State of the Art: Imaging of Occupational Lung Disease

Imaging of occupational lung disease, often perceived as a static discipline, continues to evolve with changes in industry and imaging technology. The challenge of accurately identifying an occupational exposure as the cause of lung disease demands a team approach, requiring integration of imaging features with exposure type, time course, and severity. Increasing use of computed tomography has demonstrated that specific occupational exposures can result in a variety of patterns of lung injury. The radiologist must understand the spectrum of expected imaging patterns related to known occupational exposures and must also recognize newly described occupational exposure risks, often related to recent changes in industrial practices.

Learning Objectives:
After reading the article and taking the test, the reader will be able to:
- Categorize the broad spectrum of imaging patterns from lung injury caused by occupational exposures to provide a novel framework for approaching occupational lung disorders.
- Discuss guidelines permitting the use of digital radiography in the International Labor Organization classification of pneumoconioses.
- Explain the importance of a multidiscipline approach to occupational lung disease.
- Identify newly recognized occupational lung diseases such as accelerated silicosis in denim sandblasting and deployment-related lung disease to highlight the dynamic nature of the field.

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ith advances in technology over the past 40 years, radiology has increasingly become pivotal in management of most common medical conditions, including stroke, chest pain, cancer, and trauma. In contrast, the role of radiology in the diagnosis and treatment of occupational lung disease appears at first glance to have changed very little. Despite substantial limitations, radiography remains the most widely used method for diagnosis and monitoring of many occupational lung diseases, often in conjunction with the International Labor Organization (ILO) classification system (1). In many settings, radiologists practice at a distance from their occupational medicine or pulmonary colleagues (2), relying on typical imaging appearances and the clinical history, rather than inter-disciplinary interaction, to suggest the diagnosis of occupational lung disease. Since the approach to classifying occupational lung disease is often based mainly on exposure history rather than imaging pattern (3–6), the radiologist may be at a further disadvantage in recognizing an imaging abnormality as work related.

In fact, the diagnosis and imaging of occupational lung diseases is rapidly evolving. Thin-section computed tomography (CT) of the chest continues to demonstrate previously unidentified characteristics that shape our understanding of occupational lung injury (7–11). New industrial practices and materials are increasingly recognized as causes of lung disease (12–18). Less-industrialized nations may enforce fewer protective regulations, so that “exporting” more hazardous industrial processes may result in outbreaks of occupational lung diseases that are now rare in the United States (6,17,18). Moreover, recurrent or distinct radiographic appearances in the setting of a common exposure have aided in the identification of several newer occupational exposures which cause lung disease (12–16).

In this article, we demonstrate the importance of a multidisciplinary approach to diagnosis of occupational lung disease, with particular emphasis on a radiologic pattern-based approach. We illustrate the spectrum of lung injury related to occupational exposures and discuss the imaging features of several newly described occupational diseases.

Clinical Evaluation

The Occupational and Environmental Exposure History

To recognize a lung disease as work related, the clinical findings must be informed by specifics of workplace exposures along with the relevant medical and scientific literature on causal associations between exposures and associated health effects. A detailed medical and exposure history remains the mainstay of diagnosis of exposure-related lung disease. The medical history should include the onset and timing of the patient’s chest symptoms, past medical history, review of systems, current medications, family history, and personal habits, including use of tobacco products, alcohol, and recreational drugs. The three essential components of an occupational history include (a) a chronology of current and longest held jobs, (b) a current job description, and (c) questions about symptoms during or after exposure to specific fumes, dusts, and chemicals. For example, in patients presenting with symptoms of interstitial lung fibrosis, the clinician should elicit information about previous exposure to fibrogenic dusts such as asbestos, silica, and coal mine dust. Other clinical clues that may suggest an exposure-disease link include a temporal relationship between an exposure and respiratory symptoms (eg, work-related wheezing in an auto-body worker exposed to isocyanates), a diagnosis that has been strongly linked to exposure (eg, pleural mesothelioma), or the unexpected occurrence of an illness (eg, lung cancer in a non-smoker). Such findings should prompt the physician to obtain a more thorough occupational history, with a detailed description of all jobs held, associated exposures, and presence of similar illness among coworkers. Table 1 contains some of the more common exposures associated with risk for occupational lung diseases, along with typical imaging findings.

Information on nonoccupational exposures, particularly those in the home environment and associated with particular hobbies or recreational activities, also should be elicited. The presence of animals can be important in understanding risk for hypersensitivity pneumonitis (from birds or feather furnishings) or asthma (eg, from pet dogs and cats). Use of indoor hot tubs and exposure to other

Abbreviations:
ILO = International Labor Organization
NSIP = nonspecific interstitial pneumonia
PMF = progressive massive fibrosis
UIP = usual interstitial pneumonia

Conflicts of interest are listed at the end of this article.
potentially contaminated aerosol sources (eg, those from humidifiers or moisture intrusion in the home) should be sought when hypersensitivity pneumonitis is a diagnostic consideration, as exposure abatement is necessary for disease management. Finally, the cigarette smoking history must be elicited since clinical and imaging findings may be partially or entirely explained by tobacco smoke exposure.

**Physical Examination**

Findings at lung examination are generally nonspecific and often occur late in the course of chronic occupational pulmonary diseases. For interstitial diseases, inspiratory crackles on auscultation reflect later stages of fibrosis and may be accompanied by digital clubbing and findings of right heart failure. For occupational airways diseases, physical examination findings are often normal. Wheezing may be a sign of large airways obstruction, and end-inspiratory squeaks may be heard in patients with bronchiolitis.

**Laboratory Testing**

Depending on the disease of concern, serologic and other laboratory studies may help distinguish exposure-related lung diseases from autoimmune and other conditions that may be included in the

### Table 1

<table>
<thead>
<tr>
<th>Exposure</th>
<th>Airway</th>
<th>Parenchyma</th>
<th>Pleura</th>
<th>Risk for Lung Malignancy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coal mine dust</td>
<td>Bronchial wall thickening</td>
<td>Upper lobe small nodular opacities (simple coal worker’s pneumoconiosis)</td>
<td></td>
<td>Conglomerate opacities may mimic solitary mass</td>
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<tr>
<td></td>
<td></td>
<td>Lower lobe irregular opacities</td>
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<td>Coalescent large opacities (PMF)</td>
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<td></td>
<td></td>
<td>Emphysema</td>
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<tr>
<td>Respirable silica</td>
<td>Bronchial wall thickening</td>
<td>Silicoproteinosis</td>
<td>Pleural thickening</td>
<td>Risk for lung cancer</td>
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<tr>
<td></td>
<td></td>
<td>Upper lobe small nodular opacities (simple silicosis)</td>
<td>Pleural effusions</td>
<td>Conglomerate opacities may mimic solitary mass</td>
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<td></td>
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<td>Lower lobe irregular opacities</td>
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<td>Coalescent large opacities (PMF)</td>
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<tr>
<td></td>
<td></td>
<td>Emphysema</td>
<td></td>
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</tr>
<tr>
<td>Fibrogenic asbestos</td>
<td>Bronchial wall thickening</td>
<td>Asbestosis: Lower lobe or diffuse irregular opacities</td>
<td>Pleural effusion</td>
<td>Risk for lung cancer</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Rounded atelectasis</td>
<td>Pleural plaques (calcified and noncalcified)</td>
<td>Pleural mesothelioma</td>
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<tr>
<td></td>
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<td>Diffuse pleural thickening</td>
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<td></td>
<td>(involving the costophrenic angle)</td>
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<td></td>
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<td></td>
<td>Mesothelioma (irregular nodularity of pleura)</td>
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<tr>
<td>Organic and inorganic antigens</td>
<td>Bronchial wall thickening</td>
<td>Nonfibrotic hypersensitivity pneumonitis: centrilobular nodules; ground glass opacities; thin-walled cysts; emphysema</td>
<td></td>
<td>Risk for lung cancer</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Emphysema</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Fibrotic hypersensitivity pneumonitis: volume loss; diffuse reticular opacities; modest mediastinal adenopathy; honeycombing</td>
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<tr>
<td>Beryllium</td>
<td>Airway wall thickening</td>
<td>Perilymphatic nodules</td>
<td></td>
<td>Risk for lung cancer</td>
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<td></td>
<td></td>
<td>Conglomerate opacities</td>
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<tr>
<td>Flavoring chemicals (diacetyl); oxides of nitrogen and sulfur; combustion products</td>
<td>Mosaic attenuation, air trapping</td>
<td>Centrilobular nodules</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chemical irritants and toxic fumes</td>
<td>Airway wall thickening, bronchiectasis, mosaic attenuation, air trapping</td>
<td>High dose: chemical pneumonitis</td>
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</tbody>
</table>
differential diagnosis. For example, a positive blood beryllium lymphocyte proliferation test is helpful in distinguishing chronic beryllium disease from sarcoidosis (19,20).

Pulmonary Function Testing
Pulmonary function tests are essential to determining lung disease pathophysiology, severity, management, response to treatment, prognosis, and impairment. Full pulmonary function tests include measurements of lung volume, pre- and postbronchodilator spirometry, and diffusion capacity for carbon monoxide. Occupational lung diseases are often characterized as obstructive, restrictive, or a combination of both. Cardiopulmonary exercise testing can be helpful in determining the presence and degree of ventilatory and gas exchange abnormalities, in clarifying the presence of cardiac disease as a source of chest symptoms, and in determining lung disease severity and impairment. Methacholine challenge is useful in establishing the presence of airways hyperreactivity in some occupational airways diseases (21). Interpretation of pulmonary function test results may be further enhanced when considered in relation to CT data. For example, subjects with normal spirometry findings but low lung diffusion capacity may have mixed emphysema and lung fibrosis evident on CT scans.

Bronchoscopy and Surgical Lung Biopsy
A careful exposure history in combination with imaging and pulmonary function testing is often enough to make the diagnosis of an occupational lung disease. This is particularly true for the pneumoconioses, which usually manifest with distinct imaging abnormalities and typical occupational histories. Similarly, occupational asthma and most other obstructive lung diseases are diagnosed without histologic findings. When diagnostic uncertainty remains, clinicians may need to consider lung biopsy. Fiberoptic bronchoscopy, with bronchoaveolar lavage and transbronchial biopsies, is particularly useful in the evaluation of exposure-related granulomatous lung disease such as chronic beryllium disease and hypersensitivity pneumonitis. Surgical lung biopsy may be required to confirm a diagnosis of bronchiolitis or in cases in which diagnostic certainty is important for management in the clinical setting of diffuse interstitial lung disease.

Imaging Technology
The chest radiograph remains the primary mode of screening for pneumoconiosis in the United States and elsewhere. Its advantages are relatively low cost, low radiation dose, and wide availability. However, the chest radiograph is relatively insensitive for detecting early pneumoconiosis (21–24). The chest radiograph is particularly insensitive for abnormalities such as ground-glass opacity found in granulomatous occupational diseases like hypersensitivity pneumonitis and chronic beryllium disease (25,26). An abnormal chest radiograph is also relatively nonspecific for diagnosis of pneumoconiosis, since a substantial proportion of cases identified as demonstrating pneumoconiosis on chest radiographs are found to have no evidence of pneumoconiosis at CT (27–30) or autopsy (31). This lack of specificity may in part be due to the fact that cigarette smoking causes small irregular opacities on chest radiographs (32). Additionally, the boundary between normal and abnormal chest radiographs may be subjective and difficult to define (33).

A systematic classification system for chest radiographic findings in pneumoconioses was first developed more than 50 years ago and has been repeatedly revised by the ILO with support from the National Institute for Occupational Safety and Health (NIOSH) and the American College of Radiology (1,34,35). By passing a NIOSH-administered film-based examination, physicians may become certified “B-readers,” permitting them to apply the systematic ILO classification to interpret screening chest radiographs for pneumoconiosis. The system uses a set of 22 standard radiographic images to codify and characterize the presence, pattern, and extent of pleural and parenchymal abnormalities related to occupational exposures. An important recent development is the adaptation of the analog-based system to a digital format, with the recent release of a digital set of standard images (27) and publication of proposed guidelines permitting the use of digital radiography systems for evaluation of coal miners (36). Free customized viewing software to facilitate side-by-side viewing of digital chest images and digital standards may be downloaded at the Centers for Disease Control and Prevention Web site (37).

Thin-section CT has replaced chest radiography in evaluation of nonoccupational diffuse lung diseases because of its higher sensitivity for early lung disease and greater accuracy in characterizing the pattern of disease (38,39). Despite these advantages, no country has adopted CT as a primary screening modality for pneumoconiosis, presumably because of higher cost and radiation dose compared with chest radiography. CT is commonly used as a secondary screening modality in symptomatic or physiologically impaired workers when the chest radiograph is normal or equivocal (40). CT is particularly useful in identifying and characterizing atypical presentations of occupational lung disease, as discussed below. A standardized system for scoring extent of disease on CT scans, analogous to the ILO radiographic classification system, is increasingly used (40–44) and has been shown to be associated with moderate interreader and intrareader agreement for all categories of abnormality except ground-glass abnormality (45). Reduced-dose CT (<1.5 mSv) is beginning to be used to screen for lung cancer in those with occupational exposures creating high risk for malignancy (particularly asbestos exposure) (46,47). In such individuals, CT may also be used to screen for pneumoconiosis.

As with many lung diseases, diagnosis of occupational lung disease requires a multidisciplinary approach, including the occupational medicine physician, radiologist, industrial hygienist, pneumologist, and pathologist. Traditionally, the radiologist assists in confirming a suspected diagnosis correlated with the clinical history with or without lung biopsy—an approach which has left the radiologist no-
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Acute silicosis (3,4,57); Hypersensitivity pneumonitis (4–6,60–64,77); Asbestosis (3,4,6,8,75); Acute silicosis (3,4,57); Atypical Presentation

Asbestos exposure (4,6,75)

Complicated silicosis (3,4,6,11,22,69); Acute silicosis (3,4,6,57); Classic Presentation

Chronic silicosis (4,11); Siderosis (3,6,134); Aluminosis (134)

Clinical silicosis (3,4,6,11,22,69,82); Coal worker’s pneumoconiosis (3,4,6,10,28); Chronic beryllium disease (3–5,9,67); Aluminosis (134)

Perilymphatic/subpleural

Diffuse pulmonary opacities: Consolidation

Hypersensitivity pneumonitis (4–6,60–64,77); Acute silicosis (3,4,6,57); Hard metal lung (3,4,6,134); Flock worker’s lung (15,16)

Hypersensitivity pneumonitis (5,6,57); Hard metal lung (3,4,6,66,134); Fibrosis

Hypersensitivity pneumonitis (5,6,57); Hard metal lung (3,4,6,66,134,136); Siderosis (3,6,134); Ardystil syndrome (5,6); Accelerated silicosis (49)

Table 2 (continues)

Pattern-based Approach to Differential Diagnosis of Occupational Lung Disease

<table>
<thead>
<tr>
<th>Pattern</th>
<th>Classic Presentation</th>
<th>Atypical Presentation</th>
</tr>
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<tbody>
<tr>
<td>Diffuse pulmonary opacity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Consolidation</td>
<td>Acute silicosis (3,4,6,57); Hypersensitivity pneumonitis (5,6,57); Ardystil syndrome (5,56)</td>
<td>Hard metal lung (3,4,6,134); Flock worker’s lung (15,16)</td>
</tr>
<tr>
<td>Ground glass</td>
<td>Hypersensitivity pneumonitis (4–6,60–64,77); Acute silicosis (3,4,6,57); Hard metal (3,4,6,66,134); Flock worker’s lung (15,16); Ardystil syndrome (5,56); Indium-tin oxide (12,132)</td>
<td>Chronic beryllium disease (3–5,9,67,68); Asbestosis (3,4,6,8,69); Siderosis (3,5,6,134); Accelerated silicosis (49)</td>
</tr>
<tr>
<td>Crazy paving</td>
<td>Acute silicosis (3,4,57); Indium-tin oxide (12)</td>
<td></td>
</tr>
<tr>
<td>Nodules</td>
<td>Centrilobular</td>
<td>Chronic silicosis (3,4,6,11,22,69,82); Coal worker’s pneumoconiosis (3,4,6,10,28); Chronic beryllium disease (3–5,9,67); Aluminosis (134)</td>
</tr>
<tr>
<td></td>
<td>Siderosis (3,5,6,74,134); Flock worker’s lung (15,16); Ardystil syndrome (5,56); Indium-tin oxide (12); Byssinosis (6); Accelerated silicosis (49)</td>
<td>Hard metal lung (4,65); Aluminosis (134); Accelerated silicosis (48,49); Asbestosis (4,8,75)</td>
</tr>
<tr>
<td>Perilymphatic/subpleural</td>
<td>Chronic silicosis (3,4,6,11,22,69,82); Coal worker’s pneumoconiosis (3,4,6,10,28); Chronic beryllium disease (3–5,9,67); Aluminosis (134)</td>
<td>Hard metal lung (4,65); Aluminosis (134); Accelerated silicosis (48,49); Asbestosis (4,8,75)</td>
</tr>
<tr>
<td>Fibrosis</td>
<td>UIP/NSIP</td>
<td>Asbestosis (3,4,6,8,75); Hypersensitivity pneumonitis (4–6,60–64)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Chronic silicosis (4,11); Coal worker’s pneumoconiosis (4,28); Chronic beryllium disease (3–5,9,67); Hard metal lung (3,4,6,66,136); Siderosis (3,6,134); Ardystil syndrome (5); Flock worker’s lung (15,16); Indium-tin oxide (24); Aluminosis (134)</td>
</tr>
<tr>
<td>Masses</td>
<td>PMF/conglomerate</td>
<td>Complicated silicosis (3,4,6,11,22,69); Complicated coal worker’s pneumoconiosis (3,4,6,28); Talcosis (3,5,71,135)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Siderosis (3,134); Chronic beryllium disease (3–5,67)</td>
</tr>
<tr>
<td>Round atelectasis</td>
<td>Asbestos exposure (4,6,75)</td>
<td>Silicosis (11)</td>
</tr>
</tbody>
</table>

Several occupational exposures characteristically cause ground-glass opacities on CT scans, such as hypersensitivity pneumonitis (Fig 2) (4,5,60–64) and hard metal interstitial lung disease (65,66). Other exposures like accelerated silicosis (49), asbestosis (8), and chronic beryllium...
disease (9, 67, 68) occasionally demonstrate ground-glass opacities, usually in combination with more characteristic findings. Processes that may mimic airspace patterns include conglomerate fibrosis (Fig 3), rounded atelectasis, and malignancy.

The crazy-paving pattern, described as well-defined regions of ground-glass opacity with superimposed septal thickening, scattered throughout regions of normal-appearing lung, has been associated with silicoproteinosis in some but not all studies (57).

### Table 2 (continued)

| Pattern-based Approach to Differential Diagnosis of Occupational Lung Disease |
|----------------------------------|------------------|------------------|
| **Pattern**                     | **Classic Presentation** | **Atypical Presentation** |
| Bronchogenic carcinoma          | Asbestos exposure (3,4,6,75) | Silicosis (3,4); Coal worker’s pneumoconiosis (3,4); Hard metal lung (3) |
| Airway abnormalities            |                             |                             |
| Mosaic attenuation/air trapping | Hypersensitivity pneumonitis (4,5,60–62); Flavor worker’s lung (4); Toxic fume inhalation (6,63); Occupational asthma (137) | Asbestosis (4,6,8); Silicosis (82) |
| Bronchiectasis                  | Toxic fume inhalation (6,63) | Silicosis (82) |
| Bronchial wall thickening       | Occupational asthma (138,139); Toxic fume inhalation (6); Chronic beryllium disease (3–5,9,67) | Flavor worker’s lung (4) |
| Emphysema                       | Coal worker’s pneumoconiosis (3,4,10,28,76); Talcosis (injected) (3); Siderosis (3,5,134) | Hypersensitivity pneumonitis (4,62); Silicosis (4,22,49,69,76,82); Asbestosis (8); Chronic beryllium disease (9); Hard metal lung (65,134); Talcosis (inhaled) (71,135) |
| Cysts                            | Hypersensitivity pneumonitis (4,5,60,77) |                             |
| Pleural abnormalities           |                             |                             |
| Pleural plaques/thickening      | Asbestos exposure (4,6,75); Chronic silicosis (4,11,49,69); Accelerated silicosis (49) | Calciocosis (5); Siderosis (134) |
| Pseudoplaques                   | Chronic silicosis (3,4); Chronic beryllium disease (5,9,67); Coal worker’s pneumoconiosis (4,28) |                             |
| Pleural effusion                | Asbestos exposure (4,6,75); Acute silicosis (57) | Chronic silicosis (11) |
| Mesothelioma                    | Asbestos exposure (4,6,75) |                             |
| Lymph nodes                     | Chronic silicosis (3,4,22); Chronic beryllium disease (3–5,9,67,68); Hypersensitivity pneumonitis (4); Accelerated silicosis (49) | Coal worker’s pneumonitis (3,28); Acute silicosis (57) |
| Hyperdense or calcified lymph nodes | Chronic silicosis (3,4,22); Acute silicosis (57); Chronic beryllium disease (5,9); Coal worker’s pneumoconiosis (6,28) | Accelerated silicosis (49); Aluminosis (140) |

Note.—Numbers in parentheses represent reference numbers.
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Nodular Patterns
When evaluating nodular lung disease, distinction between centrilobular versus perilymphatic or random distribution is important. Centrilobular nodularity resides within the central secondary pulmonary lobe, sparing the subpleural region. Conversely, perilymphatic nodules are distributed along the axial and peripheral interstitium, with septal beading, bronchovascular studding, and subpleural nodularity. Silicosis and coal worker’s pneumoconiosis most often produce well-defined perilymphatic nodules or combined centrilobular and subpleural nodules (3,4,22,28,49,69,70), as do most other pneumoconioses (3,5,71,72). Early pneumoconiosis tends to be more centrilobular (Fig 4) (30). Centrilobular nodularity in subjects with subacute hypersensitivity pneumonitis, acute silicosis, hard metal disease, and siderosis tends to be less well defined or ground glass in appearance (3–5,37,58,60–66,73,74). Perilymphatic distribution of nodules similar to sarcoidosis is seen with chronic beryllium disease (Fig 5) (3–5,9,67,68). Characteristic differences in the distribution of nodularity reflect important differences in pathophysiology between the various occupational lung diseases, but it is important to remember that substantial overlap may occur.

Reticular Patterns
Reticular linear opacities may represent fibrosis occurring in asbestosis (Fig 6a), chronic hypersensitivity pneumonitis, and in some patients with respirable silica or coal mine dust exposure. Typical appearances of usual interstitial pneumonia (UIP) and nonspecific interstitial pneumonia (NSIP) may be seen, but the findings in early pneumoconiosis may be less specific. Certain radiologic characteristics may help differen-
tiate potential exposures. For example, although asbestososis typically manifests as a UIP pattern, subpleural dotlike or branching opacities and parenchymal bands are more common than in idiopathic pulmonary fibrosis (8,73). Additionally, because pleural plaques occur at a much lower fiber burden than asbestosis, interstitial fibrosis from asbestos exposure is uncommon in the absence of pleural plaques (Fig 6b). The presence of lobular decreased attenuation, air trapping, or predominance of disease in the upper or midlungs should suggest fibrotic hypersensitivity pneumonitis (4,5,60,61,63).

**Emphysema and Cysts**

Several studies describe emphysema associated with occupational lung disease, particularly silicosis or coal worker’s pneumoconiosis (4,22,69,76). Centrilobular and panacinar emphysema are present in higher rates in coal workers (Fig 7) or silica exposed individuals than in the general population, even when adjusting for smoking history (69), and may be found in nonsmokers. Paracatricial emphysema, defined as emphysema resulting from fibrosis and architectural distortion, may also occur in relationship to PMF. Peripheral cystic spaces, which may represent paraseptal emphysema or fibrotic-related changes, have been reported in hard metal interstitial lung disease (65,66), and scattered cysts have been described in subacute hypersensitivity pneumonitis (Fig 8) (4,5,77). Interestingly, emphysema has also been described as one of the more common manifestations of chronic hypersensitivity pneumonitis on long term follow-up, even in nonsmokers (62,78–80).

**Airway Patterns**

Small airway disease typically manifests as centrilobular nodules, centrilobular ground-glass opacities, or mosaic perfusion with air trapping. Mosaic perfusion (sharply defined heterogeneity of lung attenuation) and air trapping suggest hypersensitivity pneumonitis or constrictive bronchiolitis (Fig 9). “Flavor worker’s lung” or “popcorn worker’s lung” associated with artificial butter flavoring results in constrictive bronchiolitis manifesting as mosaic perfusion and air trapping (4). Recently, concerns have arisen regarding risk of bronchiolitis in military personnel returning from deployment in Iraq and Afghanistan (81). Abnormalities of small airway disease have also been described in other occupational lung diseases, such as asbestosis and silicosis (4,8,82).

Large airway disease manifested by means of imaging findings of peribronchial thickening and bronchiectasis may be associated with inhalation of toxic fumes (Fig 10) and with other causes of occupational asthma (83). Large airway changes may be also found in chronic beryllium disease (3,4,9), silicosis or coal worker’s pneumoconiosis (49,76) and constrictive bronchiolitis (14), although not as the dominant pattern.

**Pleural Patterns**

Benign pleural abnormalities (including effusion, diffuse pleural thickening, or pleural plaques) are most commonly associated with asbestos exposure (3,5,73,84,85). Pleural effusions typically arise within 10–15 years and pleural thickening or plaques generally arise 20–30 years after initial exposure (Fig 11) (8). In silicosis, parenchymal pseudo-plaques secondary to coalescence of subpleural nodules may be seen (3–5), and more recently silicosis has also been shown to be associated with true pleural abnormality, including pleural thickening, effusion, and pleural invagination (11).

**Major Contemporary Occupational Lung Diseases**

**Silicosis**

Although the incidence of silicosis has fallen since its peak during World War II, it is still a major cause of pulmonary impairment in at-risk workers. Occurrence of new cases of silicosis may be due to inadequacy of current respirable dust limits or failure to control workplace exposure to respirable silica. In addition to the classic occupational settings for silicosis risk in miners, foundry workers, and sandblasters, silicosis may also occur in ceramic (86) or construction
workers (87). A newly recognized cause of accelerated silicosis and silicoproteinosis is sandblasting denim clothing, mainly in developing countries where few exposure controls exist (17,18,48,88). In a study of former denim sandblasters, chest radiographic evidence of silicosis was found in 77 of 145 subjects (18). Other newly recognized occupational groups at risk for silicosis include goldworking jewelers (89) and electric cable manufacturers (90).

Centrilobular and perilymphatic nodules are characteristic of silicosis. Nodal enlargement with or without nodal calcification may also be seen (91). Emphysema (22,92) and expiratory gas trapping (82) are important contributors to physiologic impairment. Conglomerate masses occur typically in the posterior upper lobes or superior segments of lower lobes (93) and are often hypermetabolic on positron emission tomography (PET) scans (Fig 12) (3,94,95). Slowly progressive fibrosing interstitial pneumonia with a pattern typical of UIP may occur in about 10% of silicosis patients (Fig 13) (96,97). The prevalence of pleural abnormality in silicosis has been underemphasized; silicosis is associated with unexplained pleural effusions in 11% and pleural thickening in 58% of subjects (11). Rounded atelectasis may also be seen (98,99). In acute or subacute silicoproteinosis, consolidation is characteristic. Crazy-paving pattern may or may not be seen, and other findings may include centrilobular nodules and pulmonary calcifications (57).

**Coal Worker’s Pneumoconiosis**

Despite knowledge of risk factors and implementation of dust control measures, increasing prevalence of pneumoconiosis among underground U.S. coal miners has been identified in several recent publications. Wade et al (100) reported 138 newly identified cases of coal workers pneumoconiosis—related PMF in West Virginian coal miners from 2000 to 2009, with 21 deaths. Nearly all miners in this group who developed PMF experienced exposures following implementation of federal dust regulations. Laney et al (101) also report increased prevalence of pneumoconiosis in underground coal miners from 1980 to 2008. Additionally, regional increases...
in prevalence of coal workers pneumoconiosis in Appalachia have been reported (102).

The imaging features of coal worker’s pneumoconiosis are similar to those described for silicosis. A substantial minority (10%–40%) of coal miners affected by coal worker’s pneumoconiosis develop diffuse lung fibrosis, characterized on the chest radiograph by small irregular opacities in the lower lungs (99,103,104). These irregular opacities correlate better than rounded opacities with the extent of physiologic impairment (105). On chest CT scans, this entity is characterized by reticular abnormality often associated with honeycombing, similar to UIP or NSIP (106,107). Reticular abnormality may or may not be associated with pneumoconiotic nodules. This pattern of diffuse interstitial fibrosis appears to be associated with a high prevalence of lung cancer, preferentially occurring in areas of lung fibrosis (107).

Asbestos-related Lung and Pleural Disease

Though exposure to friable asbestos has been increasingly regulated in developed countries, CT-based studies have shown substantial prevalence of asbestos-related disease in current and former asbestos workers (108,109). A recent CT study of 1011 asbestos-exposed workers showed that 47% had pleural plaques and 6% had asbestosis (109). There is increased concern about environmentally acquired asbestos-related disease. For example, in Libby, Montana, a significant increase in mortality from lung cancer, mesothelioma, and asbestosis occurred in miners and community residents owing to locally mined vermiculite contaminated with small amounts of tremolite asbestos (Fig 14) (110–112). In this population, the presence of radiographic pleural disease was associated with restrictive lung function (113). In developing countries, where hazardous occupations like ship demolition and asbestos milling are often poorly regulated, the prevalence of asbestos-related disease on radiographs remains substantial (114,115).

Hypersensitivity Pneumonitis

Hypersensitivity pneumonitis is a group of pulmonary syndromes caused by repeated inhalation of and sensitization to a wide variety of organic aerosols and some chemical antigens. Diagnosis relies on a constellation of features including antigen exposure, characteristic signs and symptoms, pulmonary function abnormalities, radiologic abnormalities, and histologic findings (116). Work-related exposures important in development of hypersensitivity pneumonitis include microbially-contaminated metal-working fluids, isocyanates used in two-part paints, and organic dusts in farming, among many others. However, no clear causal antigen can be identified in a substantial minority of cases. In a series of 85 consecutive cases of hypersensitivity pneumonitis presenting to the Mayo Clinic, avian antigens accounted for 29 cases (34%), with hot tub lung in 18 (21%) and farmer’s lung in nine (11%) (117). No cause was identified in 21 cases (25%). Regardless of the antigen, typically only a minority of exposed individuals will develop hypersensitivity pneumonitis. The clinical diagnosis of chronic hypersensitivity pneumonitis can be quite challenging because of the variety of pulmonary presentations and the diversity of potential antigenic causes, which may not be elicited in a routine medical history (118).

Imaging features of hypersensitivity pneumonitis include poorly defined centrilobular ground-glass nodules (Fig 15),
ground-glass attenuation, mosaic attenuation, expiratory gas trapping, and features of lung fibrosis, including reticular abnormality and honeycombing (119–124). Emphysema and cysts may occur in chronic cases (62,77,125). Classically, the presentation of hypersensitivity pneumonitis has been divided into acute, subacute, and chronic forms. However, the imaging findings in hypersensitivity pneumonitis do not necessarily correlate with the duration or presentation of symptoms, except for the fact that fibrosis is found only in chronic hypersensitivity pneumonitis (Fig 16). When CT signs of lung fibrosis are present, mortality is substantially increased (126,127).

**Chronic Beryllium Disease**

Beryllium-related lung disease may be acute (now very rare) or chronic. Although chronic beryllium disease technically meets the definition of pneumoconiosis, it differs from pneumoconiosis in the following ways: (a) It represents a granulomatous hypersensitivity response to inhaled beryllium, and (b) its incidence and severity are not always related to the intensity and duration of exposure; in fact, it may occur with relatively minimal exposure, particularly in those with a genetic predisposition (128).

Exposure to beryllium occurs in a variety of industries (including aerospace, ceramics, dentistry, nuclear weapons and reactors, and several others) where workers may be at risk for disease from either direct or indirect exposure to the metal. Recently a case series of community-acquired chronic beryllium disease was reported in a neighborhood surrounding a beryllium manufacturing plant (68).

The radiographic and CT appearances of chronic beryllium disease are similar to those of sarcoidosis, though mediastinal and hilar lymphadenopathy are less common, occurring in about 25% of cases (Fig 17) (129). The radiologist should include the differential diagnosis of chronic beryllium disease in every patient with imaging appearances suggestive of sarcoidosis. In a recent series of 84 patients with previously diagnosed “sarcoidosis,” the diagnosis was corrected to chronic beryllium disease in 34 (130).

**Emergence of New Occupational Lung Diseases**

Industrial progress has resulted in multiple new causes of occupational lung disease. Development of organizing pneumonia in a series of Spanish textile workers in 1992 was attributed to Acramin-FWN exposure and later termed Ardyystil syndrome (5,55,56,59). At the Ardyystil plant in Alcoy, Spain, aerosolized Acramin-FWN for use in textile printing resulted in inhalational exposure of this product designed for brush or sponge application. Romero et al (56) found that nine of 14 Ardyystil syndrome patients demonstrated bilateral, peripheral predominant, patchy airspace consolidation on CT scans.

Exposure-related outbreaks have also been reported related to the nylon flocking, artificial flavoring, and flat-panel screen industries. Initially recognized at a Canadian plant in 1995, flock worker’s lung arises from respirable flock, an ultra-fine nylon fiber used in the production of certain fabrics, and results in a nongranulomatous interstitial lung disease (131). Subsequently, eight workers at a different plant were diagnosed in 1998 with flock worker’s lung, characterized on thin-section CT scans by patchy ground-glass and consolidations or diffuse micronodularity and at pathologic evaluation by lymphoctic bronchiolitis and peribronchiolitis with lymphoid hyperplasia (15,16).

In 2000, eight workers at a microwave popcorn plant in Missouri presented with constrictive bronchiolitis associated with exposure to the artificial butter flavoring chemical, diacetyl (2,3-butanedione), and termed flavor worker’s lung (4,14).

Reports from Japan recognized the relationship of indium-tin oxide to interstitial lung disease (24,132). The properties of indium-tin oxide allow for production of transparent conductive films utilized in flat-panel screens. Two separate cases of indium-tin oxide exposure were described related to the flat-panel screen industry, both of which resulted in biopsy-proved pulmonary fibrosis. At CT imaging, the initial case in 2002 resulted in subpleural honeycombing, while the subsequent case in 2003 demonstrated upper lobe predominant fine nodular opacities, ground-glass opacities, and associated emphysema. In 2010, Cummings et al (12) described two additional cases of...
Indium-tin oxide exposure resulting in pulmonary alveolar proteinosis, with one resulting in death. Unregulated novel chemical agents may result in concentrated exposures and acute respiratory illness. Lee et al (13) reported 15 cases of 1,1-dichloro-1-fluoroethane (HCFC-141b) exposure resulting in acute pulmonary toxicity. Animal testing of HCFC-141b showed low risk for toxicity. However, when utilized in an electronics factory to clean circuit boards in the setting of limited ventilation and no respiratory protection, HCFC-141b triggered onset of respiratory symptoms in 15 workers over the course of 1 day. By the following day, all 15 workers presented to the hospital with progressive respiratory symptoms. Subsequent evaluation with imaging, bronchoscopy, and pulmonary function testing confirmed acute pulmonary toxicity, which resolved without treatment.

Unique situations such as military service in a war zone may be associated with limited ability to control hazardous exposures and substantial variability in occupational exposure risks. In a case series by King et al (81), constrictive bronchiolitis was described in soldiers returning from Iraq and Afghanistan with symptoms of unexplained shortness of breath. While less than a quarter of the soldiers demonstrated abnormalities such as centrilobular nodules or air-trapping on thin-section CT scans (Fig 18), 38 out of 49 of those who underwent surgical lung biopsy were diagnosed with constrictive bronchiolitis. Many but not all of the soldiers diagnosed with constrictive bronchiolitis reported specific exposure to combustion products from an industrial sulfur fire in Mosul, Iraq. The prevalence of and risk factors for deployment-related lung disease await further investigation. A multidisciplinary group of scientists recently recommended that postdeployment military personnel with unexplained respiratory symptoms or physiologic impairment should undergo diagnostic testing including thin-section CT (133).

**Conclusion**

The radiologist plays a pivotal role in the diagnosis of occupational lung diseases. Radiologic diagnosis of occupational lung disease requires awareness of the diverse patterns of lung injury related to occupational exposures, appropriate use of CT, and awareness of newer occupational exposures. The radiologist is often ideally placed to recognize potential occupational lung disease and question the clinician about possible exposures that, if causally relevant, may lead to more targeted medical management and prevention.

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