

CE Dyspnea

Applying Research to Bedside Practice

Nancy Spector, DNSc, RN

Maria A. Connolly, DNSc, CNE, APRN, FCCM

Karen K. Carlson, MN, RN, CCNS

ABSTRACT

Dyspnea is a common symptom in patients with acute and chronic critical illness as well as in patients receiving palliative care. While dyspnea can be found in a variety of clinical arenas and across many specialties, the mechanisms that cause dyspnea are similar. Although not often the cause for admission to critical care, it may complicate and extend length of stay. This article defines and describes dyspnea and its pathophysiology. Critical care nurses should

strive to implement interventions supported by evidence whenever possible. An evidence-based plan of care for the assessment, planning, intervention, and evaluation of the patient with dyspnea is outlined, using levels of recommendation based on the strength of available evidence. Two case studies are presented to illustrate its application to clinical practice.

Keywords: dyspnea, evidence-based practice, respiratory symptoms

In 1999, a multidisciplinary group of dyspnea experts¹ published a consensus statement on dyspnea, defining *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.”

This widely accepted definition describes the multidimensional and subjective aspects of dyspnea. The patient’s description of dyspnea is crucial, although obtaining that description can be difficult in critical care settings. Dyspnea can be influenced by psychological, physiological, social, environmental, or situational components^{2,3}; therefore, management strategies may address a wide variety of associated conditions, including anxiety or depression.^{1,4,5}

The American Thoracic Society’s consensus statement also differentiates between the sensation and the perception of dyspnea.¹ The

sensation of dyspnea arises from neural activation caused by stimulation of an afferent receptor, while the perception of dyspnea is the reaction to the sensation. Thus, the perception of dyspnea is controlled in the cortical and limbic areas of the brain, whereas the sensation of dyspnea is produced by various physiological mechanisms.

Treatment may address the sensation and/or perception of dyspnea. For example, opioids alter patients’ perception of dyspnea

Adapted with permission from Spector N, Connolly M. Dyspnea. In: Carlson KK, ed. *AACN Protocols for Practice: Symptom Management in Acute and Critical Care*. Aliso Viejo, Calif: American Association of Critical-Care Nurses; 2003.

Nancy Spector is Director of Education, National Council of State Boards of Nursing, 111 E Wacker, Suite 2900, Chicago, IL 60601 (e-mail: nspector@ncsbn.org).

Maria A. Connolly is Dean, College of Nursing and Allied Health, University of St Francis, Joliet, Ill. Karen K. Carlson is a Critical Care Clinical Specialist, Carlson Consulting Group, Bellevue, Wash.

by affecting the central interpretation of neural signals.¹ When opioids are inhaled, they may affect the sensation of dyspnea by direct action on peripheral receptors in the airways, to block cholinergic-induced bronchoconstriction and mucus secretion.^{6,7}

Clinically, the terms *breathlessness*, *dyspnea*, and *shortness of breath* are often used interchangeably and will be for this article; however, some authors have distinguished breathlessness from dyspnea. They assert that breathlessness is not always unpleasant, as with the breathlessness from excitement or exercise.^{6,8} Davis⁹ prefers the use of *breathlessness* when talking with patients because she thinks patients often better understand this term.

Dyspnea has been classified as chronic or acute^{10,11} and terminal.¹² According to Curley,¹⁰ acute dyspnea is short-term and requires medical intervention, while chronic dyspnea is long-term and more moderate in intensity. McCauley¹¹ differentiates chronic from acute dyspnea in that chronic dyspnea is persistent, with a variable intensity, whereas acute dyspnea is episodic, with high intensity. Others differentiate between acute and chronic dyspnea by the acuteness of its causes.⁶ Terminal dyspnea often occurs at the end of life¹²; in fact, in a study of 1500 cancer patients, Reuben and Mor¹³ found that 70% of patients in their study complained of shortness of breath in their last 6 weeks of life. Davis⁹ classifies dyspnea as breathlessness during exertion, breathlessness at rest, and terminal breathlessness. She further delineates breathlessness at rest as being intermittent or constant. The Davis classifications are clinically useful because patients can have acute dyspnea (high intensity) or chronic dyspnea that is intermittent or constant.

Incidence

Dyspnea is a common symptom in patients with acute and chronic critical illness and in patients receiving palliative care. Nelson et al¹⁴ studied 100 cancer patients in critical care and found that 33% were dyspneic. Others have found that more than 50% of cancer patients have dyspnea.¹⁵ Similarly, in general palliative care, dyspnea occurs in 29% to 90% of patients, depending on the diagnosis.¹⁶ Of the 14 million people in the United States with chronic obstructive pulmonary disease (COPD), 2 million have symptoms of chronic dyspnea that produce substantial disability.¹⁷

There is a knowledge gap in the understanding of dyspnea in patients receiving mechanical ventilation. Fewer than 10 studies published in English report measurement of dyspnea during mechanical ventilation.¹⁸ Studies that do address dyspnea conclude that dyspnea is common during mechanical ventilation. Powers and Bennett¹⁹ studied 28 patients receiving mechanical ventilation to evaluate 5 dyspnea-rating scales. Although this was a methodological study, an unanticipated finding was that overall patients experienced moderate to severe dyspnea. Knebel et al²⁰ and Bouley et al²¹ noted that patients experience significant dyspnea during weaning from the ventilator.

Etiology and Mechanisms

Dyspnea can be found in a variety of clinical arenas and across many specialties; however, mechanisms that cause dyspnea are similar. Table 1 displays some conditions that contribute to dyspnea. Figure 1 illustrates the multiple dimensions of dyspnea.

The 4 major mechanisms that cause dyspnea are stimulation of peripheral or central chemoreceptors, stimulation of intrapulmonary receptors, stimulation of chest wall or respiratory muscle mechanoreceptors, and increased motor command.⁶ The mechanisms are interrelated; more than 1 mechanism is often present in a clinical scenario.

Stimulation of Peripheral or Central Chemoreceptors

The effect of hypoxia on dyspnea is not clearly understood. Some research has concluded hypoxia stimulates ventilation through peripheral chemoreceptors, whereas other studies have found that hypoxia may directly stimulate dyspnea, independent of stimulating ventilation.²² Hypoxia stimulates ventilation through action on the carotid and aortic bodies, with the aortic bodies having a much larger role. This ventilatory stimulation is primarily manifested by an increased depth of breathing, which is often associated with dyspnea. Because the partial pressure of oxygen gas (PaO_2) is the specific signal sensed by the carotid bodies, conditions that lower oxygen content, but not PaO_2 , such as anemia or carbon monoxide, often do not elicit increased ventilation or dyspnea.²³

Although the central chemoreceptors have not been anatomically identified, evidence

Table 1: Conditions Contributing to Dyspnea^{1,27,48,98}

Cardiac	Pulmonary	Cardiopulmonary	Noncardiac and Nonpulmonary
Cardiomyopathy	Acute pulmonary emboli	Chronic pulmonary emboli	Amyotrophic lateral sclerosis
Central venous obstruction	Acute respiratory distress syndrome	Cor pulmonale	Anemia
Heart failure	Asbestosis	Deconditioning	Anxiety
Coronary arterial disease	Asthma	Trauma	Ascites
Dysrhythmias	Bronchiectasis		Cancer (radiation therapy)
Myocardial infarction	Chronic bronchitis		Chest wall deformity
Pericarditis	Congenital abnormalities of the lung or pulmonary arteries		Depression
Valve dysfunction	Cystic fibrosis		Fatigue
	Emphysema		Gastroesophageal reflux disease
	Fibrothorax		Hyperventilation syndrome
	Interstitial lung disease		Metabolic acidosis
	Lung cancer		Motor neuron disease
	Lymphangitis carcinomatosa		Multiple sclerosis
	Pleural effusion		Muscular dystrophy
	Pneumoconiosis		Myasthenia gravis
	<i>Pneumocystis carinii</i> infection		Obesity
	Pneumonia		Otorhinolaryngeal disorders
	Pneumothorax		Pain
	Primary pulmonary hypertension		Panic attacks
	Pulmonary edema		Phrenic nerve paralysis
	Pulmonary fibrosis		Pregnancy
	Silicosis		Psychoneurosis
	Thoracic surgery		Renal failure
	Tracheal obstruction		Systemic neurological disease
			Terminal illness
			Thyroid disease
			Vocal cord paralysis

indicates that they are located on the ventral surface of the medulla.²³ These receptors stimulate ventilation in response to acidosis and increasing partial pressure of carbon dioxide gas (PaCO₂), thereby causing dyspnea. Conversely, alkalosis and decreasing PaCO₂ inhibit ventila-

tion. As with hypoxia, some studies have concluded that hypercapnia may stimulate ventilation, independent of its effects on central chemoreceptors.²² If this finding is accurate, it would have implications for dyspnea associated with COPD.

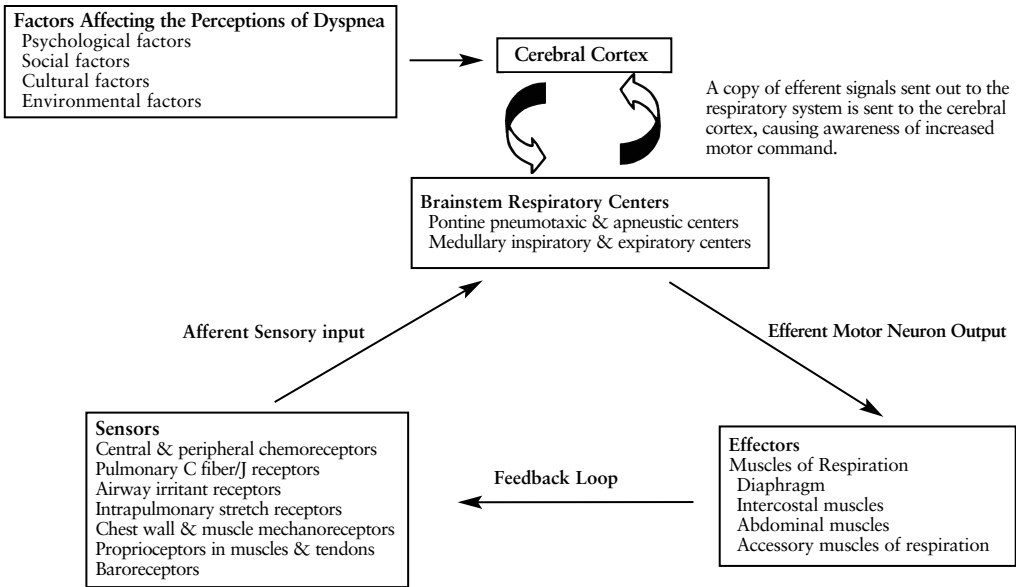


Figure 1: The multiple dimensions of dyspnea. Used with permission from Spector N, Klein D. Chronic critically ill dyspneic patients: mechanisms and clinical measurement. *AACN Clin Issues*. 2001;12:220–233.

Stimulation of Intrapulmonary Receptors

Irritant receptors, also thought to be cough receptors, lie between the epithelial cells in the airway and respond to stimuli such as smoke, dust, histamine, ammonia, and chlorine. These receptors do not appear to influence breathing under normal conditions. They are stimulated by decreased lung compliance and conditions that increase airflow resistance; therefore, their function may be to detect pathological changes in the lungs.²³ When stimulated, irritant receptors cause tachypnea, hyperventilation, and bronchoconstriction, creating a sensation of dyspnea. Stimulation of the irritant receptors causes the feeling of chest tightness, a symptom often associated with asthma.⁶

Pulmonary receptors are located in the airways and lung parenchyma, and are innervated by the vagus nerve. Stretch receptors, found in bronchial smooth muscle, are responsible for the Hering-Breuer inflation reflex, which prevents overinflation of the lungs; however, the Hering-Breuer reflex is activated only after 3 L of tidal volume is exceeded,²³ and is probably of little functional importance under normal conditions. Stimulation of these pulmonary and stretch receptors cause the sensation of dyspnea.

Lung parenchymal receptors are innervated by the C-fibers of the vagus nerve. These C-fibers (also termed J-receptors) are located in the alveolar interstitium, or the juxtacapillary region of the lung.²⁴ C-fibers respond to mechanical stimuli such as pulmonary emboli or congestion, and to chemicals such as serotonin and bradykinin.²² Stimulation of these fibers causes a pattern of rapid shallow breathing, increased secretions, and bronchoconstriction. Stimulation of C-fibers is implicated in dyspnea associated with heart failure or pulmonary edema.

Stimulation of upper-airway receptors, mediated by the trigeminal nerve distribution, may decrease the intensity of dyspnea.²² This may explain why a cool fan or open window is often effective in relieving mild dyspnea. Mechanoreceptors in muscles, joints, and tendons respond to changes in length, tension, and movement. Stimulation of these fibers reduces, rather than aggravates, dyspnea. One study on dyspneic patients with COPD showed decreased dyspnea at rest with in-phase vibration of the intercostal muscles.²⁴ This supports the premise that the sensation of dyspnea may be mediated by afferent information from chest-wall respiratory muscles to the brain and has implications for treatment.

Stimulation of Chest Wall or Respiratory Mechanoreceptors

The chest wall and respiratory musculature contain a variety of muscle and joint mechanoreceptors. These mechanoreceptors respond to movement, tension, and length, all affecting the perception of dyspnea. The dyspnea observed in neuromuscular diseases that result in impaired respiratory muscle function is the result of stimulation of these mechanoreceptors.

Increased Motor Command

An impaired sense of effort, also termed an increased motor command, has been identified as a possible cause of dyspnea.^{1,6,22} This is the patient's conscious awareness of an increased degree of motor neurosignaling to the respiratory muscles. Increased signaling creates work for the patient and causes dyspnea. The increased sense of effort is attributed to a corollary discharge, sent from the motor cortex to the sensory cortex when the outgoing command is sent to the muscles. The corollary discharge is sensed as respiratory effort or dyspnea.⁶ Specific pathways for corollary discharges have not been identified in humans, although research in animals has identified possible pathways.²⁵ Clinically, patients with morbidly obese chests and those with fatigued respiratory muscles or malnutrition might have dyspnea owing to an impaired sense of effort. Table 2 provides a summary of these mechanisms and their association with clinical conditions. Clearly, there is an interrelationship between the mechanisms.

Clinical Presentation

Although patients with dyspnea may have increased respiratory rates, dyspnea is distinct from tachypnea—an increased rate of breathing—because dyspnea is associated with discomfort. Likewise, dyspnea is distinct from hyperpnea, an increase in the depth of breathing.

Dyspnea is a subjective symptom described only by the patient. When assessing patients for dyspnea, it is important to consider all aspects of the symptom, including the psychological, social, physical, environmental, and situational interactions. For example, a patient with an elevated respiratory rate and decreased oxygen saturation may deny dyspnea. In this situation, the patient may have impaired gas exchange and an altered pattern of breathing, but should not be assumed to have dyspnea. In addition, it may be necessary to

assess the patient's anxiety level, as anxiety can affect the perception of dyspnea. As well, racial and cultural influences affect patients' descriptions of their dyspnea.²⁶ Given the complexity of dyspnea, it is easy to understand the difficulty involved in accurately assessing dyspnea in critically ill patients, a situation complicated further if communication is impaired.

A number of clues as to the cause of dyspnea⁶ can assist with assessment. Timing of dyspnea can be important. For example, paroxysmal nocturnal dyspnea and orthopnea—dyspnea when lying down—may occur with heart failure. Platypnea—dyspnea when sitting up—may be seen in patients with cirrhosis or pneumonectomy. Precipitating factors associated with dyspnea may include inhalation of irritants, which is associated with asthma. Changes in the color or consistency of sputum often indicate infection. Weight loss, seen with emphysema or acquired immunodeficiency syndrome (AIDS), is another sign associated with dyspnea. Alleviating factors such as position change or administration of nitroglycerin likewise provide clues about the cause of dyspnea.

Physical examination provides additional clues about the cause of dyspnea. On inspection, the clinician assesses the thorax for deformities such as increased anteroposterior diameter in the patient with COPD or kyphoscoliosis. Auscultation of the lungs may reveal adventitious sounds or abnormal breath sounds that could be associated with pneumonia. Cardiovascular examination may reveal signs of heart failure, such as a third heart sound or distended jugular veins. Psychiatric examination may reveal anxiety accompanied by hyperventilation²⁷ or depression.

It is important to assess the quality and intensity of dyspnea using the patient's perspective whenever possible. Intensity of dyspnea can be evaluated using scales as described below. Descriptors of dyspnea often vary, depending on the cause of dyspnea and the patient's age, gender, and culture. Duration and frequency of dyspnea should be assessed, as this influences the choice of intervention. Symptom distress is an important concept when evaluating dyspnea. The nurse asks patients how bothersome the dyspnea is to them. Often, with chronic dyspnea, the nurse may think that patients are in severe distress; yet, patients rate distress as low because they have become accustomed to dyspnea.

Table 2: Physiological Mechanisms and Associated Clinical Conditions Causing the Sensation of Dyspnea⁶

Pathophysiological Mechanisms Causing Dyspnea	Examples of Associated Conditions
<i>Stimulation of peripheral or central chemoreceptors: sensors detecting PaO₂, Paco₂, or pH</i>	Hypoxemia Hypercapnia Acidosis
<i>Stimulation of intrapulmonary receptors: airway irritant receptors, stretch receptors sensing lung volume, and C fiber/J receptors sensing edema and vascular congestion</i>	Pulmonary edema Pulmonary hypertension Pulmonary embolism COPD Pulmonary fibrosis Bronchoconstriction Lung cancer Pneumonia Acute respiratory distress syndrome Cystic fibrosis Asthma
<i>Stimulation of chest wall or respiratory muscle mechanoreceptors: sensors that respond to changes in muscle length, tension, or movement</i>	Neuromuscular diseases causing impaired respiratory muscle function Pleural effusion Fractured ribs Lung cancer Kyphoscoliosis
<i>Increased motor command: increased efferent motor neuron activity from the central nervous system to respiratory effector systems</i>	Exercise Weak respiratory muscles COPD Obesity

PaO₂ indicates partial pressure of arterial oxygen; Paco₂, partial pressure of carbon dioxide; and COPD, chronic obstructive pulmonary disease. Used with permission from Spector N, Klein D. Chronic critically ill dyspneic patients: mechanisms and clinical measurement. *AACN Clin Issues*. 2001;12:220–233.

Assessment of dyspnea is a challenge in critical care. For optimal assessment, patients must be able to communicate the many aspects of dyspnea. Many instruments are available to measure dyspnea,^{6,28,29} although most have not been validated for use in critical care and many are 1-dimensional. Numerous instruments have been used in patients with COPD but are not appropriate for use in acute or palliative care.

The 2 most common instruments used to measure dyspnea in critical care are the Visual Analog Scale (VAS)³⁰ and the modified Borg Scale³¹ (Figures 2 and 3). The VAS is a 100-mm horizontal line on which patients rate the degree of shortness of breath. The left and right endpoint anchors are 0 (no shortness of breath) and 100 (worst possible shortness of breath), respectively. The modified Borg Scale is a 12-item instrument that is anchored with

descriptors regarding the extent of dyspnea. Strong correlations have been found between the two and both are valid and reliable with critical care patients.¹⁹

Other instruments have also been used to assess dyspnea in critically ill patients. Powers and Bennett¹⁹ evaluated the Vertical Analog Dyspnea Scale (VADS), the 10-point numerical scale, and the Wong-Baker Faces Scale in a sample of 28 patients receiving mechanical ventilation and found them to have acceptable reliability and criterion validity. The VADS is a 100-mm line similar to the VAS, except that the line is vertical rather than horizontal. The numerical scale has numbers that anchor the intensity of dyspnea, with 0 representing no shortness of breath and 10 being the worst

possible shortness of breath. The Wong-Baker Faces Scale is a series of 6 pictures of faces, from happy to distressed. It was developed to assess pain in pediatric patients³²; however, it has also been used to assess pain and dyspnea in adults. Powers and Bennett¹⁹ found that 75% of critically ill patients in their study preferred the 10-point numerical scale. The use of all instruments described above requires that patients be alert and oriented. Furthermore, each instrument is 1-dimensional; only intensity or distress of dyspnea is measured.

Incorporating Evidence Into Practice

Dyspnea is a symptom seen in a wide variety of critically ill patients. The management of dyspnea should be based on the application of current scientific knowledge to clinical practice. In the following evidence-based plan of care, each recommendation is rated according to the level of evidence (ranging from I to VI) available to support the statement (Box 1).

Often, there is a clear, treatable etiology of the dyspnea. For example, dyspnea associated with asthma may require treatment with inhaled or systemic steroids. Patients with COPD may benefit from treatment with a beta 2 agonist or anticholinergic inhalers.

Borg Scale ³¹	
0	Nothing at all
0.5	Very, very slight
1	Very slight
2	Slight (light)
3	Moderate
4	Somewhat severe
5	Severe
6	
7	Very severe
8	
9	Very, very severe
10	Worst imaginable

Figure 2: Borg Scale to measure intensity of dyspnea.

Box 1: LEVELS OF EVIDENCE	
I:	Manufacturer’s recommendations only
II:	Theory based, no research data to support recommendations; recommendations from expert consensus group may exist
III:	Laboratory data only, no clinical data to support recommendations
IV:	Limited clinical studies to support recommendations
V:	Clinical studies in more than 1 or 2 different patient populations and situations to support recommendations
VI:	Clinical studies in a variety of patient populations and situations to support recommendations

Visual Analog Scale ³⁰
No shortness of breath _____
Shortness of breath as bad as can be _____

Figure 3: Visual Analog Scale to measure intensity of dyspnea.

Theophylline is sometimes used to achieve serum levels of 10 to 12 mcg/mL in these patients.⁵ Patients with heart failure are treated with diuretics and digoxin. Patients with malignant effusions often benefit from having the fluid drained and perhaps a pleurodesis or pleuroperitoneal shunt.

Although research is inconclusive regarding their efficacy, opioids remain the mainstay of treatment for dyspnea. A recent systematic review showed strong evidence for oral and parenteral opioids for palliative care treatment of dyspnea.^{3,5} Opioids are thought to decrease perception of dyspnea and to decrease ventilatory drive, thereby decreasing dyspnea. Administration of opioids should be started slowly to prevent respiratory depression. Considering the serious side effects of opioids, including respiratory depression, retention of carbon dioxide, hypoxemia, drowsiness, and nausea, they are not routinely recommended for patients with COPD. A trial might be considered, however, for patients with severe dyspnea.

Investigators are studying the efficacy of nebulized morphine for relief of dyspnea; however, present evidence does not support this route except in extraordinary circumstances.³³⁻³⁵ While nebulized morphine cannot be recommended as an evidence-based treatment now, it has been considered a promising treatment because it acts locally on opiate receptors in the airways.⁷ If the opioids are not absorbed systemically, they will not cause the unacceptable side effects that systemic opioids sometimes cause, including somnolence, constipation, and urinary retention.

Often in critical care nebulizers are used to deliver respiratory medication. However, when medicine is administered via a nebulizer, delivery to the small airways is inefficient because a large percentage of the drug is lost, either by expiration or deposition on the large airways. A mask is often better for the acutely ill or for very frail patients when using a nebulizer, because holding a mouthpiece can be tiring. Advantages of a mouthpiece are it helps avoid (1) deposition of the drug on the face, (2) feeling claustrophobic, and (3) acute angle glaucoma from anticholinergics.³⁶

Other pharmacologic interventions for dyspnea include anxiolytic and other psychoactive agents such as benzodiazepines. Studies have been contradictory with regards to efficacy and choice of agent; however, when dysp-

nea is associated with anxiety it is reasonable to try an anxiolytic agent.

Oxygen is recommended when dyspneic patients are hypoxic. Oxygen may reduce the stress and anxiety of dyspnea and provide comfort for patients at the end of life. Oxygen reduces chemoreceptor activity, thus depressing ventilation. Furthermore, the flow of oxygen across the nasal receptors may decrease dyspnea.³⁷ In patients with COPD, low-flow delivery is preferred to high-flow delivery, as it is better tolerated. Generally, oxygen masks are discouraged, as they increase the sense of breathlessness and are often poorly tolerated in critically ill patients. In patients receiving mechanical ventilation, changing ventilator settings (such as decreasing tidal volumes from 6 to 10 mL/kg of body weight) and using continuous positive airway pressure or pressure support may reduce dyspnea.

A variety of other nonpharmacologic interventions also may decrease dyspnea. A cool breeze across the face stimulates receptors in the trigeminal nerve and may decrease dyspnea. For patients with COPD, sitting forward and leaning their arms on a table may decrease dyspnea because unsupported arm movement alters the efficient use of respiratory muscles.³⁸ Pursed-lip and abdominal breathing are sometimes successful strategies for patients with COPD. However, reports are conflicting: One study reported increased dyspnea when using abdominal breathing.³⁹ Although patients with COPD may benefit from breathing strategies, their efficacy should be evaluated carefully. Likewise, exercise training and inspiratory-muscle training strategies may work for these patients, although they are not usually realistic for critical care patients. Lastly, complementary treatments such as acupuncture, acupressure, and relaxation strategies have relieved dyspnea in patients with COPD and those with end-stage malignancies.⁴⁰⁻⁴² This is an exciting new area of dyspnea management.

Evidence-based Plan of Care for the Patient With Dyspnea

Assessment

1. *Assess patients at high risk for dyspnea, including medical-surgical patients with acute*

or chronic conditions, psychiatric patients, obstetric patients, pediatric patients, patients with transplanted organs, patients receiving mechanical ventilation, and patients in palliative care (see Table 1 for specific conditions). (Level VI: Clinical studies in a variety of patient populations and situations to support recommendations)^{1,4,6,18,20,21,43–46}

Rationale: Dyspnea affects a wide range of patients.

Comments: Generally, dyspnea results from stimulation of pulmonary receptors, stimulation of chemoreceptors, and/or increased sense of effort (Table 2).

Dyspnea is reported to occur in 33% of critical care patients.¹⁴

Dyspnea is a significant public health problem. It is estimated that approximately one third of older adults (older than 70 years who live at home) experience dyspnea.

Hansen-Flaschen¹⁸ calls for a new era of “patient-centered mechanical ventilation,” where clinicians routinely measure dyspnea in patients receiving mechanical ventilation.

2. *Assess dyspnea from the patient’s perspective whenever possible. Use a dyspnea assessment instrument when appropriate.*¹⁹ Assessment should include the following: (Level V: Clinical studies in more than 1 or 2 different patient populations and situations to support recommendations)^{1,6,18,27–29,47–50}

- a. *Quality and timing of dyspnea*
- b. *Alleviating and precipitating factors*
- c. *Associated symptoms*
- d. *Physical assessment and pulmonary function measures, as indicated*
- e. *Pulmonary factors, for example, hypoxia or increased work of breathing*
- f. *Nonpulmonary factors, for example, pain, anxiety, depression, or fluid overload*

Rationale: Dyspnea is a subjective symptom that occurs with many conditions and is affected by physiological, psychological, social, and environmental factors. Because the mechanisms and causes of dyspnea are multifactorial, a good basic physical assessment is essential.

Comments: Patients often use different descriptors, depending on their condition or culture. Adequate multidimensional instruments do not exist for use with critically ill or terminally ill patients.^{6,47}

Treatment of the Underlying Disease Process

3. *Treat underlying disease processes as follows:* (Level V: Clinical studies in more than 1 or 2 different patient populations and situations to support recommendations)^{5,9,45,51–61}

- a. *COPD: inhaled anticholinergic agents and beta 2 agonists*
- b. *Asthma: inhaled steroids*
- c. *Malignant airway obstruction: stents*
- d. *Pleural effusion: pleurodesis or pleuroperitoneal shunt*
- e. *Heart failure: left ventricular unloading, diuretic agents, digoxin, beta blockers, angiotensin-converting enzyme inhibitors*

Rationale: Specific cause-focused treatment is always the first line of therapy.

Comments: Few studies show the effectiveness of bronchodilators in patients with advanced lung cancer, although they may be effective in patients with newly diagnosed lung cancer.^{9,53}

Pharmacologic Interventions

4. *Administer opioids orally, subcutaneously, or intravenously, at a dose of 2.5 to 7.5 mg every 4 hours, as needed. Titrate higher as needed to relieve dyspnea. Morphine sulfate (Roxanol SR or MS Contin), with morphine sulfate immediate release (MSIR) to treat breakthrough dyspnea, is commonly given orally.* (Level V: Clinical studies in more than 1 or 2 different patient populations and situations to support recommendations)^{1,4,5,7,9,45,62–69}

Rationale: Opioids are thought to decrease dyspnea by blunting the central perception of dyspnea and lowering the ventilatory drive.

Comments: Because of adverse drug reactions associated with opioids, routine opioid use is recommended only in the terminal phase of illness.¹ Slow-release preparations seem inferior to immediate-release preparations,^{9,68} and supplemental doses of immediate-release opioids are effective in patients receiving opioids every 4 hours. Further study of nebulized morphine is needed to determine the effectiveness of this route of administration.^{7,33–35,43,62,65,69,70}

5. *Administer anxiolytic and other psychoactive agents as follows:* (Level IV: Limited clinical studies to support recommendations)^{1,4,5,48,71–75}

- a. *Diazepam (Valium), 25 mg once a day orally*
- b. *Alprazolam (Xanax), 0.5 mg twice a day orally*
- c. *Buspirone (BuSpar), 10 mg twice a day orally*

Rationale: These agents relieve anxiety or agitation by various actions, depending on the drug class.

Comments: A limited number of studies have been published to guide the choice of pharmacologic agent for managing anxiety or agitation. Most studies have been conducted on patients with COPD. If patients have anxiety or panic attacks associated with dyspnea, a trial with an anxiolytic or psychoactive drug is reasonable.

Oxygen Administration

6. *Administer oxygen for the following indications: (Level V: Clinical studies in more than 1 or 2 different patient populations and situations to support recommendations)*^{1,4,37,45,76-82}

- a. *Hypoxia*
- b. *When improvement in functional status is required, especially exercise capacity and social functioning*
- c. *Discomfort at end of life (palliative care)*
- d. *For ventilated patients, altering mechanical ventilation settings may reduce dyspnea:*

- *Lower tidal volumes to 6 to 8 mL/kg*
- *Increase intermittent mandatory ventilation rates and/or pressure support*

Rationale: Oxygen may relieve dyspnea by depressing the hypoxic drive mediated by peripheral chemoreceptors; however, there is also evidence that oxygen reduces dyspnea by other means, such as by improving respiratory musculature function or by altering the perception of dyspnea. Furthermore, the flow of oxygen over the nasal mucosa may relieve dyspnea by stimulating nonspecific nasal receptors. Oxygen masks generally are poorly tolerated because they are uncomfortable, they must be removed to eat or drink, and heat radiates to the face around the nose and mouth.

Comments: Oxygen administration for relief of dyspnea has been primarily studied in patients with COPD, although it has also been studied in patients with advanced cancer, heart failure, and interstitial lung disease. Nonhy-

poxic patients with dyspnea may benefit from a trial of oxygen administration, which may prevent dyspnea associated with activity. Although transtracheal oxygen administration allows for reduced flow rates required to maintain oxygenation, the use of nasal prongs is more feasible with critically ill or palliative care patients.⁴⁵ High flow rates (4–6 L/min) may optimally correct hypoxia, but are impractical at home.

7. *Use fans to blow cool air across the face. (Level IV: Limited clinical studies to support recommendations)*^{1,21,45,83,84}

Rationale: Some feel this method decreases intensity of dyspnea owing to trigeminal nerve-mediated stimulation of upper-airway receptors.

Comments: Opening a window or having a fan blow across the patient's face can be used in early dyspnea relief.

Other Therapies

8. *Adjust the patient's position (to sitting, leaning forward, or resting the arms on a table in front of the patient) (Level IV: Limited clinical studies to support recommendations)*^{1,45,85-88}

Rationale: Positions that increase abdominal pressure may improve respiratory musculature function. Leaning forward may facilitate excursion of the diaphragm and supports accessory muscles so that they are more available to assist with respiration.

Comments: Studies have been done only on patients with COPD.

9. *Encourage diaphragmatic breathing with pursed lips, and slowed-pace breathing, which may temporarily relieve dyspnea. (Level IV: Limited clinical studies to support recommendations)*⁸⁹⁻⁹²

Rationale: Pursed-lip breathing slows expiration and raises intra-airway pressure, thereby preventing airway collapse. Diaphragmatic breathing may improve respiratory synchrony of the abdominal and thoracic muscles.

Comments: Studies have been done only on patients with COPD. Breathing strategies are most useful in times of respiratory distress. Rapid shallow breathing is often compensatory, so patients may quickly revert to previous

breathing patterns.¹ Controversy exists regarding the value of diaphragmatic breathing.³⁹ Improvement of dyspnea associated with diaphragmatic breathing may be due to distraction, altered pattern of breathing, or relaxation.⁹¹ Breathing strategies should be used only in those patients who show relief after a trial with these strategies.

10. Institute complementary treatment modes, such as acupuncture, acupressure, and relaxation techniques. (Level IV: Limited clinical studies to support recommendations)^{40–42,45,93–97}

Rationale: Acupuncture and acupressure use specific pressure points to relieve dyspnea. Relaxation decreases anxiety associated with dyspnea.

Comments: This is an emerging area of study. Other possible treatment modes include massage therapy, aromatherapy, guided imagery, biofeedback, and music therapy. Most studies have been done on patients with COPD or in palliative care.

Application to Practice

Dyspnea in the Patient Receiving Mechanical Ventilation

M.W., a 22-year-old African American woman, arrived at a free clinic with complaints of cough, fever, fatigue, and shortness of breath. She reported having “tough breaths.” One differential diagnosis was community-acquired pneumonia. M.W. was dyspneic with a pulse oximetry measurement (SpO₂) of 88% and, therefore, was sent by ambulance to the emergency department of a nearby hospital.

Chest radiography revealed consolidation in the right middle lobe of her lung, with a large right-side pleural effusion. M.W. never smoked but admitted to having “trouble getting air in” when she exercised. She was 1.63 m (5 ft 4 in) and weighed 49.43 kg (109 lb). Thoracentesis was performed and 300 mL of fluid was drained; a specimen was sent to the laboratory for culture. Immediately, M.W. indicated that she could breathe more easily. One hour later, she complained of increasing dyspnea and her SpO₂ decreased. Her vital signs were as follows: blood pressure, 96/60 mm Hg; heart rate, 142 beats per minute; and body temperature, 38.2°C. Auscultation revealed

crackles on the right side of the chest and expiratory wheezes.

M.W. was diagnosed with pneumonia requiring intubation and mechanical ventilation, and was admitted to critical care. Tidal volume, inspiratory flow, and level of ventilatory assistance were adjusted to alleviate her dyspnea. M.W. was anxious but alert, oriented, and able to communicate by finger movements. The critical care staff routinely assessed M.W. for dyspnea by asking 2 questions: “Are you experiencing tough breaths right now?” and, if yes, “Are your tough breaths mild, moderate, or severe?” M.W. used a 10-point numerical scale to rate the intensity of the “tough breaths.” If she rated her dyspnea a 2 or 3, morphine sulfate was administered to relieve the dyspnea.

After 2 days of aggressive intravenous antimicrobial therapy, M.W.’s condition, as revealed by chest radiography, began to improve, as did her dyspnea. She was weaned from mechanical ventilation according to protocol. Clinicians continued to monitor her dyspnea by asking the 2 questions and using the 10-point numerical scale. M.W. was moved to a medical-surgical unit and discharged home in less than 1 week. Her discharge medication was albuterol inhaler, 2 puffs twice a day. M.W. was instructed to return to the clinic for follow-up evaluation and work-up to determine if she had exercise-induced asthma.

Dyspnea in Palliative Care

J.C., a 78-year-old man, was at home under hospice care for COPD, cor pulmonale, atrial fibrillation, aortic aneurysm, and pernicious anemia. J.C. had a history of smoking 50 packs of cigarettes per year, before quitting at age 60. During his early years with COPD, he treated his mild dyspnea by using a fan to blow air across his face, using pursed-lip breathing, and sitting forward with his arms resting on a table in front of him. These strategies worked for several years, but became ineffective as his disease progressed.

While under hospice care, J.C. took the following medications daily: nitroglycerin, digoxin, furosemide (Lasix), haloperidol (Haldol), and ipratropium bromide (Atrovent) inhaler. He also used long-acting oral morphine (MS Contin) to treat dyspnea, and immediate-release morphine (MSIR Liquid) to treat breakthrough dyspneic episodes. He breathed supplemental oxygen continuously at 4 L/min via nasal cannula.

J.C. rated his dyspnea as a 3 or 4 (moderate or somewhat severe) on the Borg Scale³¹; however, owing to adverse reactions to oral opioids, the dosage could not be increased. A Foley catheter was in place to relieve urinary retention, and J.C. spent most of the day lying in the fetal position. Although he was confused, J.C. was able to recognize his family. He had unremitting constipation, such that the physician, as a last resort, ordered a colonoscopy. Because nebulized morphine has been used off-label for managing dyspnea, the hospice team decided to try it, stopping administration of all oral opioids. Administration of nebulized morphine was started at 5 mg every 4 hours, to be increased up to 40 mg every 4 hours. The dose was soon increased to 20 mg every 4 hours, and J.C. maintained a dyspnea rating of 1 or 2 (very slight or slight) on the Borg Scale. He was able to get out of bed, sit in a chair in the living room, watch television, and converse with his family. His Foley catheter was removed, and he began to have more normal bowel movements. His dyspnea was treated with nebulized morphine for 6 months until his death. He died comfortably at home with his family at his side.

[Authors' note: While controlled trials show inconsistent results with using nebulized morphine to alleviate dyspnea, in this situation it improved the quality of J.C.'s life in his disease's terminal stages. This treatment was used as a last resort by the palliative care team, after trying several other treatment modalities.]

Conclusions

Dyspnea is a common symptom seen in critically ill patients. There is evidence available to support best practice in the assessment, plan, implementation, and evaluation of care of the critically ill patient experiencing dyspnea. Utilization of these recommendations should enhance care of critically ill patients.

References

- American Thoracic Society. Dyspnea: mechanisms, assessment, and management: a consensus statement. *Am J Respir Crit Care Med*. 1999;159:321-340.
- Lenz ER, Suppe F, Gift AG, Pugh LC, Milligan RA. Collaborative development of middle-range nursing theories: toward a theory of unpleasant symptoms. *ANS Adv Nurs Sci*. 1995;17:1-13.
- Lenz ER, Pugh LC, Milligan RA, Gift A, Suppe F. The middle-range theory of unpleasant symptoms: an update. *ANS Adv Nurs Sci*. 1997;19:14-27.
- Ripamonti C. Management of dyspnea in advanced cancer patients. *Support Care Cancer*. 1999;7: 233-243.
- Runo JR, Ely EW. Treating dyspnea in a patient with advanced chronic obstructive pulmonary disease. *Best Pract*. 2001;175:197-201.
- Spector N, Klein D. Chronic critically ill dyspneic patients: mechanisms and clinical measurement. *AACN Clin Issues*. 2001;12:220-233.
- Zebraski SE, Kochenash SM, Raffa RB. Minireview. Lung opioid receptors: pharmacology and possible target for nebulized morphine for dyspnea. *Life Sci*. 2000;66:2221-2231.
- Carrieri VK, Janson-Bjerklie S, Jacobs S. The sensation of dyspnea: a review. *Heart Lung*. 1984;13:436-445.
- Davis C. Palliation of breathlessness. In: von Gunten CF, ed. *Palliative Care and Rehabilitation of Cancer Patients*. Boston, Mass: Kluwer Publishers; 1999.
- Curley FJ. Dyspnea. In: Irwin RS, Curley FJ, Grossman RF, eds. *Diagnosis and Treatment of Symptoms of the Respiratory Tract*. New York, NY: Futura Publishers; 1997:55-115.
- McCarley C. A model of chronic dyspnea. *Image*. 1999;31:231-236.
- Campbell ML. Terminal dyspnea: caring for a patient who refuses intubation or ventilation. *Dimens Crit Care Nurs*. 1996;15:4-12.
- Reuben DB, Mor V. Dyspnea in terminally ill cancer patients. *Chest*. 1986;89:234-236.
- Nelson JE, Meier DE, Oei EJ, et al. Self-reported symptom experience of critically ill cancer patients receiving intensive care. *Crit Care Med*. 2001;29: 277-282.
- Lynch MP. Dyspnea. *Clin J Oncol Nurs*. 2006;10:323-326.
- Rousseau P. Nonpain symptom management in terminal care. *Clin Geriatr Med*. 1996;12:313-327.
- Celli BR. The importance of spirometry in COPD and asthma: effect on approach to management. *Chest*. 2000;117:15S-19S.
- Hansen-Flaschen JH. Dyspnea in the ventilated patient: a call for patient-centered mechanical ventilation. *Respir Care*. 2000;45:1460-1467.
- Powers J, Bennett SJ. Measurement of dyspnea in patients treated with mechanical ventilation. *Am J Crit Care*. 1999;8:254-261.
- Knebel AR, Janson-Bjerklie SL, Malley JD, Wilson AG, Marini JJ. Comparison of breathing comfort during weaning with two ventilatory modes. *Am J Respir Crit Care Med*. 1994;149:14-18.
- Bouley GH, Froman R, Shah H. The experience of dyspnea during weaning. *Heart Lung*. 1992;21:471-476.
- Manning HL, Schwartzstein RM. Mechanisms of dyspnea. In: Mahler DA, ed. *Dyspnea*. New York, NY: Marcel Dekker Inc; 1998.
- Caruana-Montaldo B, Gleeson K, Zwillich CW. The control of breathing in clinical practice. *Chest*. 2000;117: 205-225.
- Sibuya M, Yamada M, Kanamaru A, et al. Effect of chest wall vibration on dyspnea in patients with chronic respiratory disease. *Am J Respir Crit Care Med*. 1994;149: 1235-1240.
- Chen ZF, Eldridge L, Wagner PG. Respiratory-associated thalamic activity is related to level of respiratory drive. *Respir Physiol*. 1992;90:99-113.
- Schwartzstein RM. Language of dyspnea. In: Mahler DA, O'Donnell DE, eds. *Dyspnea*. Boca Raton, Fla: Taylor & Francis; 2005.
- Morgan WC, Hodge HL. Diagnostic evaluation of dyspnea. *Am Fam Physician* [serial online]. February 15, 1998. Available at: <http://www.aafp.org/afp/980215ap/morgan.html>. Accessed December 27, 2006.
- Mahler DA. Measurement of dyspnea: clinical ratings. In: Mahler DA, O'Donnell DE, eds. *Dyspnea*. Boca Raton, Fla: Taylor & Francis; 2005.
- Van der Molen B. Dyspnea: a study of measurement instruments for the assessment of dyspnea and their application for patients with advanced cancer. *J Adv Nurs*. 1995;22:948-956.
- Aitken RCB. Measurement of feelings using visual analog scales. *Proc Royal Soc Med*. 1969;62: 989-993.

31. Borg G. Perceived exertion as an indicator of somatic stress. *Scand J Rehab Med*. 1970;2:92–98.
32. Wong DL, Baker CM. *Reference Manual for the Wong-Baker Faces Pain Rating Scale*. Duarte, Calif: Mayday Pain Resource Center; 1991.
33. Ferraresi V. Inhaled opioids for the treatment of dyspnea. *Am J Health-Syst Pharm*. 2005;62:319–320.
34. Jennings AL, Davies AN, Higgins JP, et al. Opioids for the palliation of breathlessness in terminal illness. *Cochrane Database Syst Rev*. 2001;4:CD002066.
35. Polosa R, Simidchiev A, Walters EH. Nebulised morphine for severe interstitial disease. *Cochrane Database Syst Rev*. 2002;3:CD002872.
36. Rushby I, Scullion J. Managing dyspnea in end-stage chronic obstructive pulmonary disease (COPD). *Prim Health Care*. 2004;14:43–49.
37. Booth S, Kelly MJ, Cox NP, Adams L, Guz A. Does oxygen help dyspnea in patients with cancer? *Am J Respir Crit Care Med*. 1996;153:1515–1518.
38. Cherniack N. *Chronic Obstructive Pulmonary Disease*. Philadelphia, Pa: WB Saunders; 1991.
39. Gosselink RA, Wagenaar RC, Rijswijk H, Sargeant AJ, Decramer ML. Diaphragmatic breathing reduces efficiency of breathing in patients with chronic obstructive pulmonary disease. *Am J Respir Crit Care Med*. 1995;151:1136–1142.
40. Gift A, Moore T, Soeken K. Relaxation to reduce dyspnea and anxiety in COPD patients. *Nurs Res*. 1992;41:242–246.
41. Renfro KL. Effect of progressive relaxation on dyspnea and state anxiety in patients with chronic obstructive pulmonary disease. *Heart Lung*. 1988;17:408–413.
42. Pan CX, Morrison R, Ness J, Fugh-Berman A, Leipzig RM. Complementary and alternative medicine in the management of pain, dyspnea, and nausea and vomiting near the end of life: a systematic review. *J Pain Symptom Manage*. 2000;20:374–387.
43. Allard P, Lamontagne C, Bernard P, Tremblay C. How effective are supplementary doses of opioids for dyspnea in terminally ill cancer patients? A randomized continuous sequential clinical trial. *J Pain Symptom Manage*. 1999;17:256–265.
44. Ho SF, O'Mahoney MS, Steward JA, Brey P, Buchalter M, Burr ML. Dyspnoea and quality of life in older people at home. *Age Ageing*. 2001;30:155–159.
45. Hospice Nurses Association. *Hospice and Palliative Care Clinical Practice Protocol: Dyspnea*. Pittsburgh, Pa: Hospice and Palliative Nurses Association; 1996:1–28.
46. Knebel AR. Dyspnea in the ventilator-assisted patient: evaluation and treatment. In: Mahler DA, ed. *Dyspnea*. New York, NY: Marcel Dekker Inc; 1998.
47. Birks C. Pathophysiology and management of dyspnoea in palliative care and the evolving role of the nurse. *Int J Palliat Nurs*. 1997;3:264–274.
48. Hardie GE, Janson S, Gold W, Carrieri-Kohlman V, Boushey H. Ethnic differences: word descriptors used by African-American and white asthma patients during induced bronchoconstriction. *Chest*. 2000;117:935–943.
49. Harver A, Mahler DA, Schwartzstein RM, Baird JC. Descriptors of breathlessness in healthy individuals: distinct and separable constructs. *Chest*. 2000;118:679–690.
50. Lanuza D, Lafaiver C, McCabe M, Farcas GA, Garrity E Jr. Prospective study of functional status and quality of life before and after lung transplantation. *Chest*. 2000;118:115–122.
51. Casaburi R. Exercise training in chronic obstructive lung disease. In: Casaburi R, Petty TL, eds. *Principles and Practice of Pulmonary Medicine*. Philadelphia, Pa: WB Saunders; 1993.
52. Combivent Inhalation Aerosol Study Group. In chronic obstructive pulmonary disease, a combination of ipratropium and albuterol is more effective than either agent alone: an 85-day multicenter trial. *Chest*. 1994;105:1411–1419.
53. Congleton J, Muers MF. The incidence of airflow obstruction in bronchial carcinoma, its relation to breathlessness, and response to bronchodilator therapy. *Respir Med*. 1995;89:291–296.
54. Luce JM, Luce JA. Management of dyspnea in patients with far-advanced lung disease: "Once I lose it, it's kind of hard to catch it..." *JAMA*. 2001;285:1331–1337.
55. O'Donnell DE, Webb KA, Bertley J, Chau L, Conian A. Mechanisms of relief of exertional breathlessness following unilateral bullectomy and lung volume reduction surgery in emphysema. *Chest*. 1996;110:18–27.
56. Ottanelli R, Rosi E, Romagnoli I, et al. Do inhaled corticosteroids affect perception of dyspnea during bronchoconstriction in asthma? *Chest*. 2001;120:770–777.
57. Ramirez-Venegas A, Ward J, Lentine T, Mahler DA. Salmeterol reduces dyspnea and improves lung function in patients with COPD. *Chest*. 1997;112:336–340.
58. Reid RT, Rudd RM. Management of malignant pleural effusion. *Thorax*. 1993;48:779–780.
59. Tanigawa N, Sawada S, Okuda Y, Kobayashi M, Mishima K. Symptomatic improvement in dyspnea following tracheobronchial metallic stenting for malignant airway obstruction. *Acta Radiol*. 2000;41:425–428.
60. Wong PS, Goldstraw P. Pleuroperitoneal shunts. *Br J Hosp Med*. 1991;50:16–21.
61. Yusen RD, Lefrak SS, Trulock EP. Evaluation and preoperative management of lung volume reduction surgical candidates. *Clin Chest Med*. 1997;18:199–224.
62. Dudgeon D. Management of dyspnea at the end of life. In: Mahler DA, O'Donnell DE, eds. *Dyspnea*. Boca Raton, Fla: Taylor & Francis; 2005.
63. Boyd KJ, Kelly M. Oral morphine as symptomatic treatment of dyspnoea in patients with advanced cancer. *Palliat Med*. 1997;11:277–281.
64. Bruera E, Macmillan K, Pither J, MacDonald RN. Effects of morphine on dyspnea of terminal cancer patients. *J Pain Symptom Manage*. 1990;5:341–344.
65. Farncombe M, Chater S. The use of nebulized opioids for breathlessness: a chart review. *Palliat Med*. 1994;8:306–312.
66. Light RW, Muro JR, Sato RI, Stansbury DW, Fischer CE, Brown SE. Effects of oral morphine on breathlessness and exercise tolerance in patients with chronic obstructive pulmonary disease. *Am Rev Respir Dis*. 1989;139:126–133.
67. Mazzocato C, Buclin T, Rapin CH. The effects of morphine on dyspnea and ventilatory function in elderly patients with advanced cancer: a randomized double-blind controlled trial. *Ann Oncol*. 1999;10:1511–1514.
68. Poole PJ, Veale AG, Black PN. The effect of sustained-release morphine on breathlessness and quality of life in severe chronic obstructive pulmonary disease. *J Respir Crit Care Med*. 1998;157:1877–1880.
69. Tanaka K, Shima Y, Kakinuma R, et al. Effect of nebulized morphine in cancer patients with dyspnea: a pilot study. *Jpn J Clin Oncol* [serial online]. 1999;29:600–603. Available at: <http://jjco.oxfordjournals.org/cgi/content/full/29/12/600>. Accessed December 27, 2006.
70. Nosedá A, Carpiáux JP, Markstein C, Meyvaert A, de Maertelaer V. Disabling dyspnoea in patients with advanced disease: lack of effect of nebulized morphine. *Eur Resp J*. 1997;10:1079–1083.
71. Argyropoulou P, Patakas D, Koukou A, Vasilidis P, Georgopoulos D. Bupirone effect on breathlessness and exercise performance in patients with chronic obstructive pulmonary disease. *Respiration*. 1993;60:216–220.
72. Cowcher K, Hanks GW. Long-term management of respiratory symptoms in advanced cancer. *J Pain Symptom Manage*. 1990;5:320–328.
73. Man GC, Hsu K, Sproule BJ. Effect of alprazolam on exercise and dyspnea in patients with chronic obstructive pulmonary disease. *Chest*. 1986;90:836–836.
74. McIver B, Walsh D, Nelson K. The use of chlorpromazine for symptom control in dying cancer patients. *J Pain Symptom Manage*. 1994;9:341–345.

75. Woodcock AA, Gross ER, Geddes DM. Drug treatment of breathlessness: contrasting effects of diazepam and promethazine in pink puffers. *Br Med J*. 1981;283:343-345.
76. Bruera E, de Stoutz N, Velasco-Leiva A, Schoeller T, Hanson J. Effects of oxygen on dyspnoea in hypoxaemic terminal-cancer patients. *Lancet*. 1993;342:13-14.
77. Dewan NA, Bell CW. Effect of low-flow and high-flow oxygen delivery on exercise tolerance and sensation of dyspnea: a study comparing transtracheal catheter and nasal prongs. *Chest*. 1994;105:1061-1065.
78. Garrod PR, Wedzicha JA. Supplemental oxygen during pulmonary rehabilitation in patients with COPD with exercise hypoxaemia. *Thorax*. 2000;55:539-543.
79. Knebel AR, Bentz E, Barnes P. Dyspnea management in alpha-1 antitrypsin deficiency: effect of oxygen administration. *Nurs Res*. 2000;49:333-338.
80. Moore DP, Weston AR, Hughes JM, Oakley CM, Cleland JG. Effects of increased oxygen concentrations on exercise performance in chronic heart failure. *Lancet*. 1992;339:850-853.
81. Petty TL, Casaburi R. Recommendations of the fifth oxygen consensus conference. Writing and organizing committees. *Respir Care*. 2000;45:940-943.
82. Swinburn CR, Mould H, Stone TN, Corris PA, Gibson GJ. Symptomatic benefit of supplemental oxygen in hypoxemic patients with chronic lung disease. *Am Rev Respir Dis*. 1991;143:913-915.
83. Schwartzstein RM, Lahive K, Pope A, Weinberger SE, Weiss JW. Cold facial stimulation reduces breathlessness induced in normal subjects. *Am Rev Respir Dis*. 1987;136:58-61.
84. Thompson CL. Dyspnea with and without fan blowing on face of hospitalized adults. *Crit Care Med*. 2001;29(suppl):A148.
85. Barach AL. Chronic obstructive lung disease: postural relief of dyspnea. *Arch Phys Med Rehab*. 1974;55: 494-504.
86. Eltayara L, Ghezzi H, Milic-Emili J. Orthopnea and tidal expiratory flow limitation in patients with stable COPD. *Chest*. 2001;119:99-104.
87. O'Neill S, McCarthy DS. Postural relief of dyspnoea in severe chronic airflow limitation: relationship to respiratory muscle strength. *Thorax*. 1983;38: 595-600.
88. Sharp JT, Drutz WS, Moisan T, Foster J, Machnach W. Postural relief of dyspnea in severe chronic obstructive pulmonary disease. *Am Rev Respir Dis*. 1980;122: 201-211.
89. Faling LJ. Pulmonary rehabilitation—physical modalities. *Clin Chest Med*. 1986;7:599-618.
90. Sinclair JD. The effect of breathing exercises in pulmonary emphysema. *Thorax*. 1955;10:246-249.
91. Stulberg MS, Ries AL, Belman MJ. Treatment of dyspnea: physical modalities, oxygen, and pharmacology. In: Mahler DA, ed. *Dyspnea*. New York, NY: Marcel Dekker; 1998.
92. Tiep BL, Burns M, Kao D, Madison R, Herrara J. Pursed lips breathing training using ear oximetry. *Chest*. 1986; 90:218-221.
93. Carrieri-Kohlman V, Gormley JM. Coping strategies for dyspnea. In: *Dyspnea*. New York, NY: Marcel Dekker; 1998.
94. Filshie J, Penn K, Ashley S, Davis CL. Acupuncture for the relief of cancer-related breathlessness. *Palliat Med*. 1996;10:145-150.
95. Jobst K, Chen JH, McPherson J, et al. Controlled trial of acupuncture for disabling breathlessness. *Lancet*. 1986; 2:1416-1419.
96. Maa SH, Gauthier D, Turner M. Acupressure as an adjunct to a pulmonary rehabilitation program. *J Cardiopulm Rehabil*. 1997;91:320-329.
97. McBride S, Graydon J, Sidani S, Hall L. The therapeutic use of music for dyspnea and anxiety in patients with COPD who live at home. *J Holis Nurs*. 1999;17: 229-250.
98. Carrieri-Kohlman VK, Janson-Bjerklie S. Dyspnea. In: *Pathophysiological Phenomena in Nursing: Human Responses to Illness*. Philadelphia, Pa: WB Saunders; 1993.

Test writer: Ann Lystrup, BSN, RN, CEN, CFRN, CCRN
 Contact hours: **2.0**
 Category: A
 Passing score: 10 correct (70%)

CE Test Instructions

To receive CE credit for this test (ID# CI1812), mark your answers on the form below, complete the enrollment information, and submit it with the \$12 processing fee (payable in US funds) to the American Association of Critical-Care Nurses (AACN). Answer forms must be postmarked by March 1, 2009. Within 3 to 4 weeks of AACN's receiving your test form, you will receive an AACN CE certificate.

The American Association of Critical-Care Nurses (AACN) is accredited as a provider of continuing nursing education by the American Nurses Credentialing Center's Commission on Accreditation. AACN has been approved as a provider of continuing education in nursing by the State Boards of Nursing of Alabama (#ABNP0062), California (#01036), Florida (#FBN2464), Iowa (#332), and Louisiana (#ABN12). AACN programming meets the standards for most other states requiring mandatory continuing education credit for relicensure.

AMERICAN ASSOCIATION OF CRITICAL CARE NURSES		CE Test Form	
Dyspnea: Applying Research to Bedside Practice		Test ID#: CI1812 FORM EXPIRES March 1, 2009 Fee: \$12	
Mark your answers clearly in the appropriate box. There is only one correct answer per question. You may photocopy this form.			
A	B	C	D
1. <input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
2. <input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
3. <input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
4. <input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
A	B	C	D
5. <input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
6. <input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
7. <input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
8. <input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
A	B	C	D
9. <input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
10. <input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
11. <input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
A	B	C	D
12. <input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
13. <input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
14. <input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Last name _____		First name _____ AACN Member # _____	
Address _____			
City _____		State _____ ZIP _____	
Telephone _____		E-mail _____	
State of licensure _____		License No(s). _____	
Payment by <input type="checkbox"/> Visa <input type="checkbox"/> Mastercard <input type="checkbox"/> American Express <input type="checkbox"/> Discover <input type="checkbox"/> Check			
Card # _____		Exp. Date _____	
Signature _____			
Program Evaluation			
	Yes	No	
Objective 1 was met	<input type="radio"/>	<input type="radio"/>	The level of difficulty of this test was:
Objective 2 was met	<input type="radio"/>	<input type="radio"/>	<input type="radio"/> easy <input type="radio"/> medium <input type="radio"/> difficult
Objective 3 was met	<input type="radio"/>	<input type="radio"/>	To complete this program, it took me
The content was appropriate	<input type="radio"/>	<input type="radio"/>	_____ hours/minutes.
My expectations were met	<input type="radio"/>	<input type="radio"/>	
This method of CE is effective for this content	<input type="radio"/>	<input type="radio"/>	
Mail To: AACN			
101 Columbia		Or fax to 949-362-2021	
Aliso Viejo, CA 92656		Or take test online at	
		www.aacn.org >Continuing Education	

Dyspnea: Applying Research to Bedside Practice

Objectives:

Upon completion of this article, the reader will be able to:

1. Describe the pathophysiology of dyspnea.
2. Identify the mechanisms that cause dyspnea.
3. Evaluate an evidence-based plan of care for a patient with dyspnea based upon the levels of recommendation presented.

1. Which statement best describes the findings obtained in studies of dyspnea in patients receiving mechanical ventilation?
 - a. Mechanically ventilated patients experience moderate to severe dyspnea.
 - b. Dyspnea during mechanical ventilation usually subsides within the first 12 to 24 hours.
 - c. Mechanical ventilation blocks the patient's perception of dyspnea.
 - d. Mechanical ventilation blocks the patient's sensation of dyspnea.
2. Which aspect of dyspnea arises from neural activation caused by stimulation of an afferent receptor?
 - a. The perception of dyspnea
 - b. The physiology of dyspnea
 - c. The etiology of dyspnea
 - d. The sensation of dyspnea
3. The stimulation of ventilation by hypoxia occurs as a result of action on chemoreceptors in which of the following locations?
 - a. Aortic bodies
 - b. Ventral surface of the medulla
 - c. Vagus nerve
 - d. Brain stem
4. Stimulation of C-fibers in the alveolar interstitium by serotonin and bradykinin causes which of the following responses?
 - a. Bronchodilation and rapid, shallow breathing
 - b. Rapid, deep breathing and increased secretions
 - c. Decreased secretions and bronchodilation
 - d. Rapid, shallow breathing and increased secretions
5. Central chemoreceptors inhibit ventilation in response to which parameter?
 - a. Increasing partial pressure of carbon dioxide gas
 - b. Increasing partial pressure of oxygen gas
 - c. Decreasing partial pressure of carbon dioxide gas
 - d. Decreasing partial pressure of oxygen gas
6. A patient has dyspnea if which of the following is present?
 - a. Tachypnea
 - b. Discomfort
 - c. Hyperpnea
 - d. Air hunger
7. Which dyspnea assessment instrument was preferred by 75% of critically ill patients in the study by Powers and Bennett?
 - a. Vertical Analogue Dyspnea Scale
 - b. Wong-Baker Faces Scale
 - c. Modified Borg Scale
 - d. 10-Point Numerical Scale
8. Assessment of a patient with cirrhosis is likely to reveal what finding?
 - a. Orthopnea
 - b. Paroxysmal nocturnal dyspnea
 - c. Platypnea
 - d. Tachypnea
9. What settings are recommended to reduce dyspnea in ventilated patients?
 - a. Decreased intermittent mandatory ventilation
 - b. Tidal volumes of 4 mL to 6 mL per kg
 - c. Increased inspiratory-expiratory ratio
 - d. Use of continuous positive airway pressure or pressure support
10. Oxygen decreases dyspnea by what mechanism?
 - a. Reduction of chemoreceptor activity
 - b. Reduction of nonspecific nasal receptor stimulation
 - c. Stimulation of upper airway receptors
 - d. Stimulation of motor neurosignaling activity
11. According to the evidence-based plan of care described in this article, level IV indicates the presence of which of the following?
 - a. Clinical studies in more than 1 or 2 different patient populations and situations to support recommendations
 - b. Theory-based recommendations with supporting laboratory data
 - c. Limited clinical studies to support recommendations
 - d. Expert clinical consensus group recommendations with limited supporting research data
12. The dyspnea associated with obesity is likely due to which of the following?
 - a. Stimulation of chest wall or respiratory muscle mechanoreceptors
 - b. Increased efferent motor neuron activity from the central nervous system to respiratory effector systems
 - c. Stimulation of intrapulmonary receptors
 - d. Stimulation of peripheral or central chemoreceptors
13. Opioids decrease dyspnea by which of the following mechanisms?
 - a. Bronchodilation and decreased retention of carbon dioxide
 - b. Relaxation and drowsiness
 - c. Blunted central perception of dyspnea
 - d. Decreased neuronal response of pain receptors in the pleura
14. Which of the following statements about dyspnea is true?
 - a. Patients are said to have dyspnea if they have both an increased respiratory rate (tachypnea) and an increased depth of breathing (hyperpnea).
 - b. A patient with an elevated respiratory rate and decreased oxygen saturation can be assumed to have dyspnea.
 - c. A patient with chronic dyspnea is likely to describe his or her respiratory distress as severe.
 - d. Dyspnea is a subjective symptom described only by the patient.