GOLD 1/mild COPD	FEV_1 ≥80% predicted
GOLD 2/moderate COPD	FEV_1 ≥50% but less than 80% predicted
GOLD 3/severe COPD	FEV_1 ≥30% but less than 50% predicted
GOLD 4/very severe COPD	FEV_1 <30% predicted

An FEV_1 of about 1 L (usually 50% predicted) suggests severe obstruction and, in the case of COPD, predicts a mean survival rate of 50% at 5 years. For a better predictor of mortality than FEV_1 alone, the BODE index can be used: *b*ody mass index, degree of *o*bstruction as measured by FEV_1, modified Medical Research Council *d*yspnea score, and *e*xercise capacity as denoted by 6-minute walk distance.

Lung volumes should be measured along with pulmonary function testing because the limitation to expired airflow and decreased elastic recoil lead to lung hyperinflation, as evidenced by increased RV, FRC, and, ultimately, TLC.

Destruction of alveoli decreases the surface area for gas exchange in emphysema. This loss of surface area, coupled with bronchial obstruction and altered distribution of ventilated air, results in ventilation-perfusion inequality or mismatch, a cause of hypoxemia. Hyperinflation of the lungs increases zone 1 conditions, in which alveolar pressure exceeds pulmonary arterial pressure, and this process decreases perfusion and increases physiologic dead space. Hypercarbia can be avoided by increasing the minute ventilation, even with substantial ventilation-perfusion mismatching. However, eventually, the metabolic costs of breathing become excessive, and respiratory muscles fatigue. Over time, chemoreceptors reset, allowing the level of partial pressure of carbon dioxide in arterial blood ($Paco_2$) to rise, which increases the efficiency of ventilation by eliminating a higher concentration of carbon dioxide per breath, thereby lowering the metabolic cost of breathing. Significant individual variation is observed in the degree of mechanical impairment and in the magnitude of increase in $Paco_2$. Derangements in gas exchange can be detected by measuring arterial blood gases, by showing a decrease in D_{LCO}, or by evaluating hemoglobin oxygen desaturation during exertion. The degree of decrease in D_{LCO} correlates well with the radiologic extent of emphysema in COPD.

Chest radiography may fail to reveal abnormalities during the early stages of COPD, but in later stages, radiographic studies show hyperinflation, hyperlucency, flattening of the diaphragms, and bullous changes in lung parenchyma (E-Fig. 16-6). Pleural abnormalities, lymphadenopathy, and mediastinal widening are not characteristic of emphysema and should point to other diagnoses, such as lung cancer. Computed tomography is more sensitive than plain radiography because it allows for a more detailed evaluation of the lung parenchyma and surrounding structures. Computed tomography is useful in assessing the distribution of emphysema (E-Fig. 16-7) in patients for whom operative interventions such as lung volume reduction surgery are being contemplated (see later discussion). HRCT is highly sensitive for the detection of occult emphysema and can reveal the pattern of emphysematous changes. Electrocardiography might show evidence of right ventricular strain. Echocardiography can reveal evidence of right ventricular hypertrophy or dilation and can often provide an estimate of pulmonary arterial pressures in patients with advanced COPD. A high blood hemoglobin level might reveal erythrocytosis in the setting of chronic hypoxemia, whereas increased white blood cell counts might suggest infection. The arterial blood gas analysis may show hypoxemia, hypercarbia, or both, whereas acidemia due to acute hypercarbia may be present during an exacerbation.

Differential Diagnosis

The differential diagnosis of COPD includes the other major obstructive lung disorders: asthma, bronchiectasis, and the bronchiolar disorders. Asthma can occur at any age and sometimes overlaps with COPD, such as in patients with childhood asthma who smoke as adults. However, patients with COPD are typically older than 40 years of age and have a lengthy smoking history, whereas patients with asthma often have a history of atopy, have more variable symptoms that are often worse at night, and typically have marked improvements in lung function after bronchodilator administration. Patients with asthma may have normal pulmonary function during periods in which their asthma is well controlled, whereas those with COPD demonstrate ongoing airway obstruction even during periods of relative clinical stability.

It can be difficult to distinguish COPD with chronic bronchitis from bronchiectasis, and HRCT is necessary to assess for the abnormal bronchial dilation that is diagnostic of bronchiectasis.

Bronchiolar disorders can also be difficult to distinguish from COPD but should be considered in patients with risk factors, such as connective tissue disease or occupational exposures. Again, more sophisticated testing, such as HRCT with inspiratory and expiratory views to demonstrate peripheral areas of gas trapping and centrilobular nodules consistent with mucus impaction of the small airways, or even lung biopsy, may be needed to diagnose bronchiolitis.

Nonpulmonary causes of dyspnea on exertion, such as congestive heart failure or coronary artery disease, should also be considered in the differential diagnosis of COPD.

Treatment

Prevention

Because a cure for COPD does not exist, the best approach to this condition is prevention. Most cases of COPD in the United States are caused by cigarette smoking. Therefore, an appropriate major emphasis has been placed on the development of community education programs that focus on smoking prevention and promote smoking cessation. Legislative measures banning smoking in various public settings and levying increased taxes on cigarettes have been used to diminish the effects of environmental or second-hand exposure to tobacco smoke and to discourage smoking. Although smoking cessation interventions are effective in only a minority of patients, smoking cessation decreases mortality in patients with COPD who do succeed in quitting (level 1 evidence).

Most patients who are successful at smoking cessation have had at least one prior failed attempt, so physicians should encourage smoking cessation with at least brief interventions at every opportunity, even in patients who have tried but failed to quit in the past. Long-term physician and group support increases the success of cessation attempts, and pharmacologic smoking cessation aides, including nicotine replacement with gum or