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Obstructive Lung Diseases

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INTRODUCTION

The obstructive lung diseases are a group of pulmonary disorders that result in dyspnea characterized by an obstructive pattern of expiratory airflow limitation on spirometry. These disorders include chronic obstructive pulmonary disease (COPD), asthma, cystic fibrosis (CF), bronchiectasis, and the bronchiolar disorders. In some cases, these disorders overlap clinically (Fig. 16-1), sharing several features aside from the presence of expiratory airflow limitation. These features may include symptoms of wheezing and sputum production, chronic airway-centered inflammation, presence of airway structural changes resulting in remodeling of the airways, and episodic periods of temporarily worsened clinic status, known as exacerbations. However, the causes, locations, and patterns of airway inflammatory changes and remodeling, as well as the treatments, prognoses, and natural histories, are often significantly different, making clinical distinction among these disorders important.

COPD is characterized in general by abnormal airway inflammation and abnormal lung structure in response to an inhaled irritant (typically cigarette smoke); this results in irreversible or incompletely reversible airflow limitation and is typically progressive over time. *Asthma* is distinguished from COPD by characteristic smooth muscle hyperreactivity and reversible airflow limitation, by its variable clinical course, and by its frequent association with atopy. These disorders are epidemic in the general population worldwide and account for a significant proportion of the morbidity and mortality associated with the obstructive lung diseases. *Bronchiectasis* is a permanent abnormal dilation of the bronchi that results in chronic cough, purulent sputum production, and hemoptysis; it is caused by diverse conditions, including CF, a genetic disorder resulting from mutations in the *CFTR* gene. The *bronchiolar disorders*, also called *small airways disorders*, result from inflammation and/or fibrosis of the small airways of the lung that leads to dyspnea. They may be difficult to diagnose because loss or obstruction of a majority of the small airways must occur before the appearance of expiratory airflow limitation on spirometry.

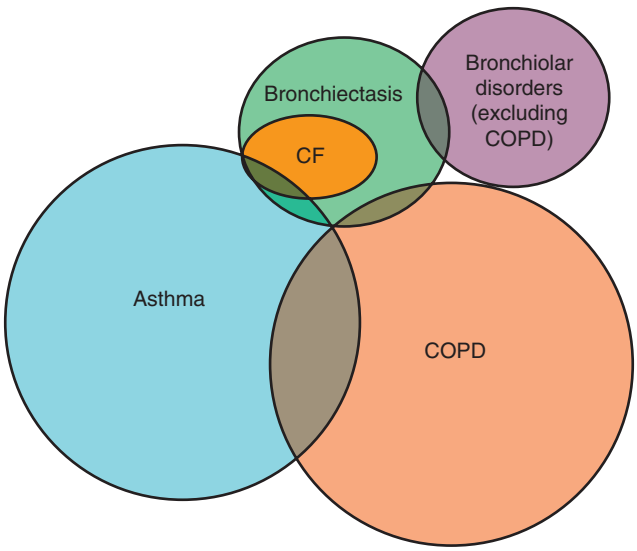

FIGURE 16-1 Classification of obstructive lung diseases. Although most patients with chronic obstructive pulmonary disease (COPD) have small airways disease, the bronchiolar disorders do not overlap with COPD. CF, Cystic fibrosis.

The basis for expiratory airflow obstruction varies among these disorders. The flow of air through the bronchial tree is directly proportional to the driving pressure and inversely proportional to the resistance. In obstructive lung disease, alterations in one or both of these processes may be present. Loss of lung elastic tissue, frequently present in COPD, results in decreased lung elastic recoil on expiration and therefore decreased driving pressure for expiratory airflow. By contrast, airflow limitation in asthma is primarily caused by smooth muscle contraction resulting in bronchoconstriction that increases airway resistance. Increases in airway resistance are also present in COPD and are related to small airway inflammation and fibrosis as well as small airway collapse due to decreased "tethering" of the airways in the setting of loss of surrounding lung elastic tissue. Mucus obstruction of airway lumens contributes to increased airway resistance in all the obstructive lung diseases.

Obstruction to airflow causes characteristic changes in lung volumes. The residual volume (RV) and functional residual capacity (FRC) are increased, whereas the total lung capacity (TLC) remains normal or is increased. Vital capacity, and particularly inspiratory capacity, is eventually reduced by the increase in RV. Several factors may contribute to the increase in FRC and RV in obstructive lung disease. Decreased lung elastic recoil in COPD increases the FRC because of reduced opposition to the outward force exerted by the chest wall. Loss of airway tone and decreased tethering by the surrounding lung in COPD, as well as bronchoconstriction and mucus plugging in acute asthma, allow airways to collapse at higher lung volumes and trap excessive air. Finally, under demands for increased minute ventilation (e.g., during exercise), the increased resistance to airflow may not allow the lungs to empty completely in the time available for expiration; this leads to so-called dynamic hyperinflation of the lungs as the volume of trapped air progressively increases while