Dyspnea is a distressing and debilitating symptom for patients with either primary or metastatic lung cancer that increases in severity with the progression of disease (Escalante et al., 1996; Vainio & Auvinen, 1996). In patients with primary lung cancer, the most common cause of dyspnea is the underlying disease—usually the cancer tumor or chronic obstructive pulmonary disease (COPD). In other cancers, the principle cause of dyspnea is lung metastases from the primary site, such as with breast, esophagus, colorectal, and prostate cancer (Heyse-Moore, Ross, & Mullee, 1991; Vainio & Auvinen). In the advanced stages of primary and metastatic lung cancer, cancer-induced complications such as pleural effusion, pericardial effusion, pulmonary embolus, pneumonitis, and superior vena cava syndrome can be the causes of dyspnea (Cowcher & Hanks, 1990). Management of dyspnea in patients with cancer requires knowledge and understanding of the underlying disease—usually the cancer tumor or COPD. This knowledge can provide the basis for successful dyspnea management in lung cancer.
of the mechanisms and pathophysiologic changes in the lungs caused by tumor involvement and treatment effects, including benefits and limitations (Dudgeon & Rosenthal, 1996). Many researchers have reported successful management of dyspnea in patients with COPD; however, few have investigated dyspnea management interventions for patients with lung cancer. Successful dyspnea management in patients with COPD logically might be applied to the management of dyspnea in patients with lung cancer because of the similarity of some causes and complications in the two diseases. The main difference between the groups is that patients with cancer have a shorter prognosis than those with COPD, but because of the similarity of symptoms, healthcare providers should consider applying the evidence on dyspnea management in COPD to lung cancer.

The main purpose of this review article is to transfer and apply the evidence base of dyspnea management used for patients with COPD to patients with lung cancer. This article presents an overview of the pathophysiologic mechanisms of dyspnea to provide a foundation for the interventions. It then offers a critical review of the evidence base for dyspnea management, including pharmacologic and nonpharmacologic management, emphasizing the application of COPD dyspnea management to dyspnea in lung cancer. Thus, the treatments to reduce cancer-induced complications, such as thoracentesis, chest tube insertion, pleurodesis for pleural effusion or pericardial window for pericardial effusion, and antibiotic for infection are not discussed. The implications of dyspnea management for oncology nursing practice also will be described. For the purpose of this article, the term lung cancer will refer to primary and metastatic lung cancers.

The Mechanisms of Dyspnea

Normally, breathing is a predominantly involuntary activity controlled by respiratory centers in the brain stem. The respiratory centers generate and regulate breathing patterns by sending nerve impulses received from various respiratory receptors through neuron pathways to respiratory muscles—the diaphragm, intercostal muscles, and accessory muscles (Fink, 1999) (see Figure 1).

Dyspnea is an abnormal breathing pattern that frequently presents in people with lung disease. The neural pathways for dyspnea are the same as those involved in normal breathing (Gift, 1990). The pathologies that produce dyspnea are classified into three major categories: chemical, neural, and emotional stimulations (Birks, 1997).

Chemical Stimulation

The central respiratory chemoreceptors are located in the medulla, and the peripheral respiratory chemoreceptors are situated in the carotid and aortic bodies. The central chemoreceptors are highly sensitive to increased arterial carbon dioxide (PaCO₂) in the blood, whereas peripheral chemoreceptors are highly sensitive to low arterial oxygen (PaO₂) (Beachey, 1998). Increased PaCO₂ or decreased PaO₂ directly stimulates the respiratory center in the brain. The reaction causes patients to breathe more forcefully and frequently to eliminate carbon dioxide.

Neural Stimulation

The neural pathways for breathing integrate the input from receptors located in the lungs and other areas, such as skin, muscles, tendons, and joints. Respiratory receptors in the lungs, called the mechanoreceptors, include stretch receptors, irritant receptors, and J receptors (Beachey, 1998; Fink, 1999).

The stretch receptors in the trachea, bronchi, and receptors in the respiratory muscles are stimulated by lung inflation. The J receptors, located in the juxta pulmonary capillary region, are stimulated by an inflammatory process in the lungs, pulmonary vascular congestion, and pulmonary edema (Beachey, 1998). The irritant receptors located in the epithelium of the small airways are stimulated by the inhalation of irritant substances or allergens such as smoke, pollens, molds, fungi, or even cold air (Beachey). Contraction of bronchial smooth muscles and stretching of the bronchial wall also stimulate the irritant receptors. The mechanoreceptors in muscles, tendons, and joints are stimulated by motion of the limbs, and the mechanoreceptors in the skin are stimulated by painful stimuli (Fink, 1999). Stimulation of mechanoreceptors causes patients to breathe rapidly.

Emotional Stimulation

Dyspnea can occur with emotional distress in the absence of any pulmonary abnormalities. This is called neurotic dyspnea. Alteration of breathing by this mechanism is the response of the higher brain center to emotional changes that stimulate the respiratory center, which, in turn, regulates the breathing pattern (Leff & Schumacker, 1993; Quinn, 1999).

Causes of Dyspnea in Chronic Obstructive Pulmonary Disease

The physiologic changes in COPD stimulate the various dyspnea mechanisms. The causes of dyspnea in COPD include progressive airway obstruction and hyperinflation resulting in

Figure 1. Breathing Pathway

Higher brain center (hypothalamus, cerebral cortex)

Mechanoreceptors (stretch receptors, irritant receptors)

Respiratory centers (medulla, pons)

Chemoreceptors

Spinal cord and nerves (C3–5, T11–12)

Respiratory muscles (diaphragm, intercostal, and accessory muscles)

Airway-controlling muscles
the well-known complications of the disease—hypoxemia, hypercapnia, pulmonary hypertension, pulmonary embolism, respiratory infection, and congestive heart failure (Phillips, Hnatiuk, & Torrington, 1997). Anxiety and depression also cause dyspnea in patients with COPD (Belville-Robertson, 1999; Rabinowitz & Florian, 1992).

**Causes of Dyspnea in Lung Cancer**

The causes of dyspnea in lung cancer can be classified as direct or indirect. Direct effects of a tumor mass that invades the lungs include airway obstruction, pleural effusion, phrenic nerve paralysis, and superior vena cava syndrome, such as pulmonary hypertension (Dudgeon & Rosenthal, 1996). Indirect causes of dyspnea in patients with cancer include treatment-related effects, anemia, pulmonary emboli, and cachexia (Dudgeon & Rosenthal). Radiation may cause radiation pneumonitis and fibrosis, whereas chemotherapy may cause pulmonary toxicity and cardiomyopathy (Wickham, 1998). In patients with lung cancer, dyspnea may not be caused directly by a tumor but rather by anxiety, depression, and comorbid diseases such as COPD, asthma, and congestive heart failure.

Dyspnea associated with COPD and dyspnea associated with lung cancer share some causative mechanisms: reduction of airflow and gas exchanges because of airway obstruction and destruction of the lung parenchyma. Pulmonary hypertension, congestive heart failure, anxiety, and depression found in patients with COPD also may be problems in patients with lung cancer (see Figure 2).

A wealth of research has been conducted in the pharmacologic (see Table 1) and nonpharmacologic (see Table 2) management of dyspnea in COPD but not in cancer. Because many of the causes of dyspnea in COPD and lung cancer are similar, applying and testing treatments found to reduce dyspnea in patients with COPD to dyspneic patients with cancer seems logical.

**Pharmacologic Management**

**Morphine**

Morphine is the drug of choice for the pharmacologic management of dyspnea and has been used successfully to relieve dyspnea for patients with COPD as well as those with lung cancer (Beauford, Saylor, Stansbury, Avalos, & Light, 1993; Farncombe & Chater). Although how morphine reduces dyspnea is not understood totally, it may decrease the response of central chemoreceptors to hypercapnia as well as decrease the responses of peripheral chemoreceptors to hypoxemia (Florez, Pazos, Hurle, & Mediavilla, 1983; Wickham, 1998). Anxiolytic effects of morphine on the receptors in the higher-brain center—hypothalamus, amygdala, and frontal cortex morphine—also might reduce dyspnea resulting from anxiety (Cowcher & Hanks, 1990). The hypotensive effect of morphine secondary to vasodilation may result in a reduction of preload that, in turn, relieves pulmonary edema and, thereby, further reduces dyspnea caused by pulmonary congestion (Cowcher & Hanks).

Morphine can be administered safely and effectively through several routes, including via nebulizer, orally, via IV (intermittent or continuous injection and infusion), subcutaneously, or per rectum (Wickham, 1998). The easiest, most convenient administration route is oral; however, subcutaneous continuous infusion is more convenient in patients who are not able to swallow or during severe episodes of dyspnea (Cohen et al., 1991; Wickham).

Studies investigating morphine for dyspnea are numerous compared with other classes of medication and nonpharmacologic treatments. Despite the quantity of the studies, they have many limitations for interpretation and utilization. The methods used included prospective designs and retrospective designs, but many relied on chart review for data collection. A few quasi-experimental studies have been reported, but insufficient sample sizes, attrition problems, and lack of control groups were common. However, the literature does indicate that morphine does not cause respiratory distress when low doses are used.

**Dosage:** Studies have not yet determined an exact dosage regimen of morphine for dyspnea management. The current recommendation is to start with a low dose and then increase if needed. The dose required for reducing dyspnea typically is lower and more standardized than that required for controlling pain (Wickham, 1998). The dosage of morphine required to reduce dyspnea varies depending on a patient’s history of morphine use for pain relief, sensitivities to morphine treatments, and routes of administration.

Oral morphine has been used successfully for reducing dyspnea and increasing exercise tolerance in patients with COPD (Farncombe, Chater, & Gillin, 1994; Light et al., 1989; Schonhofer, Suchi, Haidl, & Kohler, 2001; Young, Dvaskas, & Keena, 1989). However, the duration of action of oral morphine for dyspnea is much shorter than its analgesic effects (Bruear, Macmillan, Pither, & MacDonald, 1990). The low dose of oral morphine is 5–15 mg every four hours adjusted on an individual basis (Cowcher & Hanks, 1990). Consequently, the dose can be increased daily by 30%–50% or administered more frequently as tolerated, until patients feel comfortable or sedation becomes a problem (Rousseau, 1996; Sheehan & Forman, 1997; Wickham, 1998). The dose can be titrated as much as 15 mg every four hours in the absence of pain (Cowcher & Hanks) or as much as 10 mg every four
### Table 1. Research on Pharmacologic Treatments to Relieve Dyspnea

<table>
<thead>
<tr>
<th>Agent (Reference)</th>
<th>Subjects</th>
<th>Sample</th>
<th>Methods</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antianxiety agents (Woodcock et al., 1981)</td>
<td>Emphysema</td>
<td>18</td>
<td>Single group, double blind, placebo controlled, crossover between diazepam and pro-methazine</td>
<td>Diazepam had no effect on breathlessness and noticeably reduced exercise tolerance; promethazine reduced breathlessness and improved exercise tolerance without altering lung function.</td>
</tr>
<tr>
<td>Corticosteroids (Koyama et al., 1992)</td>
<td>Chronic obstructive pulmonary disease (COPD)</td>
<td>29</td>
<td>Single group, pre- and post-test</td>
<td>Increased FEV₁ (forced expiratory volume in one second)</td>
</tr>
<tr>
<td>Inhaled short-acting β2-agonists (Sestini et al., 2000)</td>
<td>COPD</td>
<td>13</td>
<td>Cochrane Systematic Reviews</td>
<td>FEV₁, peak expiratory flow rates, and breathlessness scores were improved; no improvement in exercise performance; no report of serious short-term side effects</td>
</tr>
<tr>
<td>IV morphine (Cohen et al., 1991)</td>
<td>Advanced lung cancer</td>
<td>8</td>
<td>Single group, multiple post-tests</td>
<td>Decreased dyspnea and increased oxygen saturation</td>
</tr>
<tr>
<td>Nebulized morphine (Farncombe et al., 1994)</td>
<td>Advanced lung cancer</td>
<td>3</td>
<td>Case study</td>
<td>Decreased dyspnea and increased exercise tolerance with no adverse effects</td>
</tr>
<tr>
<td>Nebulized morphine (Tanaka et al., 1999)</td>
<td>Advanced lung cancer of which the causes of dyspnea were progressive lung tumor (10), pneumonia (5), carcinomaous lymphangitis (3), pleural effusion (3), and airway obstruction (2). Some causes overlapped.</td>
<td>15</td>
<td>Single group, pre- and post-test</td>
<td>Decreased dyspnea without respiratory depression, nausea, or vomiting</td>
</tr>
<tr>
<td>Nebulized morphine (Young et al., 1989)</td>
<td>COPD (8) Idiopathic pulmonary fibrosis (2)</td>
<td>10</td>
<td>Single group, double blind, randomized, and crossover to receive morphine and placebo</td>
<td>Morphine improved exercise tolerance time with no side effects.</td>
</tr>
<tr>
<td>Nebulized morphine (Zeppetella, 1997)</td>
<td>Advanced lung cancer: primary lung cancer (13), lung metastases (2), and mesothelioma (2)</td>
<td>17</td>
<td>Single group, one pretest and two post-tests</td>
<td>Most patients experienced less dyspnea after 24 hours of morphine administration, but this effect lasted for only 48 hours; three patients stopped using the nebulizer because they did not tolerate it.</td>
</tr>
<tr>
<td>Oral morphine (Boyd &amp; Kelly, 1997)</td>
<td>Primary lung cancer (7) Lung metastases (8) 9 of 15 completed the study.</td>
<td>15</td>
<td>Uncontrolled, single group, repeated measures</td>
<td>Improved dyspnea in 3 of 9 patients who completed the study; sedation was the main side effect in the first 48 hours; dizziness was the second most common side effect.</td>
</tr>
<tr>
<td>Oral morphine (Light et al., 1989)</td>
<td>COPD</td>
<td>13</td>
<td>Single group, pre- and post-test, compared morphine and placebo</td>
<td>Increased exercise capacity</td>
</tr>
<tr>
<td>Oral morphine (Schonhofer et al., 2001)</td>
<td>Emphysema</td>
<td>661</td>
<td>Descriptive, retrospective (chart review)</td>
<td>Patient record charts showed dyspnea improvement after morphine administration; 10% stopped treatment because of severity of side effects.</td>
</tr>
<tr>
<td>Oxygen (Swinburn et al., 1991)</td>
<td>Chronic lung diseases: interstitial lung disease (10) and COPD (12)</td>
<td>22</td>
<td>Single group, double blind, randomized, compared oxygen and normal air via facemask</td>
<td>Dyspnea score was reduced and saturated oxygen was increased during oxygen breathing</td>
</tr>
<tr>
<td>Oxygen (Brüera, de Stoutz et al., 1993)</td>
<td>Advanced lung cancer: lung tumor (5), lung metastases (6), pleural effusion (2), and carcinomaous lymphangitis (1)</td>
<td>14</td>
<td>Single group, double blind, crossover between oxygen and normal air administered via facemask</td>
<td>Oxygen saturation, respiratory effort, respiratory rate, and the visual analog scale for dyspnea all were significantly better with oxygen than with air.</td>
</tr>
</tbody>
</table>

(Continued on next page)
hours over and above the dose required for pain relief. For older adult patients and those with renal failure, administration of morphine should start at 2.5 mg and be administered less frequently than every four hours. Symptoms of renal insufficiency and accumulation of morphine-6-glucuronide should be monitored appropriately (Cowcher & Hanks).

Morphine injection (via IV or subcutaneously) is an effective therapy for severe dyspnea in patients with cancer (Cohen et al., 1991). The starting dose of subcutaneous morphine for morphine-naive patients should be 2.5–5 mg every four hours (LeGrand & Walsh, 1999). The suggested dose for IV injection is a bolus dose 1–2 mg every 5–10 minutes until dyspnea is relieved, followed by morphine infusion with an hourly dose of 50% of the cumulative bolus dose (Cohen et al.).

Nebulized morphine is a safe route for morphine administration. An advantage of the nebulized route is that its action is local rather than systemic. Some patients, however, report discomfort using the mask (Farncombe et al., 1994; Zeppetella, 1997). To administer nebulized morphine, morphine solution usually is combined with 2 ml of sterile normal saline delivered using compressed oxygen at 5–6 L per minute. The dose of nebulized morphine to reduce dyspnea in COPD is 2.5 mg every four hours (Farncombe & Chater, 1993). A dose of 20 mg every four hours is helpful for reducing dyspnea in patients with lung cancer (Tanaka et al., 1999; Zeppetella). More evidence is needed regarding nebulized morphine for the relief of dyspnea in patients with COPD and lung cancer.

**Side effects:** Many studies have shown that using low doses of oral and injected morphine does not cause respiratory depression (Bruea et al., 1990; Sodha & Frampton, 1995). Doses higher than 15 mg may cause side effects, such as sedation and dizziness, without further improvement of dyspnea (Cowcher & Hanks, 1990). Nebulized morphine is a safe route of administration compared to oral or injected morphine. However, morphine remaining in the nebulizer and accumulating steadily over time may lead to overdose (Wood, Wilson, & Bray, 1986).

**Nursing interventions:** The side effects of morphine—dysphoria, dizziness, drowsiness, urinary retention, constipation, nausea, vomiting, and dry mouth—should be monitored (Birks, 1997; Boyd & Kelly, 1997). In addition, the provision of adequate and accurate information to patients and families is necessary to allay concerns that patients will become addicted. Before beginning therapy, nurses should provide a clear explanation of possible side effects. Patients should be reassured that morphine will help them rest and sleep well without the feeling of suffocation associated with dyspnea, that it is not dangerous, and that it does not cause chemical dependency (Wickham, 1998). Patients must understand that although morphine relieves dyspnea, it does not eliminate the causes of dyspnea. Therefore, when patients stop taking morphine, dyspnea returns.

**Corticosteroids**

For patients with advanced cancer, high-dose corticosteroids (dexamethasone and prednisolone) can be used to relieve dyspnea caused by superior vena cava syndrome and the inflammatory process induced by radiation or chemotherapy (LeGrand & Walsh, 1999; Wickham, 1998). No controlled studies have used corticosteroids for dyspnea treatment for patients with lung cancer; however, high-dose prednisolone successfully reduces the inflammatory process in COPD (LeGrand & Walsh). Because some patients with lung cancer also have inflammation in the lungs, administering high-dose corticosteroids to such patients is logical.

**Dosage:** The suggested dose for relief of dyspnea with dexamethasone is 4–8 mg twice daily (LeGrand & Walsh, 1999; Rousseau, 1996). However, controlled studies are needed to support the recommendation.

**Side effects:** Gastric toxicity and fluid retention are usual side effects of high-dose corticosteroids (Ahmedzai, 1998). The side effects should be monitored carefully. To prevent adverse effects, corticosteroids should be titrated down to the minimally effective dose after starting with a high dose to treat inflammation (Cowcher & Hanks, 1990; Rousseau, 1996). Short-term side effects, such as hyperglycemia and oral candidiasis, should be monitored (Rousseau).

**Bronchodilators**

Short-acting bronchodilators—β2-agonist stimulators (nebulized albuterol) and methylxanthines (oral theophylline or aminophylline)—are standard treatments for COPD and have been used for symptomatic treatment in patients with lung cancer (Dudgeon & Rosenthal, 1996; Wickham, 1998). Because of their bronchodilation effects, the medications are more beneficial for patients with cancer who have COPD,

<table>
<thead>
<tr>
<th>Agent (Reference)</th>
<th>Subjects</th>
<th>Sample</th>
<th>Methods</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subcutaneous morphine (Bruea et al., 1990)</td>
<td>Advanced cancer: primary lung (8), breast (4), ovarian (4), colon (2), stomach (1), and esophageal (1)</td>
<td>20</td>
<td>Single group, pre- and post-test</td>
<td>Improved dyspnea without a significant deterioration in respiratory function</td>
</tr>
<tr>
<td>Subcutaneous morphine (Bruea, MacEachern, et al., 1993)</td>
<td>Advanced cancer: progressive lung tumor (3), lung metastases (4), pleural effusion (1), and carcinomatous lymphangitis (1)</td>
<td>9</td>
<td>Single group, placebo controlled, crossover</td>
<td>Improved dyspnea without change in respiratory rate or saturated oxygen level</td>
</tr>
<tr>
<td>Theophylline (Murciano et al., 1989)</td>
<td>COPD</td>
<td>60</td>
<td>Randomized, controlled, double-blind crossover between theophylline and placebo</td>
<td>Improved respiratory muscle performance, respiratory function, and dyspnea</td>
</tr>
</tbody>
</table>
### Table 2. Research on Nonpharmacologic Management of Dyspnea in Chronic Obstructive Pulmonary Disease

<table>
<thead>
<tr>
<th>Reference</th>
<th>Intervention</th>
<th>N</th>
<th>Design</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sharp et al., 1980</td>
<td>Leaning forward</td>
<td>17</td>
<td>Crossover</td>
<td>Seven patients felt more comfortable leaning forward.</td>
</tr>
<tr>
<td>O’Neill &amp; McCarthy, 1983</td>
<td>Leaning forward</td>
<td>40 with lung disease; 140 normal people</td>
<td>Stratified sampling, quasi-experimental</td>
<td>Leaning forward was the optimum posture to generate maximum inspiratory pressures and to relieve dyspnea.</td>
</tr>
<tr>
<td>Ries et al., 1988</td>
<td>Upper-extremity exercise twice a week for eight weeks</td>
<td>E = 17; C = 11</td>
<td>Randomized, controlled</td>
<td>Improved dyspnea with increased exercise capacity in an experimental group</td>
</tr>
<tr>
<td>Breslin, 1992</td>
<td>Pursed-lip breathing</td>
<td>13</td>
<td>Single group, crossover</td>
<td>Pursed-lip breathing led to increased rib cage and accessory muscle recruitment during inspiration and expiration, decreased work of inspiration muscle, decreased respiratory rate, and improved saturated oxygen.</td>
</tr>
<tr>
<td>O’Donnell et al., 1993</td>
<td>Upper and lower limb exercises three times a week for six weeks</td>
<td>E = 23; C = 13</td>
<td>Random assignment using a staggered basis</td>
<td>Reduced dyspnea score, increased the distance of walking, and increased forced vital capacity in an experimental group</td>
</tr>
<tr>
<td>Song et al., 1993</td>
<td>Parenteral nutritional support</td>
<td>E = 12; C = 13</td>
<td>Randomized, controlled</td>
<td>After 10–20 days, an experimental group showed improved nutritional status, increased body weight, and decreased arterial carbon dioxide.</td>
</tr>
<tr>
<td>Ganzoni et al., 1994</td>
<td>High-calorie nutrition</td>
<td>E = 14; C = 16</td>
<td>Randomized, controlled</td>
<td>After increasing caloric intake to 45 kcal/kg of body weight for 12 months, patients experienced increased weight, skin fold thickness, and six-minute walking distance; and improved function of respiratory muscle exercise tolerance in a controlled group.</td>
</tr>
<tr>
<td>Ries et al., 1995</td>
<td>Lower-extremity exercise twice a week for eight weeks</td>
<td>E = 57; C = 62</td>
<td>Randomized clinical trial</td>
<td>Increased maximal exercise tolerance, maximal oxygen uptake, exercise endurance, symptoms of perceived breathlessness, self-efficacy for walking in an experimental group; decreased muscle fatigue and shortness of breath in an experimental group</td>
</tr>
<tr>
<td>Vitacca et al., 1998</td>
<td>Diaphragmatic breathing</td>
<td>25</td>
<td>Descriptive correlational, no control group</td>
<td>Diaphragmatic breathing was associated with increased transcutaneous partial pressure of oxygen and decreased partial pressure of carbon dioxide, increased tidal volume, reduced respiratory rate, reduced dyspnea, and increased inspiratory muscle effort.</td>
</tr>
<tr>
<td>Vermeeren et al., 2001</td>
<td>Part 1: high-calorie nutrition; Part 2: compared a fat-rich and a carbohydrate-rich supplement</td>
<td>Part I = 14; Part II = 11</td>
<td>Part I: single group; Part II: randomized, double blind, crossover</td>
<td>High-energy nutritional supplement increased respiratory quotient*, no significant difference in metabolism or exercise capacity after a fat-rich or carbohydrate-rich supplement</td>
</tr>
</tbody>
</table>

E—experimental subjects; C—control subjects

*Respiratory quotient is “the ratio of carbon dioxide molecules produced to oxygen molecules consumed in the tissue” (Beachey, 1998, p. 331).

bronchitis, and a history of smoking (Wickham). No studies of the effects of bronchodilators on dyspnea in patients with cancer were found.

**Dosage:** The therapeutic dose of theophylline is 200–400 mg per day to maintain a plasma level of theophylline between 8–12 mcg/ml (Couser, 2001). To reduce the side effects of theophylline, the plasma theophylline level should be maintained below 20 mcg/ml (LeGrand & Walsh, 1999).

**Side effects:** A high plasma concentration of theophylline may cause vomiting, hypokalemia, hyperglycemia,

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ONCOLOGY NURSING FORUM – VOL 32, NO 4, 2005

790
tachycardia, cardiac dysrhythmias, neuromuscular irritability, and seizures (Couser, 2001). The symptoms should be monitored, as well as plasma theophylline levels.

**Antianxiety Drugs**

Benzodiazepines (lorazepam and diazepam) and phenothiazines (chlorpromazine) have been suggested for the relief of dyspnea in lung cancer (Birks, 1997; Cowcher & Hanks, 1990; Dudgeon & Rosenthal, 1996; LeGrand & Walsh, 1999; Rousseau, 1996). The medications do not have direct effects on the lungs, but their sedative action is very helpful in reducing anxiety that may induce or exacerbate dyspnea (Quinn, 1999; Rousseau). Because dyspnea intensity is believed to be highly correlated with anxiety (Bruera, Schnitz, Pither, Neumann, & Hanson, 2000), sedatives or anxiolytic drugs may lessen the distress associated with dyspnea. However, few clinical studies have used such medications to treat dyspnea.

**Side effects:** Benzodiazepines may cause excessive sedation, impair thinking, and cause respiratory distress (Cohen et al., 1991; Cowcher & Hanks, 1990). Nurses should monitor the side effects carefully, especially in older adults and in patients with poor kidney or liver function (Macklon, Barton, James, & Rawlins, 1980).

**Nebulized Local Anesthetics**

Local anesthetic agents (lignocaine and bupivacaine) administered via nebulizer have been suggested for dyspnea treatment in patients with advanced cancer (Cowcher & Hanks, 1990). The effectiveness of lignocaine has been examined, with nonsignificant changes in dyspnea after drug administration. Bupivacaine has not been tested in patients with lung cancer. Similar to morphine administered via nebulization, anesthetic agents administered using this method may remain in the nebulizer after administration, accumulate over time, and lead to overdose. The drugs are not used often to treat dyspnea because of their side effects. Dudgeon and Rosenthal (1996) noted that inhaled anesthetics could not be recommended for the treatment of dyspnea in patients with lung cancer because of their bronchospasm effect. However, no studies have examined the effectiveness of local anesthetic agents and their side effects in patients with COPD or cancer. Controlled studies with larger sample sizes are needed to determine effective dosages and side effects of nebulized local anesthetics.

**Dosage:** Cowcher and Hanks (1990) suggested using 5 ml of 2% lignocaine administered via nebulizer to reduce dyspnea in patients with cancer. However, they did not offer a description of the effectiveness of the dose.

**Side effects:** The only concern about side effects of local anesthetics administered via nebulizer is depression of the cough and gag reflex (Cowcher & Hanks, 1990). Patients should avoid eating and drinking for several hours after use (Quinn, 1999). Other side effects are minimal. Patients might experience unpleasant taste or an uncomfortable feeling in their mouths because of anesthetic actions on the oral mucosa (Cowcher & Hanks).

**Oxygen Therapy**

Oxygen therapy commonly is used in dyspnea management. Oxygen can be administered in several ways, but administration via nasal cannula is believed to be more comfortable than via facemask. Masks can be noisy and can create a psycho-logical barrier between patients and other people (Wickham, 1998). Oxygen therapy increases oxygen saturation (SaO2) and reduces dyspnea, respiratory rate, and respiratory effort for patients with primary lung cancer and advanced disease (Bruera, de Stoutz, Velasco-Leiva, Schoeller, & Hanson, 1993; Swinburn, Moold, Stone, Corris, & Gibson, 1991).

Some arguments remain against the use of oxygen therapy for patients with cancer. Some researchers assert that oxygen is not beneficial unless patients are hypoxemic, whereas others suggest that oxygen is very beneficial (Rousseau, 1996). When hypoxemia occurs, it can be relieved with oxygen administration (Bruera, de Stoutz, et al., 1993; Turner, 1992). When no hypoxemia exists, psychological benefits are gained by reducing the fear of air hunger associated with dyspnea and hypoxia. Yet the effectiveness of oxygen and the mechanisms of dyspnea alleviation in nonhypoxic patients are not clear. Dunlop (1998) argued that hypoxia might not be a cause of breathlessness, and most patients with advanced cancer and breathlessness are not hypoxic; thus, to relieve dyspnea in such patients, other dyspnea-inducing factors such as fear, depression, and anxiety should be given priority rather than giving oxygen.

Whether oxygen actually diminishes the sensation of dyspnea in nonhypoxic patients is less important than whether patients report less dyspnea with the use of oxygen. This may be sufficient rationale to provide oxygen for palliation.

**Nonpharmacologic Management**

Nurses have a unique role in multidisciplinary teams in institutional and community settings (Birks, 1997; Grey, 1995). Being with patients frequently, nurses have a chance to observe and monitor patients’ problems to assess their needs; therefore, they can identify, interpret, and intervene in dyspnea management (Roberts, Thorne, & Pearson, 1993). Nonpharmacologic management of dyspnea, such as breathing techniques and positioning, is useful; however, although nonpharmacologic techniques have been used successfully in reducing dyspnea in patients with COPD, very few of the techniques have been applied to patients with lung cancer with dyspnea.

**Breathing Techniques**

Pursed-lip and diaphragmatic breathing have been found to have beneficial results for dyspneic patients, including reduced respiratory rate and control of dyspnea. In addition, pursed-lip and diaphragmatic breathing decrease functional residual capacity, increase respiratory muscle recruitment during inspiration and expiration, reduce the work of the diaphragm, increase tidal volume and alveolar ventilation, improve the ability to perform effective coughing, and improve blood gases (Breslin, 1992; Coppola & Wood, 2001; Sexton, 1990; Vitacca, Clin, Bianchi, & Ambrosio, 1998). The mechanism for breathing techniques for reducing dyspnea can be explained by physiologic changes during performance of breathing. Deep inhalation through the nose followed by slow exhalation through pursed lips increases lung expansion, and decreased time with the airway constricted improves gas exchange in the lungs. When patients perform proper diaphragmatic breathing, abdominal muscle contraction moves the diaphragm downward, providing more space for lung expansion and, therefore, increased gas exchange in the lungs.
Although not been much evidence supports this intervention for patients with lung cancer, the physiologic explanation is sufficient to encourage patients with dyspnea to learn and practice the techniques. Because this is such a simple and cost-effective strategy for reducing dyspnea, nurses should teach and encourage patients to practice breathing techniques. However, future research is needed to examine the effectiveness of pursed-lip and diaphragmatic breathing techniques for patients with cancer.

Positioning

Several comfortable positions can help to reduce dyspnea in patients with COPD. Examples include sitting on the edge of a bed with arms folded on a pillow on the bedside table and sitting in a chair with feet wide apart and elbows resting on knees (Foote, Sexton, & Pawlik, 1986; Rifas, 1980; Sheehan & Forman, 1997). At present, no studies have examined the effectiveness of the positions for patients with lung cancer.

Patients with COPD usually feel more comfortable sitting and leaning forward. In such a position, the abdominal wall can move outward and less transdiaphragmatic pressure occurs, thus providing more space for lung expansion and gas exchange (Sharp, Drutz, Moisan, Foster, & Machnach, 1980). Increased space for lung expansion and gas exchange related to transdiaphragmatic pressure is a logical physiologic explanation for why patients feel more comfortable sitting and leaning forward.

Nurses should teach a variety of helpful positions to patients because some positions may be better suited for one person than another and one position may be better suited for a particular activity. Several comfortable positions are necessary for daily living activities. For example, if dyspnea occurs when patients are climbing stairs, they should lean on a banister. If they experience dyspnea while shopping, they should lean on a shopping cart (Sexton, 1990). The principle is to try various positions that allow the lungs to expand with less transdiaphragmatic pressure.

Exercise

Patients with dyspnea generally are sedentary because of their fear and anxiety associated with the sensation of dyspnea caused by exertion. Decreasing activity and being sedentary may lead to progressive deconditioning and, consequently, disability, isolation, and death (Garvey, 1998). Although lung damage is irreversible and dyspnea can be alleviated by medical therapy, exercise may help patients maintain their ability to function with as much independence as possible.

Types of exercise: Nurses should instruct patients to exercise leg and arm muscles using several techniques such as lifting weights, climbing small sets of stairs, walking on a treadmill, and using a cycling ergometer. Patients exercising at home can use a can of food for arm exercises rather than a dumbbell. Compared to lower-extremity exercise, upper-extremity exercise is more beneficial for improving respiratory muscle strength and reducing dyspnea, which, in turn, improves performance of activities that need the work of the upper body, such as taking a bath, washing dishes, and getting dressed. Nurses should encourage patients with cancer who are able to exercise to do so regularly. Patients should start with low-intensity exercise and increase the intensity as tolerated. Consultation with a physical therapist is important for determining the optimal intensity of exercise.

To gain the most benefit from exercise, patients should perform stretching and strengthening exercises—head circling, shoulder shrugging, elbow circling, shoulder stretching, side bending, and weight lifting. Some simple techniques that provide benefit for lower-extremity stretching and strengthening are hamstring and quadriceps stretches, sitting leg flexions, and abdominal crunches (Ries et al., 2001). Performing upper-extremity and lower-extremity exercises is more beneficial in improving exercise endurance than doing only upper- or lower-extremity exercises (Lake, Henderson, Briffa, Openshaw, & Musk, 1990).

Frequency and duration: For exercise to be effective in improving physical endurance and reducing dyspnea, patients with COPD should exercise at least twice a week for eight consecutive weeks. Randomized, controlled studies have shown improvement of dyspnea, forced vital capacity, and exercise capacity after exercise training (O’Donnell, Webb, & McGuire, 1993; Ries, Ellis, & Hawkins, 1988; Ries, Kaplan, Limberg, & Prewitt, 1995). The studies provide essential guidelines for replicating COPD exercise research for patients with cancer if they are capable of exercise. Studies are required to determine whether exercise—even if not the same level of intensity, duration, or frequency as exercise for patients with COPD—can improve dyspnea in patients with lung cancer.

Energy Conservation and Work Simplification

Activity-induced dyspnea is a common problem in patients with lung disease; therefore, patients need to learn how to conserve energy. Teaching energy-conservation strategies to patients can help them plan their daily activities and maintain their normal lives.

Nurses should help patients learn new ways to perform their usual activities of daily living (ADL). Energy conservation is necessary to help patients maintain independence in performing ADL. Several strategies can conserve energy while patients perform ADL by limiting unnecessary effort and balancing rest and activity (Carriero-Kohlman & Stulberg, 2001; Coppola & Wood, 2001). Patients can limit unnecessary effort by placing a chair in the bathroom for showering and dressing, using no-iron fabrics to avoid ironing clothes, and air-drying dishes rather than towel-drying them. To work in the kitchen, patients should place frequently used items in easy reach and sit on a chair or stool at the kitchen counter. In addition, patients should plan their ADL and allow sufficient time to complete them so as not to feel rushed beyond their limitations. If important activities are scheduled for the afternoon, patients should rest in the morning to conserve energy for accomplishing desired afternoon activities.

Environmental Adjustment

Environmental issues related to dyspnea in patients with lung cancer have not been discussed in the literature. To get enough air, patients with COPD tend to breathe through their mouths (Sexton, 1990) instead of their noses, where air is warmed and humidified before entering the trachea (Beachey, 1998). Nurses should inform patients that breathing cold or dry air directly through the mouth stimulates the irritant receptors that provoke the cough reflex and allergic reactions, which makes dyspnea worse (Des Jardins & Burton, 2002). When exposure to cold or windy weather is unavoidable, patients should protect themselves by wearing a warm coat, scarf, and...
cap. Moreover, breathing dry air (low humidity) through the mouth impairs cilia movement and intensifies mucus-plug formation, causing airway obstruction and dyspnea. In the winter, when heating systems are operating, patients should use a humidifier to maintain optimal humidity (40%) (Sexton).

A cool environment with gentle air movement is the most comfortable environment for patients with dyspnea. Cool air from an open window or an oscillating fan set on a low speed and directed toward a patient’s face may lessen the perception of dyspnea (Rousseau, 1996; Wickham, 1998). Several years ago, studies of the effect of cool air to improve dyspnea were conducted in the COPD population with good results. Studies in this regard have not been performed for patients with lung cancer, but anecdotal evidence supports the use of cool, gently moving air.

Nutrition Management

Researchers have studied the relationship among respiratory muscle function, exercise tolerance, and nutritional depletion in patients with COPD. They have proposed that malnutrition affects respiratory muscle function, exercise performance, and mortality rates (Chapman & Winter, 1996; Landbo, Prescott, Lange, Vestbo, & Almdal, 1999). To prevent debilitation caused by malnutrition, nurses should be aware of nutritional problems and always incorporate nutritional management into nursing care plans.

One cause of decreased dietary intake for patients with COPD is that they consume less food because chewing and swallowing alter their breathing patterns, thereby decreasing SaO2 (Schols & Wouters, 2000). Furthermore, patients may avoid eating or eat less because gastric filling reduces the functional residual capacity in the lungs, which, consequently, causes dyspnea while they are eating. Also, large meals increase energy expenditure, increase carbon dioxide production and oxygen consumption, and limit movement of the diaphragm, which inhibits lung expansion and induces dyspnea (Ryan, Road, Buckley, Ross, & Whittaker, 1993).

The causes of malnutrition in patients with lung cancer may be similar to those in patients with COPD. However, other cancer-related symptoms (e.g., nausea and vomiting caused by chemotherapy) (Cleeland, 2000) and other disease-related symptoms (e.g., loss of appetite, anorexia, diarrhea, pain, taste changes, mucositis) also reduce patients’ ability to consume an adequate diet (Grant & Kravits, 2000; Sheehan & Forman, 1997; Turner, 1992). Large meals and difficulty coordinating breathing with chewing and swallowing are very relevant issues and must be addressed.

Mealt ime and food pattern: Depending on the causes of nutritional depletion, to improve nutritional status, nurses should instruct patients to adjust meal times, simplify food patterns, and balance energy expenditure to reduce the impact of eating on dyspnea. Sexton (1990) suggested that avoiding food that requires a good deal of chewing is one way to help patients’ ability to eat more. Six small, easily digestible meals per day should be better than three larger ones. The consumption of small but frequent meals helps reduce the impact of eating on breathing patterns. Patients also should avoid gas-producing foods because they may cause discomfort when the stomach becomes extended, interferes with the breathing pattern, and, possibly, induces or exacerbates dyspnea.

Calories and nutrients: Adequate caloric intake and sufficient nutrients are a hallmark of preventing dyspnea caused by malnutrition. Nurses should encourage patients to increase caloric intake by eating energy-dense food or supplementing with oral or parenteral nutrition to improve respiratory muscle strength, increase body weight, and increase activity performance (Ganzoni et al., 1994; Wilson, Rogers, Sanders, Pennock, & Reilly, 1986).

A high-calorie diet is necessary to produce energy. However, effects of such a diet may adversely affect breathing patterns. Patients should avoid consuming a high-carbohydrate diet because it induces carbon dioxide production and minute ventilation; thus, more ventilation is required to eliminate carbon dioxide (Covelli, Black, Olsen, & Beekman, 1981; Goldstein et al., 1989). Rather, patients should choose foods rich in fat, which produce less carbon dioxide, thus avoiding hypercapnia and hyperventilation (Akrabawi, Mobarhan, Stoltz, & Ferguson, 1996). However, some researchers have suggested that fat-rich meals require more time for digestion. Thus, consuming fat-rich meals may extend the duration of abdominal distension, which, in turn, affects mobility of the diaphragm, possibly inducing dyspnea (Ferreira, Brooks, Lacasse, & Goldstein, 2000). Thus, recommending high-carbohydrate or high-fat foods should depend on patient comfort. Moderate-fat foods should be chosen compared to high-fat foods, which require more time to digest (Akrabawi et al.). Research investigating the effects of specific nutrients on patients with dyspnea, with or without cancer, was not found.

Mouth breathing and increased sputum production associated with dyspnea commonly are associated with poor appetite; thus, nurses should encourage patients to rinse their mouths frequently to improve oral hygiene. In addition, fluid loss from mouth breathing can increase the risk of dehydration; hence, if no reasons exist to restrict fluids, patients should drink 3 L of fluid per day (Chapman & Winter, 1996).

Relaxation Techniques

Relaxation techniques, including controlled breathing, may help patients control dyspnea and decrease anxiety, thus stopping the vicious cycle of anxiety and dyspnea (Cowcher & Hanks, 1990; Wickham, 1998). Complete muscular relaxation is associated with decreased oxygen consumption, decreased carbon dioxide production, and decreased respiratory rate (Sexton, 1990). Tai chi and yoga are two relaxation techniques that have been suggested for dyspnea management in patients with COPD.

Tai chi involves slow movements and integrates concentration and body movement to enhance muscle relaxation and control breathing. Controlling breathing patterns also is integral to yoga. During yoga, respiratory rate slows and breathing becomes deeper (Kornfeld, 1995). Many authors have suggested the techniques for dyspnea management in patients with COPD, but no studies that examined the effectiveness of the techniques for patients with either COPD or cancer were found. Relaxation can be successful in reducing dyspnea and anxiety, and this finding has been reported in patients with COPD and other patients (Gift, Moore, & Soeken, 1992). Again, patients with lung cancer may try these techniques, particularly when the primary cause of dyspnea is airway obstruction.

Conclusion

The aim of dyspnea management is to alter, whenever possible, underlying causes, reduce the impact of symptoms,
promote independence, and improve quality of life. Management should include integration of pharmacologic and nonpharmacologic treatments.

All of the management strategies discussed in this article have been employed for relief of dyspnea in patients with COPD. The mechanisms associated with dyspnea, whether caused by chronic pulmonary disease or lung cancer, are similar. Thus, research evidence demonstrating the effectiveness of dyspnea management in patients with COPD could be applied to patients with cancer.

Author Contact: Peeranuch Jantarakult, PhD, RN, can be reached at peeranuch_j@payap.ac.th, with copy to editor at rose_mary@earthlink.net.

References


The continuing education examination and test form for the preceding article appear on the following pages.