Chronic respiratory diseases of occupational or environmental origin, and those associated with cigarette smoking are the leading causes of respiratory disability in the United States and throughout the world [1]. The interplay between occupational and environmental exposures and concomitant tobacco exposure, genetic susceptibility, and comorbid diseases complicates diagnosis, prevention, and the response to illness [2]. As a consequence, the physician is frequently called upon to assist in the assessment of the impact of these diseases on the well-being and livelihood of the affected individuals. Impairment and disability evaluation of the respiratory system is frequently done in the context of a patient’s application for benefits under a disability or entitlement program or for legal proceedings. Therefore, along with the medical system, legal and administrative systems also have important roles in this determination.

This article reviews the evaluation of impairment and disability caused by respiratory diseases, including common occupational and environmental respiratory diseases. It is intended to provide a framework by which practicing physicians may understand their role and responsibilities in the determination of disability. The respiratory system differs from many other organ systems; objective measures of pulmonary function exist and are readily applicable to the evaluation.

Definitions

Impairment is defined as any loss or abnormality of psychological, physiological, or anatomical structure or function that is objectively measured; it may be temporary or permanent, and may be variable in severity. Impairment assessment is a medical evaluation and the physician’s role is to determine the presence and severity of a pulmonary disorder using objective criteria. Disability is defined as any restriction or lack of ability to perform an activity in the manner or within the range considered normal for a human being [3]. An impairment or a disability can be temporary or permanent, depending on whether it is expected to improve with time or treatment. To aid in the determination of impairment, several professional organizations such as the American Thoracic Society (ATS), the American Medical Association (AMA), and the European Society for Clinical Respiratory Physiology have developed detailed guidelines for the evaluation of impairment [4–6].

Clinical approach to evaluating pulmonary impairment

The evaluation of patients for impairment caused by respiratory diseases requires a comprehensive history and physical examination, followed by appropriate standardized testing such as spirometry, diffusing capacity, and often, chest radiography to establish a diagnosis and determine the severity of the disease.
In the minority of cases, more comprehensive cardiopulmonary exercise testing may be indicated to define the extent of impairment.

**History**

The primary symptoms of respiratory disorders are cough, dyspnea, wheezing, and chest tightness or pain. These symptoms need to be correlated with physical findings and objective testing to assess their importance and implication.

Cough is an important defense mechanism that helps to clear excessive secretions, irritants, and foreign materials from the airway. Cough is a nonspecific symptom that can be caused by a multiplicity of disorders in various locations. Virtually any condition that stimulates cough receptors or afferent nervous pathways is capable of producing cough [7]. Cough can be categorized as acute or chronic based on the duration of symptoms. Acute cough is generally considered as less than 3 weeks’ duration and typically of minor consequence (eg, viral syndrome, acute exposure to airway irritants). Rarely, acute cough may signal a potentially life-threatening condition (eg, pulmonary embolism, congestive heart failure). Cough is considered chronic if it lasts longer than 3 weeks. The most common causes of chronic cough are asthma, chronic bronchitis, bronchiectasis, postinfectious cough, postnasal drip syndrome, and gastroesophageal reflux disease. These conditions account for more than 95% of the cases of chronic cough [8–10]. Other less common conditions associated with cough include bronchogenic carcinoma, chronic interstitial pneumonia, sarcoidosis, left ventricular failure, and aspiration caused by pharyngeal dysfunction [10,11].

A wheeze is a continuous, musical sound that is produced by a large number of conditions associated with obstruction at various levels of the airway [12,13]. Wheezing originates in airways of any size, from the large extrathoracic upper airway to intra-thoracic small airways. The pathophysiologic mechanisms that generate wheezing are not fully understood.

An appropriate history should include questions related to the frequency of duration, triggers producing symptoms, and the response of symptoms to intervention including trigger avoidance or medication. For example, asthma should be considered likely when patients present with episodic wheezing triggered by exposure to cold, infection, irritants, allergens, or other workplace sensitizing agents such as latex, isocyanates or organic antigens [14–16].

Dyspnea, or shortness of breath, is a distressing sensation of difficult, labored, or unpleasant breathing. Dyspnea is a sensitive, but not specific, symptom of respiratory disease. Most dyspnea seems to be caused by five major etiologies including cardiac disease, pulmonary disorders, psychogenic/hyperventilation, gastroesophageal reflux disease, and deconditioning disorders. The majority of patients can be categorized into one of four conditions; chronic obstructive lung disease, asthma, interstitial lung disease, or cardiomyopathy [17–19].

A detailed occupational and environmental exposure history is required to establish a diagnosis of an occupational or environmental lung disease. This history should include all employments in chronological order, including part-time jobs and hobbies. History of exposure to specific respiratory agents in the workplace like dusts, fumes, and vapors should prompt further questioning to clarify the timing, duration, and dose of exposure when possible, and the use of respiratory protection. Presence of similar complaints or diseases in coworkers also is helpful in establishing an occupational etiology. An environmental exposure history should include a discussion of potential environmental triggers that include pets in the household, exposures to birds or other organic antigens, such as molds, as well as seasonal or temporal variability of symptoms.

Smoking can be the primary cause or a major contributing factor to many respiratory diseases, therefore a detailed smoking history should be obtained. This should include types of tobacco smoked, quantity, and duration of smoking, estimated in pack-years, as well as second-hand smoke exposure.

**Physical examination**

A physical examination, that is primarily focused on the cardiopulmonary system, should be performed. This should include the height, weight, and vital signs at rest. A description of the patient’s breathing pattern, and presence or absence of labored breathing at rest should be recorded. The timing and quality of breath sounds suggest various processes. For example, early coarse inspiratory crackles may be heard in diseases of airflow obstruction with excess mucus production or airway collapse, whereas late inspiratory crackles or fine end inspiratory crackles may be present in patients with chronic interstitial diseases or congestive heart failure [12].

An important extra pulmonary manifestation of pulmonary diseases is digital clubbing. Clubbing is believed to be a consequence of chronic hypoxia, but other unidentified factors likely play a role, as well [20,21]. Respiratory diseases associated with clubbing include pulmonary fibrosis, bronchiectasis, bronchogenic carcinoma, and pleural tumors [22–24].
The patient also should be evaluated for clinical evidence of right heart failure associated with advanced and chronic respiratory disease (cor pulmonale). Typical findings include distended neck veins, dependent edema, enlarged liver, which may be accompanied by ascites, a right ventricular early diastolic gallop rhythm along the left sternal border or epigastrium that increases with inspiration, or a right ventricular heave [25–27].

Pulmonary function tests

Spirometry

Spirometry is the most commonly used objective measure of respiratory limitation. Forced vital capacity and forced expiratory volume in the first second are effort-dependent, expiratory maneuvers that require patient cooperation, but no specific training of the patient. These tests may be performed reliably and reproducibly on relatively simple equipment, at low cost, and spirometry is available in many physicians’ offices. Portable equipment allows for workplace evaluation of pulmonary function.

Valid pulmonary function testing requires that the tests be performed by trained technicians in strict accordance with standardized techniques [28, 29]. The AMA guidelines for disability evaluation recommend obtaining three spirometric tracings with reasonable uniformity of flow pattern and exhibiting concordance of at least two values within 5% [4]. Tests may either be performed off medication or after administration of a bronchodilator, dependent on the question to be answered (see later discussion). The largest value, either pre- or postbronchodilator, should be used to quantify impairment. The values obtained for FVC and FEV₁ may be compared with normative data described by various investigators, that provide predicted values on the basis of age, gender, and height [30]. The ATS and AMA guidelines suggest the use of normal values of Crapo [4, 31]. Racial differences in normal values were reported. Adjustments for race are available for blacks, but no reliable data yet exist for other racial groups. Although some controversy exists regarding race corrections, the AMA guide suggests multiplication of predicted normal FVC and FEV₁ values by 0.88 for blacks, before calculation of the value as a percent of predicted. Measurements should always be compared with the patient’s previous testing, especially if tests were performed before a specific injury or exposure.

Spirometry can distinguish obstructive from restrictive lung physiology and provide quantification of the degree of pulmonary dysfunction. On the basis of FVC, FEV₁, and diffusing capacity, classification of degree of respiratory impairment can be made using the AMA recommended guidelines (Table 1) [4]. The AMA classes of respiratory impairment refer to values as percent of predicted for spirometry and diffusing capacity, but do not differentiate whether the values are reflective of obstructive or restrictive lung disease.

Measurements that are readily available from routine spirometry, but not recommended for use in evaluation of impairment, include the midflow rates

<table>
<thead>
<tr>
<th>Pulmonary function measurement</th>
<th>Class 1 0%–9% Impairment of the whole person</th>
<th>Class 2 10%–25% Impairment of the whole person</th>
<th>Class 3 26%–50% Impairment of the whole person</th>
<th>Class 4 51%–100% Impairment of the whole person</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC</td>
<td>≥ lower limit of normal, and</td>
<td>≥ 60% of predicted and &lt; lower limit of normal, or</td>
<td>51%–59% of predicted, or</td>
<td>≤ 50% of predicted, or</td>
</tr>
<tr>
<td>FEV₁</td>
<td>≥ lower limit of normal, and</td>
<td>≥ 60% of predicted and &lt; lower limit of normal, or</td>
<td>41%–59% of predicted, or</td>
<td>≤ 40% of predicted, or</td>
</tr>
<tr>
<td>FEV₁/FVC</td>
<td>≥ lower limit of normal, and</td>
<td>≥ 60% of predicted and &lt; lower limit of normal, or</td>
<td>41%–59% of predicted, or</td>
<td>≤ 40% of predicted, or</td>
</tr>
<tr>
<td>DL_CO₂sb</td>
<td>≥ lower limit of normal, or</td>
<td>≥ 60% of predicted and &lt; lower limit of normal, or</td>
<td>41%–59% of predicted, or</td>
<td>≤ 40% of predicted, or</td>
</tr>
<tr>
<td>VO₂max</td>
<td>≥ 25 ml/kg/min, or ≥ 7.1 METS</td>
<td>20–25ml/kg/min, or 5.7–7.1 METS</td>
<td>15–20 mL/kg/min, or 4.3–5.7 METS</td>
<td>&lt; 15 mL/kg/min, or 4.3 METS</td>
</tr>
</tbody>
</table>

Adapted from: American Medical Association Guides to the Evaluation of Permanent Disability, 5th edition, 2000; with permission.

Abbreviations: METS, metabolic units.
(ie, flow measured at the mid-portion of the expiratory loop [FEF_{25–75%} and FEF_{50%}]). These measurements are relatively effort-independent, and are believed by some, to reflect small airways disease. The measurements are highly sensitive, but correlate poorly with maximal achievable oxygen consumption or dyspnea, among subjects with normal FEV_{1} and FVC [32]. In smokers, the midflow rates are directly related to the FEV_{1} and diffusing capacity, but they add no additional information when the conventional forced expiratory measures are abnormal [33]. The usefulness of midflow rate measurement is debated. It was suggested that abnormality of the small airways, as reflected by FEF_{25–75%}, precedes the development of clinically significant obstructive airways disease. Thus, abnormality of the midflow rates may reflect a “probabilistic estimate of future risk” [32]; without other predictors for development of functional impairment, it is problematic to use midflow rate abnormality to assign disability of the basis of an unknown probability of future risk.

Diffusing capacity

The measurement of the diffusing capacity for carbon monoxide (CO) is routinely performed as a single breathhold maneuver, (DL_{CO sb}), and is measured as mL/min/mmHg at standard temperature pressure dry conditions. The diffusing capacity for CO reflects the ease with which oxygen moves from the alveolus into the red blood cells in the pulmonary capillaries. In actuality, the usefulness and the limitation of the diffusing capacity measurement derive from the fact that the measurement integrates a number of variables into a single value; it is dependent on the surface area available for gas exchange, the thickness of the interstitium, as well as the diffusivity of CO through each of the epithelial and endothelial interfaces from alveolus to red blood cell. In addition, lung volume, distribution of ventilation, volume of plasma, and hemoglobin concentration all have an impact on the measured diffusing capacity. As a consequence of these variables, the measurement is highly nonspecific, but can be sensitive to a variety of factors. For a full discussion of diffusing capacity see a recent review by Crapo and colleagues [34].

Performance of the diffusing capacity test requires careful attention to standards such as those published by the American Thoracic Society [28]. Accurate instrument calibration and precise execution of the diffusing capacity test is critical, and the test should be performed by experienced pulmonary function technicians. Patients should be instructed to refrain from smoking for at least 8 hours before testing to minimize blood content of CO [4]. Elevated levels of carboxyhemoglobin (COHb) may decrease the diffusing capacity by reducing the availability of hemoglobin binding sites and creating back pressure of CO in the plasma. Venous COHb may be measured if smoking abstinence is in doubt. Apart from forming a firm seal around the mouthpiece, the test does not require particular training or cooperation on the part of the patient. Normative reference values based on age, height, and gender are well-established for diffusing capacity [4]. It is recommended that predicted normal values be multiplied by 0.93 for blacks, but no adjustments are recommended for other racial groups [4].

The diffusing capacity is routinely used in conjunction with spirometry to classify impairment for respiratory disorders (see Table 1). It has particular usefulness in evaluation of restrictive ventilatory defects and is more sensitive than lung volume measurement as a screening tool for patients at risk for pneumoconiosis [35].

Cardiopulmonary exercise tests

Standard pulmonary function testing with measurement of FVC, FEV_{1}, and DL_{CO sb} is sufficient for assessment of the majority of patients who are undergoing disability evaluation [4]. Certain subgroups of patients, however, may require more extensive and sophisticated evaluation. Relationships between simple spirometric measurements and DL_{CO sb} and dyspnea on exertion, oxygen consumption and work capacity have been established [36–39]. On the basis of these studies, the AMA, ATS, and the American College of Occupational and Environmental Medicine recommended the use of spirometry and diffusing capacity as the key objective elements of assessment of respiratory impairment.

The relationship between simple pulmonary function testing and exercise limitation, however, is not strong. For example, Cotes and colleagues [40] tested the hypothesis that exercise limitation associated with respiratory dysfunction may be accurately predicted by measurement of dynamic lung volumes and diffusing capacity. Using the maximal oxygen uptake on exertion (VO_{2} max) as the “gold standard” of exercise limitation of respiratory origin, they found that the predictive value of the individual spirometric indices (FVC, FEV_{1}, and FEV_{1}/FVC) or diffusing capacity was poor, accounting for only 15% to 25% of the variance in VO_{2} max. The predictive power of these values was improved by using them in combination and further enhanced by incorporating the ventilatory capacity during submaximal exercise into the analysis. Similarly, not everyone who has gas
exchange abnormalities during exercise testing will have a reduced DL_{CO2}; the diffusing capacity is, in some cases, an insensitive predictor of gas exchange abnormality, although its specificity is good [41].

As a result of limitations inherent to simple pulmonary function testing, there has been increased interest in the role of cardiopulmonary exercise testing (CPET) in disability evaluations. The measurement of oxygen consumption during CPET is considered to be the “gold standard” assessment of the subject’s ability to perform work. When performed properly, the CPET can provide a quantifiable measure of exercise capacity, as well as offer the examiner a means of differentiating respiratory impairment from deconditioning or cardiac dysfunction.

An advantage of measuring oxygen consumption in a particular subject, is that the individual’s maximal work capacity may be quantified and compared with the established oxygen requirements of his or her job. This comparison relies on the assumption that, if appropriately conditioned, a worker is generally able to sustain 40% to 60% of his or her maximal oxygen consumption over the majority of a work day. Near maximal oxygen consumption may only be sustained for shorter times, at intervals throughout the day. Oxygen consumption and energy expenditure were determined for work of various intensities and data exist for the typical energy expenditure imposed by specific employment categories. Such data can be a useful reference tool, but energy expenditure during the performance of work is dependent on a variety of factors; specific comparisons of an individual with normative values are limited by the nature of the data. For example, jobs of the same title may vary considerably from one work site to another. Workers will have different oxygen consumption depending on their physiologic characteristics such as body size, obesity, age, muscular energetics, and muscle length–tension relationships. Workers are unlikely to perform tasks in a stereotypical fashion throughout the workday and considerable interworker variability likely exists that depends on work experience, efficiency, and organization. Additionally, tolerance of dyspnea likely differs from one person to another. In one study, general attitude explained more of the variance in dyspnea and oxygen consumption than did FEV₁ [42]. A more detailed discussion of the complexities of oxygen consumption and disability is available in an excellent review by Harber [32].

The addition of cardiopulmonary exercise testing to routine evaluation of impairment is appropriately left to the discretion of the examining physician. If the examining physician believes that standard pulmonary function testing does not accurately reflect the degree of ventilatory impairment of an individual, consideration may be given to CPET; VO₂ max can be used to assess the degree of impairment under certain guidelines (see Table 1). The ATS statement on evaluation of impairment and disability in patients with respiratory disorders suggested that consideration should be given to CPET in persons whose work necessitates sustained moderate exertion or frequent heavy exertion, and is accompanied by significant dyspnea [31].

**Chest radiograph**

The chest radiograph is a standard element of the clinical evaluation of patients with respiratory disease. In most cases, the assessment of respiratory impairment and disability does not rely on the chest radiograph, because the correlation between radiographic abnormality of the chest and physiologic dysfunction is imperfect. For example, among patients with obstructive lung disease, chest radiography may correlate poorly with the degree of abnormality of lung mechanics. An asthmatic patient with severe, partially irreversible airflow obstruction may have only minimal abnormality of the chest radiograph. Conversely, patients with a significant amount of bullous emphysema may not have a comparable degree of airflow limitation. Nonetheless, a subject with a chest radiograph that shows hyperinflation, diaphragmatic flattening, bullae, and prominence of the pulmonary arteries (suggesting cor pulmonale) is very likely to have significant ventilatory impairment and disability. Thus, chest imaging, whether by simple chest radiograph or CT scan, may be an ancillary tool in the evaluation of pulmonary diseases, despite the absence of specific guidelines about how to incorporate the findings into the assessment of impairment.

Abnormalities of the chest radiograph may be used to assign disability in selected cases. Among patients with pneumoconioses (inorganic dust-related pulmonary interstitial diseases such as asbestosis, silicosis, and coal workers’ pneumoconioses), the importance of the radiographic findings is heightened. In an attempt to objectively characterize and communicate the extent of radiographic abnormalities in the pneumoconioses, the International Labor Organization (ILO) devised a classification scheme for the standardized interpretation of chest radiographs [43]. The National Institute for Occupational Safety and Health (NIOSH) trains and certifies physicians as “B readers” who are deemed proficient in the use of this classification system. The ILO
classification system is designed as a tool for description rather than diagnosis of a disease; findings are to be reported as “consistent with” rather than “diagnostic of” a specific pneumoconiosis. The reporting scheme rates small opacities by shape, size, and profusion on posteroanterior view of the chest radiograph (i.e., rounded opacities are designated as $p$, $q$, or $r$ and linear opacities are designated as $s$, $t$, or $u$ by size, and the extent of the opacities is rated as 0, 1, 2, or 3 with intermediate scores such as 1/2 assigned to images with profusion of opacities greater than 1, but less than 2). The system also allows for characterization of the sites, extent, and calcification of pleural thickening and plaques. The clinical significance of the chest radiograph may follow from the detailed description.

The AMA Guide to the Evaluation of Permanent Impairment recommends that those who develop pneumoconiosis should limit further exposure to the offending agent, “particularly if radiographic changes have occurred at a relatively young age or if there is associated physiologic impairment” [4]. It follows that an older patient who is nearing retirement, with minimal radiographic change after a long history of exposure, could elect to continue in the workplace, under the assumption of a lower risk of development of disabling disease. Such discretionary decisions should be made with informed discussion between the patient and physician.

**Computed tomography**

Specific guidelines for the use of computed tomography in disability assessment have not been established. Computed tomography (CT) scans offer considerable insight into the precise anatomic changes that accompany a disease state, but offer little to the evaluation of physiologic impairment. The greater resolution of high resolution CT compared with simple chest radiography, allows for identification of more subtle interstitial changes. The argument may be made that earlier identification of subtle disease could prompt removal of the individual from exposure, thus decreasing the risk of disease progression. There are no recommendations at present for CT-screening of populations at risk for occupational interstitial lung disease.

**Arterial blood gas measurement**

Arterial blood gas measurement of oxygen and carbon dioxide tension is considered to be an adjunctive tool in the assessment of impairment [4]. The results of arterial blood gas measurements are surprisingly variable within the individual. No validated relationship has been defined between resting arterial oxygenation and physiologic impairment, although extremely low oxygen tensions and oxygen desaturation with exertion are undoubtedly significant. These caveats, along with the invasive nature of the test, limit the usefulness of routine arterial blood gas measurement in assessing pulmonary impairment.

**Disease specific impairment assessment**

**Obstructive lung disease**

**Asthma**

Evaluation of impairment in the setting of asthma is one of the more problematic aspects of disability assessment in respiratory disease. Asthma is defined as an inflammatory disorder of the airways accompanied by variable airflow limitation, which is at least partially reversible, and airways hyperresponsiveness. Asthma may be categorized as allergic (extrinsic), intrinsic, and occupational. Each of these subtypes poses a specific challenge to the physician who performs the evaluation of impairment and potential disability. Because of the complexities involved with asthmatic patients, the ATS developed specific guidelines for the evaluation of impairment and disability for the asthmatic population [44].

The variable nature of asthma is the most evident problem that impedes disability assessment. As a result of the variability, a patient’s lung function on the day of evaluation may not be an accurate reflection of the patient’s usual condition, but may be atypically bad or atypically good. Additionally, the impact of medical treatment of asthma may be substantial, and what is considered as adequate treatment may vary from one practitioner to another. In addition to these treatment goals, nonpharmacologic interventions must be addressed, such as control of environment triggers, patient and family education, and compliance. Efforts must be made to ensure that evaluation of impairment is performed, with the patient as well-treated and clinically stable as possible [4].

As with all evaluations of respiratory impairment, spirometry is the key objective measurement of function. The initial goal of evaluation is to document the presence of asthma. It is recommended that spirometry be performed after withholding inhaled, short-acting bronchodilators for at least 6 hours before testing and long-acting agents (including theophylline) for 24 hours. A notation should be made if the patient is unable to withhold medication use as directed. Anti-inflammatory agents should not be
restricted before testing. When the FEV₁/FVC ratio is below the lower limit of normal, a short-acting β-adrenergic agonist should be acutely administered, in an effort to identify reversible airflow obstruction. Presence of reversibility (defined as a 12% increase in FEV₁ and an increase of ≥200 mL) suggests the diagnosis of asthma. Patients without reversibility should be trialed with inhaled or oral steroids in an attempt to achieve a 20% increase in FEV₁, and thus, a diagnosis of asthma. Patients with a history that is suggestive of asthma and who have normal spirometry in the absence of medication for asthma, may be tested for airway hyperresponsiveness with nonspecific bronchodilator challenge [45]. The usefulness of diffusing capacity measurement is to exclude conditions other than asthma that exhibit airflow obstruction (eg, emphysema, or combined obstructive and restrictive ventilatory defects).

After a diagnosis of asthma is established with reasonable certainty, evaluation of the degree of impairment may follow. If current treatment is optimal, as outlined by the guidelines for the treatment of asthma discussed earlier, then the classification of impairment may be made as dictated by the scoring scheme in Tables 2 and 3. Patients who have not yet achieved optimal control may be rated on a temporary basis and then re-evaluated for long-term impairment and disability.

The current ATS impairment classification scheme differs from the previous recommendation [31,44]. In the older recommendations, the frequency of acute exacerbations and treatment in emergency departments and hospitals were incorporated into the evaluation of impairment. Most physicians now agree that frequent exacerbations do not necessarily suggest more severe disease, but may imply inadequate control, and potentially the need for more aggressive management of inflammation and bronchospasm (eg, increased use of inhaled corticosteroids or addition of long-acting bronchodilators).

Occupational asthma narrows the definition of asthma to disease that is attributable to a specific workplace exposure(s). It is now the most frequently reported occupational lung disease in the industrialized world. Occupational asthma caused by workplace exposures should be distinguished from work-aggravated asthma (ie, pre-existing asthma which is aggravated by workplace exposures). Occupational asthma may be either immunologic or nonimmunologic in origin. Nonimmunologic, or irritant-induced, asthma occurs following high-concentration occupational exposures to a variety of particulate or toxic irritants. Immunologically-mediated occupational asthma is characterized by immune sensitization over a period of time. After an individual is sensitized, re-exposure, even to very low levels, can result in asthma (eg, isocyanate and latex-induced asthma). Early diagnosis and elimination of exposure will decrease the risk of progressive pulmonary dysfunction [46]; thus, complete removal from the sensitizing agent is recommended. It is important to distinguish immunologic occupational asthma from irritant-induced asthma, where continued work may be possible if irritant exposures are reasonably controlled.

Evaluation of impairment in patients with occupational asthma is similar to that for patients with atopic asthma. If impairment is identified, temporary disability may be assigned. Reassessment should be made 2 years after cessation of exposure, when the anticipated improvement is likely to have plateaued [47]. It is estimated that between 60% and 90% of subjects will fail to completely resolve their asthma after exposure is eliminated. Many of these patients will continue to have nonspecific airways hyperresponsiveness in the absence of the agent to which they were originally exposed. A given individual’s disability that is related to occupational asthma can vary greatly depending on the patient’s occupation and work exposures (see later discussion).

Chronic obstructive pulmonary disease

Patients with chronic obstructive pulmonary disease constitute a specific subset of patients with airflow obstruction. The AMA guidelines suggest the use of the impairment classification (see Table 1) for the rating of impairment in patients with COPD [4]. The guidelines provide no specific recommendations for the evaluation of patients with COPD; however, some recent data suggested that work-related exercise capacity may not be adequately predicted by standard pulmonary function tests alone. Among a cohort of 216 workers with COPD, tested with routine spirometry and CPET, only 30% of subjects were similarly classified by the two techniques. Cardiopulmonary exercise testing suggested less impairment than spirometry in 61% of the subjects and more impairment in 9% [48]. Cardiopulmonary exercise testing is not routinely available, is not easily performed, and is costly. Currently, recommendations for evaluation of impairment in patients with COPD include routine testing with spirometry and diffusing capacity alone, with exceptions as noted earlier.

Upper airway obstruction

Upper airway obstruction is also associated with airflow limitation that leads to respiratory impairment. Such obstruction may be caused by upper
airway tumors, compression of the airway by extrinsic masses, narrowing of the airway by laryngeal or tracheal sarcoidosis, or vocal cord dysfunction that leads to airway obstruction. Spirometry, with particular attention to the shape of the flow volume loop, can elucidate whether large airway obstruction is intra- or extrathoracic, fixed or variable. Truncation of the expiratory limb suggests intrathoracic large airway obstruction, whereas truncation of the inspiratory limb is associated with extrathoracic obstructing lesions. There are no specific guidelines for evaluating the degree of impairment in these cases, although the disability may be substantial because of airflow limitation and the impact of irritants on the large airway lesions.

Restrictive lung disease

Many conditions produce restrictive lung physiology, including idiopathic pulmonary fibrosis and interstitial lung disorders associated with collagen vascular disease, sarcoidosis, or drug-induced conditions [49]. Occupational and environmental exposures are notorious causes of interstitial disease and its associated restrictive ventilatory defect. Inorganic dust exposure produces the pneumoconioses, which include asbestosis, silicosis, and coal workers’ pneumoconiosis, each with fairly characteristic radiographic appearances. Exposure to beryllium or hard metals also is associated with pulmonary fibrosis. Organic dust exposures (eg, among bird breeders and

Table 2
Impairment classification of asthma severity

<table>
<thead>
<tr>
<th>Score</th>
<th>Post bronchodilator FEV₁, % of predicted</th>
<th>% change in FEV₁</th>
<th>Degree of airway Hyper-responsiveness, PC₂₀ (mg/mL)</th>
<th>Minimal medication needs</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>≥ 80%</td>
<td>&lt;10%</td>
<td>&gt; 8</td>
<td>None</td>
</tr>
<tr>
<td>1</td>
<td>70%–80%</td>
<td>10%–19%</td>
<td>8–&gt;0.6</td>
<td>Occasional bronchodilator or cromolyn</td>
</tr>
<tr>
<td>2</td>
<td>60%–69%</td>
<td>20%–29%</td>
<td>0.6–&gt;0.125</td>
<td>Daily bronchodilator or daily cromolyn or daily corticosteroid (CS)³</td>
</tr>
<tr>
<td>3</td>
<td>50%–59%</td>
<td>&gt;30%</td>
<td>≤0.125</td>
<td>Bronchodilator prn and daily high-dose CS³, or 1–3 courses of oral CS per year</td>
</tr>
<tr>
<td>4</td>
<td>&lt; 50%</td>
<td></td>
<td></td>
<td>Bronchodilator prn and daily high-dose CS³, and daily or QOD oral CS</td>
</tr>
</tbody>
</table>

PC₂₀ (mg/mL): provocation concentration which causes a 20% fall in FEV₁. QOD, alternate day.

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a ≤800 mcg beclomethasone or equivalent
b >800 mcg beclomethasone or equivalent
c >1000 mcg beclomethasone or equivalent

Table 3
Impairment rating for asthma

<table>
<thead>
<tr>
<th>Class</th>
<th>Impairment of the whole person</th>
<th>Total asthma score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class 1</td>
<td>0%</td>
<td>0</td>
</tr>
<tr>
<td>Class 2</td>
<td>10%–25%</td>
<td>1–5</td>
</tr>
<tr>
<td>Class 3</td>
<td>26%–50%</td>
<td>6–9</td>
</tr>
<tr>
<td>Class 4</td>
<td>51%–100%</td>
<td>10–11, or asthma not controlled despite maximal treatment</td>
</tr>
</tbody>
</table>

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a FEV₁ <50% of predicted despite use of ≥20 mg/day of prednisone or equivalent
farmers) may precipitate hypersensitivity pneumonitis and interstitial disease on that basis.

High resolution CT imaging may allow for identification of subtle degrees of interstitial disease among exposed workers who do not yet exhibit restrictive patterns on pulmonary function testing; it is not used to assess impairment. Patients with restrictive lung disease are classified by the same schema used for those who have obstructive lung disease, relying primarily on spirometry and diffusing capacity reduction (see Table 1). The limitations of routine spirometry and diffusing capacity apply to subjects with restrictive lung disease, as well as to those with obstructive physiology.

Obstructive sleep apnea

Obstructive sleep apnea is a disorder that is characterized by intermittent, repetitive occlusion of the large airway during sleep and resultant nocturnal desaturation. Obstructive sleep apnea may result in impairment caused by daytime hypersomnolence and in some cases, cor pulmonale. Although polysomnography can identify the severity of obstructive events, there is no recommendation for impairment classification based on polysomnographic findings; the assessment of impairment is left to the physician’s judgement. Hypersomnolence is of particular concern in patients for whom constant workplace vigilance is essential (eg, vehicle and machine operators). Obstructive sleep apnea is a treatable disorder. The ultimate management strategy is weight loss, but use of continuous positive airway pressure mask ventilation is effective in eradicating the apneas and the resultant hypersomnolence. Re-evaluation of impairment after treatment is, therefore, essential.

Disability evaluation

Disability evaluation is the determination of the impact of impairment on an individual’s ability to meet the demands of his or her own life. This can be assessed in terms of compromised work capacity (ie, evaluating the impact of respiratory impairment on a specific occupation or trade or any reasonable occupation). Disability evaluation has a broader focus than specific occupation or trade or any reasonable occupation. This can be determined using the legal standard of “more probable than not”, or greater than 50% probability of a disorder being attributable to work-place exposure [50]. This process requires identification of one or more of the known etiologic factors for a specific disease, an understanding of the natural history of the disease as it relates to exposure, and an assessment of the extent of the patient’s relevant work exposures and other host factors. Establishing a temporal relationship between a specific exposure and onset of the disease is critical (eg, exposure to western red cedar in a carpenter and the development of asthma). Appropriate latency between initial exposure to an

without reference to a specific occupation. Impairment does not necessarily imply disability. For example, although two individuals with asthma caused by sensitization to a specific agent (eg, animals) may have the same impairment, the individual who is an animal handler by trade may be completely disabled from his or her own occupation, whereas the other individual (an office worker) may have no disability.

Information provided by the clinician on a patient’s impairment due to respiratory disorders is used by administrators to determine the extent of disability and is translated into financial reimbursements. [50] The process by which disability is assigned varies according to the criteria of eligibility and entitlement for the specific program under which the evaluation is performed. Examples of entitlement programs include social security insurance, workers’ compensation, state and federal based eligibility programs including the Department of Veteran’s Affairs and personal or employer offered disability insurance. [51,52] Benefits are provided to individuals who meet the program- specific criteria for disability given the severity of impairment, and in the case of workers’ compensation, criteria for attributing the cause of impairment to a workplace exposure.

Physician’s report

Evaluations of impairment require a comprehensive report of the patient’s history, physical examination, and objective tests. The assessment should include a summary of the questions being addressed, the diagnosis, and the degree of impairment referencing the impairment scheme used (eg, AMA or ATS guidelines). Work-related respiratory disorders seldom present as a clinically distinct respiratory disease (ie, lung cancer or asthma). Attribution of causality is a complex process in which a physician attempts to determine whether a disorder was caused or made worse by the patient’s workplace exposures.

Causality in worker compensation cases is usually determined using the legal standard of “more probable than not”, or greater than 50% probability of a disorder being attributable to work-place exposure [50].
agent and development of disease is also important in determining causality. Most individuals are exposed to multiple agents in the workplace; therefore, knowledge of the interactions of these agents is essential. The physician may also be asked to determine apportionment, an estimation of the contribution of multiple diseases or risk factors, to the final impairment. These determinations rely on the judgment of the examining physician.

Summary

Respiratory disorders, including occupational and environmental lung diseases, are prevalent. Physicians are frequently called upon to determine impairment and aid in the assessment of disability caused by these conditions, either as the treating physician or as an independent medical examiner. In this article we reviewed the role of physicians in determining the presence and severity of pulmonary disorders. A comprehensive clinical assessment and appropriate standardized tests, to objectively characterize the severity of impairment, are the key elements of the evaluation. This assessment may also include the physician’s opinion regarding causative factors. Finally, disability determination is made by nonclinicians, through administrative means, based on the degree of impairment and a review of circumstances specific to the individual. Knowledge of these components of disability evaluation will help physicians to better serve their patients and supply appropriate data to the adjudicating system.

References


