

1662 the right side of the heart, patients may also present with symptoms of cor pulmonale, including abdominal bloating or distention and pedal edema (Chap. 279).

Additional History A thorough social history is an essential component of the evaluation of patients with respiratory disease. All patients should be asked about current or previous cigarette smoking, as this exposure is associated with many diseases of the respiratory system, most notably COPD and bronchogenic lung cancer but also a variety of diffuse parenchymal lung diseases (e.g., desquamative interstitial pneumonitis and pulmonary Langerhans cell histiocytosis). For most disorders, longer duration and greater intensity of exposure to cigarette smoke increases the risk of disease. There is growing evidence that “second-hand smoke” is also a risk factor for respiratory tract pathology; for this reason, patients should be asked about parents, spouses, or housemates who smoke. Possible inhalational exposures should be explored, including those at the work place (e.g., asbestos, wood smoke) and those associated with leisure (e.g., excrement from pet birds) (Chap. 311). Travel predisposes to certain infections of the respiratory tract, most notably the risk of tuberculosis. Potential exposure to fungi found in specific geographic regions or climates (e.g., *Histoplasma capsulatum*) should be explored.

Associated symptoms of fever and chills should raise the suspicion of infective etiologies, both pulmonary and systemic. A comprehensive review of systems may suggest rheumatologic or autoimmune disease presenting with respiratory tract manifestations. Questions should focus on joint pain or swelling, rashes, dry eyes, dry mouth, or constitutional symptoms. In addition, carcinomas from a variety of primary sources commonly metastasize to the lung and cause respiratory symptoms. Finally, therapy for other conditions, including both irradiation and medications, can result in diseases of the chest.

Physical Examination The clinician’s suspicion of respiratory disease often begins with a patient’s vital signs. The respiratory rate is often informative, whether elevated (tachypnea) or depressed (hypopnea). In addition, pulse oximetry should be measured, as many patients with respiratory disease have hypoxemia, either at rest or with exertion. The classic structure of the respiratory examination proceeds through inspection, percussion, palpation, and auscultation as described below. Often, however, auscultatory findings will lead the clinician to perform further percussion or palpation in order to clarify these findings.

The first step of the physical examination is inspection. Patients with respiratory disease may be in distress, often using accessory muscles of respiration to breathe. Severe kyphoscoliosis can result in restrictive pathophysiology. Inability to complete a sentence in conversation is generally a sign of severe impairment and should result in an expedited evaluation of the patient.

Percussion of the chest is used to establish diaphragm excursion and lung size. In the setting of decreased breath sounds, percussion is used to distinguish between pleural effusions (dull to percussion) and pneumothorax (hyper-resonant note).

The role of palpation is limited in the respiratory examination. Palpation can demonstrate subcutaneous air in the setting of barotrauma. It can also be used as an adjunctive assessment to determine whether an area of decreased breath sounds is due to consolidation (increased tactile fremitus) or a pleural effusion (decreased tactile fremitus).

The majority of the manifestations of respiratory disease present as abnormalities of auscultation. Wheezes are a manifestation of airway obstruction. While most commonly a sign of asthma, peribronchial edema in the setting of congestive heart failure can also result in diffuse wheezes, as can any other process that causes narrowing of small airways. For this reason, clinicians must take care not to attribute all wheezing to asthma.

Rhonchi are a manifestation of obstruction of medium-sized airways, most often with secretions. In the acute setting, this manifestation may be a sign of viral or bacterial bronchitis. Chronic rhonchi suggest bronchiectasis or COPD. Stridor, a high-pitched, focal inspiratory wheeze, usually heard over the neck, is a manifestation of upper airway obstruction and should prompt expedited evaluation of the patient,

as it can precede complete upper airway obstruction and respiratory failure.

Crackles, or rales, are commonly a sign of alveolar disease. A variety of processes that fill the alveoli with fluid may result in crackles. Pneumonia can cause focal crackles. Pulmonary edema is associated with crackles, generally more prominent at the bases. Interestingly, diseases that result in fibrosis of the interstitium (e.g., IPF) also result in crackles often sounding like Velcro being ripped apart. Although some clinicians make a distinction between “wet” and “dry” crackles, this distinction has not been shown to be a reliable way to differentiate among etiologies of respiratory disease.

One way to help distinguish between crackles associated with alveolar fluid and those associated with interstitial fibrosis is to assess for egophony. *Egophony* is the auscultation of the sound “AH” instead of “EEE” when a patient phonates “EEE.” This change in note is due to abnormal sound transmission through consolidated parenchyma and is present in pneumonia but not in IPF. Similarly, areas of alveolar filling have increased whispered *pectoriloquy* as well as transmission of larger-airway sounds (i.e., bronchial breath sounds in a lung zone where vesicular breath sounds are expected).

The lack or diminution of breath sounds can also help determine the etiology of respiratory disease. Patients with emphysema often have a quiet chest with diffusely decreased breath sounds. A pneumothorax or pleural effusion may present with an area of absent breath sounds.

Other Systems Pedal edema, if symmetric, may suggest cor pulmonale; if asymmetric, it may be due to deep venous thrombosis and associated pulmonary embolism. Jugular venous distention may also be a sign of volume overload associated with right heart failure. *Pulsus paradoxus* is an ominous sign in a patient with obstructive lung disease, as it is associated with significant negative intrathoracic (pleural) pressures required for ventilation and impending respiratory failure.

As stated earlier, rheumatologic disease may manifest primarily as lung disease. Owing to this association, particular attention should be paid to joint and skin examination. Clubbing can be found in many lung diseases, including cystic fibrosis, IPF, and lung cancer. Cyanosis is seen in hypoxemic respiratory disorders that result in >5 g of deoxygenated hemoglobin/dL.

DIAGNOSTIC EVALUATION

The sequence of studies is dictated by the clinician’s differential diagnosis, as determined by the history and physical examination. Acute respiratory symptoms are often evaluated with multiple tests performed at the same time in order to diagnose any life-threatening diseases rapidly (e.g., pulmonary embolism or multilobar pneumonia). In contrast, chronic dyspnea and cough can be evaluated in a more protracted, stepwise fashion.

Pulmonary Function Testing (See also Chap. 307) The initial pulmonary function test obtained is spirometry. This study is an effort-dependent test used to assess for obstructive pathophysiology as seen in asthma, COPD, and bronchiectasis. A diminished-forced expiratory volume in 1 sec (FEV_1)/forced vital capacity (FVC) (often defined as <70% of the predicted value) is diagnostic of obstruction. In addition to measuring FEV_1 and FVC, the clinician should examine the flow-volume loop (which is effort-independent). A plateau of the inspiratory and expiratory curves suggests large-airway obstruction in extrathoracic and intrathoracic locations, respectively.

Spirometry with symmetric decreases in FEV_1 and FVC warrants further testing, including measurement of lung volumes and the diffusion capacity of the lung for carbon monoxide (D_LCO). A total lung capacity <80% of the predicted value for a patient’s age, race, sex, and height defines restrictive pathophysiology. Restriction can result from parenchymal disease, neuromuscular weakness, or chest wall or pleural diseases. Restriction with impaired gas exchange, as indicated by a decreased D_LCO , suggests parenchymal lung disease. Additional testing, such as measurements of maximal expiratory pressure and maximal inspiratory pressure, can help diagnose neuromuscular weakness. Normal spirometry, normal lung volumes, and a low D_LCO should prompt further evaluation for pulmonary vascular disease.