by patients assist those who have difficulty describing their breathing sensations.

Sensory Intensity A modified Borg scale or visual analogue scale can be utilized to measure dyspnea at rest, immediately following exercise, or on recall of a reproducible physical task, such as climbing the stairs at home. An alternative approach is to gain a sense of the patient's disability by inquiring about what activities are possible. These methods indirectly assess dyspnea and may be affected by nonrespiratory factors, such as leg arthritis or weakness. The Baseline Dyspnea Index and the Chronic Respiratory Disease Questionnaire are commonly used tools for this purpose.

Affective Dimension For a sensation to be reported as a symptom, it must be perceived as unpleasant and interpreted as abnormal. Laboratory studies have demonstrated that air hunger evokes a stronger affective response than does increased effort or work of breathing. Some therapies for dyspnea, such as pulmonary rehabilitation, may reduce breathing discomfort, in part, by altering this dimension.

DIFFERENTIAL DIAGNOSIS

Dyspnea most often results from deviations from normal function in the cardiovascular and respiratory systems. These deviations produce breathlessness as a consequence of increased drive to breathe; increased effort or work of breathing; and/or stimulation of receptors in the heart, lungs, or vascular system. Most diseases of the respiratory system are associated with alterations in the mechanical properties of the lungs and/or chest wall, and some stimulate pulmonary receptors. In contrast, disorders of the cardiovascular system more commonly lead to dyspnea by causing gas-exchange abnormalities or stimulating pulmonary and/or vascular receptors (Table 47e-2).

Respiratory System Dyspnea • ***DISEASES OF THE AIRWAYS*** Asthma and COPD, the most common obstructive lung diseases, are characterized by expiratory airflow obstruction, which typically leads to dynamic hyperinflation of the lungs and chest wall. Patients with moderate to severe disease have both increased resistive and elastic loads (a term that relates to the stiffness of the system) on the ventilatory muscles and experience increased work of breathing. Patients with acute bronchoconstriction also report a sense of tightness, which can exist even when lung function is still within the normal range. These patients are commonly tachypneic; this condition leads to hyperinflation and reduced respiratory system compliance and also limits tidal volume. Both the chest tightness and the tachypnea are probably due to stimulation of pulmonary receptors. Both asthma and COPD may lead to hypoxemia and hypercapnia from ventilation-perfusion ($\dot{V}/Q$) mismatch (and diffusion limitation during exercise with emphysema); hypoxemia is much more common than hypercapnia as a consequence of the different ways in which oxygen and carbon dioxide bind to hemoglobin.

DISEASES OF THE CHEST WALL Conditions that stiffen the chest wall, such as kyphoscoliosis, or that weaken ventilatory muscles, such as myasthenia gravis or the Guillain-Barré syndrome, are also associated with an increased effort to breathe. Large pleural effusions may contribute to dyspnea, both by increasing the work of breathing and by stimulating pulmonary receptors if there is associated atelectasis.

DISEASES OF THE LUNG PARENCHYMA Interstitial lung diseases, which may arise from infections, occupational exposures, or autoimmune disorders, are associated with increased stiffness (decreased compliance) of the lungs and increased work of breathing. In addition, $\dot{V}/Q$ mismatch and the destruction and/or thickening of the alveolar-capillary interface may lead to hypoxemia and an increased drive to breathe. Stimulation of pulmonary receptors may further enhance the hyperventilation characteristic of mild to moderate interstitial disease.

Cardiovascular System Dyspnea • ***DISEASES OF THE LEFT HEART*** Diseases of the myocardium resulting from coronary artery disease and nonischemic cardiomyopathies cause a greater left-ventricular end-diastolic volume and an elevation of the left-ventricular end-diastolic as well as pulmonary capillary pressures. These elevated pressures lead to interstitial edema and stimulation of pulmonary receptors, thereby causing dyspnea; hypoxemia due to $\dot{V}/Q$ mismatch may also contribute to breathlessness. Diastolic dysfunction, characterized by a very stiff left ventricle, may lead to severe dyspnea with relatively mild degrees of physical activity, particularly if it is associated with mitral regurgitation.

DISEASES OF THE PULMONARY VASCULATURE Pulmonary thromboembolic disease and primary diseases of the pulmonary circulation (primary pulmonary hypertension, pulmonary vasculitis) cause dyspnea via increased pulmonary-artery pressure and stimulation of pulmonary receptors. Hyperventilation is common, and hypoxemia may be present. However, in most cases, use of supplemental oxygen has only a minimal impact on the severity of dyspnea and hyperventilation.

DISEASES OF THE PERICARDIUM Constrictive pericarditis and cardiac tamponade are both associated with increased intracardiac and pulmonary vascular pressures, which are the likely cause of dyspnea in these conditions. To the extent that cardiac output is limited (at rest or with exercise) metaboreceptors may be stimulated if cardiac output is compromised to the degree that lactic acidosis develops; chemoreceptors will also be activated.

Dyspnea with Normal Respiratory and Cardiovascular Systems Mild to moderate anemia is associated with breathing discomfort during exercise. This symptom is thought to be related to stimulation of metaboreceptors; oxygen saturation is normal in patients with anemia. The breathlessness associated with obesity is probably due to multiple mechanisms, including high cardiac output and impaired ventilatory pump function (decreased compliance of the chest wall). Cardiovascular deconditioning (poor fitness) is characterized by the early development of anaerobic metabolism and the stimulation of chemoreceptors and metaboreceptors. Dyspnea that is medically unexplained has been associated with increased sensitivity to the unpleasantness of acute hypercapnia.

TABLE 47e-2 MECHANISMS OF DYSPNEA IN COMMON DISEASES

Disease	↑ Work of Breathing	↑ Drive to Breathe	Hypoxemia[a]	Acute Hypercapnia[a]	Stimulation of Pulmonary Receptors	Stimulation of Vascular Receptors	Metaboreceptors
COPD	•		•	•			
Asthma	•	•	•	•	•		
ILD	•	•	•	•	•		
PVD		•	•			•	
CPE	•	•	•		•	•	•
NCPE	•	•	•		•		
Anemia							•
Deconditioning							•

[a]Hypoxemia and hypercapnia are not always present in these conditions. When hypoxemia is present, dyspnea usually persists, albeit at a reduced intensity, with correction of hypoxemia by the administration of supplemental oxygen.

Abbreviations: COPD, chronic obstructive pulmonary disease; CPE, cardiogenic pulmonary edema; ILD, interstitial lung disease; NCPE, noncardiogenic pulmonary edema; PVD, pulmonary vascular disease.