High-resolution CT (HRCT) has become a valuable tool for the evaluation of patients with diffuse pulmonary diseases. HRCT is now widely recognized as more sensitive and specific than chest radiography for the assessment of such patients, and it has been integrated into the diagnostic algorithms for the assessment of a number of diffuse lung processes, most notably the idiopathic interstitial pneumonias, eosinophilic lung diseases, and obstructive lung diseases. Furthermore, HRCT has become a front-line test for the evaluation of patients with a number of very common clinical complaints, including patients with chronic cough and progressive shortness of breath or exertional dyspnea. Because HRCT is a commonly requested imaging technique, familiarity with the basis of interpretation of HRCT images is critical for accurate diagnosis.

In essence, HRCT imaging, by use of narrow collimation and high spatial frequency reconstruction algorithms, seeks to maximize spatial resolution and thereby approach a pathologic representation of a disease process. Maximizing spatial resolution allows HRCT findings frequently to correlate closely with pathologic findings. As stated in the forward to the first edition of *High-Resolution CT of the Lung* by Webb et al [1], “...to the extent that gross pathology can be used to diagnose lung disease, HRCT can as well.” In some circumstances, this observation may even be extended to the histopathologic level. For this reason, much of the HRCT literature has focused on radiologic–pathologic correlations for various pulmonary diseases to improve appreciation for disease patterns on imaging. It is clear that detailed knowledge of normal pulmonary anatomy and an understanding of how normal anatomy is altered in disease states are required to appreciate fully HRCT findings in patients with pulmonary disease. With such a foundation, a pattern approach to HRCT interpretation may be used successfully.

**Pulmonary anatomy: the requisites for high-resolution CT interpretation**

**The pulmonary interstitium**

A basic understanding of pulmonary anatomy is required for accurate HRCT interpretation. Pulmonary anatomy may be broadly divided into the pulmonary gas exchange units and the pulmonary interstitium. The pulmonary interstitium may be further subdivided into the central peribronchovascular interstitium and the peripheral centrilobular interstitium; these two fiber networks are continuous with one another. The central peribronchovascular inter-
stitium invests the larger central bronchi and vessels near the pulmonary hilum and courses peripherally, producing the peripheral centrilobular interstitium, eventually merging with the subpleural interstitial fiber network. The latter is located immediately beneath the visceral pleura and extends into the underlying lung parenchyma at various intervals to produce interlobular septa.

In the peripheral lung, the components of the pulmonary gas exchange units, including the respiratory ducts, alveolar ducts, and alveoli, are suspended from the interlobular septa and the peripheral centrilobular interstitium by the intralobular interstitium. Fibers of the intralobular interstitium consist of a very fine web of connective tissue that is not routinely visible on HRCT studies.

The secondary pulmonary lobule

The secondary pulmonary lobule is defined as the smallest unit of lung function marginated by connective tissue septa; these connective tissue septa are the interlobular septa [1]. Pulmonary veins course within the interlobular septa at the edges of a secondary pulmonary lobule (Fig. 1). Secondary pulmonary lobules vary in size from 1 to 2.5 cm and are usually most easily visible over the upper lobes, the anterior and lateral aspects of the right middle lobe and lingula, and over the diaphragmatic surfaces of the lower lobes, where they are the most well developed. On average, each secondary pulmonary lobule contains 12 or fewer pulmonary acini. Secondary pulmonary lobules are supplied by an artery and bronchus, termed the “centrilobular artery and bronchus.” The centrilobular artery and bronchus branch dichotomously within the secondary pulmonary lobule, successively producing intralobular arteries and bronchi, acinar arteries, and respiratory bronchioles, eventually terminating in pulmonary gas exchange units.

Intralobular anatomy

The centrilobular artery and bronchus are approximately 1 mm in diameter and are located about 5 to 10 mm from the visceral pleural surface [1]. Intralobular arteries are slightly smaller, and smaller still are the acinar arteries, which vary in size from 0.3 to 0.5 mm (see Fig. 1) [1–3]. Acinar arteries may be visible on HRCT scans as a small dot positioned about 3 to 5 mm from interlobular septa or the visceral pleural surface [2,3]. Although very small arteries within the secondary pulmonary lobule are often visible on clinical HRCT scans, the resolution of HRCT for tubular structures, such as bronchi, is considerably less. Bronchi within the secondary pulmonary lobule are not normally visible on HRCT.
High-resolution CT technique

Narrow collimation and the use of a high spatial frequency reconstruction algorithm are the two most important technical factors that distinguish a thoracic CT examination as an HRCT study. Other technical modifications may be used to enhance the quality of an HRCT examination, such as targeted reconstructions and higher kilovolt (peak) or milliamperage values [4], but these techniques are not required to produce diagnostic-quality HRCT images. In fact, in recent years increasing awareness of the radiation dose attributable to diagnostic imaging, in particular CT, has led a number of investigators to reduce kilovolt (peak) and milliampere to limit patient radiation dose [5–11]. In general, it has been shown that diagnostic-quality HRCT examinations may be obtained with substantially decreased radiation doses compared with standard-dose HRCT examinations [8,11]. Nevertheless, because some subtle abnormalities may be less visible on HRCT examinations performed with reduced-dose technique [5], and because optimal low-dose HRCT techniques likely
vary among patients and indications and have not yet been established, several investigators have advocated that initial HRCT examinations be performed with standard-dose techniques and that low-dose HRCT be reserved for following patients with known abnormalities or screening large numbers of patients at high risk for a particular disease [1].

Display parameters, especially window width and level, are crucial for accurate HRCT interpretation. In general, window levels ranging from −600 to −700 HU and window widths ranging from 1000 to 1500 HU are appropriate for displaying “lung windows.” Window widths below 1000 HU produce too much contrast for optimal viewing, whereas excessive window widths inappropriately decrease the contrast between and adjacent structures and can render fine detail inconspicuous. Once proper display parameters are chosen, the same parameters should be used when evaluating serial studies for a particular patient. Viewing serial imaging using different display parameters makes determination of interval change difficult and can contribute to diagnostic inaccuracy.

Collimation

HRCT imaging requires narrow collimation, usually on the order of 1 mm, to achieve maximal spatial resolution, and is typically performed at full inspiration. Usually, HRCT imaging is performed using

<table>
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<td><strong>HRCT finding</strong></td>
<td><strong>Further pattern subclassification</strong></td>
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<td><strong>Increased lung capacity</strong></td>
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<td>Nodules</td>
<td>Centrilobular, perilymphatic, random</td>
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<td>Linear abnormalities</td>
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<td>Reticular abnormalities</td>
<td>Coarse or fine reticulation, intralobular interstitial thickening</td>
</tr>
<tr>
<td>Ground-glass opacity</td>
<td>Must be based on clinical history and associated scan findings</td>
</tr>
<tr>
<td>Consolidation</td>
<td>Must be based on clinical history and associated scan findings</td>
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<tr>
<td><strong>Decreased lung capacity</strong></td>
<td></td>
</tr>
<tr>
<td>Areas of decreased attenuation with walls (cysts or cysticlike appearance)</td>
<td>Cyst shape, distribution, wall thickness, pattern of organization</td>
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<tr>
<td>Areas of decreased attenuation without walls</td>
<td>Emphysema, mosaic perfusion</td>
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</table>

Fig. 4. HRCT findings in patients with diffuse lung disease. Centrilobular nodules with (small black arrow) and without (double small black arrows) tree-in-bud; perilymphatic nodules (black arrowhead); peribronchovascular nodules (double black arrowheads); ground-glass opacity (G); consolidation (C); lobular low attenuation representing mosaic perfusion (white arrow); parenchymal bands (white double arrows); subpleural lines (white arrowhead); paraseptal (double white arrowheads) and centrilobular emphysema; and lung cysts (*).
axial technique. The rapid acquisition times provided by multislice CT (MSCT) have allowed the relatively recent development of volumetric HRCT, a technique that is described later.

Typical HRCT protocols (Table 1) use 1- to 1.5-mm collimation every 10 to 20 mm throughout the thorax, which effectively images only approximately 10% of the lung parenchyma. Because HRCT is typically used for the assessment of diffuse lung disease, such a sampling technique provides adequate representation of the disease process while minimizing the radiation dose delivered to the patient.

Supine HRCT protocols, often used for the assessment of patients with suspected obstructive lung diseases (including bronchiectasis, emphysema, and bronchiolitis obliterans), patients with suspected opportunistic infections (eg, *Pneumocystis jiroveci* pneumonia), and patients with suspected cystic lung disease (see Table 1) typically use 10-mm spacing between images (interslice gap). HRCT protocols using both supine and prone imaging often use a 20-mm interslice gap; the radiation dose associated with supine and prone HRCT imaging is nearly equivalent to that delivered by supine HRCT protocol.

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**Fig. 5.** Nodules on HRCT: distribution within the secondary pulmonary lobule. *(A)* Perilymphatic nodules. Nodules are immediately in contact with interlobular septa and the visceral pleura. *(B)* Centrilobular nodules. Nodules are positioned 5 to 10 mm from costal and visceral pleural surfaces and interlobular septa. Note that peribronchovascular nodules are also often present with pathologic processes that involve the lymphatic tissues, and may be part of the spectrum of perilymphatic nodules. For this reason, centrilobular nodules are seen with processes producing perilymphatic nodules, but they are not the predominant pattern. *(C)* Random nodules. Random nodules show no obvious relationship to any secondary pulmonary lobular structures. They are found in relation to the visceral pleura, interlobular septa, and center of the lobule roughly equally.
Supine and prone HRCT imaging is often used for the evaluation of patients with suspected idiopathic interstitial pneumonias or patients with restrictive patterns on pulmonary function testing (see Table 1). Prone imaging is essential in this context for distinguishing dependent density (atelectasis) from pulmonary inflammation or fibrosis-atelectasis detected on supine imaging resolves with prone imaging (Fig. 2), whereas alveolitis or fibrosis persists on prone imaging (Fig. 3) [12–14]. Furthermore, the relatively small interslice gap used with supine HRCT protocols allows one to track abnormalities more easily sequentially from image to image (eg, ectatic bronchi in patients with bronchiectasis) than do prone protocols that use a wider interslice gap.

Expiratory scanning
Expiratory scanning is a critical component to any HRCT protocol [15]. Expiratory HRCT may be performed using static HRCT methods or dynamic expiratory HRCT (imaging during a forced vital capacity maneuver). Static methods of expiratory scanning image the patient’s lungs at or near functional residual capacity, and are performed by imaging after complete exhalation (“take a deep breath in and blow it all out”) or using lateral decubitus CT [16]. The latter is particularly useful when expiratory imaging is desired but patient cooperation with specific breathing instructions is not ensured, as is often the case when language barriers are present.

Fig. 6. Centrilobular nodules: *Mycobacterium avium*—complex infection. (A) Axial CT shows right middle lobe and lingular predominant bronchiectasis with bronchiolar impaction, representing tree-in-bud. The findings present within the rectangle enclosing part of the lingula are detailed in B. (B) Schematic view of A details the centrilobular nodules. The branching nodules represent bronchiolar impaction (tree-in-bud), with smaller, clustered nodules representing rosettes. (C) Gross specimen shows centrilobular nodules (arrows). The thin rim of lung peripheral to the nodules is consistent with a centrilobular position. Note ectatic bronchus (arrowhead), confirming airway disease. (D) Histopathologic specimen shows centrilobular nodules (arrows) and abnormally dilated airway (arrowhead). Note position of nodules from visceral pleura at top of image.
Dynamic expiratory HRCT is performed by imaging the patient during a forced vital capacity maneuver, using either a spiral CT scanner or an electron-beam CT scanner [17–19]. Dynamic expiratory HRCT is also performed easily with MSCT scanners. Images are acquired at user-selected levels with imaging performed in cine mode (without table increment), usually for six to eight images per level [17,19]. Dynamic expiratory HRCT provides a greater overall increase in lung attenuation compared with static expiratory methods and may be more sensitive for the detection of subtle or transient air trapping than static expiratory methods. Dynamic expiratory HRCT may be performed using low-dose techniques with no compromise in diagnostic quality [19].

**Volumetric (multislice) high-resolution CT**

Volumetric HRCT has been performed using several different methods, including clustered axial scans at user-selected levels [20]; single breathhold single-slice CT [21,22]; and, most recently, entire-thorax MSCT-HRCT [23–27]. Although volumetric HRCT of the chest was performed with some success using conventional and single-slice CT methods, until the introduction of MSCT, the difficulty inherent in imaging the entire thorax with these older methods limited their use. MSCT, particularly scanners with 16 detectors or greater, easily allows imaging of the entire thorax using 1-mm collimation within a single breathhold. The volumetric dataset obtained with current MSCT scanners allows near-isotropic imaging, which provides the ability to view the dataset in any desired plane and for the creation of maximum intensity or minimum intensity projected images, also in any desired plane or level. For example, volumetric MSCT-HRCT may provide improved assessment of the distribution of parenchymal lung abnormalities in patients with diffuse lung diseases [24,28], and volumetric MSCT-HRCT may allow for simultaneous assessment of small airway and large airway pathology [28]. The major drawback to the widespread use of volumetric MSCT-HRCT is the increased radiation dose: volumetric MSCT-HRCT studies may deliver more than five times the radiation dose compared with routine axial HRCT techniques [23].

**High-resolution CT patterns of disease**

An organized approach to HRCT scan findings is critical to successful interpretation. Although simple pattern recognition can often provide the correct diagnosis in a number of cases, a firm understanding of the pathologic presentations of disease on HRCT is far more rewarding in many circumstances.

HRCT scan findings may be broadly classified into findings of increased lung opacity (Table 2) and decreased lung opacity (Table 2 and Fig. 4). HRCT disease patterns, both those manifesting as increased lung opacity and those manifesting as decreased lung opacity, may be further subclassified to facilitate organization and differential diagnosis. Occasionally, findings of increased and decreased opacity may be present on the same imaging study, either reflecting the presence of two or more diseases or, in certain cases, a pathologic process that manifests with both an infiltrative and obstructive process.

**High-resolution CT scan findings manifesting as increased lung opacity**

HRCT scan findings manifesting as increased lung opacity may be further subclassified into nodular abnormalities, linear abnormalities, reticular ab-

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**Table 3**

<table>
<thead>
<tr>
<th>Nodule distribution on HRCT</th>
<th>Relevant secondary pulmonary lobular anatomic structures</th>
<th>Representative diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Centrilobular</td>
<td>Centrilobular artery and bronchus</td>
<td>Infectious bronchiolitis, diffuse panbronchiolitis, hypersensitivity pneumonitis, respiratory bronchiolitis, lymphocytic interstitial pneumonia, pulmonary edema, vasculitis, plexogenic lesions of pulmonary hypertension, metastatic neoplasms</td>
</tr>
<tr>
<td>Perilymphatic</td>
<td>Interlobular septa, subpleural interstitium, centrilobular bronchus</td>
<td>Sarcoidosis, lymphangitic carcinomatosis, amyloidosis</td>
</tr>
<tr>
<td>Random</td>
<td>All structures of the lobule</td>
<td>Hematogenously disseminated infections and neoplasms</td>
</tr>
</tbody>
</table>
normalities, ground-glass opacity, and consolidation (see Table 2).

Nodules

A pulmonary nodule may be broadly defined as any relatively sharply defined, discrete, nearly circular opacity within the lung, ranging in size from 2 to 30 mm. Nodules are usually further characterized with respect to size, border definition, density, number, and location. The term “micronodule” is occasionally used when describing HRCT findings, usually referring to nodules less than 3 to 7 mm in size, but the significance of this designation is uncertain [29].

![Image](image_url)

Fig. 7. Centrilobular nodules: *Mycobacterium tuberculosis* infection. (A) Axial HRCT image through the upper lobes shows peripheral, branching opacity consistent with bronchiolar impaction (tree-in-bud) (arrow). (B) Sagittal maximum intensity projected image from Fig. 7A demonstrates centrilobular nodules (arrows), some showing tree-in-bud (arrowhead). Note that the nodules approach, but do not contact, pleura, consistent with a centrilobular distribution. (C) Gross specimen shows tree-in-bud opacity (arrows).

The diagnostic value of HRCT for the assessment of diffuse nodular diseases relies heavily on the distribution of the nodules relative to the secondary pulmonary lobule, a diagnostic approach that was first recognized as valuable for interpretation of biopsy and surgical histopathologic specimens. HRCT technique allows imagers to extrapolate these pathologic findings to imaging findings [30]. Histopathologically, at least four nodule distributions within the secondary pulmonary lobule are recognized: (1) bronchiolocentric, (2) angiocentric, (3) lymphatic, and (4) random [30]. Nodules that are bronchiolocentric in distribution are related to the centrilobular and lobular bronchi, and angiocentric nodules are related...
to the pulmonary arteries within the secondary pulmonary lobule; because the artery and bronchus are in close proximity to one another, both nodule types are located in or very near the center of the secondary pulmonary lobule. These two distributions are not readily distinguished from one another on HRCT, so bronchiolo-centric and angiocentric nodule histopathologic distributions are grouped together as centrilobular nodules, and three distributions of nodules within the secondary pulmonary lobule are recognized on HRCT: (1) centrilobular, (2) perilymphatic, and (3) random (Fig. 5).

Centrilobular nodules. Centrilobular nodules are distributed primarily within the center of the secondary pulmonary lobule (see Figs. 5B and 6). Centrilobular nodules range in size from a few millimeters to slightly greater than 1 cm, and may be well-defined or ill-defined, depending on the underlying disease process. A centrilobular nodular distribution may be recognized when nodules are roughly evenly spaced from one another and approach, but do not contact, visceral pleural surfaces (see Figs. 4, 5B, and 6); the nodules are usually positioned about 5 to 10 mm from the visceral pleural surface. Because the centrilobular artery and bronchus are the structures that predominate in the center of the pulmonary lobule, diseases affecting these two anatomic structures account for most processes that produce centrilobular nodules on HRCT (Table 3).

Centrilobular nodules may be further characterized by the presence or absence of a branching configuration, so-called “tree-in-bud.” Tree-in-bud reflects the presence of impaction of the centrilobular bronchus with mucous, pus, or fluid, resulting in dilation of the bronchus, associated with peribronchial inflammation (see Figs. 6A, 6B, and 7) [31]. Dilated, impacted bronchi produce Y- or V-shaped structures on HRCT imaging, and have been likened to a budding tree in spring [32,33], hence the term “tree-in-bud.” The presence of centrilobular nodules with tree-in-bud morphology is very diagnostically useful, because this finding is almost always seen with pulmonary infections. When centrilobular nodules are present but tree-in-bud morphology is absent, infections remain a consideration, but the differential diagnosis must be expanded (Box 1) to include several noninfectious etiologies and certain vascular lesions. Despite the relatively large and varied differential diagnosis that requires consideration when centrilobular nodules without tree-in-bud are encountered, other features of the nodules themselves may provide useful information. For instance, poorly defined centrilobular nodules distributed evenly from pulmonary apex to base are characteristic of subacute hypersensitivity pneumonitis, whereas well-defined centrilobular nodules in the posterior portions of the upper lobes, associated with nodules in the subpleural region of lung, are suggestive of silicosis (Fig. 8). Additionally, other findings are often present to assist in generating a sensible differential diagnosis. For example, well-defined centrilobular nodules, associated with bizarre-shaped cysts distributed primarily within the upper lobes in a smoker, are characteristic of Langerhans’ cell histiocytosis [29,34].

Perilymphatic nodules. Perilymphatic nodules are seen with diseases that preferentially involve lymphatic structures, such as sarcoidosis, lymphangitic carcinomatosis, lymphoproliferative disorders, and amyloidosis (Box 2). Pulmonary lymphatics are normally found within the visceral pleura, within the interlobular septa, and along the veins and bronchovascular bundles, so diseases involving the lym-
phatics may produce nodules in relation to these structures (see Fig. 5A). Perilymphatic nodules are recognized on HRCT as nodules that abut costal and fissural pleural surfaces, usually in a patchy distribution (Fig. 9). Note that because pulmonary lymphatics are present along bronchovascular bundles, centrilobular nodules are commonly also seen in diseases producing perilymphatic nodules; however, the nodules are predominately found along interlobular septa and the visceral pleura and not within the center of the lobule.

Random nodules. Random nodules show no definable distribution relative to the secondary pulmonary lobule; nodules are seen in the center of the lobule and in contact with interlobular septa and visceral pleural surfaces (see Fig. 5C). The differential diagnosis of randomly distributed nodules on HRCT is listed in Box 3. Random nodules, in contrast to perilymphatic nodules, usually do not show a patchy distribution in the lung parenchyma; rather, random nodules are usually distributed uniformly throughout the lung parenchyma in a bilaterally symmetric distribution (Fig. 10).

Anatomic nodule localization. Localizing nodules on HRCT begins first with assessing whether or not subpleural nodules are present (nodules in contact with the visceral pleura surfaces, either costal pleura or fissures) (Fig. 11). If subpleural nodules are absent, then the nodules are centrilobular in location (see Figs. 5B and 6–8). Once nodules are identified as centrilobular, one should search for tree-in-bud. The presence of tree-in-bud opacity essentially limits the differential diagnosis to infections (see Box 1). If subpleural nodules predominate, then either a perilymphatic or random distribution is present. In this case, the overall distribution of nodules, with reference to the upper, mid, and lower lungs, should be assessed. If the nodules are patchy in distribution, a perilymphatic distribution is present (see Fig. 9). If the nodules are scattered rather evenly throughout the upper, mid, and lower lungs, a random distribution is present (see Fig. 10).

When small nodules detected on HRCT are approached using the anatomic localization method just described, HRCT possesses a high diagnostic

**Box 2. Perilymphatic nodules: diagnostic considerations**

- Sarcoidosis
- Lymphangitic carcinomatosis
- Follicular bronchiolitis and lymphocytic interstitial pneumonia
- Lymphoproliferative disorders
- Amyloidosis
accuracy. In a study by Gruden et al [34], anatomic nodule localization using the method described previously allowed the proper classification of the nodule distribution in 94% of cases with high interobserver agreement.

Linear abnormalities

A number of linear abnormalities may be evident on HRCT scans of the thorax, including interlobular septal thickening, parenchymal bands, subpleural lines, and irregular linear opacities. Among these linear findings on HRCT, interlobular septal thickening is the most diagnostically useful.

Interlobular septal thickening. Interlobular septa are normally about 0.1 mm thick and are just at the threshold for detection on HRCT imaging. Visualization of a few interlobular septa, usually anteriorly or along the mediastinal pleural surfaces, is normal, but visualization of numerous septa indicates an abnormal condition. Thickening of interlobular septa may be seen in conditions associated with dilatation of the pulmonary veins; infiltration of the pulmonary lymphatics; or with infiltration of the pulmonary interstitium by cells, fluid, or fibrosis. Thickened septa should be characterized as smooth, nodular, or irregular (Box 4). Smooth interlobular septal thickening is commonly seen with pulmonary edema (Fig. 12) and pulmonary alveolar proteinosis, among other etiologies (see Box 4). Lymphangitic carcinomatosis also often produces smooth interlobular septal thickening, although nodular interlobular septal thickening is more characteristic.

Nodular interlobular septal thickening is typical of diseases that involve the pulmonary lymphatics, particularly lymphangitic carcinomatosis (Fig. 13) and sarcoidosis. Both of these diseases may also produce perilymphatic nodules, but sarcoidosis is often associated with architectural distortion, reflecting underlying pulmonary fibrosis, whereas lymphangitic carcinomatosis is a nonfibrosing process and does not produce architectural distortion.

Irregular interlobular septal thickening is usually seen in fibrosing lung diseases (Fig. 14). Generally, the presence of irregular interlobular septal thickening is of limited diagnostic value and usually other findings are present on HRCT to assist in generating a differential diagnosis.
Parenchymal bands. The term “parenchymal band” refers to a nontapering linear or reticular opacity ranging from 2 to 5 cm in length, usually perpendicular to and in contact with the pleural surfaces (Fig. 15). Parenchymal bands vary in thickness from

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**Box 4. Interlobular septal thickening: differential diagnostic considerations**

**Smooth**

- Pulmonary edema
- Pulmonary alveolar proteinosis
- Lymphangitic carcinomatosis
- Pulmonary hemorrhage
- Lymphoproliferative disease
- Infections (especially subacute Pneumocystis jiroveci pneumonia)
- Amyloidosis

**Nodular**

- Lymphangitic carcinomatosis
- Sarcoidosis
- Lymphocytic interstitial pneumonia
- Amyloidosis
- Lymphoproliferative diseases

**Irregular**

- Chronic hypersensitivity pneumonitis
- Sarcoidosis
- Silicosis/coal worker’s pneumoconiosis
- Asbestosis
- Usual interstitial pneumonia

---

Fig. 11. Anatomic nodule localization on HRCT. ABPA, allergic bronchopulmonary aspergillosis; BAC, bronchioloalveolar carcinoma; CF, cystic fibrosis; COP, cryptogenic organizing pneumonia; EG, Langerhans’ cell histiocytosis; HP, hypersensitivity pneumonitis; infxn, infection (bacterial, fungal, or viral); LIP, lymphocytic interstitial pneumonia; MAC, Mycobacterium-avium–complex; PLC, pulmonary lymphangitic carcinomatosis; PNA, pneumonia (most commonly bacterial); RB-ILD, respiratory bronchiolitis–interstitial lung disease; TB, Mycobacterium tuberculosis; tib, tree-in-bud.

Fig. 10. Random nodules: miliary tuberculosis. (A) Axial HRCT image shows multiple nodules scattered uniformly throughout the lung parenchyma. Some nodules show a centrilobular distribution (arrow), whereas others are located in the subpleural regions of lung (arrowhead) or along interlobular septa, consistent with a random nodule distribution. (B) Gross specimen shows numerous, widely scattered small nodules, some of which are centrilobular in location (arrow), whereas others are subpleural in location (arrowhead), consistent with a random distribution.
one to several millimeters and are most commonly encountered in patients with atelectasis or fibrosing lung diseases. Occasionally, a parenchymal band represents several contiguous interlobular septa. Parenchymal bands have been reported to occur frequently in patients with asbestos exposure [13], although they may be encountered in a wide variety of fibrotic lung processes and are ultimately nonspecific.

Subpleural lines. A subpleural line is a curvilinear opacity measuring less than 10 mm in thickness that parallels the pleura (Fig. 16). Subpleural lines are nonspecific, and usually represent atelectasis, fibrosis, or inflammation. Subpleural lines were first described in patients with asbestosis, and are seen more commonly in this disease than other fibrotic lung diseases, but they are not exclusive to patients with asbestosis.

Irregular linear opacities. Irregular linear opacities are nonspecific linear structures that cannot be classified as a parenchyma band, subpleural line, or interlobular septa. They range in thickness from 1 to

Fig. 12. Smooth interlobular septal thickening: pulmonary edema. Axial HRCT shows extensive smooth interlobular septal thickening (arrows) in the lung bases of a patient with congestive heart failure. Note presence of centrilobular nodules without tree-in-bud opacity.

Fig. 14. Irregular interlobular septal thickening in a patient with pulmonary fibrosis of uncertain etiology. Axial HRCT shows peripheral reticulation and irregular linear opacities and several irregularly thickened interlobular septa (arrows).

Fig. 13. Nodular interlobular septal thickening: lymphangitic carcinomatosis. (A) Axial HRCT image shows smooth interlobular septal thickening (arrow) in the anterior right lung (more commonly seen with lymphangitic carcinomatosis), with nodular interlobular septal thickening (arrowheads) in the posterior right lung base (more characteristic of lymphangitic carcinomatosis). Note presence of ipsilateral pleural effusion and the marked asymmetry of the process. (B) Gross specimen of a patient with lymphangitic carcinomatosis shows nodular interlobular septal thickening (arrows).
3 mm and are most commonly encountered in fibrotic lung diseases, and are quite nonspecific.

Reticular abnormalities

Reticular opacities represent linear opacities that intersect one another at various angles, producing a netlike pattern. The most important form of reticular opacity encountered on HRCT imaging is intralobular interstitial thickening. Intralobular interstitial thickening reflects infiltration and thickening of the interstitial framework of the secondary pulmonary lobule and may be caused by pulmonary fibrosis or inflammation in the absence of fibrosis. When underlying fibrosis is present, the reticulation often appears coarse, and traction bronchiolectasis and architectural distortion may also be seen [2,35].

Intralobular interstitial thickening is a common finding in patients with usual interstitial pneumonia—idiopathic pulmonary fibrosis, and may be the predominant finding before honeycombing is evident (Fig. 17). Intralobular interstitial thickening is also a common finding in patients with nonspecific interstitial pneumonitis and pulmonary disease associated with collagen vascular diseases (Fig. 18) [36–39]. Intralobular interstitial thickening may also be seen in other idiopathic interstitial pneumonias, pulmo-
nary infections, pulmonary edema, and lymphangitic carcinomatosis.

Ground-glass opacity
Ground-glass opacity is defined as hazy increased attenuation that does not obscure visibility of the underlying vasculature (see Fig. 4). Ground-glass opacity is a nonspecific finding that may reflect volume averaging of abnormalities that cannot be completely resolved with HRCT technique, a purely interstitial abnormality, a purely alveolar abnormality, or a disease process that involves both the pulmonary interstitium and the air spaces [40,41]. A study by Leung et al [41] of the histopathologic correlates of ground-glass opacity on HRCT showed that 54% of patients had a primarily interstitial abnormality, 32% had a mixed interstitial and alveolar process, and 14% of patients had primarily an alveolar process.

The significance of ground-glass opacity depends on the patient’s symptoms (acute versus chronic, and the actual presenting symptoms); the distribution of the ground-glass opacity on HRCT; and the presence or absence of other findings on the HRCT study. In severely immunocompromised patients with a clinical presentation suggesting infection, ground-
glass opacity often reflects active infection, such as viral pneumonia or *P jiroveci* pneumonia (Fig. 19). In patients presenting with hemoptysis, falling hematocrit, or pulmonary capillaritis, multifocal ground-glass opacity often reflects pulmonary hemorrhage (Fig. 20). For patients with a suggestive antigen exposure, multifocal ground-glass opacity reflects the histopathologic presence of poorly formed granulomas, cellular bronchiolitis, and interstitial inflammation caused by hypersensitivity pneumonitis (Fig. 21).

The presence of ground-glass opacity in patients with idiopathic interstitial pneumonias often, but not invariably, reflects active pulmonary inflammation and potentially reversible disease. In the study by Leung et al [41] cited previously, 82% of patients with ground-glass opacity on HRCT had reversible disease shown on lung biopsy. Similarly, Remy-Jardin et al [40] showed that ground-glass opacity on HRCT corresponded to active, reversible pulmonary inflammation in 65% of patients undergoing biopsy. In this same study, however, 22% of patients with ground-glass opacity on HRCT had lung biopsies showing more fibrosis than inflammation, and in 13% of patients only fibrosis was found on biopsy. For these latter patients, ground-glass opacity on HRCT represented fibrosis below the limit of HRCT resolution. Ground-glass opacity should be interpreted as active, potentially reversible disease when unaccompanied by other findings suggestive of fibrosis, such as traction bronchiectasis and honeycombing (Fig. 22) [40,42]. In patients with ground-glass opacity in some areas of lung but findings suggesting fibrosis in other areas, biopsy should be directed toward the areas of ground-glass opacity and away from areas more suggestive of fibrosis [40,42].

**Consolidation**

Consolidation is defined as increased attenuation, which results in obscuration of the underlying vasculature, usually producing air bronchograms (see Fig. 4). The presence of consolidation implies that the air within affected alveoli has been replaced by another substance, such as blood, pus, edema, or cells. When consolidation is evident on a chest radiograph, HRCT does not usually provide additional diagnostically useful information. HRCT may detect consolidation earlier than chest radiography, however, and in certain circumstances may provide useful information regarding the distribution of con-
solidation and detect findings diagnostically important but not visible radiographically.

The differential diagnosis of consolidation is extensive, and requires integration of clinical history with other relevant scan findings to become manageable. One circumstance where HRCT is quite valuable in the assessment of consolidation is the determination of the distribution of findings. Although many etiologies of consolidation may often be indistinguishable from one another on HRCT imaging, consolidation in a peripheral or subpleural distribution should evoke a specific differential diagnosis (Box 5). Recognition of this particular distribution of consolidation can be diagnostically quite useful (Fig. 23).

High-resolution CT scan findings manifesting as decreased opacity

**Bronchiectasis**

Bronchiectasis is defined as localized, irreversible dilation of the bronchial tree. There are numerous

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Characteristic disease distribution</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Postinfectious (bacterial and viral)</td>
<td>Lower lobe</td>
<td>—</td>
</tr>
<tr>
<td>AIDS-related airway disease</td>
<td>Lower lobe</td>
<td>—</td>
</tr>
<tr>
<td>Cystic fibrosis</td>
<td>Upper lobe</td>
<td>Mucoid impaction</td>
</tr>
<tr>
<td>Allergic bronchopulmonary aspergillosis (see Fig. 26)</td>
<td>Central, upper lobe</td>
<td>Mucoid impaction (may be high attenuation)</td>
</tr>
<tr>
<td>Williams-Campbell syndrome</td>
<td>Central</td>
<td>—</td>
</tr>
<tr>
<td>Mycobacterium avium complex infection (see Fig. 6A)</td>
<td>Right middle lobe, lingula</td>
<td>Older women</td>
</tr>
<tr>
<td>Immotile cilia syndromes</td>
<td>Right middle lobe, lingula, lower lobes</td>
<td>May be accompanied by situs inversus in Kartagener’s syndrome</td>
</tr>
<tr>
<td>Hypogammaglobulinemia (neoplasm, stricture)</td>
<td>Right middle lobe, lingula</td>
<td>—</td>
</tr>
<tr>
<td>Airway obstruction</td>
<td>Focal, distal to obstruction</td>
<td>Often shows a lobar distribution</td>
</tr>
</tbody>
</table>

Distribution reflects predominant areas of involvement. Other regions may also be simultaneously involved.

**Table 4**

Causes of bronchiectasis and characteristic disease distribution

**Fig. 24.** HRCT assessment of bronchiectasis: the “signet ring” sign. Coned axial HRCT image shows a dilated bronchus (arrow) in cross-section. Note that the internal diameter of the bronchus exceeds the diameter of the adjacent pulmonary artery.

**Fig. 25.** HRCT assessment of bronchiectasis: lack of bronchial tapering. Coned axial HRCT image shows bronchial dilation with lack of tapering (arrows). Bronchial morphology is consistent with varicose bronchiectasis.
causes of bronchiectasis. Several of the most common etiologies are listed in Table 4. Bronchography was traditionally performed to confirm the diagnosis of bronchiectasis, but now has been replaced by HRCT. HRCT findings of bronchiectasis include increased bronchoarterial ratios, lack of appropriate airway tapering, bronchial wall thickening and irregularity, mucoid impaction, and mosaic perfusion with air trapping.

**Bronchial dilation (increased bronchoarterial ratio).** Bronchial dilation is the most specific finding for bronchiectasis. In general, bronchiectasis

![Fig. 26. HRCT assessment of bronchiectasis: mucoid impaction. (A) Axial HRCT image shows extensive, bilateral mucoid impaction (arrows). Bilateral inhomogeneous lung opacity represents mosaic perfusion caused by large and small airway obstruction. Small centrilobular nodules are visible in the right lower lobe. (B) Sagittal maximum intensity projected image shows the mucoid impaction (arrows) to advantage; “finger-in-glove” appearance is clear.](image)

![Fig. 27. Pitfalls in the HRCT assessment of bronchiectasis: wide collimation and respiratory motion. (A) Coned axial CT image obtained with 7-mm collimation and degraded by respiratory motion fails to demonstrate bronchiectasis clearly. (B) Coned axial HRCT image clearly shows bronchiectasis in the right middle lobe (arrows) and the right lower lobe.](image)
is present when the bronchoarterial ratio (the ratio of the internal diameter of the bronchus to its adjacent pulmonary artery) exceeds 1. When an increased bronchoarterial ratio is seen in cross-section, it has been termed the “signet ring” sign (Fig. 24). This sign is suggestive of bronchiectasis, but it does have limitations. The ratio may not be perceptibly increased in areas of consolidation, and patients who live at altitudes well above sea level or asthmatics often have bronchoarterial ratios greater than 1 [43].

Lack of bronchial tapering. Perhaps the earliest sign of cylindrical bronchiectasis, lack of bronchial tapering, is often subtle, but is important to appreciate. It is probably easiest to see in longitudinal section (Fig. 25). In cross-section, with noncontiguous HRCT images, the finding is difficult to assess. One indication of this finding in cross-section is lack of change in the size of an airway over 2 cm after branching.

Visualization of peripheral airways. Visualization of an airway within 1 cm of the costal pleura is abnormal and indicates potential bronchiectasis. Airways may be seen within 1 cm of the mediastinal pleura, but should never be seen actually to abut the mediastinal pleura.

Mucoid impaction. Fluid- or mucous-filled, dilated bronchi are usually easily appreciated on HRCT. They may be seen as branching structures when imaged in longitudinal section, or as nodules when imaged in cross-section (Fig. 26).

Ancillary findings. Ancillary findings of bronchiectasis include indicators of bronchiolectasis (with mucoid impaction and tree-in-bud); mosaic perfu-

<table>
<thead>
<tr>
<th>Table 5</th>
<th>Pitfalls in high-resolution CT diagnosis of bronchiectasis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pitfall</td>
<td></td>
</tr>
<tr>
<td>Inadequate HRCT technique-collimation too wide, large interslice gap (see Fig. 27)</td>
<td>Use narrow collimation; interslice gap should not exceed 1 cm</td>
</tr>
<tr>
<td>Respiratory (see Fig. 27) and cardiac motion</td>
<td>Usually maximal near heart in right middle lobe and lingula. May mimic or obscure (see Fig. 27A) diagnosis of bronchiectasis. Look for motion elsewhere on the scan</td>
</tr>
<tr>
<td>Consolidation</td>
<td>Avoid diagnosing bronchiectasis when active infection or significant atelectasis is present; obtain follow up imaging</td>
</tr>
<tr>
<td>Cystic lung diseases</td>
<td>Use narrow collimation and narrow interslice gap to facilitate appreciation of airway origin of pulmonary cystic lucency (cystic lung diseases lack tubular morphology). To suggest bronchiectasis, look for constant relationship of cysts to pulmonary arteries and for “cluster of grapes” morphology</td>
</tr>
<tr>
<td>Increased bronchoarterial ratios in normal patients</td>
<td>Obtain history of asthma or residence at altitude</td>
</tr>
<tr>
<td>Traction bronchiectasis (see Fig. 28)</td>
<td>Look for other findings of fibrosis</td>
</tr>
</tbody>
</table>

Fig. 28. Pitfalls in the HRCT assessment of bronchiectasis: traction bronchiectasis. Axial HRCT image shows extensive lower lobe bronchial dilation (arrows). Note corrugated appearance of bronchi; this morphology is characteristic of traction bronchiectasis. Surrounding architectural distortion, ground-glass opacity, and coarse reticulation are present and suggest presence of fibrotic lung disease. Bronchi are also visible in the immediate subpleural regions of lung. This finding indicates an abnormal condition, and may be seen with traction bronchiectasis or primary large and small airway diseases.
Pitfalls in the high-resolution CT diagnosis of bronchiectasis. Pitfalls in the diagnosis of bronchiectasis (Figs. 27 and 28) are listed in Table 5. Careful attention to technique is essential for avoiding some of these pitfalls. MSCT-HRCT imaging of the thorax may play a role in the evaluation of suspected bronchiectasis because volumetric imaging obtained in a very brief time period avoids several of the pitfalls in bronchiectasis imaging that may occur with routine HRCT technique.

Use of high-resolution CT for the diagnosis of the etiology of bronchiectasis. Bronchiectasis may be classified, in ascending order of severity, as cylindrical (discussed later); air trapping; and bronchial wall thickening.

Emphysema

Emphysema is defined as a condition “of the lung characterized by permanent, abnormal enlargement of the airspaces distal to the terminal bronchiole, accompanied by destruction of the air space walls” [48,49]. Emphysema results from an imbalance between proteolytic and antiproteolytic enzymes, and the balance is shifted toward proteolysis by smoking or enzymatic deficiencies, such as $\alpha_1$-antiprotease deficiency [50].

Classification of emphysema: approach using high-resolution CT. Emphysema may be classified into centrilobular, panlobular, and distal acinar (paraseptal) patterns using both histopathologic techniques and HRCT imaging (Table 6). Centrilobular emphysema (Fig. 30) is found most commonly in the upper lobes and manifests as multiple small areas of low attenuation without a perceptible wall, producing a

<table>
<thead>
<tr>
<th>Pattern</th>
<th>Distribution</th>
<th>Appearance</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Centrilobular</td>
<td>Upper lobe</td>
<td>Rounded low attenuation with minimal or no perceptible wall. May see centrilobular artery within area of low attenuation</td>
<td>Smokers</td>
</tr>
<tr>
<td>Panlobular</td>
<td>Generalized or lower lobe</td>
<td>Lobular low attenuation, diffuse low attenuation with simplification of pulmonary architecture</td>
<td>$\alpha_1$-Antiprotease deficiency. May be difficult to appreciate until moderate or severe</td>
</tr>
<tr>
<td>Paraseptal</td>
<td>Upper lobe</td>
<td>Thin-walled cyst in subpleural regions of upper lobes. Spontaneous pneumothorax</td>
<td>Smokers, associated with other forms of emphysema. May be seen in nonsmokers</td>
</tr>
<tr>
<td>Irregular air space enlargement</td>
<td>None (more commonly upper lobe)</td>
<td>Irregular low attenuation in proximity to scars or progressive massive fibrosis</td>
<td>Silicosis, sarcoidosis</td>
</tr>
</tbody>
</table>

Fig. 29. HRCT assessment of bronchiectasis: cystic bronchiectasis. Axial HRCT imaging shows extensive bilateral cystic bronchiectasis (arrows), consistent with the diagnosis of tracheobronchomegaly. Note dilated trachea (T).
punched-out appearance. Often the centrilobular artery is visible within the center of these lucencies. On occasion, a very thin, barely perceptible, wall may be encountered in patients with centrilobular emphysema, probably related to some degree of surrounding fibrosis. When centrilobular emphysema becomes more pronounced, areas of confluent low attenuation become evident. This appearance has been referred to as “confluent centrilobular emphysema” (Fig. 31).

In contrast to centrilobular emphysema, panlobular emphysema is histopathologically characterized by complete destruction of the entire pulmonary lobule, and shows either diffuse or lower lobe predominance. This is the pattern of emphysema commonly present in patients with α1-antiprotease deficiency [50]. On HRCT, panlobular emphysema appears as extensive low attenuation that manifests as diffuse “simplification” of pulmonary architecture.
and the pulmonary vessels appear stretched and attenuated in the presence of panlobular emphysema. Centrilobular emphysema and paraseptal emphysema are uncommonly associated with panlobular emphysema.

Distal acinar, or paraseptal, emphysema commonly occurs in smokers and shows upper lobe predominance, although paraseptal emphysema may be seen associated with other types of emphysema and even in nonsmokers. Because this pattern of emphysema preferentially destroys the distal portion of the pulmonary acinus, findings on HRCT are characteristically peripheral in distribution (Fig. 33). Paraseptal emphysema appears on HRCT imaging as multiple areas of low attenuation with thin, definable, uniform walls distributed in the subpleural regions of lung, forming a single layer. Spontaneous pneumothorax may occur in association with paraseptal emphysema.

Finally, cicatricial emphysema, now more properly termed “irregular air space enlargement,” may be recognized in association with parenchymal scars, especially in the setting of progressive massive fibrosis in patients with pneumoconiosis. Irregular emphysema appears on HRCT as low attenuation in immediate proximity to progressive massive fibrosis or pulmonary scars (Fig. 34).

Honeycomb lung

Honeycomb lung represents the presence of end-stage lung and may occur from a wide variety of insults. Pathologically, honeycomb cysts consists of air-containing spaces with thick walls that are lined with bronchiolar epithelium and fibrous tissue. The HRCT demonstration of honeycomb cysts allows for a confident diagnosis of a fibrosing pulmonary process, and the specific distribution of the honeycomb cysts may be a clue to the etiology of the fibrotic lung disease (Table 7). Note, however, that microscopic honeycomb cysts may be shown on surgical lung biopsy in patients without clear evidence of honeycomb cysts on HRCT.

Table 7
Honeycomb cysts: diagnostic utility of distribution of honeycomb cysts on high-resolution CT imaging

<table>
<thead>
<tr>
<th>Disease etiology</th>
<th>Distribution</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>UIP</td>
<td>Lower lobe</td>
<td>Includes idiopathic pulmonary fibrosis (UIP), asbestosis, aspiration, and connective tissue disorders</td>
</tr>
<tr>
<td>NSIP</td>
<td>Lower lobe</td>
<td>Includes fibrotic NSIP, connective tissue disorders</td>
</tr>
<tr>
<td>Other idiopathic interstitial pneumonias</td>
<td>Variable</td>
<td>Associated with respiratory failure, diffuse ground-glass opacity and consolidation</td>
</tr>
<tr>
<td>AIP</td>
<td>Variable</td>
<td>Smokers with multifocal or diffuse ground-glass opacity [61,62]</td>
</tr>
<tr>
<td>DIP</td>
<td>Variable</td>
<td>Tends to spare extreme bases, unlike UIP [63]</td>
</tr>
<tr>
<td>Hypersensitivity pneumonitis</td>
<td>Mid-lungs</td>
<td>May see associated perilymphatic nodules (see Fig. 15)</td>
</tr>
<tr>
<td>Sarcoaidosis</td>
<td>Upper lobe</td>
<td>Depends on port; may recognize abrupt margins and nonanatomic distribution</td>
</tr>
<tr>
<td>Radiation injury</td>
<td>Variable</td>
<td>Posterior atelectasis in ARDS may be protective from oxygen toxicity, allowing preferential damage to anterior lung [64]</td>
</tr>
</tbody>
</table>

Abbreviations: AIP, acute interstitial pneumonia; ARDS, adult respiratory distress syndrome; DIP, desquamative interstitial pneumonia; NSIP, nonspecific interstitial pneumonia; UIP, usual interstitial pneumonia.
Honeycomb cysts on HRCT appear as cystic areas with clearly definable walls, ranging from a few millimeters to several centimeters in size. Honeycomb cysts may form a single layer in the subpleural lung, although when the disease process becomes more advanced, honeycomb cysts stack on one another in several layers (Fig. 35A); this allows them to be readily distinguished from bullae in patients with paraseptal emphysema. Honeycomb cysts usually share walls with one another, and associated findings of fibrosis (architectural distortion, coarse reticulation, intralobular interstitial thickening, and traction bronchiectasis) are also commonly present (Fig. 35).

The determination of the presence or absence of honeycombing on HRCT in patients with idiopathic interstitial pneumonia is of great importance. The confident diagnosis of lower lobe, subpleural honeycombing in such patients strongly suggests usual interstitial pneumonia–idiopathic pulmonary fibrosis, and obviates the need for surgical lung biopsy (see Fig. 35). In a group of patients from multiple centers selected on the basis of the suspicion of idiopathic pulmonary fibrosis, thoracic radiologists confidently diagnosed usual interstitial pneumonia in nearly 60% of patients, with a positive predictive value of 96% [52]. In a subsequent analysis of these data, the presence of either of two HRCT features (upper lobe reticulation and lower lobe honeycombing) was found to increase the probability of a histopathologic diagnosis of usual interstitial pneumonia by fivefold to sixfold [53]. Similarly, in another study of patients suspected of having an idiopathic interstitial pneumonia, the presence of honeycombing on HRCT in at least one lobe had a positive predictive value of 92%

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**Fig. 35.** HRCT demonstration of honeycomb lung in patients with idiopathic pulmonary fibrosis–usual interstitial pneumonia (UIP). (A) Axial HRCT image through the lung bases shows multiple cystic structures in the subpleural regions of lung (arrows), consistent with honeycomb lung. Note how the cysts stack on themselves in layers and share walls with one another. (B) Axial HRCT image through the lung bases shows subpleural reticulation (arrow), suggestive of fibrotic lung disease. Several honeycomb cysts are present within the right lower lobe, allowing the diagnosis of UIP to be suggested on the basis of imaging.

**Inhomogeneous Lung Opacity**

- normal vessels
- decreased vessel size in areas of ↓ attenuation
  - headcheese
  - HP, infxn
  - RB-ILD
  - edema
- ↑ & ↓ opacity
  - GGO
- ↑ air trapping
  - BO
  - HP
  - asthma bronchitis
- no air trapping
  - vascular
  - PE
  - PA HTN

**Fig. 36.** Inhomogeneous lung opacity: differentiating between infiltrative and obstructive etiologies. BO, bronchiolitis obliterans; GGO, ground-glass opacity; HP, hypersensitivity pneumonitis; infxn, infection (bacterial or viral); PA HTN, pulmonary hypertension; PE, pulmonary embolism; RB-ILD, respiratory bronchiolitis–interstitial lung disease.
The diagnosis of usual interstitial pneumonia cannot be confidently offered on HRCT imaging when honeycomb cysts are not seen. Some patients without HRCT evidence of honeycombing subsequently have usual interstitial pneumonia diagnosed on surgical lung biopsy (see Fig. 17) [52].

Mosaic perfusion and inhomogeneous lung opacity

Pulmonary tissue density is in part determined by the blood volume present within lung tissue. Any pathologic process that disturbs the distribution of pulmonary blood volume may alter pulmonary parenchymal attenuation. Alterations in pulmonary parenchymal attenuation that are seen on HRCT imaging that either result from infiltration of the lung parenchyma or from disturbances in pulmonary blood volume may be collectively referred to as “inhomogeneous lung opacity.” When infiltrative pathology is the cause of inhomogeneous lung opacity, either ground-glass opacity or consolidation is seen; when alterations in pulmonary blood distribution are the cause of inhomogeneous lung opacity, decreased lung opacity is encountered and the term “mosaic perfusion” may be used. The alterations in lung parenchymal perfusion that result in mosaic perfusion produce both areas of relatively increased attenuation (hyperperfused lung) and areas of relatively decreased attenuation (hypoperfused lung), although the latter are more striking on HRCT imaging.

Two major categories of pathologies producing mosaic perfusion are recognized: airway obstruction and vascular occlusion. Obstructive airway lesions that may produce mosaic perfusion include large airway diseases, such as bronchiectasis, and the various forms of bronchiolitis (particularly chronic bronchiolitis with hypersensitivity pneumonitis and constrictive bronchiolitis). Vascular occlusion is commonly the result of chronic thromboembolic dis-
ease, pulmonary hypertension, or capillaritis caused by vasculitis.

**Inhomogeneous lung opacity: distinguishing between ground-glass opacity and mosaic perfusion**

When inhomogeneous lung opacity is encountered on HRCT, infiltrative pathology must be distinguished from obstructive pathology; occasionally both patterns may be present. An algorithmic approach to the assessment of inhomogeneous lung opacity facilitates accurate diagnosis (Fig. 36). Mosaic perfusion may be differentiated from ground-glass opacity by the observation that vessels within the areas of relatively decreased lung attenuation are abnormally small (Fig. 37), whereas in the cases of ground-glass opacity, vessels are equal in size throughout all areas of inhomogeneous lung opacity.

![Fig. 39. Mosaic perfusion caused by airway obstruction: value of expiratory imaging with HRCT in a patient with hypersensitivity pneumonitis. (A) Axial HRCT image through the lower lobes shows bilateral inhomogeneous lung opacity consistent with a combination of increased lung attenuation, representing ground-glass opacity, and decreased lung attenuation, representing mosaic perfusion (arrows). (B) Axial low-dose expiratory HRCT image shows accentuation of the inhomogeneous lung opacity. There is failure of the low-attenuation areas seen on the inspiratory image (arrows) to increase in attenuation appropriately, indicating air trapping.](image)

Fig. 40. Air trapping on expiratory imaging in the absence of inspiratory scan findings in a patient with bronchiolitis obliterans. (A) Axial inspiratory image through the lower lobes shows no clear evidence of inhomogeneous lung opacity. (B) Axial expiratory image shows abnormal low attenuation (arrows) caused by air trapping, representing failure of the expected increase in lung attenuation that should normally occur with expiratory imaging. (Fig. 40)
The small size of the vessels within areas of mosaic perfusion reflects the diminished blood flow within these areas of lung. The presence of lobular low attenuation, in which the outlines of individual secondary pulmonary lobules may be recognized, also favors the presence of mosaic perfusion over ground-glass opacity (Fig. 38) [56]. Lobular low attenuation is encountered when the small lobular bronchioles are diseased.

Once mosaic perfusion is diagnosed, airway and vascular pathologies must then be distinguished; this may be accomplished with expiratory imaging [55,57]. When the cause of mosaic perfusion is vascular, the inhomogeneous opacity seen on the inspiratory image remains roughly similar on the expiratory image. When the cause of the mosaic perfusion on the inspiratory image is related to bronchiolitis, however, the appearance of the inhomogeneous lung opacity is accentuated (Fig. 39) [55,58]. This occurs because lung parenchymal attenuation increases with expiratory imaging as air within the lung is exhaled. For vascular causes of mosaic perfusion, air trapping is not present and all areas of lung increase in attenuation in a similar fashion. With airway causes of inhomogeneous opacity, however, air trapping impedes the escape of air from some areas of lung, whereas other areas decompress normally. This results in an accentuation of the inhomogeneous opacity with expiratory imaging. As a general rule, small airway causes of mosaic perfusion are far more common than vascular etiologies [57,58].

Normal inspiratory scans with air trapping on expiratory imaging

Most patients with air trapping seen on expiratory scans have inspiratory scan abnormalities, such as bronchiectasis, mosaic perfusion, airway thickening, or nodules with or without tree-in-bud that suggest the proper differential diagnosis. Occasionally, air...
trapping may be the sole abnormal finding on an HRCT study; the inspiratory scan is normal (Fig. 40) [57]. In this situation, expiratory HRCT techniques are valuable for demonstrating the presence of an underlying airway abnormality. This circumstance may reflect less extensive physiologic derangements than conditions in which abnormalities are visible on the inspiratory images. The differential diagnosis of this air trapping on expiratory imaging in the presence of normal inspiratory scan findings includes constrictive bronchiolitis (bronchiolitis obliterans); asthma; chronic bronchitis; and hypersensitivity pneumonitis [57].

The “head-cheese” sign

The head-cheese sign represents the simultaneous presence of ground-glass opacity, normal lung, and mosaic perfusion on inspiratory HRCT images (Fig. 41). This finding is produced by mixed infiltrative and obstructive diseases, and carries the name “head-cheese sign” because of its resemblance to the variegated appearance of sausage made from the parts of the head of a hog [59]. The differential diagnosis of this observation includes hypersensitivity pneumonitis (especially chronic disease); sarcoidosis; and atypical infections (eg, *Mycoplasma pneumoniae*). Respiratory bronchiolitis–interstitial lung disease may also produce this sign.

**Cystic lung diseases**

Lung diseases characterized by cysts include Langerhans’ cell histiocytosis (Fig. 42), lymphangioleiomyomatosis (Fig. 43), lymphocytic interstitial pneumonia (Fig. 44), postinfectious pneumatoceles, and amyloidosis (Table 8). Recently, lung cysts have
been reported in association with hypersensitivity pneumonitis [60]. The various features on HRCT that allow these disorders to be distinguished are outlined in Table 8.

Table 8
Cystic lung diseases: distinguishing features on high-resolution CT

<table>
<thead>
<tr>
<th>Disease</th>
<th>Clinical</th>
<th>HRCT features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Langerhans’ cell histiocytosis</td>
<td>Smoker</td>
<td>Upper lobe predominance</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Centrilobular nodules</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bizarre-shaped cysts</td>
</tr>
<tr>
<td>Lymphangioleiomyomatosis</td>
<td>Women of childbearing age</td>
<td>Diffuse distribution</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pleural effusion</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Uniformly shaped cysts</td>
</tr>
<tr>
<td>Lymphocytic interstitial pneumonia</td>
<td>Connective tissue disorders (especially Sjögren’s syndrome)</td>
<td>Cyst size range: 1–30 mm</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Septal thickening</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Centrilobular nodules</td>
</tr>
<tr>
<td>Postinfectious pneumatoceles</td>
<td>Children with <em>Staphylococcus aureus</em> pneumonia</td>
<td>Cyst evolves with bronchopneumonia</td>
</tr>
<tr>
<td></td>
<td>Severely immunocompromised patients (AIDS, <em>Pneumocystis jiroveci</em> pneumonia)</td>
<td>Multifocal ground-glass opacity, pneumothorax</td>
</tr>
<tr>
<td></td>
<td>Infection in endemic region (coccidioidomycosis)</td>
<td>Cyst evolves from a nodule</td>
</tr>
</tbody>
</table>

Summary

HRCT is a very powerful tool in the assessment of patients with diffuse lung disease. Complete appreciation of the diagnostic capabilities of HRCT requires a firm understanding of pulmonary anatomy, particularly the anatomic arrangement of the secondary pulmonary nodule, and the technical factors required for optimal HRCT imaging. With such knowledge, abnormalities on HRCT may be effectively approached by using an organized, algorithmic method. Rigorous application of an ordered, pattern approach to HRCT abnormalities allows for reproducible and accurate interpretation.

References


