Airway disorders can be categorized into those that involve the trachea, those that involve the bronchi, and those that involve the bronchioles, the smallest branching airways leading to alveoli. Many disorders can, and frequently do, involve more than one airway compartment. Tracheal disorders will be discussed first, and disorders involving both the bronchi and bronchioles will be discussed together. The reader is referred to Chapter 1 for a discussion of normal anatomy of the airways.

TRACHEAL DISORDERS

Tracheal shape varies, depending on the phase of the respiratory cycle. The intrathoracic trachea is round or elliptic on inspiration images and flat or horseshoe-shaped during and at the end of a forced exhalation as a result of anterior bowing of the posterior noncartilaginous tracheal membrane during exhalation (1). Upper limits of normal for coronal and sagittal tracheal dimensions, respectively, as determined by chest radiographs, are 25 and 27 mm for men and 21 and 23 mm for women. The lower limit of normal for both dimensions is 13 mm in men and 10 mm in women (2). Mean measurements on computed tomography (CT) of anteroposterior (AP) and transverse diameters of the extrathoracic trachea, respectively, are 20.1 and 18.4 mm (3); these can increase by as much as 15% in men with aging (4).

CT is superior to chest radiography for detection of abnormalities of the trachea and main bronchi; sensitivities in detecting disease on chest radiographs and CT are 66% and 97%, respectively (5). Spiral multidetector CT, which allows for the acquisition of a whole thoracic volume during a single breath-hold, eliminating respiratory motion, is the technique of choice for noninvasive imaging of the airways. Volume acquisition with multidetector CT has fostered a renewed interest in two-dimensional and three-dimensional (3D) reconstructions applied to the tracheobronchial tree. Potential clinical applications of 3D reconstructions, such as shaded surface display and volume rendering, include assisting with diagnoses, replacing bronchoscopy in some instances, and helping in surgical planning and endobronchial treatments (6). In the case of a lesion that completely obstructs an airway, CT allows visualization of the airway beyond the obstruction. However, CT’s virtual bronchoscopy is currently unable to show mucosal detail, and 3D postprocessing methods are time-consuming to perform and rarely performed in routine clinical practice.

Patients with tracheal disease can be asymptomatic or may present with cough, dyspnea, wheezing, or stridor. Because of the variety of conditions that can cause wheezing, a misdiagnosis of asthma is common (7). Tracheal disorders are generally organized into those that cause tracheal widening and those that cause narrowing. CT can demonstrate the degree of widening or narrowing, in addition to the location and extent of tracheal abnormality; it can also demonstrate the presence of associated extraluminal disease, postobstructive atelectasis, and pneumonia. Magnetic resonance imaging (MRI) is a valuable method for observing the trachea because of its multiplanar capabilities.
demonstration of the airway, the mediastinal vessels, and the other structures simultaneously, without the need for contrast medium or exposing the patient to radiation. MRI is particularly useful in children and in patients with either vascular rings or tracheal compression by the innominate artery.

Disorders That Cause Tracheal Widening
Congenital or nonacquired diffuse tracheal widening is much less common than tracheal narrowing and has a more limited differential diagnosis. The Mounier–Kuhn syndrome, which affects primarily men in the fourth and fifth decades, accounts for almost all cases of nonacquired tracheal widening (8). Thought to be congenital (9), it is an abnormality of the trachea and main bronchi characterized by atrophy or absence of elastic fibers and thinning of muscle, which allows the trachea and main bronchi to become flaccid and markedly dilated on inspiration, with narrowing or excessive collapse on expiration and cough. The abnormal airway dynamics and pooling of secretions in broad outpouchings, or diverticula, of redundant musculomembranous tissue between the cartilaginous rings predispose patients to the development of chronic pulmonary suppuration, bronchiectasis, emphysema, and pulmonary fibrosis (10). The trachea is involved from the subglottic region to the carina. A tracheal diameter greater than 3 cm is required for diagnosis, and tracheal widths up to 5.5 cm have been recorded (8). The radiographic and CT features of the condition include marked dilatation of the trachea and mainstem bronchi, tracheal diverticulosis, and a variable incidence of bronchiectasis and chronic pulmonary parenchymal disease (11,12).

Several conditions can result in acquired tracheobronchomegaly that may closely resemble that seen in Mounier–Kuhn syndrome (Table 13.1). Some degree of tracheal dilatation may be seen with aging (4,13) and in musicians who play wind instruments (13). Chronic

### Table 13.1 DISORDERS THAT CAUSE TRACHEOBRONCHOMEGALY

<table>
<thead>
<tr>
<th>Nonacquired</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mounier–Kuhn syndrome</td>
</tr>
<tr>
<td>Acquired</td>
</tr>
<tr>
<td>Common</td>
</tr>
<tr>
<td>Normal aging</td>
</tr>
<tr>
<td>Chronic airway infection</td>
</tr>
<tr>
<td>Cigarette smoking</td>
</tr>
<tr>
<td>Chronic bronchitis</td>
</tr>
<tr>
<td>Emphysema</td>
</tr>
<tr>
<td>Cystic fibrosis</td>
</tr>
<tr>
<td>Diffuse pulmonary fibrosis</td>
</tr>
<tr>
<td>Uncommon</td>
</tr>
<tr>
<td>Playing wind instruments</td>
</tr>
<tr>
<td>Inhalation of noxious fumes</td>
</tr>
<tr>
<td>Chronic intubation</td>
</tr>
<tr>
<td>Ehlers–Danlos syndrome</td>
</tr>
<tr>
<td>Cutis laxa</td>
</tr>
<tr>
<td>Marfan syndrome</td>
</tr>
<tr>
<td>Ataxia-telangiectasia</td>
</tr>
<tr>
<td>Ankylosing spondylitis</td>
</tr>
<tr>
<td>Kenny–Caffey syndrome</td>
</tr>
<tr>
<td>Brachmann–de Lange syndrome</td>
</tr>
<tr>
<td>Connective tissue diseases</td>
</tr>
<tr>
<td>Bruton-type agammaglobulinemia</td>
</tr>
<tr>
<td>Light-chain deposition disease</td>
</tr>
</tbody>
</table>

### Table 21.3 DISORDERS THAT CAUSE TRACHEAL NARROWING

<table>
<thead>
<tr>
<th>Extrinsic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Masses (e.g., thyroid, aberrant vessels, enlarged nodes)</td>
</tr>
<tr>
<td>Fibrosing mediastinitis</td>
</tr>
<tr>
<td>Intrinsic</td>
</tr>
<tr>
<td>Congenital tracheal narrowing</td>
</tr>
<tr>
<td>Infection</td>
</tr>
<tr>
<td>Granulomatous disorders (e.g., granulomatosis with polyangiitis [Wegener granulomatosis], sarcoidosis)</td>
</tr>
<tr>
<td>Neoplasms</td>
</tr>
<tr>
<td>Trauma, including intubation</td>
</tr>
<tr>
<td>Amyloidosis</td>
</tr>
<tr>
<td>Relapsing polychondritis</td>
</tr>
<tr>
<td>Tracheobronchopathia osteochondroplastica</td>
</tr>
<tr>
<td>Saber-sheath trachea</td>
</tr>
<tr>
<td>Idiopathic</td>
</tr>
</tbody>
</table>
Amyloidosis of the respiratory tract, both primary and secondary, is a rare condition that produces focal or diffuse irregular narrowing of the airway by submucosal deposits of amyloid (22). Both radiography and CT of the chest can demonstrate diffuse narrowing or show nodular protrusions into the tracheal lumen that can be calcified (Fig. 13.1) (8).

Tracheobronchopathy osteochondromatosa is a rare, benign condition characterized by multiple submucosal osteocartilaginous growths along the inner anterolateral surfaces of the trachea (31,32). Although the etiology is unknown, theories have linked this disorder to chronic inflammation, degenerative processes, amyloidosis, and neoplasia (33–35). Radiography and CT of the chest show multiple sessile nodular tumors, with or without calcification, extending over a long segment of the trachea and into the main bronchi. In contrast, the nodules in amyloidosis may be circumferential and can be distinguished from those of tracheobronchopathy osteochondromatosa, in which there is always sparing of the posterior membranous wall.

Granulomatosis with polyangiitis (Wegener granulomatosis) is characterized by granulomatous vasculitis of the upper and lower respiratory tract, usually in conjunction with renal and other organ involvement. The CT appearance of airway involvement includes circumferential narrowing of the airway lumen, abnormal soft tissue within the tracheal rings, and dense irregular calcification of the tracheal cartilages (36).

Sarcoidosis is another granulomatous disorder that may rarely involve the trachea and bronchi. Granulomatous sarcoid lesions may exist intrinsically in the airway, or enlarged hilar nodes may compress the bronchi extrinsically (37,38).

Many viral, bacterial, or fungal diseases can involve the trachea. In North America, most cases of laryngotracheobronchitis are viral in nature; subglottic or laryngeal narrowing is common, but radiographically demonstrable tracheal narrowing is unusual (7).

**Tracheobronchial Filling Defects**

In adults, tracheobronchial filling defects are usually produced by mucus (Fig. 13.2) or neoplasms. Less common causes include pneumonia, infections (Fig. 13.3), foreign bodies, broncholiths, and other miscellaneous disorders (Figs. 13.4 to 13.6). If mucus is suspected on a CT scan, it can be helpful to selectively repeat the scan at the region of interest after the patient has coughed.

Ninety percent of all adult primary tracheal tumors are malignant (39). Squamous cell carcinoma is the most common tracheal and bronchial tumor (Fig. 13.7). The most common imaging appearance is a small sessile or polypoid lesion in the lower third of the trachea or an occluding mass within a main bronchus (Figs. 13.8 and 13.9). Adenoid cystic carcinoma is the second most common tracheal tumor. Nearly half of patients are younger than 30 years. On CT, it appears as a smooth, focal mass in the trachea or main bronchi (Figs. 13.10 and 13.11). The longitudinal extent is typically greater than the cross-sectional area. The most common airway tumor in adolescents and young adults is endobronchial carcinoid tumor. On CT it appears as a well-defined spherical or oval central bronchial mass with a slightly lobulated border and marked contrast enhancement (Fig. 13.12). Respiratory papillomatosis results from infection of the upper respiratory tract by the human papilloma virus and rarely occurs in adults (40). It predominately affects the larynx but may spread into the trachea, bronchi, and even lung (Fig. 13.13). The most common primary tumors that spread hematogenously to the large airways are adenocarcinomas (e.g., breast, colon kidney, lung) and melanoma (41) (Figs. 13.14 and 13.15).
FIG. 13.2 ● Mucous plugging. A: PA chest radiograph of a 45-year-old woman with shortness of breath after cervical spine fusion shows abnormal opacities in both lower lobes and volume loss in the left lower lobe. B: CT shows low-attenuation material in both lower lobe segmental bronchi (solid arrows), postobstructive atelectasis of the left lower lobe (A), and posteromedial displacement of the left major fissure (dashed arrow).

FIG. 13.3 ● Endobronchial blood clot. CT of a 10-year-old girl with leukemia and Rhizopus necrotizing pneumonia shows a filling defect occluding the bronchus intermedius (arrow). The right middle and lower lobes were surgically resected; pathology showed pulmonary artery and vein thromboses, diffuse pneumonia and pulmonary hemorrhage, and clotted blood in the bronchus intermedius.

Foreign-body aspiration is more common in children than adults (42). In adults, the foreign body is usually aspirated food (Figs. 13.16 and 13.17). Aspiration of teeth or restorative dental material can be related to trauma, iatrogenic, or otherwise (Fig. 13.18).

Broncholithiasis is characterized by compression on or erosion into an airway by calcified peribronchial lymph nodes (Figs. 13.19 and 13.20). Patients may occasionally present with expectorations of stones (lithoptysis). On CT, the broncholith is almost always accompanied by other calcified peribronchial, hilar, or mediastinal lymph nodes.

Tracheoesophageal Fistulas
Tracheoesophageal fistulas in adults are almost exclusively acquired lesions. They occur as a complication of intrathoracic malignancies (accounting for 60% of cases), infection, and trauma (43,44). The diagnosis is usually made with a fluoroscopic contrast study but can be made, in some cases, with CT. In addition to demonstrating the site of a fistula, CT can suggest the etiology and detect pulmonary and mediastinal complications (45).
**FIG. 13.5 • Endobronchial blood clot.** A: CT of an 18-year-old woman with hemoptysis shows a circumscribed mass in the left upper lobe bronchus (arrow). B: CT at a level superior to (A) shows consolidation and ground-glass opacity in the left upper lobe. Bronchoscopy confirmed a blood clot in the bronchus and hemorrhage in the lung.

**FIG. 13.6 • Endobronchial lipoma.** CT of a 65-year-old man with recurrent right middle lobe pneumonia shows a small circumscribed mass in the right middle lobe bronchus (arrow). The diagnosis was confirmed bronchoscopically.

**FIG. 13.7 • Endobronchial squamous cell carcinoma.** CT of a 63-year-old woman with cough shows a soft tissue mass that almost completely obliterates the lumen of the bronchus intermedius (arrow).

**FIG. 13.8 • Endobronchial squamous cell carcinoma.** CT of a 73-year-old man with cough shows a mass obstructing the right lower lobe bronchus (arrow) and postobstructive pneumonia in the right lower lobe.

**FIG. 13.9 • Endobronchial squamous cell carcinoma.** CT of a 52-year-old man with chest pain and 20-lb weight loss shows cutoff of the right middle lobe bronchus (arrow) and a surrounding mass in the right middle lobe.
FIG. 13.10  Tracheal adenoid cystic carcinoma. A: PA chest radiograph of a 59-year-old man with recurrent right middle lobe pneumonia and cough shows a linear band of opacity in the right midlung (arrow). B: Lateral view shows an oblique band of opacity paralleling the inferior aspect of the major fissure (arrow). C: Coronal reformatted CT shows a lobular soft tissue mass (arrow) almost completely obstructing the lumen of the trachea just above the level of the carina. D: Axial CT shows that the mass (arrow) almost completely fills the lumen of the trachea. E: CT at the level of the inferior pulmonary veins shows postobstructive atelectasis and pneumonia in the right middle lobe.
FIG. 13.11  •  Tracheal adenoid cystic carcinoma. A: Coned-view dual-image chest radiograph of a 31-year-old woman with shortness of breath, wheezing, and hemoptysis shows a mass overlying the trachea (solid arrow). Note an incidental accessory azygos fissure (dashed arrows). Coronal (B) and sagittal (C) reformat ted CT images confirm the endotracheal mass (arrows). The patient was treated for asthma for a year prior to the diagnosis of adenoid cystic carcinoma.

FIG. 13.12  •  Endobronchial carcinoid. CT shows a circumscribed mass in the left upper lobe bronchus (arrow).

FIG. 13.13  •  Tracheal papillomatosis. CT of a 28-year-old man with cough shows a polypoid mass in the trachea (arrow).
FIG. 13.14 ● Tracheal metastasis. A: PA chest radiograph of a 75-year-old woman with endometrial carcinoma shows a mass adjacent to the right hilum. B: Lateral view shows the mass projected over the heart. The contours of the mass suggest that it is related to the inferior edge of the major fissure. C: Axial CT shows low-attenuation material obliterating the lumen of the right middle lobe bronchus (arrow). Note that the bronchial wall is outlined by calcium. Tumor is growing through the bronchus into the right middle lobe. The opacity seen on the chest radiograph represents tumor and collapsed right middle lobe.

FIG. 13.15 ● Tracheal metastasis. CT of a 64-year-old woman with renal cell carcinoma shows a soft tissue mass adjacent to the anterior wall of the trachea (arrow), representing one of many biopsy-proven metastases to the trachea. Mucus could have a similar appearance, but it will usually clear with a repeated scan after the patient clears the throat.

FIG. 13.16 ● Endobronchial foreign body. CT shows a radiopaque foreign body (arrow) in the left main bronchus. Note the hyperlucency and hyperinflation of the left upper and lower lobes secondary to air trapping. A chicken bone was removed from the airway.
FIG. 13.17 • Endobronchial foreign body. CT with lung windowing (A) and soft tissue windowing (B) of a 42-year-old man with cough, dyspnea, wheezing, and fever show a round mass occluding a left lower lobe segmental bronchus (arrow). C: CT at a level inferior to (A) shows postobstructive pneumonia in the left lower lobe. An unpopped popcorn kernel was endoscopically removed from the bronchus.

FIG. 13.18 • Aspirated tooth. PA (A) and lateral (B) chest radiographs show a radiopaque foreign body in the right lower lobe (arrow), representing a tooth that was dislodged during attempt at intubation.
**FIG. 13.19 • Broncholithiasis.** CT of a 72-year-old man with cough, wheezing, and shortness of breath shows large calcifications in the left hilum and left lower lobe bronchus (arrows) and postobstructive atelectasis of the left lower lobe.

**FIG. 13.20 • Broncholithiasis.** A: PA chest radiograph of a 51-year-old man with cough shows abnormal opacification obscuring the right heart border. B: Lateral view shows the abnormal opacity projected over the heart, limited by the minor fissure superiorly and the lower aspect of the right major fissure inferiorly. C: CT shows a large calcification occluding the right middle lobe bronchus (arrow) and postobstructive atelectasis and pneumonia in the right middle lobe.
Congenital Tracheobronchial Anomalies

Congenital tracheobronchial anomalies can present as life-threatening emergencies at birth, or they may go undiagnosed for years. Clinical symptoms are often nonspecific, and radiographic evaluation is frequently required to localize and characterize the lesion before endoscopy, surgery, or medical management. The radiologist must be on the alert for unsuspected associated anomalies involving Airways, lungs, great vessels, and the esophagus, which occur with relative frequency.

Tracheal webs produce localized areas of narrowing with no associated deformity of the underlying cartilage. The thickness of the webs determines the severity of obstruction and the therapeutic approach (46). Congenital tracheal stenosis may occur in any portion of the trachea, usually involving more length and depth of the trachea than webs, and is more likely to require resection rather than dilatation alone. Stenosis secondary to long-term compression by a dilated esophagus, abnormal great vessels, or cervicomediastinal masses results in a focal fibrous and cartilaginous deformity that persists for some time after the mass is removed. Congenital tracheal stenosis is frequently associated with bronchial stenosis; pulmonary hypoplasia or agenesis; tracheal bronchus; tracheoesophageal fistula; tracheomalacia; anomalies of vertebrae, ribs, and thumbs; and cardiac anomalies.

Tracheomalacia is an abnormally flaccid trachea that may involve all or part of the trachea and results in abnormal anteroposterior tracheal collapse during expiration of greater than or equal to 50% of cross-sectional area. The innominate artery compression syndrome can result in secondary tracheomalacia, in which there is persistent narrowing of the anterior tracheal wall at the level of the thoracic inlet. Short trachea, which occurs when there are 15 or fewer tracheal rings, can be diagnosed on CT when the tracheal bifurcation lies above the fourth thoracic vertebral body in children younger than 2 years or above the fifth thoracic vertebral body thereafter (47).

A tracheal bronchus (so-called “pig bronchus,” or bronchus suis) is the most common anomalous airway pattern, reported in 2% of children during bronchoscopy examination (48). It occurs most commonly in boys, arising most often from the right lateral tracheal wall within 2 cm of the carina. A tracheal bronchus can be a true supernumerary bronchus or anatomical variant, with a normal but displaced upper lobe, segmental, or subsegmental bronchus. It can be asymptomatic, or it may result in right upper lobe infection, atelectasis, or bronchiectasis, usually from a stenotic bronchial segment and poorly cleared secretions. The CT appearance is that of a bronchus arising from the trachea in a section more cephalad than the carina (Fig. 13.21) (49).

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

COPD refers to a group of disorders characterized by chronic or recurrent obstruction to airflow. Five principal disorders fall under this heading (Table 13.3), although the term is commonly used clinically to refer to pulmonary emphysema. Because these disorders are sometimes difficult to distinguish from one another on chest radiography, the term COPD should not be restricted to pulmonary emphysema.

Asthma

There is no universally accepted definition of asthma; it may be regarded as a diffuse, obstructive lung disease with hyperreactivity of the Airways to a variety of stimuli and a high degree of reversibility of the obstructive process, which may occur either spontaneously or as a result of treatment. Asthma is a complex disorder involving biochemical, autonomic, immunologic, infectious, endocrine, and psychological factors in varying degrees in different individuals. Both large and small Airways may be involved, again to varying degrees. The three elements that contribute to Airways obstruction in asthma are (i) spasm of smooth muscle; (ii) edema and inflammation of the mucous membranes lining the Airways; and (iii) intraluminal exudation of mucus, inflammatory cells, and cellular debris. Asthma can be a benign, self-limiting problem; it can lead to acute respiratory failure; or it can be a chronic, recurrent disease that leads to debilitating, irreversible airflow obstruction and COPD. Emphysema is not a prominent finding in the lungs of nonsmoking patients with asthma, even in those with severe disease (50).

Chest radiographs of patients with asthma can be normal, show increased lung markings and hyperinflation, or show low lung volumes and multifocal atelectasis. CT findings may include bronchiectasis involving mostly subsegmental and distal bronchi, bronchial wall thickening, small centrilobular opacities, and decreased lung attenuation (51). Allergic bronchopulmonary aspergillosis (ABPA) occurs with a greater prevalence in patients with asthma and CF (52) (Figs. 13.22 and 13.23). Central bronchiectasis on CT is the hallmark of ABPA. Bronchial wall thickening in asthma (which is assessed subjectively on CT) may reflect bronchial and peribronchial inflammation as well as increased smooth muscle, mucous gland, cartilage, and submucosal
areas (53,54). Areas of hyperlucency are caused by decreased lung perfusion secondary to reflex vasoconstriction in hypoventilated areas, and by air trapping (55) (Fig. 13.24). Emphysema seen on the CT scans of patients with asthma is attributed to cigarette smoking (56). Small centrilobular opacities may correspond to plugging or to thickening of the bronchiole walls (53). Because central airway lesions and mitral stenosis can produce symptoms attributed to asthma, the airways, cardiac silhouette, and pulmonary vasculature should always be evaluated closely on every chest radiograph when the clinical history is “asthma.” The chest radiograph should also be assessed for evidence of pneumonia, which is known to exacerbate asthma, and pneumomediastinum (Fig. 13.25) and pneumothorax, as evidence of alveolar rupture that can be caused by wheezing and coughing (Table 13.4).

**FIG. 13.22** Allergic bronchopulmonary aspergillosis. PA chest radiograph of a 64-year-old woman with a long history of asthma shows multiple tubular opacities in the left upper lobe (arrows), representing dilated bronchi filled with mucus, debris, and fungal hyphae.

**FIG. 13.23** Allergic bronchopulmonary aspergillosis. CT shows central bronchial dilatation and impaction (solid arrow). Peripheral nodular and ground-glass opacities (dashed arrow) represent impaction of small airways and peribronchiolar inflammation.

**FIG. 13.24** Asthma. A: Inspiratory CT of a 34-year-old woman with steroid-dependent asthma is normal. Note the round contour of the trachea. B: Expiratory image at the same level as (A) shows areas of lucency (L), representing air trapping. Note the flattened posterior tracheal contour on expiration.

**FIG. 13.25** Pneumomediastinum. PA chest radiograph of a patient with asthma shows mediastinal air outlining the heart (dashed arrow) and extending into the neck bilaterally (solid arrows).
Bronchiectasis

Bronchiectasis, defined as irreversible dilatation of the bronchial tree, can cause chronic sputum production and hemoptysis, and it can be described morphologically as cylindric, varicose, cystic, or traction in type (57). Cylindric bronchiectasis, the mildest form, is characterized by smooth, uniformly dilated bronchi (Figs. 13.26 and 13.27). Sectioned lengthwise, these bronchi resemble nontapering “tram tracks”; sectioned crosswise, the bronchi appear round or oval. Beaded dilatation of bronchi describes the varicose type (Fig. 13.28); cystic bronchiectasis, the most severe type, is characterized by cysts in clusters, often with air–fluid levels (Figs. 13.29 to 13.31). Traction bronchiectasis refers to irreversible dilatation of bronchi and bronchioles in areas of pulmonary fibrosis. It occurs predominantly in the peripheral portions of lung, where bronchi contain less supporting cartilage (see Fig. 3.4B) (58). There are numerous causes of bronchiectasis; these can be remembered with the mnemonic “BRONCHIECTASIS” (Table 13.5).

Table 13.4 THINGS TO LOOK FOR ON THE CHEST RADIOGRAPH WHEN THE PATIENT HISTORY IS “ASTHMA”

<table>
<thead>
<tr>
<th>“PHAME”</th>
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</thead>
<tbody>
<tr>
<td>Pneumothorax</td>
</tr>
<tr>
<td>Pneumomediastinum</td>
</tr>
<tr>
<td>Hyperinflation</td>
</tr>
<tr>
<td>Atelectasis</td>
</tr>
<tr>
<td>Mucous plugging (ABPA)</td>
</tr>
<tr>
<td>Mitral stenosis (patients can present with symptoms of “asthma”)</td>
</tr>
<tr>
<td>Endotracheal lesion (e.g., patients with carcinoid tumor can present with symptoms of “asthma”)</td>
</tr>
</tbody>
</table>

ABPA, allergic bronchopulmonary aspergillosis.

FIG. 13.26 • Cylindric bronchiectasis. Coronal reformatted CT shows smooth, uniformly dilated bronchi (arrows), predominantly in the lower lungs.

FIG. 13.27 • Atypical mycobacterial bronchiolitis. A: CT shows tree-in-bud opacities in the periphery of the right lower lobe (arrows). B: Thin-section CT (1.25 mm) shows cylindric bronchiectasis in the right lower lobe (arrow). Note bronchiolar opacities in the left lower lobe as well.

FIG. 13.28 • Varicose and cystic bronchiectasis. CT of a 66-year-old man shows dilated bronchi and bronchioles. In profile, some of the bronchiectatic airways have the “beaded” appearance of varicose bronchiectasis (straight arrow); in cross section, some are grouped together like a “cluster of grapes,” as is seen with cystic bronchiectasis (curved arrow). The bronchial and bronchiolar walls are thickened. Some of the dilated bronchioles are filled with mucus, forming peripheral nodular opacities (arrowhead).
Although patients with bronchiectasis rarely have a normal chest radiograph (59), the chest radiographic findings are neither sufficiently sensitive nor specific enough to be of value in the accurate assessment of bronchiectasis, and they are unreliable in determining the severity and extent of the disease (59–61). The common radiographic findings are loss of definition and increase in number and size of the bronchovascular markings (caused by peribronchial inflammation/fibrosis and the presence of retained secretions), tram tracking, tubular or ring-shaped opacities with central lucency if the airways are air-filled, central opacity if there is mucoid impaction, and cystic spaces that can be up to 2 cm in diameter.

Early studies assessing the accuracy of conventional CT in diagnosing bronchiectasis resulted in sensitivities of 60% to 80% and specificities of 86% to 100% (61–64). With the use of 1.5-mm collimation at
10-mm intervals, sensitivity with CT improved to a range of 96% to 98%, with specificity of 93% to 99% (65,66). Thin-section CT is now the accepted gold standard for diagnosing bronchiectasis. With current state-of-the-art multidetector CT, routine imaging of the lungs with 3-mm collimation and 1.25-mm reformatting will allow for diagnosis of most cases of bronchiectasis, even very mild cases. The most reliable finding for the diagnosis of cylindrical bronchiectasis is visualization of bronchi within 1 cm of costal or paravertebral pleura, or visualization of bronchi abutting the mediastinal pleura. Although lack of normal bronchial tapering and increased bronchoarterial ratios are helpful in the diagnosis of bronchiectasis, these findings can also be seen in 10% to 20% of healthy subjects.

Despite the ease with which bronchiectasis can be identified on CT in most cases, there are a number of potential pitfalls (67). These include artifacts from both respiratory and cardiac motion, and inappropriate collimation and electronic windowing. A number of diffuse lung diseases can simulate bronchiectasis, especially cystic bronchiectasis; these include Langerhans cell histiocytosis, lymphangioleiomyomatosis, cystic changes related to connective tissue diseases or lymphocytic interstitial pneumonia or in patients with acquired immunodeficiency syndrome (AIDS) and Pneumocystis jiroveci pneumonia, emphysema, and cystic metastases. The characteristic combination of “cyst” paired with the accompanying pulmonary artery is sometimes helpful in confirming bronchiectasis, as is variation in the sizes of bronchiectatic “cysts” with inspiration and expiration, a feature that is not usually seen with other types of cystic lesions. Following cystlike lesions from one CT scan section to another and noting their relationship to central airways and their “tubular” nature also allow for accurate distinction between bronchiectasis and other cystic diseases, in most cases. This can be facilitated by reconstructing multidetector CT data to create both maximum- and minimum-intensity-projection images and coronal reformatations.

The dyskinetic cilia syndrome, first described in 1976 (68), represents a spectrum of genetically determined defects in ciliary structure and function that interfere with mucociliary clearance. Although the term immotile cilia syndrome has been used, in many cases the cilia demonstrate some motility (although dyskinetic). Described conditions include (a) situs inversus, paranasal sinusitis, and bronchiectasis (the three major components of Kartagener syndrome) (Figs. 13.32 and 13.33); (b) recurrent upper and lower respiratory tract infections; and (c) immotile sperm and infertility.

**Cystic Fibrosis**

CF is a relatively common genetic disorder that affects the upper and lower respiratory tracts, pancreas, liver and gallbladder, intestines, and genital tract. Approximately 1 in 1,600 live births is affected by this...
autosomal recessive disease, which occurs predominantly in Caucasians. In 1985, the CF defect was determined to be located on chromosome 7 (69), and 4 years later the CF gene was identified by positional cloning (70–72). This new knowledge gave rise to new therapies, including in vivo gene therapy (73). The median survival age rose from about 18 years in 1976 to 29 years in the early 1990s (74,75), with some living for many more decades.

Chest radiographic findings in adult patients with CF include hyperinflation and atelectasis as well as bronchiectasis (76). CT shows the presence, severity, and extent of bronchiectasis, peribronchial thickening, mucous plugging, abscesses, bullae, lung collapse, and dense parenchymal opacification (77) (Fig. 13.34). In early stages of the disease, the upper lungs are involved to a greater extent than the lower lungs (Fig. 13.35). As the disease progresses, the process becomes more diffuse and an upper lung–predominant pattern may not be appreciated.

Chronic Bronchitis
Chronic bronchitis is defined clinically as a chronic or recurrent increase in the volume of mucoid bronchial secretions sufficient to cause expectoration, occurring on most days during at least three consecutive months for no less than 2 consecutive years (78). It is common among cigarette smokers. The diagnosis is based on the presence of chronic productive cough in the absence of any specific cause, such as bronchiectasis or chronic infection. The radiographic features are nonspecific and include tubular shadows, thickened bronchial walls, hyperinflation of the lungs, and areas of pulmonary oligemia. The term dirty lung has been used to describe the increase in bronchovascular markings. Hyperinflation and oligemia are probably a result of associated pulmonary emphysema. Findings of centrilobular emphysema can predominate on CT of patients with chronic bronchitis. Radiographic and CT features are insensitive and nonspecific, and a high degree of interobserver variability further limits the diagnostic capabilities of imaging.

Bronchiolitis
Evaluation of the bronchioles, defined as peripheral airways that do not contain cartilage, requires an understanding of the anatomy of the secondary pulmonary lobule, the smallest portion of lung that is surrounded by connective tissue septa. The lobular bronchioles measure no more than 1 mm in diameter (79), and their walls are less than 0.1 mm thick. Normal bronchioles are generally not seen on thin-section CT. However, bronchiolar abnormalities may be detected when there is thickening of the bronchiolar wall, peribronchiolar
FIG. 13.34  ●  Cystic fibrosis.  
\( \textbf{A:} \) CT of a 22-year-old man with cystic fibrosis shows extensive bronchiectasis and bronchial wall thickening in the upper lobes. 
\( \textbf{B:} \) CT at level inferior to (A) shows extensive involvement of the small airways.

FIG. 13.35  ●  Cystic fibrosis.  
\( \textbf{A:} \) Chest radiograph of a 35-year-old woman with CF shows bilateral upper lung bronchiectasis. 
\( \textbf{B:} \) CT shows bronchiectasis, bronchiolectasis, airway wall thickening, and airway impaction. 
\( \textbf{C:} \) CT at a level inferior to (B) shows normal lower lungs. 
\( \textbf{D:} \) CT 1 month prior to (B) shows pneumonia in the left upper lobe (arrow).
inflammation and fibrosis, and bronchiolectasis with or without filling of the dilated bronchiole with secretions (80).

Another CT feature of bronchiolar (small-airway) disease is a mosaic pattern of lung attenuation, which can also be seen with pulmonary vascular and infiltrative lung diseases. In cases of small-airway disease, areas of variable lung attenuation that form a mosaic pattern, which is accentuated during forced exhalation, represent air trapping, hypoxic vasoconstriction, and mechanical pressure on blood vessels (81). On CT obtained at end-expiration, air trapping is recognized as diminished increase in attenuation or a lack of decrease in volume of areas of abnormally lucent lung. In small-airway disease, the size and number of vessels in the abnormally lucent area of lung are decreased relative to areas of higher-attenuation lung. Air trapping can also be seen with pulmonary vascular diseases but not with infiltrative diseases.

Constrictive bronchiolitis (CB) is defined pathologically as irreversible fibrosis of small-airway walls that causes the airway lumina to become narrow or obliterated (82). The clinical criteria used for the diagnosis of CB are irreversible airflow limitation, with a forced expiratory volume in 1 second (FEV₁) that is less than 60% of the predicted value, in the absence of emphysema, chronic bronchitis, asthma, or other cause of airway obstruction (83). CB and cryptogenic organizing pneumonia are not thought to be related, although they can occur as a result of similar etiologic factors. Both are commonly idiopathic. CB is a common sequel of heart or lung transplantation—representing chronic rejection in lung transplantation—and bone marrow transplantation, where CB represents chronic graft-versus-host disease. CB is a component of Swyer–James syndrome related to childhood viral infection (Fig. 13.36).

Chest radiographs are usually normal in CB but can show slowly progressive hyperinflation. The CT findings include bronchiolectasis, centrilobular branching structures and nodules caused by peribroncholar thickening and bronchiolectasis with secretions (80), and mosaic lung attenuation (84–87) (Fig. 13.37). Interpretation of air trapping must be made with caution, because occasional isolated areas of lobular air trapping can be seen in healthy individuals (55).

Pathologic changes occur in the small airways of essentially all smokers (88,89). Respiratory bronchiolitis, also referred to as smoker’s bronchiolitis (90,91), involves the respiratory bronchioles and is characterized by mild chronic inflammation of the bronchioles associated with accumulation of pigmented macrophages in respiratory bronchioles and adjacent alveoli. The condition may be severe enough to produce clinical symptoms of cough and shortness of breath and to produce CT abnormalities, including areas of ground-glass attenuation, centrilobular micronodules, and air trapping (92,93). The abnormalities usually involve predominantly the upper lungs (a distribution similar to that of smoking-related centrilobular emphysema) but may be diffuse.

Diffuse panbronchiolitis is an inflammatory lung disease of unclear etiology that is prevalent in Asians and rare in Europeans and North Americans. Histologically, there is thickening of the walls of respiratory bronchioles and associated peribronchiolitis, and, in advanced stages, bronchiolectasis (94). The chest radiograph can show disseminated small nodular opacities up to 2 mm in size (95). The findings on CT have been classified into four types: (a) nodules alone, (b) nodules associated with branching linear opacities, (c) nodules with ring-shaped or small, tubular opacities (probable bronchiolectasis), and (d) large, cystic opacities accompanied by dilated proximal bronchi (94,96) (Figs. 13.38 and 13.39).

Bronchopneumonia, regardless of the type of infectious agent, can result in centrilobular nodules or branching structures on CT, which are related to peribronchiolar consolidation or pus-filled small airways (95), and it is the most common cause of the “tree-in-bud” pattern
Airways

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seen on CT (97,98) (Figs. 13.40 to 13.43). Aspiration of infected or other material into the small airways is another common cause of the tree-in-bud pattern (Fig. 13.44).

Pulmonary Emphysema

Pulmonary emphysema, as defined by the National Heart, Lung, and Blood Institute, is “an abnormal permanent enlargement of the airspaces distal to the terminal bronchioles, accompanied by destruction of the alveolar walls, and without obvious fibrosis” (99). Three different morphologic subtypes of emphysema have been described according to their location in the secondary pulmonary lobule: centrilobular, panlobular, and paraseptal (distal lobular) (Table 13.6). A fourth type of emphysema, paracatricial emphysema, results from and is always associated with pulmonary fibrosis and therefore does not meet the strict definition of emphysema.

Emphysema is found at autopsy in up to 66% of adult patients (100,101), but clinical detection of disease during life is difficult unless the condition is advanced. The presence of airflow obstruction alone is a sensitive indicator of the presence of emphysema but is not specific, because asthma, irreversible small-airway disease, and certain forms of interstitial lung disease may also result in decreased FEV₁ (99,102,103).

Evidence of impairment in gas transfer, as assessed with carbon monoxide diffusing capacity, is more sensitive than abnormal spirometry for the diagnosis of emphysema; it is also nonspecific, however, and patients may have up to 30% of their lung involved with emphysema but have no evidence of functional impairment (104). The accuracy of diagnosis based on findings from chest radiographs depends on the severity of parenchymal destruction (105,106). CT findings correlate with the presence and severity of morphologic emphysema better than chest radiographic findings or results of pulmonary function tests (107,108), although several studies that assessed CT with 10- and 1-mm collimation concluded that CT consistently underestimates the extent of centrilobular and panlobular emphysema and the severity of
emphysema when compared with pathologic assessment (109–112). In spite of these limitations, CT is currently the best way to detect emphysema in living patients. New and exciting quantitative imaging techniques for evaluating patients with emphysema are on the horizon.

The most common form of emphysema, centrilobular emphysema, is strongly associated with cigarette smoking, with the severity of emphysema increasing with the number of cigarettes smoked (113–114). Centrilobular emphysema results from destruction of alveoli around the proximal respiratory bronchiole and characteristically has a predominantly upper lung distribution. Although the upper lungs are more severely affected by emphysema, the degree of emphysema in the lower lungs has a stronger correlation with pulmonary function abnormalities. This indicates that the upper lungs are physiologically a relatively silent region, where extensive destruction may occur before functional abnormalities become detectable (115).

Panlobular emphysema has a characteristic lower lobe–predominant distribution; this is the type of emphysema seen in

Table 13.6 FEATURES OF THREE MORPHOLOGIC SUBTYPES OF EMPHYSEMA

<table>
<thead>
<tr>
<th>Centrilobular</th>
<th>Panacinar</th>
<th>Paraseptal</th>
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</thead>
<tbody>
<tr>
<td>Involves central portion of secondary pulmonary lobule</td>
<td>Involves entire secondary pulmonary lobule</td>
<td>Bullae or air cysts in a subpleural location</td>
</tr>
<tr>
<td>Usually a result of cigarette smoking</td>
<td>Seen in α-1-antitrypsin deficiency</td>
<td>Associated with spontaneous pneumothorax</td>
</tr>
<tr>
<td>“Swiss cheese” appearance on CT (early on)</td>
<td>Lower lung–predominant distribution</td>
<td>Most common in lung apices</td>
</tr>
<tr>
<td>“Lung simplification” appearance on CT</td>
<td></td>
<td>CT, computed tomography.</td>
</tr>
</tbody>
</table>
patients with $\alpha$-1-antitrypsin deficiency (Fig. 13.45). In panlobular emphysema, the alveoli are destroyed throughout the secondary pulmonary lobule. The same findings of basilar emphysema can be seen in patients who intravenously inject methylphenidate (crushed Ritalin tablets) (Fig. 13.46) (116).

Paraseptal emphysema is a focal or multifocal abnormality involving the periphery of the pulmonary lobule that is almost always seen in the periphery of the lung along the fissures and at sharp pleural reflections. Coalescence of paraseptal emphysema leads to the formation of bullae and is important in the development of spontaneous pneumothorax (117,118). Paraseptal emphysema should not be confused with honeycombing, which has thicker walls and is associated with fibrosis (119).

Criteria for chest radiographic diagnosis of emphysema include two or more of the following:

1. Depression and flattening of the diaphragm on the posteroanterior (PA) chest radiograph and blunting of costophrenic angles, with the actual level of the diaphragm not as significant as the contour (this can be determined from a straight line connecting the costophrenic junction to the vertebrophrenic junction on each side; if the highest level of the diaphragm contour is less than 1.5 cm above this line, the diaphragm can be recorded as flat).
2. Irregular radiolucency of the lung, as a result of irregularity in distribution of the emphysematous tissue destruction.

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**FIG. 13.45 • Alpha-1-antitrypsin deficiency.** A: PA chest radiograph of a 43-year-old man shows hyperinflation and hyperlucency in the lower lungs. B: Lateral view shows increased retrosternal lucency and flattening of the diaphragm. C: CT shows bullous emphysema in the lower lungs. D: CT at a level superior to (C) shows less severe emphysema. Compared with smoking-related centrilobular emphysema, emphysema caused by $\alpha$-1-antitrypsin deficiency, although diffuse, is more severe in the lower lungs.
3. Increased retrosternal radiolucency, as seen on the lateral view, measuring 2.5 cm or more from the sternum to the most anterior margin of the ascending aorta.

4. Flattening or even concavity of the diaphragm contour on the lateral chest radiograph, as determined by the presence of a sternodiaphragmatic angle of 90 degrees or larger (100) (Fig. 13.47).

**FIG. 13.47** Emphysema. PA (A) and lateral (B) chest radiographs of a long-time cigarette smoker show flattening of the diaphragm with blunting of the costophrenic angles, increased retrosternal lucency, increased AP diameter of the chest, and enlarged central pulmonary arteries. Note that the sternodiaphragmatic angle is greater than 90 degrees, indicative of extreme flattening and even minimal concavity of the diaphragmatic contour, as seen on the lateral view.
Other findings include increased AP diameter of the chest, saber-sheath configuration of the trachea, narrow cardiomediatinal silhouette, and enlargement of the central pulmonary arteries and right ventricle when pulmonary arterial hypertension and cor pulmonale are present, respectively.

Thin-section CT shows centrilobular emphysema as focal areas of low attenuation up to 1 cm in diameter within a homogeneous background of lung parenchyma; occasionally, this results in a “Swiss cheese” appearance. These areas of low attenuation are usually round or oval, have no definable wall, and are often associated with a small centrilobular “dot” representing the normal centrilobular core structures (Fig. 13.48). The appearance of panlobular emphysema on CT is large, extensive areas of uniform low attenuation with a lower lobe–predominant distribution associated with a reduction in the size and number of pulmonary vessels. No peripheral preservation of the lobule occurs, and therefore no striking difference in density exists between affected lobules and a homogeneous background of normal pulmonary parenchyma. Because of this, mild to moderate disease can be easily missed and the extent of disease underestimated (120). Paraseptal emphysema appears as multiple small, subpleural airspaces ranging from a few millimeters to 1 cm in diameter (121).

Bullae, a term used synonymously with blebs, are described as air-filled structures greater than 1 cm in diameter, with thin walls, occurring in a subpleural or intraparenchymal location. They are usually multiple or associated with paraseptal, centrilobular, or panlobular emphysema (122). Giant bullous emphysema, or vanishing lung syndrome (Fig. 13.49), is characterized by large bullae that are several centimeters in diameter and in some cases large enough to fill an entire hemithorax. When giant bullae impair pulmonary function and are associated with compressed lung on CT, the usual method of treatment is surgical resection (bullectomy) (123). On occasion, bullae can become infected and present as cystic masses with air–fluid levels (Figs. 13.50 and 13.51).

**FIG. 13.49** Giant bullous emphysema. PA (A) and lateral (B) chest radiographs of a 47-year-old man with progressive severe shortness of breath shows marked hyperinflation and hyperlucency of the lungs. The vascular markings are sparse (so-called vanishing lung sign), with the majority of residual perfusion going to the right medial base. The crowding of vascular markings at the right lung base, from compressive emphysema, should not be mistaken for focal pneumonia. Pneumothorax can be confused with this appearance; in some cases, CT is the only way to exclude a pneumothorax. C: CT shows a huge bulla in the left upper lobe that displaces the major fissure posteriorly and mediately (arrows) and a prominent bulla in the right lower lobe. Note the abnormal lucent emphysematus spaces within the lungs (L).
The Essentials

References


**FIG. 13.50 ● Infected bulla.** A: PA chest radiograph of a 68-year-old man shows an air-fluid level in the right medial base (arrow). B: Lateral view shows a thin-walled cystic structure with an air-fluid level (arrow) in the right middle lobe. C: CT confirms a thin-walled bulla with an air-fluid level in the right middle lobe (arrow). Additional bullae were seen at several other levels on CT.

**FIG. 13.51 ● Infected bulla.** CT of a 39-year-old man with α-1-antitrypsin deficiency shows a bulla with an air-fluid level (arrow) in the left lower lobe.


**SELF-ASSESSMENT QUESTIONS**

1. What condition is associated with the syndrome shown?

   ![Image A]

   A. Sinusitis  
   B. Laryngitis  
   C. Pharyngitis  
   D. Otitis

2. The patient is an 18-year-old woman with shortness of breath and wheezing. What is the most likely diagnosis?

   ![Image B]

   A. Langerhans cell histiocytosis  
   B. Cystic fibrosis  
   C. Sarcoidosis  
   D. Mycoplasma pneumonia

**Answers**

1. A Sinusitis. The chest radiograph shows situs inversus. The CT scan shows bronchiectasis. Kartagener syndrome consists of situs inversus, bronchiectasis, and sinusitis.

2. B Cystic fibrosis. The chest radiograph shows upper lung-predominant bronchiectasis.