Chapter 5: The Pulmonary System

Introduction

This chapter provides a framework for the recognition and assessment of pulmonary impairments that affect the individual's ability to perform Activities of Daily Living (ADLs). Pulmonary assessment requires clinical evaluation, measurement of pulmonary function, analysis of the relevant data and then comparison of this clinical information to the *Guides'* criteria to arrive at an impairment rating.

This chapter provides a brief overview of the principles of pulmonary assessment. The most recently published guidelines from the American Thoracic Society (ATS) and the European Respiratory Society (ERS) are the primary references used in this chapter. Additionally, a brief discussion is provided on the use of contemporary diagnostic testing, including the use of specific laboratory data and imaging studies. For a more detailed review of pulmonary assessment, the reader should refer to the numerous textbooks available on the subject.

~~Substantial transformation is apparent throughout this edition of the *Guides*.~~ The reader is encouraged to read Chapters 1 and 2 in their entirety and understand the key concepts and philosophy of the AMA *Guides* before reading this chapter.

~~The functional impairment classes have been standardized. In addition, the Table of Permanent Pulmonary Impairment has been updated to reflect consistency to the extent that is practical with the common impairment rating grid and to comply with the goal to create uniformity and internal consistency across various chapters. In this update to Chapter 5, key changes are the use of pulmonary function reference equations in a way to promote health equity across racial/ethnic categories, ages, and sexes; changes from blaming to patient-centered language; and recognition of the skin pigmentation bias of pulse oximetry.~~

Updates to this chapter aim to enhance consistency, replace vague symptom terms with descriptive language, and update medication criteria to reflect current practices, while ensuring consistency across chapters and promoting health equity. The revisions introduce a new diagnostic row framework to streamline the rating process.

~~A relatively new~~The concept of, ~~"Burden of Treatment Compliance," has also been introduced~~ updated into the Sixth Edition and relates well to impairment ratings for asthma and other pulmonary disorders. As the treatment options increase for a variety of pulmonary disorders, individuals are able to live longer with less dysfunction, ~~*albeit* at the expense of rigid compliance with the treatment. Most disease survivors are therefore receiving treatment and lead productive lives, yet without treatment, members of this group would have limited function leading to greater impairment. Any Burden of Treatment for pulmonary disease is already accounted for in the various impairment tables in this chapter and must not be the basis for additional impairment by itself.~~

5.1 Assessing the Pulmonary System

The goals of the impairment assessment of the pulmonary system should be to determine if a permanent pulmonary impairment exists, ~~quantify~~ estimate its severity, assess ~~its~~ expected impact on the ability to perform ADLs, and, when possible, identify the cause of the abnormality and

recommend measures to prevent progression of impairment and ensure optimum function. ~~The use of pPulmonary function testing reference equations to quantify pulmonary function, —is the core objective measure for theimpairment rating of impairment, —helps allocates resources equitably by estimating the likelihood of an individual being observed in the reference population and the The published evidence shows that the use of race-neutral rather than race-specific reference equations leads to a more similar association of pulmonary function withlikelihood that an individual's pulmonary function is associated with respiratory symptoms, respiratory events, structural abnormalities in the respiratory system, and all-cause mortality across race categories (ATS statement). Therefore, race-neutral pulmonary function reference equations more equitably allocate resources compared to race-specific reference equations. Furthermore, race categories are imprecise when applied to individuals from real populations which are heterogeneous both at one point in time and across time.~~

A detailed clinical evaluation of the pulmonary system should begin with a comprehensive history. An inquiry into specific symptom severity, duration, and manner of onset guides the initial evaluation. ~~Personal habits~~Individual-level exposures~~, especially such as the use of combustablecombustible~~ tobacco ~~cigarettes, e-cigarettes, or other inhaled substances and drugs, must be ascertained. An individual's~~ birth history ~~(e.g. premature birth), childhood~~ respiratory ~~illnesses, environmental exposures (including second hand smoke), and previous chest diseases, surgery, or trauma to the chest may be of consequence.~~ Individual-level exposures, such as the use of combustible tobacco cigarettes, e-cigarettes, or other inhaled substances, must be ascertained. An individual's birth history (e.g. premature birth), childhood respiratory illnesses, environmental exposures (including second hand smoke), and previous chest diseases, surgery, or trauma may be of consequence. A detailed work history is of critical importance. Workplace exposures to potentially toxic substances might explain or contribute to respiratory symptoms. When work exposures are thought to be a potential cause for impairment, a systematic review of all jobs and their possible exposures beginning with employment during the worker's teen years may well be relevant. Each of a worker's jobs should be described in detail, because often specific environmental exposures may be associated with just one of the many occupations in a workplace. A thorough history enables the examiner to direct the physical examination to areas of concern and identify the most appropriate diagnostic studies. The physician then evaluates the structural or movement abnormalities of the chest and its contents. Important features of the examination are detailed in Section 5.4.

~~Although i~~Imaging techniques provide information on the radiographic ~~severity~~ structure of a pulmonary abnormality, ~~they~~ and are most helpful in identifying and diagnosing lung disease. Radiologic techniques provide visual evidence of internal anatomic abnormalities that are not apparent by external inspection, palpation of the chest wall, or percussive or auscultatory assessment. Advanced radiographic techniques may be necessary. For example, a high-resolution computed tomography (CT) scan of the chest may help elucidate anatomic abnormalities recognized in interstitial lung diseases, and a CT scan with pulmonary embolism protocol may identify obstructive abnormalities in the larger pulmonary arteries.

Pulmonary function testing is the core objective measure for impairment rating. Appropriate measurement techniques and reference equations are discussed further in Section 5.4d. These tests are most helpful in classifying the severity of physiological impairment.

~~Pulmonary function tests are the most reliable for assessment of functional changes in the lungs and pulmonary interstitium. The appropriate techniques are discussed in Section 5.4. These tests are most helpful in addressing classifying the category of respiratory disease physiological impairment and its severity. and the extent of impairment.~~

5.2 Clinical Presentation of Pulmonary Disease

Symptoms and signs associated with pulmonary dys-function include dyspnea, cough, sputum production, hemoptysis, wheezing, chest pain or tightness, and night sweats. ~~Although q~~Quantification of some of these indicators of pulmonary abnormalities may be difficult in view of their subjective nature. ~~,~~ ~~t~~The severity, duration, and manner of onset of each of these specific symptoms should be explored during history taking.

5.2a Dyspnea

Dyspnea is the most common presenting symptom in patients with pulmonary impairment. Its importance is matched only by its non-specificity and resistance to objective quantification. Dyspnea can be caused by deconditioning, anxiety, or diseases of cardiac, hematologic, metabolic, or neurologic origin.

Various schemes have been developed to grade dyspnea. The most widely used classification system is based on the ATS Lung Diseases Pulmonary Symptom Questionnaire[1] (Table 5-1). ~~This classification is an attempt to provide a reasonable means to compare an individual's symptoms with objective measurements of pulmonary function.~~ Although generally helpful,[2] in some instances, there may be a poor correlation between ~~lung~~ pulmonary function (as measured by spirometry) and symptoms~~subjective complaints~~. Examples when this might occur could include individuals with lung function within the reference range but with pulmonary vascular disease; an individual with asthma who has recovered from an acute episode; or an individual who had baseline above-average lung function. but loses function after onset of another disease, thereby leaving them with lung function within the reference range despite marked structural abnormality. If there is a great disparity between the subjective complaints and the objective findings, a more complete and detailed investigation may be necessary.[3] ~~Examples when this might occur could include individuals with normal lung function within the reference range but with pulmonary vascular disease, ; or an individual with an asthmatic asthma who has recovered from an acute episode; or an individual who had baseline above-average lung function. but loses function after onset of another disease, thereby leaving them with lung function within the reference range despite marked structural abnormality, or an applicant for disability who exaggerates his or her symptoms. If there is a great disparity between the subjective complaints and the objective findings, a more complete and detailed investigation may be necessary.~~[3]

Table 5-1 Impairment Classification of Dyspnea (Adapted)[a]

Severity	Definition and Question
Mild	Do you have to walk more slowly on level ground than people of your age because of breathlessness?

Severity	Definition and Question
Moderate	Do you have to stop for breath when walking at your own pace on level ground?
Severe	Do you ever have to stop for breath after walking about 90 m (100 yd) or for a few minutes on level ground?
Very severe	Are you too breathless to leave the house, or breathless on dressing or undressing?

[a] Adapted from Ferris BG. Epidemiology standardization project: American Thoracic Society. *Am Rev Respir Dis*. 1978;118(6, pt 2): 1–120. The person's lowest level of physical activity and exertion that produces breathlessness denotes the severity of dyspnea.

5.2b Cough, Sputum Production, and Hemoptysis

Although cough is considered to be an important indicator of lung disease,[2] there are other explanations for cough, including such non-pulmonary illnesses as gastroesophageal reflux,[3] medication,[4] postnasal drip, and esophageal dysfunction.[5] ~~Although a~~A recent review has suggested that computerized cough counting over a 24-hour period is an effective way to determine the severity of cough, ~~it~~however, this ~~is a~~ is currently a research tool and not widely available.[6] ~~For these reasons, the~~The presence of cough is not considered an objective determinant of pulmonary impairment. Nonetheless, it is incumbent on the physician to document its presence or absence, associated sputum production, duration, and any associated hemoptysis. The purpose of this documentation is to identify individuals who require further evaluation.

Once non-pulmonary causes of cough are ruled out, an acute, self-limited cough most commonly is reasonably attributed to infection or airway irritation. A subacute or recurrent non-productive cough may be a manifestation of asthma and should be investigated further with pulmonary function testing. A chronic, productive cough is often a marker of bronchitis. ~~According to ATS criteria, t~~The term *chronic bronchitis* is used to describe a cough productive of sputum that occurs on most days for at least 3 consecutive months per year, for at least 2 years in succession.[7] In some individuals with a chronic productive cough, bronchiectasis should be considered.[8]

Hemoptysis frequently accompanies bronchitis and pneumonia, usually in the form of blood-streaking of the sputum. Although a common cause of hemoptysis is bronchitis, hemoptysis may be life-threatening, with serious causes including bronchogenic carcinoma, pulmonary embolism, bronchiectasis, tuberculosis, vasculitis, and arteriovenous malformations. The presence of hemoptysis requires evaluation to determine whether this finding indicates a disease that might lead to impairment. In particular, hemoptysis in male smokers over the age of 50 years carries a high risk of lung cancer, and bronchoscopy is indicated.[9]

5.2c Wheezing

High-pitched, musical sounds often are described as wheezing by patients who have partial airway obstruction. These sounds can be generated at any point along the airway from the glottis to the bronchioles. Identification of the part of the respiratory cycle where the wheeze is identified is important. Inspiratory wheeze, or stridor, suggests laryngeal disease, whereas expiratory wheeze can be a feature of bronchospasm or bronchitis and suggest airway secretions with localized

bronchial narrowing. Seasonal occurrence of wheezing suggests allergy. Intermittent wheezing suggests a bronchospastic, allergic, or asthmatic cause,[10] whereas persistent wheezing raises the suspicion of a fixed bronchial obstruction. Wheezing and/or cough occurring primarily in the workplace, or having a definite temporal relationship to work, suggest occupational or work-exacerbated asthma.[11] Wheezing that ~~follows~~ starts after several minutes of exercise and either continues for several minutes or gets worse after cessation of exercise suggests exercise-induced asthma.[12] Finally, wheezing that usually accompanies pulmonary tract infections, typically in a person with~~n~~ asthma~~tic~~, can be classified as asthmatic bronchitis.[13]

Although these different varieties of asthma are commonly described as separate entities, the clinical presentations are similar, attributable to the common underlying mechanism of airway hyperresponsiveness.

5.3 Environmental Exposures~~, Lifestyle Choices,~~ and Pulmonary Disease

5.3a Tobacco Use

The most common cause of pulmonary impairment is combustible tobacco cigarette smoking. Although there is variable individual susceptibility to the adverse effects of combustible tobacco cigarette smoke, a discernible dose-response relationship is commonly recognized.[14] The examining physician should standardize data collection regarding combustible tobacco cigarette use by inquiring about the age when the patient started smoking, age at quitting or current age if the smoking continues, and the average number of packs smoked per day. Multiplying the number of years of smoking by the number of packs smoked per day produces a commonly expressed measure for cigarette use, pack-years. This information can be used in assessing the impact of ~~personal habits~~individual exposures on pulmonary impairment and may aid in the apportionment of pulmonary abnormality among various deleterious factors. Combustible tobacco ~~C~~cigarette smoking is the most significant causative factor in the development of chronic bronchitis, emphysema, and lung cancer. Chronic exposure to environmental tobacco smoke (ETS, or "second-hand" smoke) ~~appears~~ may also ~~to~~ be carcinogenic,[15] promulgates adverse asthma outcomes in younger individuals,[16] and potentiates the risks for atherosclerotic vascular disease.[17] There is also evidence linking ETS with COPD).[70] Smoking cessation in individuals with underlying lung disease can be a life-saving intervention.[18]

5.3b Occupational History

Environmental exposure in the workplace often is cited as a causative or contributory factor in the development of pulmonary impairment. To evaluate the possible effects of such exposure, it is important to obtain a complete occupational history. A major part of the history consists of a chronological description of work activities beginning with the year of first employment. This description includes the names of employers, the specific types of work performed, the material or materials used, and the potentially toxic material that the worker is able to identify in the workplace. Employers are required to maintain a list of potentially toxic materials used in the workplace. This is available to the employee and the treating physician in the form of Material Safety Data Sheets. Such information includes the chemical descriptions of the agent under consideration, and the physical and health hazards. This information can aid the examiner in directing the assessment. An estimate of frequency, duration, and intensity of exposure to each

substance is needed to assess its significance. Information about the use of pulmonary protective devices is important and should be elicited. The number of years the devices were used with each employer should be recorded.

In addition to information regarding workplace environmental exposures, information should be collected about hobbies or leisure activities that might involve exposure to potential respiratory toxins. Home and environmental exposures (encountered in the enjoyment of a hobby or during a leisure activity) to organic and inorganic agents such as allergens, bioaerosols, paints, glues, or pesticides may be more potent causative agents of a pulmonary disorder than the putative agents present in the workplace. As examples, in the home, exposure to pets and the use of cool-mist vaporizers, humidifiers, and indoor hot tubs may be associated with pulmonary ~~disease~~impairment.

There are a number of ways that the lung can be injured and ~~disabilities~~ impairment can develop. The outcome of the exposure to the toxic agent, irritant, or sensitizing material varies based on whether the exposure is acute or chronic in nature, the amount of the agent in the ambient environment at the time of exposure, and the physical characteristics of the agent.

An acute, excessive irritant gas exposure can be associated with several outcomes. Acute pulmonary parenchymal injury may result from the inhalation of a highly irritant gas, fume, mist, or vapor. This is recognized as noncardiogenic pulmonary edema or the acute respiratory distress syndrome (ARDS). If the individual survives the acute pulmonary injury, the healing process may produce diffuse pulmonary fibrosis or obliterative bronchiolitis, both of which may lead to functional impairment. If the outcome of such an exposure is persistent cough and asthma, this is described as reactive airways dysfunction syndrome (RADS).

Recurrent inhalation of gases or fumes at irritant exposure levels can result in persistent airway irritation and cause chronic bronchitis. If the worker is exposed to agents with a sensitizing potential, airway hyperresponsiveness can develop and the signs and symptoms of asthma develop. An irritant exposure can also exacerbate (i.e., cause a temporary worsening that returns to baseline) a worker's preexisting, underlying condition such as asthma, chronic bronchitis, or emphysema. Inhalation of organic material or certain types of reactive chemicals can cause hypersensitivity pneumonitis or asthma through an immune-mediated pulmonary mechanism. Inhalation of fibrogenic dusts, typically over a protracted time, can cause pneumoconiosis (e.g., silicosis, asbestosis, or coal workers' pneumoconiosis). Workplace exposures to a variety of carcinogens, such as asbestos, can cause lung cancer.

5.4 Clinical Evaluation, Imaging Studies, and Other Tests for Evaluating Pulmonary Disease

5.4a Physical Examination

~~Although a~~ A thorough physical examination is an important component of ~~in~~ judging pulmonary impairment. ~~, it may not be sensitive in early stages of pulmonary disease, or it may be normal in a disease such as asthma during the nonacute phase.~~ A thorough physical examination should include:

- Vital signs measured after the patient has had an opportunity to relax and become accustomed to the surroundings.

- A detailed chest examination. The physician should note the use of accessory muscles on respiration and the patient's body habitus. A breathing pattern characterized by pursing the lips during expiration suggests chronic obstructive pulmonary disease (COPD). The thoracic cage should be inspected for vertebral or rib cage deformity, wasting of the intercostal muscles, features of a barrel-shaped chest that may indicate hyperinflation, and adequacy of the movement of the ribs with inspiration and expiration.
- Percussion of the chest is carried out to ascertain hyperresonance or consolidation and to assess diaphragmatic motion.
- In a healthy chest, auscultation reveals vesicular breath sounds throughout the lung, with bronchovesicular sounds over the trachea. Adventitious sounds include decreased breath sounds, crackles, wheezes, and rhonchi. The intensity, quality, and location of wheezing, rhonchi, and rales should be described, as well as whether they are heard during inspiration, expiration, or both. In bronchitis, coarse sounds attributable to airway secretions change location with cough. Crackles are typically present in individuals with inter-stitial disease; these usually occur during late inspiration. Early inspiratory crackles may be heard in bronchiolitis obliterans. ~~The presence of wheezing cannot be excluded until the physician performs auscultation during both quiet breathing and forced expiration.~~ Diffuse, bilateral, expira-tory wheezing indicates generalized broncho-spasm, whereas unilateral or localized wheezing may be caused by partial bronchial obstruction.

Cyanosis, indicated by a ~~bluish~~ discoloration of the lips, is a striking but unreliable indicator of impairment. Its presence can be attributed to anemia, changes in skin pigmentation, heart disease associated with right to left shunt, and severe pulmonary impairment. Poor lighting in the examination room and variations in skin pigmentation can interfere with assessing its magnitude. Suspicion of cyanosis calls for measurement of oxygen saturation by pulse oximetry or arterial blood gas analysis. Measurement error of pulse oximetry in people with darker skin pigmentation should also be acknowledged and mitigated whenever possible.[59] Although less likely, also consider methhemoglobinemia ~~or carboxyhemoglobinemia~~.

Hypercarbia may be suspected in an individual with a substantially impaired respiratory status; however, accurate assessment of this abnormality requires arterial blood gas determination. Alternatively, metabolic testing can measure end-tidal CO_2 and provide a reliable assessment.

Digital clubbing is characterized by loss of the angle at the junction of the cuticle and the nail, softening of the nail bed, increased curvature of the nail, and widening of the distal portion of the fingers or toes. It is usually a sign of advanced disease. Digital clubbing needs to be differentiated from pseudoclubbing. This can generally be done by examination of the Lovibond angle, which is ≥ 180 degrees with clubbing, and <180 degrees with pseudoclubbing. Diseases of the chest associated with clubbing include pulmonary fibrosis, bronchiectasis, bronchogenic carcinoma, pleural tumors, lung abscess, empyema, and cyanotic congenital heart disease.

5.4b Imaging Studies of the Chest

Chest Radiography: ~~Roentgenograms~~

The initial chest radiographic examination should include posteroanterior and lateral views of the chest taken in full inspiration. Chest radiographic findings often correlate poorly with physiologic findings ~~in diseases with~~of airflow limitation, such as asthma and emphysema~~, particularly in the tall and slender individual~~. Persistent abnormalities of the chest radio-graph may be classified as parenchymal, vascular, pleural, or osseous. Inspection of the mediastinum and trachea and the major airways may identify abnormalities. Terms used to describe parenchymal changes include *hyperinflation, fibrosis, cavitary*, or *cystic*.

Briefly, airway obstruction as seen in asthma, emphysema and bronchitis may show hyperinflation of the lungs with accentuated bronchi. Asthma and emphysema are associated with parenchymal destruction identified as flattening of the diaphragms, vascular attenuation, an increased anteroposterior diameter of the chest, and increased retrosternal airspace. In addition, the plain chest radiograph can provide evidence of pulmonary vascular abnormalities associated with chronic pulmonary disease. Pulmonary hypertension is indicated by bilateral enlargement of the main pulmonary arteries and rapid tapering of the peripheral vessels. Cor pulmonale is suggested by enlargement of the right ventricle and the changes of pulmonary hypertension. The presence of pulmonary hypertension and cor pulmonale should be confirmed by additional clinical and laboratory tests.

The radiographic features of fibrotic lung diseases may be localized or diffuse, reflecting the distribution of such anatomic features in the lung. Because of the redundancy of pulmonary tissue in the normal individual, diffuse fibrotic disease is much more likely to cause impairment than is localized disease. Diffuse, fibrotic abnormalities assume the characteristic radiologic appearance of rounded (nodular) or linear (reticular). Specific diagnostic information is obtained by describing both the type and the predominant location of fibrotic changes observed on the chest radiograph. As examples, sarcoidosis, coal workers' pneumoconiosis, the miliary distribution of fungal or acid-fast organisms (e.g., miliary tuberculosis), and silicosis all may present with a distribution of small lung nodules (typically in the upper zones with silicosis) and not infrequently with additional radiographic abnormalities. Interstitial lung diseases with irregular or linear abnormalities are typically distributed in the lower zones and include scleroderma, idiopathic pulmonary fibrosis, and asbestosis. Pleural abnormalities, such as pleural plaques or diffuse pleural thickening, may also be identified in individuals with asbestosis or may be the sole manifestation of past asbestos exposure. In the absence of pulmonary fibrosis, asbestos pleural plaques are not associated with pulmonary impairment unless extensive, diffuse, massive pleural thickening causes lung entrapment.

A standardized scheme of classifying radiographic abnormalities associated with fibrotic diseases caused by the pneumoconioses was adopted by the International Labor Office (ILO) in 1950 and was most recently revised in 2000.[19] Although not designed to be used in the context of impairment assessment, this radiograph evaluation and grading process has become a key part of the medical legal system. It is worth noting that the purported objective nature of the ILO classification system notwithstanding, the correlation of interpretations and readings with physiologic measures of impairment is poor. Some reports address the interrater and even intrarater reliability of the ILO classification system, particularly in the medicolegal context.[20] The US National Institute for Occupational Safety and Health (NIOSH) regularly administers an examination to certify knowledge and proficiency in the use of this method.[21] Those competent in this evaluation process are

identified as “B” readers. In association with the American College of Radiology, NIOSH provides hands-on training seminars as well as a self-study program.

Computed Tomography

Computed tomography and high-resolution computed tomography (HRCT) scans are radiographic techniques that may augment the standard chest radiograph. A conventional CT scan is obtained by averaging the signals of 10-mm-thick sections through various lung fields. This technique is good for identifying anatomic features and for recognizing nodules with high radiographic attenuation. Because HRCT consists of 1- to 2-mm-thick sections through the affected part of the lung, it provides a clearer definition of the parenchyma; it is most useful for addressing interstitial lung disease. There is no standardized process for the evaluation of interstitial abnormalities found by HRCT comparable to that used in the evaluation of the chest radiograph.[22] The standard CT and/or HRCT can provide greater accuracy as part of a thorough assessment of the pulmonary parenchyma. ~~With its fewer sections, the HRCT delivers significantly less whole body effective dose radiation than does thestandard CT.~~ If HRCT is performed by skipping large portions of lung, it can deliver significantly less whole-body effective dose radiation than conventional CT. On many modern machines and centers, conventional CT scans consist of < 2 mm-thick sections, and HRCT does not skip portions of the lung.

Also, HRCT is helpful in the detection of early changes in the lung consistent with focal emphysema; regional air trapping associated with small airway disease, such as obliterative bronchiolitis; and large airway abnormalities, such as bronchiectasis. For example, air trapping of the type seen with obliterative bronchiolitis is best demonstrated by comparing full inspiratory and full expiratory scans. Prone and supine position scans also are helpful in distinguishing atelectasis and hydrostatic changes related to blood volume that are transient and can occur in the dependent position of the lungs from fixed parenchymal abnormalities.[23]

5.4c Spine and Other Musculoskeletal Abnormalities Affecting Pulmonary Function

Thoracic cage and osseous spine abnormalities may produce pulmonary impairment due to mechanical factors that affect the size of the chest cavity and restrict rib motion. Kyphoscoliosis, the most common of these abnormalities, is characterized by curvature of the vertebral column from side to side in the frontal plane (scoliosis) and from the dorsal to the ventral aspect in the sagittal plane (kyphosis). Although not always interpreted in a uniform manner, the Cobb method is a commonly used measurement tool for curvature severity.[24] With this method, the posteroanterior and lateral spinal radiographs measure the curvature angles. Only severe curvature angles—Cobb angles that are greater than 100°—are likely to lead to pulmonary failure. Even when there are severe spinal deformities, pulmonary decompensation usually does not occur until middle age or later.

With severe spinal abnormalities, pulmonary compromise is produced by the combined effects of restricted lung volume, decreased cross-sectional area of the vascular bed, and age-related decrease in chest wall compliance. Progressive stiffness of the chest wall with advancing age increases the work of breathing and leads to ~~hyperventilation~~hypoventilation, which produces hypoxia and hypercapnia. Hypoxia is a powerful pulmonary vasoconstrictor and further decreases the vascular cross-sectional area, eventually leading to cor pulmonale. Judge the severity of

pulmonary impairment on the criteria described in the sections on forced pulmonary maneuvers, diffusing capacity for carbon monoxide, and the criteria for rating impairment due to pulmonary disease in this chapter.

5.4d Physiologic Tests of Pulmonary Function

Pulmonary function studies including spirometry, diffusing capacity of the lung for carbon monoxide (DLco) and measurements of exercise capacity such as oxygen consumption per unit time (Vo_2), performed on standardized equipment with validated administration techniques, provide the quantitative measurement on which the pulmonary impairments tables are based in this chapter. It is critical that the technician be appropriately trained and knowledgeable regarding the contraindications of performing spirometry, ~~optimally measuring patient features such as~~accurate measurements of height and weight, knowing how to position the individual to optimally perform lung function tests, and protecting the patient from infection.[25]

~~Spirometric~~ Pulmonary function testing ~~equipment, calibration, and administration techniques~~ must conform to the ~~guidelines~~ standards ~~presented in~~of the ATS/ERS ~~Standardization of Spirometry~~[26] ~~report~~.[60] When the clinician desires to document ~~reversible~~ responsive airway obstruction, the subject should undergo baseline testing if not taking any drugs before the test. Short-acting drugs (albuterol, salbutamol, and ipratropium) should not be used within 4 hours of testing. Longer acting β-agonists (salmeterol, formoterol), oral aminophylline, leukotriene receptor antagonists, tiotropium, or slow-release-agonists should be withheld for at least 12 hours.[60] Cigarette smoking should be avoided for at least 1 hour before testing.

Forced Pulmonary Maneuvers on Ventilatory Study (Simple Spirometry)

An acceptable forced expiratory maneuver has a maximal inspiration, a satisfactory start of test, an ~~smooth~~ expiratory effort free from artifact (cough, glottic closure), and a plateau at the end of test. Measurements available from the forced expiratory maneuver and relevant to the assessment of impairment include the forced vital capacity (FVC), forced expiratory volume in the first second (FEV_1), and the ratio of these measurements (FEV_1/FVC).

Interpretation of lung function tests involves three tasks. The first is the comparison of an individual's measured values to a set of reference values; the second the classification of the physiological impairment; and the third is the clinical diagnosis which considers the individuals medical history, symptoms, and other clinical measures. A measured volume is compared with the expected range of a population of individuals of the same biological sex, height, and age.

~~Interpretation of lung function tests involves three 2 tasks. The first is the comparison of the an tested individual's measured individual's values to a set of reference values, ; and the second is an interpretation of the values that were measured the classification of the physiological impairment,; and the third is the clinical diagnosis which considers the individuals medical history, symptoms, and other clinical measures. A measured volume is compared with the expected range of a population of individuals of the same biological sex, height, and age. Recommendations for selecting reference values include the following: using sex (gender), age, and height, and matching age-range and height ranges, and racial or ethnic background. There should be similar lung function instruments and lung function testing protocols in the tested groups compared with the~~

~~reference group. All parameters should be taken from the same reference source. Differences in the assessment of lung function using different sets of reference equations have been recognized.[27] A difference in prediction values for different races has been identified.[28] Specifically, North American whites have larger spirometric values for a given age, height, and sex than North American blacks, with a similar tendency noted for Hispanics, Native Americans, and Asians; in the past, adjustments were made in the prediction equations based on race. Currently, the Third National Health and Nutrition Examination Survey (NHANES III) has ethnically appropriate reference equations that are recommended for people aged 8 to 80 years.[29] Reference values for whites, African Americans, and Mexican Americans aged 8 to 80 years were developed from 7429 asymptomatic, lifelong nonsmokers. Reference values and lower limits of normal were derived using a model with age and height as predictors. These reference values encompass a wide age range for 3 racial or ethnic groups and make it unnecessary to use adjustments for race.~~

~~Measurement of total lung capacity can be an important part of lung function assessment. A number of different approaches to measuring lung volumes have been put forward, including body plethysmography (using various methods), nitrogen washout, gas dilution, and radiographic imaging. More recently, measurement of lung volumes using imaging techniques such as CT or magnetic resonance imaging (MRI) have been added. There are inadequate data to recommend one approach over another.[30] The readers are reminded that a restrictive ventilatory defect is characterized by the reduction in total lung capacity below the fifth percentile of the predicted value, and a "normal" FEV_1/FVC. Although a restrictive defect can be suspected when the FVC is reduced and the FEV_1/FVC is normal or increased, this is not always the case. A reasonable approach to the clinical indications for the measurement of total lung capacity is presented by Aaron et al[31] and Gladys and colleagues.[32]~~

Although there is no clear consensus as to when to ~~measure lung function after~~compare changes in lung function after administration of bronchodilator inhalation, reasonable indications would include an FEV_1/FVC below 0.70, wheezing on physical examination, or a history suggestive of asthma. When the pre-bronchodilator spirometry demonstrates ~~airway~~ airflow obstruction (FEV1/FVC below 0.70, an FEV1 <80% predicted) wheezing on physical examination, or a history suggestive of asthma, then albuterol should be given.

For purposes of impairment rating determination, consistency in the type of bronchodilator, its dose, time for administration, and time for testing is paramount. To improve interrater reliability, the following steps should be followed:

- ~~and spirometry repeated 10 to 15 minutes later (post-BD).~~ A total of 360-400 mcg of albuterol should be inhaled by the ~~patient~~ examinee, one deep inhalation at a time, with 30-60 seconds between each of the four puffs.
- A volume spacer should be used between the metered dose inhaler and the person. ~~At least 3 acceptable FVC maneuvers should be repeated post-BD.~~
- Spirometry repeated 20 minutes after the final administration of inhaled bronchodilator.
- At least 3 acceptable ~~FVC~~spirometry maneuvers should be repeated post-bronchodilator administration~~BD.~~ To demonstrate repeatability, the second highest FEV1 should match

the highest FEV1 within 0.15 liters. The highest FEV1 should be reported. Increments of less than 8% of 150 mL are likely to be within measurement variability.

A change exceeding 12% or 200 mL compared with baseline suggests a "significant" bronchodilation. Changes in postbronchodilator FEV_1 and FVC values can be helpful in understanding the potential medication responsiveness and prognosis, but it is not reasonable to assume that the absence of a significant change means that bronchial responsiveness is absent (e.g., in the well-controlled ~~asthmatic~~ patient with asthma there may be no responsiveness).

A comprehensive interpretation of lung function tests is beyond the scope of this discussion, and the reader is advised to ~~read~~ consult any of a number of ~~books~~ resources that address this topic. However, the following discussion ~~would~~ will be helpful in understanding the current literature on the topic.

~~Use of Predicted Values From NHANES III~~Reference Ranges

Historically, race-specific reference equations (or "race correction factors") were used to interpret lung function measurements. The rationale for race-specific approaches were to account for population-level differences in lung function. Race is a social construct that is based on appearance and reflects social values, structures, and practices. Classification of people into racial and ethnic groups differs geographically and temporally. These considerations challenge the notion that racial and ethnic categories have biological meaning and question the use of race in PFT interpretation.

In 2020, the American Thoracic Society convened a workshop to critically appraise available evidence. The use of race-specific approaches may mask the effects of differential exposures on lung health, and may contribute to health disparities by norming population differences in lung function.[61] Consequently, the ATS now recommends race-neutral PFT interpretation approaches. Race-neutral approaches include~~,~~ (1) interpretation of the ratio of FEV to FVC, which is relatively constant between populations and racial and ethnic groups, (2) interpretation of measured FEV or FVC values using a race-neutral prediction equation (i.e., the Global Lung Function Initiative reference equations), and (3) tracking changes in absolute values over repeated measurements.[62] Multiple studies have established that the use of race-neutral rather than race-specific reference equations leads to a more similar association of pulmonary function with respiratory symptoms, respiratory events, structural abnormalities in the respiratory system, and all-cause mortality across race categories.[58, 71-74, 76,] Therefore, race-neutral pulmonary function reference equations more equitably allocate resources compared to race-specific reference equations. Subsequently, the AMA has adopted a policy to eliminate the use of race adjustment measurements in medical diagnostic equations, consistent with this approach. As a result, The Guides will now utilize the Global Lung Function Initiative (GLI) normative values for purposes of determining impairment ratings in this chapter.

Normative values can be determined using the calculator tools located at https://gli-calculator.ersnet.org/

~~Multiple studies have now established the superiority of a race-neutral prediction equations over race-specific equations for achieving equity in the association between lung function and symptom~~

~~burden, structural changes in the lungs, and survival (can just use ATS statement or Baugh; McCormack; Elmaleh-Sachs; Liu; and so on but now there are so many!).~~

~~In the past, reliable population data were not available for ethnic groups, such as Hispanics, Native Americans, and Asians. Studies recognized that whites had greater mean FVC and FEV_1 value than did Mexican Americans and African Americans across the entire age range. For these ethnic groups, the values for North American whites had been used, yet adjustments were necessary.[28] For the same height, the lung function of African Americans was less, resulting in adjustments in predicted FVC and FEV_1 because African Americans had a smaller trunk-to-leg ratio compared with whites. Whites and~~

~~Mexican Americans had similar FEV_1 and FVC values with respect to height. The ATS Task Force for Interpretation of Pulmonary Function recommended an adjustment on a population basis for predicted lung function in blacks by multiplying values for predicted normal FVC and FEV_1 by 0.88, and for normal single-breath DLco by 0.93.[33]~~

~~The NHANES III, reported in 1999, was the first report to compare a large number of lung function values for healthy whites, Mexican Americans, and African Americans. It measured FVC, FEV_1, forced expiratory volume in 6 seconds (FEV6), peak expira-tory flow rate (PEF), and forced expiratory flow rate at 25% to 75% of FVC (FEF_{25-75}). No adjustments for race were needed. Since that time, the reference values for spirometry have become the standardized reference values in US pulmonary function laboratories.[34]~~

~~The NHANES prediction equations for men and women of the different age groups and 3 races are readily available[34] if hand calculation is desired; however, we recognize that this set of prediction equations is often used as the "computerized" comparison values when lung function tests are performed. In addition, we recognize that few physicians will hand calculate the relationship between the values generated by the patient and compare the patient's values to predicted values. Data from NHANES prediction equations as tables could be entered in a spreadsheet, and the computer quickly and accurately generates the comparison of the patient's values to the reference value. Most of the standard spirometers currently in use have the associated software with the ability to compute the predicted values for various pulmonary functions. For the purpose of determining pulmonary impairment using Table 5-4, pulmonary impairment classification, such use of computer-assisted comparison values for lung function tests is useful and appropriate.~~

Diffusing Capacity for Carbon Monoxide

The single-breath DLco method is the most accepted method used in addressing carbon monoxide (CO) uptake in the lung. The DLco measurement provides information about gas transfer efficiency across the lungs. The CO uptake is determined by the alveolar-capillary interface. This is dependent on the structural and functional properties of the alveolar and vascular walls, available gas exchange surface area, gas solubility, pulmonary capillary blood volume, hematocrit, CO concentration gradient across the alveolar-capillary membrane, ~~and hemoglobin-binding site availability~~ventilation heterogeneity, as well as other parameters.[35] In addition, ventilation and

perfusion matching in the lung plays an important role in determining the ability of CO to transfer from the alveolar space into the vasculature.

DLco is considered repeatable if 2 test values are within 2 units. ~~Measurement of DLco is technically difficult, and it may take numerous efforts to gain reproducibility~~repeatability~~. As an example, the DLco is considered reproducible~~ repeatable if 2 test values are within 3 2 units, ~~whereas the FEV_1 and FVC tests must be within 5%~~0.15 L ~~to be considered reproducible~~repeatable~~.~~ Mechanical factors that affect DLco results include test gas inhalation speed, inspiration depth, period of breath holding, and expiration speed. Although mechanical factors generally are controlled by DLco test automation, extrapulmonary factors are important to ascertain proper interpretation. For example, cigarette smoking can elevate the blood~~'s~~ CO level~~s~~, causing as much as 10% to 12% hemoglobin saturation with CO and decreasing DLco. It is reasonable that the test subject not smoke for at least 8 hours before the test. Regardless, the time of the last cigarette smoked should be noted and adjustments in CO back pressure made for heavy or recent cigarette smoking. Anemia also decreases the DLco by reducing CO uptake. When the DLco is reduced, corrections should be made for anemia and carboxyhemoglobin.[~~35~~ 69]

~~Selecting reference values for DLco is more complex than selecting values for spirometry because inter-laboratory differences are much larger for DLco.[36,37] Commonly used DLco prediction equations include those of Crapo and Morris.[38] It is the recommendation of the authors that the Crapo reference values for DLco be used for everyone. Caucasians, American Indians, and Hispanic-Americans. For African-Americans and Asian-Americans, a correction factor of 0.93 should be applied to the DLCO predicted values from Crapo (until studies of DLCO from healthy samples of these ethnic groups become available). It is reasonable for laboratories to compare their measured values against the published reference values for population-based predicted normal diffusing capacity.~~The American Thoracic Society and European Respiratory Society currently recommend the Global Lung Function Initiative reference equations for DL_{CO}.[58]

Static Lung Volumes

Measurement of total lung capacity can be an important part of lung function assessment. A number of different approaches to measuring lung volumes have been put forward, including body plethysmography ~~(using various methods)~~, nitrogen washout, gas dilution, and radiographic imaging. More recently, measurement of lung volumes using imaging techniques such as CT or magnetic resonance imaging (MRI) have been added.[58] For individuals with airflow obstruction, plethysmography is currently the preferred method of lung volume measurement.[58] ~~There are inadequate data to recommend one approach over another.[30]~~ The readers are reminded that a restrictive ventilatory defect is characterized by the reduction in total lung capacity below the fifth percentile of the predicted value~~, and a "normal" FEV_1/FVC~~. The American Thoracic Society and European Respiratory Society currently recommend the Global Lung Function Initiative reference equations for static lung volumes. Although a restrictive defect can be suspected when the FVC is reduced and the FEV_1/FVC is normal or increased, this is not always the case.

Cardiopulmonary Exercise Testing

Diseases that affect the heart, lungs, circulation, or blood will cause an abnormal response to exercise. Exercise testing is useful to help evaluate the cause of shortness of breath that otherwise

cannot be determined at rest (heart vs lungs). It also may be diagnostic in recognizing myocardial ischemia, abnormal blood pressure response to exercise, poor circulation, loss of the vasculature of the lung (e.g., pulmonary embolism or other vasculature obliterative diseases), exercise-induced asthma, lack of fitness, and hyper-ventilation syndromes.

The cardiopulmonary exercise gas-exchange measurement, often referred to as metabolic studies, can be an additional means of assessing the severity and cause of exercise intolerance. Simultaneous measurement of oxygen consumption, carbon dioxide (CO_2) production, minute ventilation, and heart rate allows determination of whether exercise capacity limitation is due to cardiac, pulmonary, peripheral vascular, muscular, or coexisting impairments. More invasively, measuring arterial blood gases and lactate levels can aid in patient evaluation. When properly performed and interpreted, these tests can help differentiate pulmonary impairment from cardiac impairment or physical deconditioning effects.

Exercise capacity is measured by Vo_2 in milliliters per kilogram ~~multiplied by~~per minute~~s~~ (mL/[kg/min]) or in metabolic equivalents (METs), a unit of expended energy equal to 3.5 mL/(kg/min) oxygen consumption. METs are discussed in Chapter 4, in Section 4.2. Generally, an individual can sustain a work level equal to 40% of his or her measured maximum Vo_2 for an 8-hour period.[39]

Table 5-2 shows the relationship between work intensity and oxygen consumption.[40]

Table 5-2 Impairment Classification for Prolonged Physical Work Intensity by Oxygen Consumption[a]

Work Intensity for 70-kg Person[a]	Oxygen Consumption
Light (2 METs)	7 mL/kg/min; 0.5 L/min
Moderate (2-4 METs)	8–15 mL/kg/min; 0.6–1.0 L/min
Heavy (4-6 METs)	16–20 mL/kg/min; 1.1–1.5 L/min
Very heavy (6-8 METs)	21–30 mL/kg/min; 1.6–2.0 L/min
Arduous (>8 METs)	>30 mL/kg/min; >2.0 L/min

[a] Adapted from Astrand and Rodahl.[47] METs indicate~~s~~ metabolic equivalents (multiples of resting oxygen uptake). This example is for a 70-kg ~~person (presumably male)~~ male and should be interpreted cautiously, avoiding strict use of these cut-offs, as it is unclear if these ranges ~~apply generally~~ are generalizable to all people.

Exercise testing is infrequently needed in the investigation of pulmonary impairment. These studies can be difficult to perform, add considerable cost to the assessment, and may be more invasive than conventional tests. Typically, the clinical assessment of the individual, spirometry and diffusion capacity, and specialized cardiac tests, if necessary, usually provide sufficient

information to determine whether impairment is present and the apparent explanation for this. Exercise testing has traditionally been most useful in addressing the clinical situation where the individual's complaints are out of proportion to ~~his or her~~ their ~~static~~ lung function ~~test~~ measurements. ~~abnormalities.~~ In addition, exercise testing is used in situations where additional information is needed to clarify the nature and severity of impairment,[41, 42] where concurrent illnesses (heart disease)[43] or other factors (smoking) may limit exercise,[44] or when an understanding of the job-related energy requirements are needed to determine whether the worker can meet the energy demand of employment.[45]

Not surprisingly, an individual's cardiac and conditioning status must be considered in performing the test and in interpreting the results. Do not use exercise capacity measurements to study patients with medical contraindications such as unstable cardiac disease.

5.5 Methodology for Determining ~~the Grade in an~~ Impairment ~~Class~~ (Except for Asthma / Hyperresponsive Airway Diseases)

The largely uniform impairment rating methodology ~~grid~~ developed for this edition of the *Guides* is designed to create a standard platform across which organ systems can be rated in a consistent reproducible manner. Pulmonary impairment ratings in this chapter follow the International Classification of Functioning, Disability, and Health (ICF) scheme of 5 functional classes (0 to 4).

Each impairment class will have an assigned range of the whole person impairment percentage values based primarily on the severity of the pulmonary function ~~loss~~ impairment on various objective test results ~~(the key factor used in this chapter)~~ for each condition being rated. In each class there are ~~5~~3 different possible impairment grades, designated A, B, and C. ~~The median g~~Grade A is the ~~default~~ rating ~~for initial impairment determination and may be adjusted on either side of the median but only in the same impairment class, based on the non-key factors according to history and physical exam. The general steps for determining impairment class, and grade within class are outlined according to the example in Table 5-3~~which reflects the most common and expected clinical presentation for examinees within that Class. Grades B and C reflect worsening severities within the Class.~~.~~

~~**Table 5-3** Methodology for Determining the Grade in an Impairment Class~~

IMPAIRMENT CLASS	CLASS 0	CLASS 1	CLASS 2	CLASS 3	CLASS 4
SEVERITY GRADE		1 2 3 4 5	6 7 8 9 10	11 12 13 14 15	16 17 18 19 20
(%)		(A B **C** D E) ↑ Class 1 Default	(A B **C** D E) ↑ Class 2 Default	(A B **C** D E) ↑ Class 3 Default	(A B **C** D E) ↑ Class 4 Default

In order to consistently determine the appropriate impairment ~~grade~~ for a given class, the following procedure is recommended:

1. Determine the impairment class (IC) first, according to the "key factor" for that particular impairment grid, which is determined by objective test results as listed in Table 5-~~4~~3.
2. Compare the specific individual elements (SIEs) of history and physical examination within each Class in order to determine the appropriate Grade and subsequent impairment rating value according to the diagnostic rows below Table 5-3.

~~to the middle ("C") grade position for that ICCompare the specific individual elements (SIEs) of history and physical examination within each Class in order to determine the appropriate Grade and subsequent impairment rating value according to the classification rows below Table 5-4.~~

~~For the first remaining (non-key) factor, determine the most appropriate IC position and record the number difference to the key factor IC.~~

~~Repeat step 3 for each remaining (non-key) factor.~~

~~Summate the IC column differences and add or subtract the final number from the default identified in step 1 to determine the final impairment grade.~~

~~To illustrate, if the key factor identifies IC 3 (default to 3C), and non-key factors identify IC 1 and 4, this would produce differences of −2 and +1, respectively. These summate to −1. Subtracting 1 grade from IC/grade 3C gives a final IC/grade of 3B.~~

~~In this example, if the non-key factors both identified IC 1, you would summate the differences to −4. Since this procedure does not allow jumping from one IC to a lower (or higher) IC, you would subtract the maximum allowable 2 grades, for a final IC/ grade of 3A.~~

~~If the key factor indicated class 4C, and both nonkey factors were also IC 4, the differences would summate to zero, and IC/grade 4D or 4E would not be possible. In order to correct this deficiency, if the key factor identifies IC4C, automatically add +1 difference to the value of each non-key factor. For example, if the key factor identifies IC 4, and the first non-key factor was IC 3, the second was IC 4, the differences are −1 and zero. Adding +1 to each of these yields zero and 1, which summates to 1. Consequently, the final IC/grade is 4D.~~

~~Most pulmonary impairments can be rated according to Table 5-4, which is the Standard Impairment Classification Table.~~

The examiner should note that throughout this chapter the objective test results are used as the primary or "key" factor in the impairment rating for the condition, or range of conditions. Well-validated organ-specific functional test measures exist for the pulmonary system that correlate well with levels of impairment.[33] It is therefore appropriate to choose "objective test results" as the primary determinant of the impairment class rating in this chapter. The examiner assigns the grade by assessing the SIEs. If the SIEs are consistent with the expected presentation for the class, the rating remains at Grade A. If they suggest greater severity, the grade is adjusted to B or C accordingly. However, the impairment class itself must not change based on non-key factors, even if they differ significantly from the key factor.

~~1. Each impairment class in Table 5-4 has a corresponding range of available impairment ratings for each of grades A to EC; for example, cClass 1 corresponds to a rating that ranges from 2% to 10% of whole person impairment. The examiner should consider the range in each class as divisible into 53 subsections, each equidistant from the bottom and top of the range. For example, if the examiner determines that the key impairment factor places~~

~~an examinee into class 1, the choices for the impairment rating will be 2%, 4%, 6%, 8%, or 10%, for grades A to E, respectively.~~

~~Using the key impairment factor (objective test results), the examinee is assigned an impairment class, with severity grade in median as the default position. This is midway between the top and bottom of the range (C is midway between A and E). Continuing with our example, class 1C = 6%. After the key impairment factor has led to a preliminary impairment rating, it will be adjusted based on the results from rating the other factors.~~

~~2. The examiner will then assign grades (A to EC) for factors ***other*** than that considered "key." However, only the key factor can be used to assign the impairment class (IC). When non-key factors , called SIEs, such as history and physical exam are relevant to the rating, they are each assigned a relative class valueconsidered, which in turn is used to move the impairment rating up or down in the same class (class 1 in this example)determine the appropriate Grade. So in our example, if the examiner determines that the other factors affecting the rating are in the same relative consistent with the expected presentation of the examinee's class (class 1) that had been used as the baseline rating, the final rating will stay in the middle of that classbe Grade A. On the other hand, if the relative classes SIEs chosen by the non-key factors such as including the history and physical exam are higher more severe than what would typically be expected or lower than that used for the baseline impairment rating, the level will be moved proportionally to the right or to the left Wither Grade B or C to reflect the collective value of these non-key factors. Regardless of discrepancy of impairment classes between the key factor and non-key factors, the impairment rating should never move out of the class to which it was initially assigned, using only the key factor.~~

The classification of objective test results for FVC, FEV_1, FEV_1/FVC, and DLco is ~~given in Table 5-4~~also considered as an objective key factor. The DLco is primarily of value for persons with parenchymal lung disease. In evaluating the cause of abnormality in any of the listed measures, the physician should also consider the extrapulmonary factors contributing to pulmonary system impairment. For example, chest wall muscle weakness or obesity may decrease the FVC, and anemia may decrease the DLco. However, only the valid pulmonary dysfunction consistent and concordant with the validated pathology should be considered in evaluating impairment according to ~~Table 5-4~~this Section.

~~Table 5-4~~The below impairment rating rows present~~s~~ criteria for estimating the extent of permanent impairment. Spirometry and DLco must be performed on each individual being studied. The cardiopulmonary exercise study to measure $\dot{V}o_2$ max is not typically performed as it is not often necessary for identifying classes of impairment. If the individual is to be considered to have no impairment, all the listed criteria except for $\dot{V}o_2$ max must be met. For all other classes, at least one of the listed criteria must be fulfilled.

Impairments of other organ systems may be evaluated according to the criteria given in other *Guides'* chapters and then *combined* with the pulmonary system impairment using the Combined Values Chart (found in the Appendix).

DBI Table 5-3 Pulmonary Dysfunction Impairment

~~**Table 5-4**~~ Criteria for Rating Permanent Impairment due to Pulmonary Dysfunction~~a~~[a]

CLASS	CLASS 0	CLASS 1	CLASS 2	CLASS 3	CLASS 4
WHOLE PERSON IMPAIRMENT RATING (%)	**0%**	**2%-10%**	**11%-23%**	**24%-40%**	**45%-65%**
OBJECTIVE TESTS FVC FEV_1 FEV_1/FVC (%) DLco $\dot{V}o_2$ max	FVC ≥80% of predicted ***and*** FEV_1 ≥80% of predicted ***and*** FEV_1/FVC (%) >lower limits of normal and/ or (>75% of predicted) ***and*** DLco ≥75% of predicted ***or*** >25mL/(kg/-min) or >7.1 METs	FVC between 70% and 79% of predicted ***or*** FEV_1 between 65% and 79% of predicted ***or*** DLco between 65% and 74% of predicted ***or*** between 22 and 25 mL/(kg-/min) ***or*** 6.1–7.1 METs	FVC between 60% and 69% of predicted ***or*** FEV_1 between 64% and 55% of predicted ***or*** DLco between 55% and 64% of predicted ***or*** between 21 and 18 mL/(kg/-min) ***or*** 5.1–6.0 METs	FVC between 50% and 59% of predicted ***or*** FEV_1 between 45% and 54% of predicted ***or*** DLco between 45% and 54% of predicted ***or*** between 17 and 15 mL/(kg/-min) ***or*** 4.3–5.0 METs	FVC below 50% predicted ***or*** FEV_1 below 45% of predicted ***or*** DLco below 45% of predicted ***or*** <15mL/(kg /min) ***or*** <4.3 METs

[a] FVC indicates forced vital capacity; FEV_1, forced expiratory volume in the first second; DLco, diffusion capacity for carbon monoxide; Vo_2 max, maximum oxygen consumption; and METs, metabolic equivalents (multiples of resting oxygen uptake).

Class 1 Grade ~~Aa~~A (2% WPI)

- No dyspnea
- No physical examination findings
- PRN use of medication only

Class 1 Grade ~~Ab~~B (4% WPI)

- No dyspnea
- No physical examination findings

- Continuous daily ~~Daily~~ use of medication

Class 1 Grade ~~Ba~~C (6% WPI)

- Intermittent mild dyspnea as defined by having to walk more slowly on level ground than people of your age because of breathlessness
- No physical examination findings
- Continuous daily use of medication

Class 1 Grade ~~Bb~~Aa (8% WPI)

- Intermittent mild dyspnea as defined by having to walk more slowly on level ground than people of your age because of breathlessness
- One physical examination finding
    - Abnormal breath sounds (wheezes, rales, rhonchi, crackles)
    - Cyanosis
    - Clubbing
    - Witnessed cough
    - Accessory muscle use
- Continuous daily use of medication

Class 1 Grade ~~C~~Bb (10% WPI)

- Intermittent mild dyspnea as defined by having to walk more slowly on level ground than people of your age because of breathlessness
- Two or more physical examination findings
    - Abnormal breath sounds (wheezes, rales, rhonchi, crackles)
    - Cyanosis
    - Clubbing
    - Witnessed cough
    - Accessory muscle use
- Continuous daily use of medication

Class 2 Grade A (13%WPI)

- Constant mild dyspnea as defined by having to walk more slowly on level ground than people of your age because of breathlessness and/or intermittent moderate dyspnea defined by having to stop for breath when walking at your own pace on level ground
- No physical examination findings
- Continuous use of daily medications

Class 2 Grade B (18%WPI)

- Constant mild dyspnea as defined by having to walk more slowly on level ground than people of your age because of breathlessness and/or intermittent moderate dyspnea defined by having to stop for breath when walking at your own pace on level ground
- One physical examination finding
    - Abnormal breath sounds (wheezes, rales, rhonchi, crackles)

    - Cyanosis
    - Clubbing
    - Witnessed cough
    - Accessory muscle use
- Continuous use of daily medications

Class 2 Grade C (23% WPI)

- Constant mild dyspnea as defined by having to walk more slowly on level ground than people of your age because of breathlessness and/or intermittent moderate dyspnea defined by having to stop for breath when walking at your own pace on level ground
- Two or more physical examination findings
    - Abnormal breath sounds (wheezes, rales, rhonchi, crackles)
    - Cyanosis
    - Clubbing
    - Witnessed cough
    - Accessory muscle use
- Continuous use of daily medications

Class 3 Grade A (26% WPI)

- Constant moderate dyspnea as defined by having to stop for breath when walking at your own pace on level ground and/or intermittent severe dyspnea as defined by having to stop for breath after walking about 90 m (100 yd) or for a few minutes on level ground
- None or one physical examination finding
    - Abnormal breath sounds (wheezes, rales, rhonchi, crackles)
    - Cyanosis
    - Clubbing
    - Witnessed cough
    - Accessory muscle use
- Continuous use of daily medications

Classe 3 Grade B (33% WPI)

- Constant moderate dyspnea as defined by having to stop for breath when walking at your own pace on level ground and/or intermittent severe dyspnea as defined by having to stop for breath after walking about 90 m (100 yd) or for a few minutes on level ground
- Two physical examination findings
    - Abnormal breath sounds (wheezes, rales, rhonchi, crackles)
    - Cyanosis
    - Clubbing
    - Witnessed cough
    - Accessory muscle use
- Continuous use of daily medications

Class 3 Grade C (40% WPI)

- Constant moderate dyspnea as defined by having to stop for breath when walking at your own pace on level ground and/or intermittent severe dyspnea as defined by having to stop for breath after walking about 90 m (100 yd) or for a few minutes on level ground
- Two or more physical examination finding
    - Abnormal breath sounds (wheezes, rales, rhonchi, crackles)
    - Cyanosis
    - Clubbing
    - Witnessed cough
    - Accessory muscle use
- Continuous use of daily medications

Class 4 Grade A (45% WPI)

- Constant severe dyspnea as defined by having to stop for breath after walking about 90 m (100 yd) or for a few minutes on level ground and/or being too breathless to leave the house, or breathless on dressing or undressing.
- One physical examination finding
    - Abnormal breath sounds (wheezes, rales, rhonchi, crackles)
    - Cyanosis
    - Clubbing
    - Witnessed cough
    - Accessory muscle use
- Continuous use of daily medications

Class 4 Grade B (55% WPI)

- Constant severe dyspnea as defined by having to stop for breath after walking about 90 m (100 yd) or for a few minutes on level ground and/or being too breathless to leave the house, or breathless on dressing or undressing.
- Two physical examination findings
    - Abnormal breath sounds (wheezes, rales, rhonchi, crackles)
    - Cyanosis
    - Clubbing
    - Witnessed cough
    - Accessory muscle use
- Continuous use of daily medications

Class 4 Grade C (65% WPI)

- Constant severe dyspnea as defined by having to stop for breath after walking about 90 m (100 yd) or for a few minutes on level ground and/or being too breathless to leave the house, or breathless on dressing or undressing.
- Three or more physical examination findings
    - Abnormal breath sounds (wheezes, rales, rhonchi, crackles)
    - Cyanosis
    - Clubbing

- Witnessed cough
- Accessory muscle use

- Continuous use of daily medications

5.6 Asthma and Other Hyperreactive Airway Diseases

5.6a Diagnosis

The diagnosis of asthma requires both relevant clinical symptoms (current or historic~~),~~) and consistent features on the physical examination~~, and pulmonary function tests~~. ~~The latter should reveal~~Pulmonary function tests ~~either the~~ can also measure ~~presence of~~ airflow ~~limitation~~ obstruction that is partially or completely ~~reversible~~ responsive either spontaneously or with treatment, or the presence of airway hyperresponsiveness to methacholine or histamine ~~in the absence of airflow limitation~~. ~~Most~~ Many, but not all, ~~patients~~people with asthma have a significant postbronchodilator response on spirometry, ~~indicating airway hyperresponsiveness~~, yet, as a caution, the presence bronchodilator response, or a positive methacholine by itself, ~~of such a response by itself~~ is not diagnostic of asthma.[64] ~~46,47~~

5.6b Evaluation of Impairment and Disability

~~Although the diagnosis of asthma may be straightforward, a~~Assessment of impairment and disability from asthma is complex due to the condition's variable nature. ~~The ATS in 1993 provided the first guidelines to classify impairment due to asthma. This classification was categorical and without numerical estimates. The first numerical estimates for impairment from asthma were reported in 1997.[48] We have again used this approach in putting forward the~~Assessment of impairment and disability from asthma is complex due to the condition's variable nature. The following protocol for the evaluation of disability in asthma is recommended:

1. Confirm the presence of asthma.
2. Determine the severity of asthma.
3. Estimate the permanent whole person impairment.
4. Assess work-related asthma.

Confirm the Presence of Asthma

Asthma usually can be confirmed by relevant symptoms (shortness of breath, wheezing, chest tightness, coughing, and sputum production) and by the presence of ~~reversible~~ responsible airflow ~~limitation~~ obstruction or by the presence of airway hyperresponsiveness to methacholine ~~in the absence of airflow limitation~~.[49] The physician should follow ~~the~~ national guidelines for the diagnosis of asthma.[77-79] ~~11~~

Determine the Severity of the Asthma

Physiologic measures and clinical parameters can determine the asthma's severity. The physiologic measures in Table 5-5 include maximum postbronchodilator FEV_1. This should be measured after optimal therapeutic goals are achieved (i.e., minimum medication that obtains the best overall outcome). The lower the postbronchodilator FEV_1, despite optimal treatment, the

greater the severity of asthma. A second measure of asthma severity is the percentage of FEV_1 change or the extent of ~~reversibility~~ response of FEV_1. This percentage is defined by subtracting the baseline FEV_1 from the post-bronchodilator value, then dividing it by the baseline FEV_1, and multiplying it by 100.[64]

In the absence of ~~reversible~~ responsive airflow ~~limitation~~obstruction, the measurement of airway hyperresponsiveness should be measured by histamine/methacholine inhalation challenge testing by standard methods. To measure the degree of airway hyperresponsiveness, use the dose of methacholine (provocative concentration) that results in a 20% decline in FEV_1 compared with the baseline value upon provocation with less than or equal to 400 mcg ~~8 mg/mL~~ of methacholine using the tidal breathing method.[~~49~~ 65, 66]

Many clinical parameters have been used in the past to assess the severity of asthma in disability rating schemes. For example, the frequency of acute exacerbations requiring urgent intervention has been suggested as a measure of severity of impairment. However, given the current knowledge of the patho-physiology of asthma, many experts now believe that frequent exacerbations of asthma probably represent suboptimal treatment, although ongoing exposure to an occupational or environmental trigger, or severe uncontrollable asthma despite maximal/optimal treatment are also possibilities. Given this, many specialists believe that the number and types of medications required for both Maximum Medical Improvement (MMI) and the best outcome (balancing side effects) are better indicators of the severity of asthma than the frequency of attacks. If frequent exacerbations persist, one should determine whether the individual is receiving maximal medical therapy (including, when possible, removal from asthma-producing agents) before the impairment evaluation.

Estimate Permanent Whole Person Impairment

Table 5-5 lists the classes for whole person impairment based on pulmonary impairment, incorporating the parameters from the ATS "Guidelines"[~~46~~ 64] into those of the *Guides*. Prior to this current approach, physiologic measurements using standard techniques were required during the 12 weeks preceding medical evaluation. However, the examiner must determine that the ~~patient~~ examinee is receiving optimal therapy before considering the clinical parameters. The ~~patient~~examinee must be clinically stable from a pulmonary perspective. ~~The framework of Table 5-5 has the similar key elements as Table 5-4.~~

Note that in the absence of airflow limitation with asthma treatment, Table 5-5 may not be used to determine impairment for airway hyperresponsiveness (specific or nonspecific) alone. The individual with airway hyperresponsiveness may have no measurable impairment (solely determined on the basis of lung function test values) but may still have disability for specific jobs.

DBI Table 5-~~4~~5 Criteria for Rating Permanent Impairment due to Asthma[a]

CLASS	CLASS 0	CLASS 1	CLASS 2	CLASS 3	CLASS 4
WHOLE PERSON IMPAIRMENT RATING (%)	0	2%–10%	11%–23%	24%–40%	45%–65%

CLASS	CLASS 0	CLASS 1	CLASS 2	CLASS 3	CLASS 4
MAXIMUM POSTBRONCHODILATOR FEV_1 PERCENTAGE PREDICTED[b, c]	>80%	70%–80%	60%–69%	50%–59%	<50%
	or	**or**	**or**	**or**	**or**
OBJECTIVE TESTS FOR DEGREE OF AIRWAY HYPERRESPONSIVENESS PC_{20} mg/mL[b]	6–8	3–5	3–>0.5	0.5–0.25	0.24–0.125

[a] ~~a~~ Modified from Ranavaya, MI. The challenge of evaluating asthma impairment and disability. *AMA* Guides *Newsletter*. May-June 1997:1-4.[48]

[b] The "key" factor PC_{20} indicates and measures the degree of airway hyperresponsiveness. Alternatively the postbronchodilator FEV_1 percentage predicted is used as Key factor

[c] Percent predicted FEV_1 after albuterol therapy

Once the Class of impairment is determined by using the objective key factor of maximum post bronchodilator FEV1 response or histamine/methacholine challenge, the evaluator then determines the Grade and its associated impairment value using the Table below. For purposes of this section, low dose inhaled steroid is defined as less than 500 mcg of beclomethasone or its equivalent, and high dose inhaled steroid is defined as greater than 500 mcg of beclomethasone or its equivalent. Consistent with the previous evaluation methodology in this chapter, the Grade A level is the typically expected clinical presentation for each Class, with Grades B and C representing worsening clinical scenarios:

Class 0 (0% WPI)

Class 1 Grade A (4% WPI)

- Occasional bronchodilator use

Class 1 Grade B (7% WPI)

- Daily low dose inhaled steroid and occasional bronchodilator use

Class 1 Grade C (10% WPI)

- Daily high dose inhaled steroid and daily long-acting inhaled bronchodilator coupled with frequent rescue short acting bronchodilator use, may also include immunomodulators or scheduled daily systemic steroids or frequent short course systemic steroids

Class 2 Grade A (13% WPI)

- Daily low dose inhaled steroid and occasional bronchodilator use

Class 2 Grade B (18% WPI)

- Daily high dose inhaled steroid and daily long-acting inhaled bronchodilator coupled with frequent rescue short acting bronchodilator use, with occasional short course systemic steroids

Class 2 Grade C (23% WPI)

- Daily high dose inhaled steroid and daily long-acting inhaled bronchodilator coupled with frequent rescue short acting bronchodilator use, plus immunomodulators or scheduled daily systemic steroids or frequent short course systemic steroids

Class 3 Grade A (26% WPI)

- Daily high dose inhaled steroid and daily long-acting inhaled bronchodilator coupled with frequent rescue short acting bronchodilator use, with occasional short course systemic steroids

Class 3 Grade B (33% WPI)

- Daily high dose inhaled steroid and daily long-acting inhaled bronchodilator coupled with frequent rescue short acting bronchodilator use, plus immunomodulators or scheduled daily systemic steroids or frequent short course systemic steroids

Class 3 Grade C (40% WPI)

- Asthma not controlled by maximum treatment

Class 4 Grade A (45% WPI)

- Daily high dose inhaled steroid and daily long-acting inhaled bronchodilator coupled with frequent rescue short acting bronchodilator use, plus immunomodulators or scheduled daily systemic steroids or frequent short course systemic steroids

Class 4 Grade B (55% WPI)

- Asthma not controlled by treatment

Class 4 Grade C (65%WPI)

- Asthma not controlled by treatment
- Frequent hospitalizations and/or reliance upon others for most ADLs

Assess Work-Related Asthma

Although different categories of asthma can be described, they all share an underlying commonality of airway hyperresponsiveness. There are 3 recognized variants of asthma in the workplace: occupational, work-aggravated, and irritant-induced. Occupational asthma represents a special subset of asthma subjects. Occupational asthma is defined as a reversible airflow limitation caused by a specific agent in the workplace.[50,51] Occupational asthma has now

surpassed pneumoconiosis as the most commonly reported occupational lung disease linked to a particular occupational or environmental agent. In addition, besides directly causing occupational asthma de novo, work exposures can also acutely exacerbate a preexisting underlying asthmatic condition, which typically returns to baseline status with removal from exposure. Such events are recognized as work-aggravated asthma. Although potentially very dangerous, this exacerbation is temporary. Irritant-induced asthma, known as RADS (reactive airways dysfunction syndrome),[52] may result from a single massive high-level exposure to a highly irritating gas, mist, or vapor.

A variety of sensitizers (allergens) or irritants can cause occupational asthma. Sensitizers are classified as either high molecular weight or low molecular weight. High-molecular-weight sensitizers of animal or plant origin include animal dander or grain dust. Such agents are of similar molecular weight to the common antigens associated with exacerbations of asthma outside of the workplace. Low-molecular-weight sensitizers, typically organic or inorganic chemicals, include di-isocyanates. These agents are often peculiar to the workplace. Low-molecular weight sensitizers generally require a latency period for the development of immunologic responsiveness. This latency period may last from a few months to several years after first exposure.

There is substantial evidence to show that the best prognosis is attained through early diagnosis and prompt removal from further exposure as soon as possible after the diagnosis. Yet, not all workers leave the workplace after receiving a diagnosis of occupational asthma. In sensitized workers who remain in the workplace, asthma typically persists, with the potential for severe and even life-threatening exacerbations of asthma upon re-exposure. Workers who leave the workplace may improve, yet improvement is not always predictable. More than 50% of workers with occupational asthma fail to recover completely, even after 2 or more years since the last exposure and complete avoidance of the workplace. In those workers in whom asthma persists, a physician needs to monitor the worker's course of asthmatic symptoms.

For individuals with occupational asthma that occurs de novo in the workplace—those with work-aggravated asthma and in those with asthma after an acute inhalation injury (RADS) —the issues of employability in certain jobs and job accommodation are separate issues from an impairment rating. Follow-up studies of occupational asthma cases document that recovery is gradual, but most people with asthma related to the workplace have a plateau in their symptoms and lung function about 2 years after removal from exposure to putative agents. It is prudent that final recommendations for permanent impairment in occupational asthma cases be made at least 2 years after the initial diagnosis and removal from exposure.

~~Physicians struggle to manage individuals with occupational asthma who refuse to leave the workplace.~~ Continued exposures to sensitizing agents in the work environment or irritants at work or otherwise lead to a more permanent change (aggravation) in ~~an asthmatic~~ someone with asthma ~~individual~~. This likely increases the chance for an impairment to develop, which persists even after removal from exposure. If an individual's asthma is worsening, it is important to remove the individual from exposure at least temporarily or, at a minimum, to reduce exposure and reevaluate the worker's condition when it has stabilized. Although prevention is optimal, medication including inhaled steroids can modify symptoms and the clinical course of asthma.

5.7 Hypersensitivity Pneumonitis

Hypersensitivity pneumonitis, also known as extrinsic allergic alveolitis, is a granulomatous interstitial and bronchiolar lung disease caused by immune sensitization to organic dusts and some low-molecular-weight chemical antigens.[53] A wide variety of antigenic substances are known to cause this disease. The acute disease is characterized by the onset of respiratory and constitutional symptoms beginning 4 to 8 hours after exposure to the offending material. Symptoms include chest tightness, cough, dyspnea, fever, chills, malaise, and myalgias.

Pulmonary function tests in the acute phase of the disease show volume restriction and decreased diffusing capacity. Hypoxia may be demonstrated by pulse oximetry or arterial blood gas testing. Measurement of oxygen saturations in people with darker skin pigmentation is demonstrably biased.[59] Caution is required to interpret pulse oximetry. Chest radiographs may be normal but often show diffuse micronodular changes in the pulmonary parenchyma. When the person is removed from exposure, the symptoms, physiologic changes, and chest radio-graphic abnormalities begin to resolve in 1 to 2 days, although they may take 4 to 6 weeks for complete resolution. In the subacute and chronic presentations of hypersensitivity pneumonitis, the predominant symptoms include exertional dyspnea and cough; some patients report sputum production, anorexia, fatigue, and weight loss. Pulmonary function studies often show mixed restriction and obstruction, with isolated obstructive changes in some individuals.

With repeated exposures, pulmonary fibrotic changes may occur, and the parenchymal abnormalities become chronic and irreversible with respiratory impairment and limitations on other types of employment.[54] If pulmonary fibrosis has not yet occurred, normal pulmonary function may be reestablished. Once the acute episode has resolved and the condition is stable, the examiner may rate the degree of permanent impairment according to the criteria given in Table 5-3~~4~~.

Asthma, pneumoconiosis, and hypersensitivity pneumonitis may require that the person refrain from working in a specific occupational setting where he or she is exposed to the offending agent. If reassigned to a site where no ongoing exposure occurs, the individual may not have a permanent respiratory impairment.

5.8 Pneumoconiosis

Pneumoconiosis is a term used to describe diseases resulting from the inhalation of inorganic dusts such as silica, coal, asbestos, and metals such as cobalt and beryllium. The radiologic and pathologic patterns of pneumoconiosis from these dusts are usually quite distinct and beyond the scope of this chapter. Latency between exposure to these dusts and development of disease varies, but disease can occur anywhere from 10 to 30 years after initial exposure.

The severity of impairment related to pneumoconiosis depends on the characteristics of the specific dust inhaled, the dust burden retained in the lungs, the susceptibility of the individual, and the length of time since first exposure. Under some circumstances, the parenchymal changes on chest radio-graphs may be progressive, even after removal from exposure, and may or may not be associated with physiologic impairment. Individuals in whom pneumoconiosis develops 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. In those with silicosis, there is an increased risk of mycobacterial diseases. However, these individuals may be capable of

working at other jobs where the offending dust is not present. Impairment due to pneumoconiosis should be addressed with the use of Table 5-34.

5.9 Lung Cancer

AllMost persons with lung cancer are considered to be severely impaired at diagnosis. At reevaluation 1 year after the diagnosis is established, if the patient is found to be free of all evidence of tumor recurrence, that person is evaluated according to criteria listed in Table 5-43.

If there is still evidence of tumor, the patient is considered to be severely impaired (class 4 impairment); if the tumor recurs, the person will also be considered to be severely impaired (class 4 impairment). The Karnofsky's index of performance status (KPS) and the Eastern Cooperative Oncology Group Performance Status Scale (ECOG PS) are widely used methods of assessing the functional status of cancer patients. Table 5-56 (the ECOG PS Karnofsky scale with cross referenced values to the Karnofsky scale), has been specifically developed to describe the capabilities of individuals with cancer,[5555]67,68 and may be used to further describe the capabilities of a person with lung cancer and enable categorization in a particular class. The ECOG PS is favored for purposes of determining impairment, given that its 5 grade designations coincide with the 5 Class designations used to categorize impairments.

~~The scale below is not standard to my knowledge. Why not use the ECOG performance scale which is known and used by virtually everyone taking care of patients with cancer?~~

Table 5- —5 S6 Scale for Judging Capabilities of Subjects With Cancer[a]

ECOG Grade (PS)	Definition ~~should be KPS to be consistent~~
0	Fully active, able to carry on all pre-disease activities without restriction (KS 90-100)
1	Restricted in physically strenuous activity but ambulatory and able to carry out work of a light or sedentary nature (KS 70-80)
2	Ambulatory and capable of all self-care but unable to carry out any work activities. Out of bed > 50% (KS 50-60)
3	Capable of only limited self-care , confined to bed or chair > 50% waking hours (KS 30 - 40)
4	Completely disabled, cannot carry on any self-care, totally confined to bed or chair (KS 10-20)

~~Grade~~	~~Description~~
~~0~~	~~Fully active; able to carry on all predisease activities without restrictions~~

Grade	Description
1	Restricted in physically strenuous activity but ambulatory and able to carry out light tasks, such as light work in home or office
2	Requires occasional to considerable care for most needs and frequent medical care
3	Capable only of limited self-care and confined to bed or chair at least half of waking hours
4	Almost totally impaired; cannot care for self, and totally confined to bed or chair

* Adapted from Moossa et al.[55]

5.10 Sleep Disorders and Other Impairments Related to Pulmonary System

Certain pulmonary conditions may cause impairment that is not readily quantifiable by measuring spirometry, diffusing capacity, or exercise testing. Sleep disorders are examples of conditions of this type.

Although the pulmonary system may be structurally and functionally normal while the individual is awake, the breathing pattern during sleep is altered and periods of apnea occur throughout the night. In the normal individual, alterations in respiratory drive and ventilatory mechanics occur during the various stages of sleep. In normal non—rapid eye movement (NREM) sleep, there is an overall reduction in respiratory drive. In rapid eye movement (REM) sleep, the respiratory drive is irregular and the muscle tone of the rib cage and upper airways is decreased. In patients with disordered breathing during sleep, periods of apnea occur and can lead to episodes of hypoxia and hypercapnia. Arousal from sleep under these circumstances is effected by reticular and cortical activation in the brain. While arousal is a protective response, the result is fragmentation of normal sleep, with a morning headache, daytime sleepiness, intellectual impairment, and personality changes.

Two major subgroups of sleep apnea are recognized: obstructive sleep apnea (OSA) and central sleep apnea. Obstructive sleep apnea is characterized by occlusion of the upper pulmonary tract by sleep-induced relaxation of the oropharyngeal muscles. This allows the tongue and palate to rest against the posterior pharyngeal wall. Air ceases to flow through the nose and mouth despite continued pulmonary efforts by muscles of the chest wall and diaphragm. Arousal from sleep leads to improvement in muscle tone of the upper airway, which dilates the pharynx and allows passage of air into the lungs. The apneic period is usually terminated by loud snoring. Approximately 75% of patients with OSA are obese. The observation that weight loss decreases the severity of OSA supports the concept that obesity narrows the upper airway.[56]

The second major subgroup of these disorders is central sleep apnea. Episodic apneas during sleep are characterized by a total cessation of pulmonary effort rather than an obstruction to airflow. Although the genesis of the disorder is in the central nervous system, the clinical and physiologic effects of sleep disruption are similar to the effects seen in OSA. The manifestations of the abnormality are chronic alveolar hypoventilation with persistent arterial blood gas abnormalities, pulmonary hypertension, and cor pulmonale. It is possible to see the obstructive and central types of sleep apnea in the same individual.

Untreated sleep apnea is a cause of impairment. Daytime sleepiness, intellectual impairment, and personality changes can affect an individual's gainful employment. People affected by OSA are at significantly increased risk of being involved in motor vehicle collisions.[57] Severe daytime somnolence may prevent them from functioning adequately. Subtle changes in neuropsychological function include memory abnormalities and worsened motor coordination and mood that may affect the person's daily life.

A diagnosis of OSA is confirmed by nocturnal polysomnography in an accredited sleep laboratory. Grading OSA severity depends on the number of apnea and hypopnea episodes observed in polysomnography and the severity of hypoxia caused by these episodes. There are no standard, well-documented criteria for determining the level of impairment based on the results of polysomnography. Ideally, patients with documented sleep apnea should receive effective therapy, pursue weight loss, and then be reevaluated by polysomnography before they are judged to be impaired. The impairment rating should be based on clinical and physiologic parameters which are not readily measurable by the tables included here. The rating physician is encouraged to document pathology associated with OSA and affecting other organ systems (ie, corpulmonale; polycythemia) and rate these according to the appropriate organ system where applicable. Any add-on for strictly pulmonary impairment must be determined by an appropriately qualified, accredited, and experienced sleep specialist physician, and should not exceed 3% of the whole person impairment.

5.11 Examples of Impairment due to Pulmonary Disorders

Class 0

0% Impairment of the Whole Person

~~Example~~ Vignette 5-1:

Inadequate Cardiac Output

Subject: A 50-year-old male delivery truck driver.

History: Truck driver for the past 25 years; was referred because he had become too short of breath to carry 3 boxes up a flight of stairs. Three months earlier he had been hospitalized for treatment of an anteroseptal myocardial infarction. He was allowed to return to work after beginning a progressive exercise program. He had smoked cigarettes since age 18 years, averaging 1 pack per day (total exposure, 32 pack-years), but he stopped soon after the myocar~~-~~dial infarction.

Current Symptom: Dyspnea.

Physical Examination: Patient was 180 cm (6 ft) tall, weighed 99 kg (220 lb), and had a BMI of 30 kg/m^2. Chest and cardiac examinations were normal. Chest roentgenogram showed left ventricular enlargement and normal lung parenchyma. Technically acceptable p~~P~~ulmonary function studies, ~~which met ATS criteria~~are technically acceptable~~,~~ showed an FVC 85% of predicted, FEV_1/FVC ratio of 0.75~~%~~, FEV_1 80% of predicted, and DLco 75% of predicted.

A Bruce protocol stress test was performed. Maximum exercise could not be achieved due to fatigue and chest discomfort. Echocardiogram showed ejection fraction was 40%.

Diagnosis: Inadequate cardiac output resulting from myocardial infarction.

Impairment Rating: Class 0, no impairment (for pulmonary impairment).

Comment: Although the patient was a smoker, pulmonary function studies indicate he is still in class 0 and has no pulmonary impairment (see Table 5-4). However, he is limited by cardiac impairment. Further cardiac evaluation is recommended.

Class 1

2% to 10% Impairment of the Whole Person

Example Vignette 5-2:

Chronic Bronchitis

Subject: A 46-year-old female attorney.

History: Has had intermittent morning cough productive of sputum for several years. She uses over-the-counter medications that help the cough somewhat, and an albuterol inhaler typically twice per day. She has no very intermittent dyspnea, chest pain, or hemoptysis but notes wheezing, especially with colds. She finds difficulty in keeping up with her friends and coworkers while walking. She is a smoker and has smoked 1 pack per day since age 18 years. There is no history of asthma or pneumonia. She knows of no occupational or environmental exposures to respiratory hazards.

Current Symptoms: Intermittent morning cough productive of sputum.

Physical Examination: Patient's height was 168 cm (5 ft 7 in), and her weight was 58.5 kg (130 lb). There was an expiratory wheeze with forced exhalation. Otherwise, results of the physical examination were normal. Chest roentgenogram was normal. Pulmonary function studies, which were found to be valid based on a ATS criteriatechnically acceptable test, revealed FVC 85% of predicted, FEV_1 70% of predicted, and DLCO 75% of predicted.

Clinical Studies: Cardiopulmonary stress test was performed, and her Vo_2 at maximal exercise was 25 mL/kg/min.

Diagnosis: Chronic bronchitis with mild airflow obstruction.

Impairment Rating: Class 1, 6% whole person impairment (Table 5-4).

Pulmonary function studies (key factor in Table 5-4) indicated she was in cClass 1 (ie, FEV_1 70% of predicted). Maximal exercise Vo_2 of 25 mL/kg/min also places her in cClass 1. Note that one needs only 1 of the key objective findings to be qualified for a class. using Table 5-4. Exercise testing is not needed to make this determination of impairment. Her non-key criteria of impairmentspecific individual elements (SIEs) indicate that she has intermittent dyspnea, one abnormal physical exam finding, and uses daily medication, which would place her in Grade Bb(history and physical

~~findings) also place her in class 1~~. Therefore, in the final analysis she ~~remains in the middle of class 1, with~~ would be given an ~~6~~8% whole person impairment.

Class 2

11% to 23% Impairment of the Whole Person

~~Example~~ Vignette 5-3:

Occupational Asthma

Subject: A 28-year-old male auto body worker.

History: Had no history of asthma at the time of hiring. He had been spray painting for 10 years with polyurethane paints containing an asthma-causing diisocyanate. Over the first several years of employment, he noticed a gradual onset of chest tightness, with a nonproductive cough. This occurred mostly at work and gradually improved when away from work, on weekends and during vacations. Three years earlier he had been admitted to the hospital with severe dyspnea and wheezing; a diagnosis of asthma was made and asthma therapy was initiated. He requires daily high-dose inhaled corticosteroid therapy and ~~occasional use of an~~long acting inhaled β-agonist bronchodilator. On occasion, a rescue inhaler is necessary when exposed to irritants.

Current Symptoms: ~~None, a~~After 2 years of avoidance of spray painting and faithfully following his medication regimen, she does reasonably well except for wheezing and coughing when exposed to perfumes, tobacco smoke, or hairspray.

Physical Examination: Normal.

Clinical Studies: Spirometry reveals reversible obstructive ventilatory defect with maximal post-bronchodilator FEV_1 of 69% predicted. His FEV_1 improved 15% with bronchodilator compared with baseline.

Diagnosis: Occupational asthma due to occupational exposure to polyurethane paints.

Impairment Rating: The ~~W~~worker was evaluated for permanent impairment approximately 2 years after leaving the workplace. ~~Now i~~It is reasonable to consider his lung function and symptoms under the best control possible. Objective testing for the degree of airway hyperresponsiveness (key factor ~~using Table 5-5~~) shows that this worker's impairment is in ~~the middle of c~~Class 2 (the maximum postbronchodilator FEV_1 of 69% predicted places him in ~~c~~Class 2). ~~However, t~~The ~~clinical parameters~~ specific individual element (SIE) in this case is the medication use requirement, which indicates ~~document~~ that he requires daily high-dose inhaled corticosteroids and ~~occasional~~ daily use of a long acting inhaled-agonist bronchodilator with occasional rescue inhaler for his lung function to remain stable. These values would place him in ~~class 3 of Table 5-5~~Class B. Therefore, in the final analysis, he ~~remains in the class 2 but 1 grade higher than the default, for class 2D. This is a 20% whole person impairment.~~has an impairment rating of 18% whole person.

~~**Comment:** Further exposure to diisocyanate should be discouraged, as the evidence suggests that individuals with immune-mediated asthma do better when they are diagnosed early and removed from exposure. Recommend a reference for this. The reference I looked it which looked specifically~~

~~at diisocyanate asthma cases dd not mention this (J Occ Env Hygeine, 10:597-608), but did say that dermal and indirect exposure could cause asthma.~~

Class 3

24% to 40% Impairment of the Whole Person

~~Example~~ Vignette 5-4:

Occupational Pneumoconiosis (Asbestosis)

Subject: A 52-year-old man.

History: An examinee ~~C~~complains of increasing dyspnea of 5 years' duration that now affects his ADLs, making it difficult for him to participate in basic ADLs. He has difficulty keeping up with others of his age, and he usually has to stop and rest before climbing a flight of stairs. He says his coworkers have to help him with lifting and carrying at work; otherwise, he would lose his job. He denies cough, wheezing, or chest pain. He has worked as an insulator for 35 years; had mixed powdered asbestos with water and applied it to pipes and steel beams for the first 20 years of his working life. He reports to be a lifelong nonsmoker.

Current Symptom: Dyspnea.

Physical Examination: Examination disclosed that the patient was 170 cm (5 ft 8 in) tall and weighed 63 kg (140 lb). There was finger clubbing. He was noted to frequently cough during the history and exam. He had bilateral end-inspiratory, fine crackles~~, signs~~, and signs of severe interstitial lung disease. The results of the cardiac examination were normal. Chest ~~roentgeno-gram~~x-ray showed moderately pronounced, small, linear, irregular opacities at the lung bases. Small, bilateral pleural plaques were present.

Clinical Studies: Pulmonary function studies, which based on ~~ATS criteria~~a technically acceptable test were found to be valid, showed FVC 55% of predicted, FEV_1/FVC ratio 75%, FEV_1 60% of predicted and DLco to be 50% of predicted. Exercise testing showed the maximal Vo_2 to be 16 mL/kg/min.

Diagnosis: Occupational pneumoconiosis (asbestosis).

Impairment Rating: Class 3 Grade C, 40% whole person impairment ~~(Table 5-4)~~. This man's interstitial lung disease is moderately impairing his lung capacity. The crackles on chest auscultation, presence of finger clubbing, and witnessed cough are SIEs that satisfy the criteria for Grade C. Although typically a daily medication requirement is necessary to qualify for this Class and Grade, asbestosis does not usually respond to medications, and this requirement is disregarded in this scenario. The decreased vital capacity, and decreased gas exchange by diffusion capacity measurement are consistent with interstitial lung disease and restriction of lung volumes. The ~~P~~pulmonary function studies ~~(key factor using Table 5-4)~~ showing FVC to be 55% of predicted indicate he has ~~c~~Class 3 impairment.~~, which by default places him in the middle of the class.~~ Maximal exercise Vo_2 value of 16 mL/kg/min also places him in ~~the middle (median as default) of~~ ~~C~~class 3. (Again, note that only one of the objective findings is needed to be qualified for a class ~~under Table 5-4,~~ and exercise testing is usually unnecessary to determine this extent of

impairment.) The next step is to review the ~~non-key criteria (history and physical findings), both of~~ specific individual elements (SIEs) which place him in ~~impairment class 4~~Grade C~~. The relative difference is 2 intervals greater than the default, which places him in class 3E,~~ for a final impairment rating of 40% whole person impairment.

Class 4

45% to 65% Impairment of the Whole Person

~~Example~~ Vignette 5-5:

Severe Emphysema

Subject: A 57-year-old female law professor.

History: Shortness of breath gradually developed during a 10-year period. Dyspnea became so severe that she is unable to perform routine daily activities, such as driving to and from work, walking on level ground, taking a shower, or self-dressing. She has no wheezing, chest pain, or hemoptysis. She reports smoking regularly 2 1/2 packs a day for the past 40 years and was able to stop smoking 6 months previously. There is no history of asthma or pneumonia. She knows of no respiratory exposure to occupational or environmental hazards. She has been using a combination inhaler of a high dose corticosteroid, long-acting beta agonist, and ipratropium for several years, and has had an average of two to three emergency room visits per year for the last four years.

Current Symptoms: Dyspnea; occasional, nonproductive cough.

Physical Examination: She was found to be 163 cm (5 ft 5 in) tall and weigh 116 lb. She is barrel-chested, occasionally using accessory muscles, and breath sounds were barely audible. No crackles or wheezes were heard, but cyanosis is present. The ~~Ca~~cardiac examination was unremarkable.

Clinical Studies: Chest roentgenogram showed hyperinflated lungs, narrowed mediastinum, wide retrosternal space, emphysematous bullae, and lack of truncated vascular markings. Pulmonary function studies, which based on ~~ATS~~ valid and acceptable criteria, ~~were found to be valid,~~ showed FVC of 65% of predicted, FEV_1/FVC ratio of 40%, FEV_1 of 31% predicted, and DLco of 37% of predicted.

Diagnosis: Severe emphysema.

Impairment Rating: Class 4 Grade C, 65% whole person impairment ~~(Table 5-4). According to Table 5-4, t~~The key factor (objective test results) places her in ~~c~~Class 4; her specific individual elements (SIEs) indicate three abnormal examination findings, significant limitation on ability to walk, and daily medication use, qualifying for Grade C. ~~non-key factors (functional history and physical findings) also place her in class 4. The relative difference is 2 units greater than the "default" (add 1 to each non-key factor when key factor is in class 4), which places her in final impairment class/grade 4E.~~ This results in a final impairment rating of 65% whole person impairment.

5.12 Pulmonary Impairment Evaluation Summary

Table 5-~~7~~6 provides a summary of pulmonary impairment evaluation.

Table 5-~~6~~7 Pulmonary Impairment Evaluation Summary

Disorder	History, Including Selected Relevant Symptoms	Examination Record	Assessment of Pulmonary Function	End-Organ Damage	Diagnosis	Degree of Impairment
General	Respiratory symptoms (eg, cough); general symptoms Impact of symptoms on function and ability to do daily activities; prognosis if change anticipated Review medical history	Comprehensive physical examination; detailed respiratory system assessment	Data derived from relevant studies (eg, pulmonary function tests)	Include assessment of sequelae, including end-organ damage and impairment	Record all pertinent diagnosis(es); note if they are at maximal medical improvement; if not, discuss under what conditions and when stability is expected	Criteria outlined in this chapter, see Tables 5-1, 5-2, 5-3 ~~and 5-4~~
Obstructive Disorders	Dyspnea; cough; sputum production; infections; medication use; exercise tolerance	Note breath sounds, wheeze, loud P_2, jugular vein distention, right heart prominence	Pulmonary function: spirometry, lung volumes, diffusing capacity, methacholine challenge, radiographs	Assess relevant organs (eg, cardiac function, cor pulmonale)	Asthma; chronic bronchitis and emphysema; other obstructive diseases	See Table 5-4~~5~~ for asthma, see Table 5-3~~4~~ for other diseases
Restrictive Disorders	Dyspnea; cough; fatigue; sputum;	Chest wall excursion;	Pulmonary function: spirometry, lung volumes,	Assess cardiac function	Idiopathic pulmonary fibrosis; asbestosis;	

Disorder	History, Including Selected Relevant Symptoms	Examination Record	Assessment of Pulmonary Function	End-Organ Damage	Diagnosis	Degree of Impairment
	exercise tolerance	crackles; clubbing	diffusing capacity, imaging studies		pneumoconiosis; chest wall disorders; others	
Cancer	Exercise tolerance; dyspnea; chest pain; fatigue; weight loss; tobacco use; environmental exposures	Chest wall excursion; crackles; clubbing; adenopathy	Bronchoscopy; pulmonary function tests; biopsy	Assess other organ function; signs of metastases	Squamous, adeno, small cell, etc	See Table 5-3~~4~~ and Table 5-5~~6~~

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