47e Dyspnea

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DYSPNEA

The American Thoracic Society defines *dyspnea* as a "subjective experience of breathing discomfort that consists of qualitatively distinct sensations that vary in intensity. The experience derives from interactions among multiple physiological, psychological, social, and environmental factors and may induce secondary physiological and behavioral responses." Dyspnea, a symptom, can be perceived only by the person experiencing it and must be distinguished from the signs of increased work of breathing.

MECHANISMS OF DYSPNEA

Respiratory sensations are the consequence of interactions between the *efferent*, or outgoing, motor output from the brain to the ventilatory muscles (feed-forward) and the *afferent*, or incoming, sensory input from receptors throughout the body (feedback) as well as the integrative processing of this information that we infer must be occurring in the brain (Fig. 47e-1). In contrast to painful sensations, which can often be attributed to the stimulation of a single nerve ending, dyspnea sensations are more commonly viewed as holistic, more akin to hunger or thirst. A given disease state may lead to dyspnea by one or more mechanisms, some of which may be operative under some circumstances (e.g., exercise) but not others (e.g., a change in position).

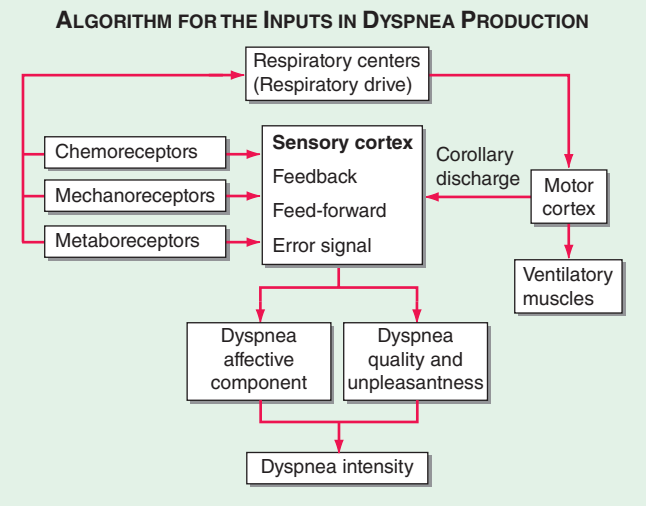

FIGURE 47e-1 Hypothetical model for integration of sensory inputs in the production of dyspnea. Afferent information from the receptors throughout the respiratory system projects directly to the sensory cortex to contribute to primary qualitative sensory experiences and to provide feedback on the action of the ventilatory pump. Afferents also project to the areas of the brain responsible for control of ventilation. The motor cortex, responding to input from the control centers, sends neural messages to the ventilatory muscles and a corollary discharge to the sensory cortex (feed-forward with respect to the instructions sent to the muscles). If the feed-forward and feedback messages do not match, an error signal is generated and the intensity of dyspnea increases. An increasing body of data supports the contribution of affective inputs to the ultimate perception of unpleasant respiratory sensations. *(Adapted from MA Gillette, RM Schwartzstein, in SH Ahmedzai, MF Muer [eds]. Supportive Care in Respiratory Disease. Oxford, UK, Oxford University Press, 2005.)*

Motor Efferents Disorders of the ventilatory pump—most commonly, increased airway resistance or stiffness (decreased compliance) of the respiratory system—are associated with increased work of breathing or the sense of an increased effort to breathe. When the muscles are weak or fatigued, greater effort is required, even though the mechanics of the system are normal. The increased neural output from the motor cortex is sensed via a *corollary discharge*, a neural signal that is sent to the sensory cortex at the same time that motor output is directed to the ventilatory muscles.

Sensory Afferents *Chemoreceptors* in the carotid bodies and medulla are activated by hypoxemia, acute hypercapnia, and acidemia. Stimulation of these receptors and of others that lead to an increase in ventilation produce a sensation of "air hunger." *Mechanoreceptors* in the lungs, when stimulated by bronchospasm, lead to a sensation of chest tightness. J-receptors, which are sensitive to interstitial edema, and pulmonary vascular receptors, which are activated by acute changes in pulmonary artery pressure, appear to contribute to air hunger. Hyperinflation is associated with the sensation of increased work of breathing, an inability to get a deep breath, or an unsatisfying breath. *Metaboreceptors*, which are located in skeletal muscle, are believed to be activated by changes in the local biochemical milieu of the tissue active during exercise and, when stimulated, contribute to breathing discomfort.

Integration: Efferent-Reafferent Mismatch A discrepancy or mismatch between the feed-forward message to the ventilatory muscles and the feedback from receptors that monitor the response of the ventilatory pump increases the intensity of dyspnea. This mismatch is particularly important when there is a mechanical derangement of the ventilatory pump, as in asthma or chronic obstructive pulmonary disease (COPD).

Contribution of Emotional or Affective Factors to Dyspnea Acute anxiety or fear may increase the severity of dyspnea either by altering the interpretation of sensory data or by leading to patterns of breathing that heighten physiologic abnormalities in the respiratory system. In patients with expiratory flow limitation, for example, the increased respiratory rate that accompanies acute anxiety leads to hyperinflation, increased work and effort of breathing, and the sense of an unsatisfying breath.

ASSESSING DYSPNEA

Quality of Sensation Like pain assessment, dyspnea assessment begins with a determination of the quality of the patient's discomfort (Table 47e-1). Dyspnea questionnaires or lists of phrases commonly used

TABLE 47e-1 ASSOCIATION OF QUALITATIVE DESCRIPTORS, CLINICAL CHARACTERISTICS, AND PATHOPHYSIOLOGIC MECHANISMS OF SHORTNESS OF BREATH

Descriptor	Clinical Examples	Pathophysiology
Chest tightness or constriction	Asthma, CHF	Bronchoconstriction, interstitial edema
Increased work or effort of breathing	COPD, asthma, neuromuscular disease, chest wall restriction	Airway obstruction, neuromuscular disease
"Air hunger," need to breathe, urge to breathe	CHF, PE, COPD, asthma, pulmonary fibrosis	Increased drive to breathe
Inability to get a deep breath, unsatisfying breath	Moderate to severe asthma and COPD, pulmonary fibrosis, chest wall disease	Hyperinflation and restricted tidal volume
Heavy breathing, rapid breathing, breathing more	Sedentary status in healthy individual or patient with cardiopulmonary disease	Deconditioning

Abbreviations: CHF, congestive heart failure; COPD, chronic obstructive pulmonary disease; PE, pulmonary embolism.