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## Chapter 11 Dyspnea, Orthopnea, and Paroxysmal Nocturnal Dyspnea

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### Definition

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*Dyspnea* refers to the sensation of difficult or uncomfortable breathing. It is a subjective experience perceived and reported by an affected patient. Dyspnea on exertion (DOE) may occur normally, but is considered indicative of disease when it occurs at a level of activity that is usually well tolerated. Dyspnea should be differentiated from tachypnea, hyperventilation, and hyperpnea, which refer to respiratory variations regardless of the patients' subjective sensations. *Tachypnea* is an increase in the respiratory rate above normal; *hyperventilation* is increased minute ventilation relative to metabolic need, and *hyperpnea* is a disproportionate rise in minute ventilation relative to an increase in metabolic level. These conditions may not always be associated with dyspnea.

*Orthopnea* is the sensation of breathlessness in the recumbent position, relieved by sitting or standing. *Paroxysmal nocturnal dyspnea* (PND) is a sensation of shortness of breath that awakens the patient, often after 1 or 2 hours of sleep, and is usually relieved in the upright position.

Two uncommon types of breathlessness are trepopnea and platypnea. *Trepopnea* is dyspnea that occurs in one lateral decubitus position as opposed to the other. *Platypnea* refers to breathlessness that occurs in the upright position and is relieved with recumbency.

### Technique

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A patient with dyspnea may say: "I feel short of breath," "I'm having difficulty breathing," "I can't catch my breath," "I feel like I'm suffocating." Because it is a subjective phenomenon, the perception of dyspnea and its interpretation vary from patient to patient. Begin with a nonleading question: Do you have any difficulty breathing? If the response is affirmative and dyspnea is established as a problem, it should be characterized in detail. When did it begin? Has the onset been sudden or insidious? Inquire about the frequency and duration of attacks. The conditions in which dyspnea occurs should be ascertained. Response to activity, emotional state, and change of body position should be noted. Ask about associated symptoms: chest pain, palpitations, wheezing, or coughing. Sometimes a nonproductive cough may be present as a "dyspnea equivalent." What other significant medical problems does the patient have, and what medications has he been taking? How much has he smoked?

Dyspnea on exertion is by no means always indicative of disease. Normal persons may feel dyspneic with strenuous exercise. The level of activity tolerated by any individual depends on such variables as age, sex, body weight, physical conditioning, attitude, and emotional motivation. Dyspnea on exertion would be abnormal if it occurred with activity that is normally well tolerated by the patient. It is helpful to ask if he has noticed any recent or progressive limitation in his ability to conduct specific tasks that he was able to perform without difficulty in the past (e.g., walking, climbing stairs, performing household chores). The degree of functional impairment can be assessed in this manner.

Additional questions should be aimed at ascertaining whether the patient has orthopnea or paroxysmal nocturnal dyspnea. Inquire about the number of pillows he uses under his head at night

and whether he has ever had to sleep sitting up. Does he develop coughing or wheezing in the recumbent position? Did he ever wake up at night with shortness of breath? How long after lying down did the episode occur, and what did he do to relieve his distress? Characteristically, the patient with left ventricular failure sits up at bedside, dangles his feet, and refrains from ambulation or other activity that is likely to worsen his symptoms.

## Basic Science

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Spontaneous respiration is controlled by neural and chemical mechanisms. At rest, an average 70 kg person breathes 12 to 15 times a minute with a tidal volume of about 600 ml. A normal individual is not aware of his or her respiratory effort until ventilation is doubled, and dyspnea is not experienced until ventilation is tripled. An abnormally increased muscular effort is now needed for the process of inspiration and expiration. Because dyspnea is a subjective experience, it does not always correlate with the degree of physiologic alteration. Some patients may complain of severe breathlessness with relatively minor physiologic change; others may deny breathlessness even with marked cardio-pulmonary deterioration.

There is no universal theory that explains the mechanism of dyspnea in all clinical situations. Campbell and Howell (1963) have formulated the "length-tension inappropriateness theory," which states that the basic defect in dyspnea is a mismatch between the pressure (tension) generated by respiratory muscles and the tidal volume (change of length) that results. Whenever such disparity occurs, the muscle spindles of the intercostal muscles transmit signals that bring the act of breathing to the conscious level. Additionally, juxtacapillary receptors (J-receptors), located in the alveolar interstitium and supplied by unmyelinated fibers of the vagus nerve, are stimulated by pulmonary congestion. This activates the Hering-Breuer reflex whereby inspiratory effort is terminated before full inspiration is achieved, resulting in rapid and shallow breathing. The J-receptors may be responsible for dyspnea in situations where pulmonary congestion occurs, such as with pulmonary edema. Other theories that have been proposed to explain dyspnea include acid-base imbalance, central nervous system mechanisms, decreased breathing reserve, increased work of breathing, increased transpulmonary pressure, fatigue of respiratory muscles, increased oxygen cost of breathing, dyssynergy of intercostal muscles and the diaphragm, and abnormal respiratory drive.

Orthopnea is caused by pulmonary congestion during recumbency. In the horizontal position there is redistribution of blood volume from the lower extremities and splanchnic beds to the lungs. In normal individuals this has little effect, but in patients in whom the additional volume cannot be pumped out by the left ventricle because of disease, there is a significant reduction in vital capacity and pulmonary compliance with resultant shortness of breath. Additionally, in patients with congestive heart failure the pulmonary circulation may already be overloaded, and there may be reabsorption of edema fluid from previously dependent parts of the body. Pulmonary congestion decreases when the patient assumes a more erect position, and this is accompanied by an improvement in symptoms.

Paroxysmal nocturnal dyspnea may be caused by mechanisms similar to those for orthopnea. The failing left ventricle is suddenly unable to match the output of a more normally functioning right ventricle; this results in pulmonary congestion. Additional mechanisms may be responsible in patients who experience paroxysmal nocturnal dyspnea only during sleep. Theories include decreased responsiveness of the respiratory center in the brain and decreased adrenergic activity in the myocardium during sleep.

Dyspnea on exertion is caused by failure of the left ventricular output to rise during exercise with resultant increase in pulmonary venous pressure. In cardiac asthma, bronchospasm is associated with pulmonary congestion and is probably precipitated by the action of edema fluid in the bronchial walls on local receptors. Trepopnea may occur with asymmetric lung disease when the patient lies with the more affected lung down because of gravitational redistribution of blood flow. It has also been reported with heart disease when it is probably caused by distortion of the great vessels in one lateral decubitus position versus the other. Platypnea was originally described in chronic obstructive pulmonary disease and was attributed to an increased wasted ventilation ratio in the upright position. Platypnea in association with orthodeoxia (arterial deoxygenation in the upright position) has been reported in several forms of cyanotic congenital heart disease. It has been proposed that this is precipitated by a slight decrease in systemic blood pressure in the upright position, resulting in increased right-to-left shunting.

### Clinical Significance

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Dyspnea may be induced in four distinct settings: (1) increased ventilatory demand such as with exertion, febrile illness, hypoxic state, severe anemia, or metabolic acidosis; (2) decreased ventilatory capacity such as with pleural effusion, pneumothorax, intrathoracic mass, rib injury, or muscle weakness; (3) increased airway resistance such as with asthma or chronic obstructive pulmonary disease; and (4) decreased pulmonary compliance such as with interstitial fibrosis or pulmonary edema.

In early left ventricular failure, the cardiac output does not increase sufficiently in response to moderate exercise; tissue and cerebral acidosis occurs, and the patient experiences dyspnea on exertion. The shortness of breath may be accompanied by fatigue or a sensation of smothering or sternal compression. In the later stages of left ventricular failure, the pulmonary circulation remains congested, and dyspnea occurs with mild exertion. Moreover, the patient may develop orthopnea or paroxysmal nocturnal dyspnea. Acute pulmonary edema is the most dramatic manifestation of pulmonary venous overload and may occur in the setting of a recent myocardial infarction or in the last stage of chronic left ventricular failure. Cardiovascular causes of dyspnea include valvular diseases (particularly mitral stenosis and aortic insufficiency), paroxysmal arrhythmia (such as atrial fibrillation), pericardial effusion with tamponade, systemic or pulmonary hypertension, cardiomyopathy, and myocarditis. Unrestricted fluid intake or administration in a patient with oliguric renal failure is also likely to precipitate pulmonary congestion and dyspnea.

Pulmonary disease constitutes another major category of conditions producing dyspnea and is discussed in [Chapter 36](#). Important pulmonary causes include bronchial asthma, chronic obstructive pulmonary disease, pulmonary embolism, pneumonia, pleural effusion, pneumothorax, allergic pneumonitis, and interstitial fibrosis. In addition, dyspnea may occur in febrile and hypoxic states and in association with some psychiatric conditions such as anxiety and panic disorder. Diabetic ketoacidosis seldom causes dyspnea but commonly induces slow, deep respirations termed *Kussmaul breathing*. Cerebral lesions or intracranial hemorrhage may be associated with intense hyperventilation and sometimes irregularly periodic breathing called *Biot's respiration*. Cerebral hypoperfusion from any cause may also result in alternating periods of hyperventilation and apnea called *Cheyne–Stokes respiration*, although no breathing difficulty may be perceived by the patient.

Diagnosis of the cause of dyspnea can be made relatively easily in the presence of other clinical signs of heart or lung disease. Difficulty is sometimes encountered in determining the precipitating cause of breathlessness in a patient with both cardiac and pulmonary conditions. An additional

diagnostic problem may be the presence of anxiety or other emotional disorder. A careful history and physical examination are always helpful, and occasionally cardiac catheterization, pulmonary function studies, or other tests may be necessary.

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