCTION

Key elements in the management of UAO include general principles, securing the airway, and specific interventions.

General Management

The primary goals in management of any patient with UAO are assurance of adequate oxygenation and ventilation and management of the underlying condition. If airway obstruction is partial, and the patient's condition is stable, close monitoring and diagnostic studies are appropriate. Depending upon the underlying etiology, temporary measures may include close observation in an intensive care unit, elevation of the head of the bed, administration of humidified oxygen, use of a helium–oxygen inhalation mixture (heliox), systemic corticosteroids, and inhaled racemic epinephrine, pending definitive medical or surgical management.

Heliox may be useful in management of UAO when the obstruction is temporary and reversible. The physiologic rationale for heliox is based upon a reduction in work of breathing achieved through administration of a low-density gas. In particular, heliox has a lower density than does oxygen, room air, or a mixture of the two, resulting in conversion of the predominantly turbulent flow at the site of obstruction to a more laminar pattern. Furthermore, since laminar flow requires a smaller pressure gradient than turbulent flow to achieve the same flow rate, the accompanying work of breathing is less (see Chapter 10). The major limitation of the modality is an inability to deliver gas with an inspiratory fraction of oxygen ($F_{I_{O_2}}$) of more than 40%. Despite physiologic evidence and clinical reports of efficacy, prospective, randomized studies demonstrating improved outcome in patients receiving heliox are lacking, as are data supporting use of corticosteroids or inhaled epinephrine in airway obstruction from a variety of causes.

Securing the Airway

Although under controlled circumstances, a significant portion of the so-called difficult airways and intubations may be identified in the course of a thorough preoperative assessment, the patient with impending airway obstruction presents a challenge. Under such circumstances, a critical first concern is deciding whether an artificial airway is needed emergently.[87] Regardless of the airway utilized, emphasis is placed on ensuring adequate oxygenation and ventilation.

Airways judged unsafe for routine management may be addressed according to the "difficult airway algorithm" recommended by the American Society of Anesthesiologists (see Chapter 146).[88] A difficult airway is defined as a clinical circumstance in which a conventionally trained anesthesiologist experiences difficulty using face mask ventilation, endotracheal intubation, or both.

Airway access in emergency situations may be challenging because the patient frequently is critically ill and can deteriorate quickly. The likelihood of a difficult intubation can be estimated by using the Mallampati score or a modification of the score to assess potential laryngeal exposure and prospects for adequate airway visualization.

A number of parameters, such as mouth opening distance, jaw size, thyromental distance, and cervical range of motion, have been incorporated into airway assessment scoring systems; each parameter has limited sensitivity and specificity. Combining scoring systems provides better accuracy of prediction. The "rule of threes," which is a useful, simple bedside tool, predicts successful direct laryngoscopy if the examiner can place three finger breadths (~6 to 7 cm) between the upper and lower teeth, the mandible and hyoid bones, and the thyroid cartilage and sternal notch. In the emergency setting of UAO, the most experienced physician available should secure the airway. Appropriate equipment and monitoring, along with backup resources for alternative and invasive airway management, should be available.

A variety of invasive and noninvasive techniques are available as alternatives to standard, laryngoscopy-guided orotracheal intubation. Invasive methods include surgical and/or percutaneous tracheostomy, surgical and percutaneous transtracheal (needle) cricothyrotomy, translaryngeal-guided or "retrograde" intubation, fiberoptic endotracheal intubation, and use of a rigid ventilating bronchoscope. Noninvasive techniques include use of specialized laryngoscope blades, Glidescope, guiding and lighted stylets, directional tip control tubes, and esophageal–tracheal (Combitube) or laryngeal mask airways. In selected circumstances, tactile intubation, nasotracheal intubation, or blind orotracheal intubation may be employed.

Cricothyroidotomy

Cricothyroidotomy (either surgical or based on Seldinger technique) has a long history of use in emergency access to the airway when more conservative approaches fail or are contraindicated. In

early reports, a high incidence of laryngeal stenosis during long-term follow-up was noted, perhaps related to the presence of infectious laryngeal disease, the use of large-bore tubes, and prolonged intubation.[89] More contemporary reviews indicate a much lower (1.1%) incidence of airway stenosis following cricothyroidotomy.[90] The procedure is useful for short-term airway control; tracheostomy should be considered if prolonged airway access is required.

Tracheostomy

Most tracheostomies are performed on intubated patients in the intensive care unit. Percutaneous tracheostomy is rapidly becoming the method of choice in the intensive care unit and is associated with acceptable intraoperative and postoperative complication rates.[91,92] Advantages of the technique over the traditional procedure include low cost, short procedure time, and elimination of the need to transport critically ill patients to the operating room. Adaptation of percutaneous techniques for emergency situations also has been described.

In a review of more than 1100 patients who underwent tracheostomy, 76% were performed in patients who required prolonged ventilation, 6% for UAO, 7% for extensive maxillofacial trauma, and 11% as an adjunct for head and neck or chest surgeries; only 0.26% were performed as emergency procedures. Overall mortality was 0.7%.[93]

Interventional Bronchoscopic Techniques

Interventional bronchoscopy is discussed in Chapter 34. Use of these techniques for managing UAO is well established and is briefly summarized in the following paragraphs.

Rigid bronchoscopy allows oxygenation, ventilation, and application of various diagnostic and therapeutic interventions, including debridement of obstructing lesions, control of bleeding, and removal of foreign bodies. Complications include anesthetic risks; barotrauma; damage to teeth, lips, or gums; airway perforation; bleeding; and mucosal injury.

Thermal modalities may be used to devitalize tissue prior to mechanical debridement or to cauterize the base of lesions following debulking. Electrocautery, laser (Nd-YAG, CO_2), and argon plasma coagulation (APC) can be applied through rigid or flexible bronchoscopy. Side effects include bleeding, perforation, airway fire, and damage to cartilage. All thermal modalities are about equally as effective at achieving airway patency, and often the method of choice is clinician preference. APC has the best hemostatic properties.

Photodynamic therapy (PDT) is based on the principle of targeted tissue apoptosis after local activation of a systemically injected photosensitive chemotherapeutic agent. In essence, PDT creates a phototoxic cell reaction when nonthermal laser light is applied by bronchoscopy and activates a drug trapped in these target cells. The major disadvantage of this technique is that the tissue necrosis occurs within 48 to 72 h and as such the ability to debride the airway is delayed for several days. Bleeding and obstruction from necrotic tumor and edema are potential immediate complications. PDT may also result in airway perforation.

Standard cryotherapy, based on repeated freeze–thaw cycles to achieve cell necrosis and tissue damage, is used in benign and malignant disorders of the upper airway. A flexible cryoprobe can be inserted via flexible or rigid bronchoscopy and uses extremely cold gas at the tip cooled to –40°C to –80°C to freeze tissue. Cryotherapy has excellent hemostatic effects; the incidence of perforation or bleeding is low. Due to the delayed beneficial effect, cryotherapy is not usually used under emergent conditions. A method of debulking obstruction uses a cryobiopsy technique in which the tissue is simply frozen to the probe and then sheared from the surface, obviating the thaw cycle. Applied in this fashion, cryotherapy can be used for the immediate treatment of airway obstruction, although when used in this fashion it has less of a hemostatic effect than the thermal modalities. In addition, cryotherapy has the advantage over other thermal modalities in that cartilage is cryoresistant whereas malignant tissue is generally cryosensitive. An endoluminal cryospray catheter has been developed and has been used effectively to treat tracheal stenosis due to both benign and malignant causes.[94]

Finally, external beam radiation and brachytherapy are useful modalities for palliative management of airway obstruction and hemoptysis. For external beam radiation, unwanted exposure of adjacent structures is a limiting factor, while hemorrhage, radiation bronchitis, and fistulae with surrounding structures are known complications of radiotherapy. The maximal effect of all radiation techniques is delayed days to weeks, and there is a risk of worsening of obstruction during therapy due to airway edema.

Airway Stents

Airway stents are used in the palliative management of both benign and malignant airway obstruction.[95] Available tracheal stents include expandable metal, silicone, and hybrid prostheses. Major complications include stent migration, granulation tissue formation, and stent interference with mucociliary clearance. In a series of more than 1500 patients who had stents placed for UAO due to benign or malignant disorders, stent migration was reported in 9.5%, granulation tissue formation in 7.9%, and obstruction in 3.6%. Due to concerns regarding stent fracture and the likelihood that stents will remain in place longer in benign disease, the FDA posed a warning on the use of metallic airway stents for this indication. Currently, metallic stents are only recommended in benign disease for patients who are not candidates for or have failed a trial of silicone stent placement. Recent advances in 3D airway modeling and printing have allowed for the development of customized patient-specific silicone stents that have had promising results in patients with complex stenosis.[96]

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