Mosaic attenuation is a CT pattern defined by the Fleischner Society glossary as a “patchwork of regions of differing attenuation seen on CT of the lungs.” It is characterized by heterogeneous lung attenuation with well-defined borders corresponding to the secondary pulmonary lobules. The name derives from mosaic artwork, which consists of variously colored glass or stone tiles inlaid in a contrasting pattern (Fig. 1). The three major pathologic causes of this pattern are pulmonary vascular, small airways, and primary parenchymal disease. These entities can be differentiated on CT by correlating inspiratory with expiratory images and evaluating the appearance of the pulmonary vasculature.

The mosaic pattern can be explained by a nonuniform distribution of disease. In pulmonary vascular disease, hypoperfused lung appears lower in attenuation than adjacent normal or hyperperfused lung. Similarly, in small airways disease, regional variations in the presence of air trapping lead to a patchwork of low-attenuation lung that is interposed with normally ventilated higher-attenuation lung. Conversely, in primary parenchymal disease, the higher-attenuation lung is abnormal and, when heterogeneously distributed, contrasts with adjacent normal lower-attenuation lung to produce a patchwork mosaic pattern.

**Pitfalls in Classification**

The recognition of a mosaic pattern of attenuation is usually straightforward, but differentiation between small airways, vascular, and primary parenchymal disease can be challenging. The presence of air trapping excludes primary parenchymal disease unless there is a suspicion of hypersensitivity pneumonitis, which has mixed features of both parenchymal and small airways disease. Contrary to prior belief, air trapping can rarely occur in pulmonary vascular disease and thus may be a confusing sign, requiring a search for ancillary features that suggest a primary vascular cause of mosaic attenuation.

Mosaic attenuation during expiratory CT sequences can be seen in normal individuals. If paired inspiratory images are normal and the extent of involvement is minor (i.e., three or fewer secondary pulmonary lobules), the presence of related small airways abnormalities should be determined to avoid an overdiagnosis of air trapping when there are physiologic small volumes of mosaic attenuation. The most common causes of mosaic attenuation and their respective CT features are summarized in Figure 2.

**Pulmonary Vascular Disease**

Pulmonary vascular disease can be difficult to detect clinically because the symptoms and signs of pulmonary hypertension are nonspecific. Echocardiography and cardiac catheterization are the most commonly used diagnostic studies. Mosaic attenuation is most frequently observed in patients with secondary pulmonary hypertension and is less common in primary pulmonary hypertension and acute pulmonary embolism.

**Pulmonary Hypertension**

The mosaic pattern related to pulmonary hypertension consists of regions of hypoattenuation due to hypoperfusion interposed with areas of hyperattenuation where there is normal or excessive perfusion (Fig. 3). Mosaic attenuation most commonly occurs in patients with severe pulmonary hypertension because of chronic pulmonary embolism, pulmonary venoocclusive disease, and pulmonary edema.
Mosaic Attenuation

disease, or collagen vascular disease. Classic CT features of pulmonary hypertension include right ventricular enlargement, bulging of the interventricular septum, and central pulmonary arterial enlargement with peripheral tapering (Fig. 3).

Small airways disease and pulmonary vascular disease can usually be distinguished on expiratory CT images because air trapping is not a typical feature of pulmonary vascular disease. However, recent data suggest that air trapping can rarely occur in pulmonary vascular disease complicated by airway involvement.

Primary Pulmonary Arterial Hypertension

Primary pulmonary arterial hypertension is a subtype of pulmonary hypertension in which no cause is identified. Typically affecting young women (20–45 years), primary pulmonary hypertension is characterized histologically by a plexiform arteriopathy that consists of intimal cellular proliferation in small and medium pulmonary arteries that interrupts the internal elastic lamina and involves the alveolar capillaries. It is occasionally identifiable on CT as peripheral serpiginous pulmonary vessels that do not arise from pulmonary veins. The typical CT features of pulmonary hypertension may also be present. The distribution of mosaic attenuation in primary pulmonary hypertension is described as heterogeneous or patchy with a perivascular distribution, which differs from the typical segmental and well-defined distribution of mosaic attenuation associated with chronic pulmonary embolism.

Fig. 1—Example of mosaic artwork.

Fig. 2—Flowchart shows causes of mosaic attenuation pattern.
Chronic Pulmonary Embolism

Chronic pulmonary arterial hypertension is reported to occur in approximately 4% of patients with acute pulmonary embolism, in whom repetitive thromboembolism leads to elevated pulmonary artery pressure. The true incidence is thought to be higher due to clinically silent acute pulmonary embolism. CT features that suggest chronic pulmonary embolism include weblike pulmonary arterial filling defects, serpiginous pulmonary arteries, and hypertrophic bronchial arteries (Fig. 4).

Lung parenchymal features include segmental and subsegmental mosaic attenuation with well-defined borders and scarring at the site of previous infarcts. Air trapping may rarely occur and is postulated to result from bronchoconstriction in hypoperfused lung mediated by increased
**Mosaic Attenuation**

expression of the potent bronchoconstrictor endothelin-1 and underexpression of the potent bronchodilator nitric oxide. Mild bronchiectasis in segments of hypoperfused lung has also been described in patients with chronic pulmonary embolism.

**Pulmonary Venoocclusive Disease**

Pulmonary venoocclusive disease is a rare subtype of pulmonary arterial hypertension. This condition has been associated with viral infection, chemotherapy, autoimmune disease, intracardiac shunts, radiation injury, and genetic predisposition. There is clinical, radiologic, and pathologic overlap between pulmonary venoocclusive disease and the angio proliferative process of pulmonary capillary hemangiomatosis. In both entities, pulmonary venous thrombosis and fibrosis cause increased pulmonary venous pressure; pulmonary edema; and, rarely, hemorrhage. Characteristic findings of pulmonary venoocclusive disease include centrilobular ground-glass opacity, septal thickening, and pleural effusions. Mosaic attenuation was described in one half of the patients in a small study of pathologically confirmed pulmonary venoocclusive disease. The characteristic radiologic features of pulmonary arterial hypertension are also observed when the disease is severe.

**Collagen Vascular Disease**

Pulmonary hypertension is most commonly described in the limited form of scleroderma: calcinosis, Raynaud phenomenon, esophageal dysmotility, sclerodactyly, telangiectasia (CREST) and in rheumatoid arthritis. In both conditions, pulmonary hypertension is thought to be a manifestation of pulmonary fibrosis rather than of vascular cause, and the appearance is identical to the previously described findings of pulmonary hypertension.

**Small Airways Disease**

Air trapping due to bronchial or bronchiolar obstruction occurs in small airways disease. The resultant mosaic attenuation pattern consists of regional hypoa attenuation where air trapping has occurred interposed with regions of hyper attenuation representing normal ventilation. Mosaic attenuation may be observed on inspiratory images and is accentuated at end-expiration. The lung that retains air does not decrease in volume relative to adjacent normal lung on paired inspiratory and expiratory images. This is in contrast to pulmonary vascular or primary parenchymal disease, in which there is a homogeneous decrease in lung volume. In severe cases of small airways disease, there is a decrease in the number and caliber of pulmonary vessels in the lucent lung compared with the adjacent normal lung, which reflects hypoxic vasoconstriction in the hypoventilated lung and relative hyperperfusion of the normally ventilated lung. According to a study of ventilation-perfusion SPECT in patients with the mosaic attenuation pattern, hypoperfusion may contribute to heterogeneous attenuation in patients with severe obstructive airways disease.

Air trapping on CT is dependent on the respiratory effort of the patient at the time of image acquisition. Therefore, it may not be reproducible, particularly in an acutely dyspneic patient. The anatomic location and extent of air trapping, which have been shown to be reproducible CT features when performed in a group of stable heart-lung transplant recipients, are therefore most reliable in functionally stable patients. In this study, air trapping in conjunction with bronchial wall thickening was found to be the most reliable indicator of obstructive airways disease when correlated with pulmonary function tests. Mosaic attenuation and mucous plugging were found to be the second most reliable indicators, with airway dilatation and centrilobular nodules the least consistent features.

**Bronchiolitis**

Two distinct entities previously shared the name “bronchiolitis obliterans,” despite being dissimilar both clinically and pathologically. Constrictive bronchiolitis is characterized by peri-bronchial fibrosis and leads to chronic airflow obstruction with resultant mosaic attenuation and air trapping. Bronchiolitis obliterans organizing pneumonia, now termed “cryptogenic organizing pneumonia,” is characterized by intraluminal granulation tissue accompanied by consolidation and is not a common cause of the mosaic attenuation pattern. Another uncommon cause of mosaic attenuation is cellular bronchiolitis, which may precede constrictive bronchiolitis or be self-limiting. It is included in this discussion because a subtype of cellular bronchiolitis, hypersensitivity pneumonitis, typically exhibits the mosaic attenuation pattern.
Idiopathic constrictive bronchiolitis is rare. Constrictive bronchiolitis is usually secondary to postinfectious scarring; noxious fume inhalation; graft versus host disease; lung transplantation; rheumatoid arthritis; or, rarely, diffuse idiopathic pulmonary neuroendocrine cell hyperplasia. It is defined histologically as concentric luminal narrowing of the membranous and respiratory bronchioles secondary to peribronchiolar inflammation and fibrosis.

**Air Trapping**

Direct signs of bronchiolitis are uncommon in the constrictive form because the amount of bronchiolar inflammation is small. Indirect signs of bronchiolitis predominate, including heterogeneously distributed mosaic attenuation, bronchial dilation, and dramatic air trapping. Viral and *Mycoplasma* pneumonia are the most common infections that can result in constrictive bronchiolitis. In lung transplant recipients, constrictive bronchiolitis is known as "bronchiolitis obliterans syndrome" and is the most common cause of chronic rejection. Typical findings include bronchial wall thickening, bronchial dilation, mosaic attenuation, and air trapping (Fig. 5).

**Cryptogenic Organizing Pneumonia**

Cryptogenic organizing pneumonia was previously termed "bronchiolitis obliterans organizing pneumonia" because of the histologic finding of granulation tissue within the lumina of bronchioles and alveolar ducts. The clinical presentation may be subacute and consists of malaise followed by dyspnea and cough. It is usually idiopathic but can occur as a reaction to infectious pneumonia, drugs, or chronic eosinophilic pneumonia or as a complication of collagen vascular disease. The characteristic CT appearance of cryptogenic organizing pneumonia includes peripheral or peribronchial consolidation, reticulonodular opacities, and bronchial dilation. Randomly distributed ground-glass attenuation with a mosaic appearance is a nonspecific feature of cryptogenic organizing pneumonia (Fig. 6).

**Cellular Bronchiolitis**

Cellular bronchiolitis is a common histologic pattern found in hypersensitivity pneumonitis and infectious, aspiration (Fig. 7), respiratory, and follicular bronchiolitides.
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Although these entities have some similarities on CT, they are usually distinguishable by clinical history. The most commonly described CT features of bronchiolitis include centrilobular and branching tree-in-bud nodules that represent cellular debris within the bronchiole. Mosaic attenuation and air trapping are described in cellular bronchiolitis of all causes but occur most typically in subacute hypersensitivity pneumonitis. This condition involves both small airways and lung parenchyma and is further discussed in the section on primary parenchymal disease.

Asthma

Asthma is a chronic immunologically mediated condition caused by a disturbance of the normal airway repair mechanism. It manifests clinically as episodic attacks of dyspnea, cough, and wheezing due to bronchoconstriction, inflammation, and airway remodeling. Although CT is not a first-line imaging modality for the investigation of asthma, features include bronchial wall thickening, bronchiectasis, and emphysema. Mosaic attenuation and air trapping are indirect features of asthma (Fig. 8). It is important to differentiate significant mosaic attenuation from minor physiologic variations in attenuation that involve three or fewer secondary pulmonary lobules because this appearance has been observed with equal frequency in asthmatics and healthy subjects (Fig. 9). More extensive mosaic attenuation and air trapping, involving more than one third of the lung, are sensitive indicators for the presence and severity of asthma and correspond with pulmonary function test abnormalities.

Fig. 7—Chemical bronchiolitis. Transverse CT image shows mosaic attenuation in patient with witnessed episode of aspiration.

Fig. 8—Air trapping in patient with acute exacerbation of asthma. A and B, Transverse inspiratory (A) and expiratory (B) CT images show mosaic attenuation in both upper lobes with marked accentuation at held expiration; More lucent lung parenchyma does not decrease in volume relative to adjacent normal lung on expiratory images.

Fig. 9—Air trapping in healthy subject. A and B, Transverse inspiratory (A) and expiratory (B) CT images show minor expiratory air trapping (arrows, B) in normal asymptomatic individual.
Primary Parenchymal Disease

Mosaic attenuation in primary parenchymal disease is due to interstitial involvement or filling of air spaces with fluid, cells, or fibrosis interposed with normal hypoattenuating lung. It is not associated with regional variations in vessel size. Mosaic attenuation is most commonly observed in *Pneumocystis jiroveci* pneumonia, hypersensitivity pneumonitis, and pyogenic pneumonia. It is a rare manifestation of nonspecific and desquamative interstitial pneumonitis. Expiratory CT scans typically show a uniform decrease in lung volume and increase in attenuation of the affected lung parenchyma. One exception is hypersensitivity pneumonitis, in which air trapping can occur.

**Pneumocystis Jiroveci Pneumonia**

This fungal pulmonary infection develops almost exclusively in immune-compromised patients, especially in those with AIDS who have a CD4 count below 200 cells/mm³. Rather than on the basis of CT features alone, the diagnosis is usually made on the basis of a high index of suspicion when clinical features of fever, hypoxia, and a dry cough develop in an immune-compromised patient. The characteristic CT feature is ground-glass attenuation, which may be diffuse or perihilar or have a mosaic pattern (Fig. 10). Mosaic attenuation has also been described in patients with pyogenic pulmonary infections.

**Hypersensitivity Pneumonitis**

Hypersensitivity pneumonitis, an immunologic reaction to inhaled organic material, is most commonly seen in bird fanciers and farmers. The symptoms of acute hypersensitivity pneumonitis, which typically develop 4–12 hours after dust exposure, include cough, fever, chills, and myalgia. The insidious onset of chronic hypersensitivity pneumonitis may prevent identification of a causative antigen. As a result, this condition may be underdiagnosed. The typical CT appearance consists of poorly defined centrilobular nodules with a lower lobe distribution in the acute phase, which can progress to heterogeneous ground-glass attenuation with upper or lower lobe fibrosis in the chronic stage. Mosaic attenuation can be seen at any stage and is the most common imaging finding (Fig. 11). Air trapping, when present, is due to cellular bronchiolitis. Hypersensitivity pneumonitis differs from other bronchiolitides in that...
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defines the nodules are less well circumscribed and
and a tree-in-bud pattern is infrequent.

**Interstitial Pneumonias**

Desquamative interstitial pneumonitis is a rare smoking-related lung disease. A mosaic pattern develops more frequently in this condition than in other idiopathic interstitial pneumonias. Other CT features include scattered ground-glass opacities and the development of cystic spaces. Mosaic attenuation may rarely be seen in heterogeneously distributed nonspecific interstitial pneumonitis and usual interstitial pneumonitis (Fig. 12).

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