

Symptom Management of Lung Cancer

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Managing the symptoms of the patient with cancer is key to an overall positive treatment outcome. Supportive interventions for specific side effects of therapy help to ensure safety, survival, and maintenance of a satisfactory quality of life (QOL). Since the late 1980s, much research has gone into the development of supportive measures for the management of disease and treatment-related symptoms. Oncology nurses have partnered with their colleagues in the search for evidence to better manage the care of patients. The result is a growing body of standards and guidelines for the supportive care of people undergoing treatment for cancer.

Symptom management is particularly poignant for those with lung cancer. The majority of patients have some symptoms at the time of diagnosis (Kvale, Simoff, & Prakash, 2003). Although treatment may relieve disease-related symptoms, treatment-related side effects result. As patients move through the continuum of the disease from aggressive therapies to palliative care, the focus of the interventions may change, but the significance of controlling symptoms remains constant. The most commonly occurring symptoms are cough, hemoptysis, dyspnea, fatigue, and pain (Hollen, Gralla, Kris, Eberly, & Potanovich, 1993). This chapter will review these most common symptoms and the related interventions. As most of the treatment side effects from chemotherapy are not specific to lung cancer, we have limited this section to thoracic radiation-induced symptoms.

Cough

Cough is one of the most frequent and distressing symptoms in patients with lung

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cancer. Technically, cough is a protective mechanism that allows people to clear secretions and inhaled particles from the airways; “pathologic cough” is the result of a disease process (McDermott, 2000). Chronic cough is defined as a cough that lasts longer than three weeks (McDermott). In patients who are current or previous smokers, cough that occurs for the first time and lasts for months or cough that changes in character may suggest bronchogenic carcinoma (Irwin et al., 1998). Cough is estimated to occur in up to 65% of patients as the initial manifestation of lung cancer (Kvale et al., 2003). Approximately 70%–90% of patients with lung carcinoma will develop cough at some time during the course of their illness (Irwin et al.). Complications of cough include musculoskeletal pain, rib fractures, hemoptysis, fatigue, and insomnia.

The cough reflex consists of three phases. The first phase occurs with deep inspiration. This is followed by closure of the glottis with a rapid increase in pleural pressure, marking the second phase of the reflex. The final phase involves the opening of the glottis with a rapid release of the pleural pressure (McDermott, 2000). The cough reflex is initiated by stimulation of neural mucosal receptors located within the nasopharynx, larynx, trachea, and bronchial tree (McDermott). The neural mucosal receptors are stimulated by mechanical or chemical irritants, resulting in transmission of impulses via cranial nerves IX and X to the cough center in the medulla. Once the cough center

is stimulated, cough occurs from the forceful contraction of the diaphragm and other expiratory muscles in the chest (McDermott).

Cough from lung cancer is more likely to occur in patients with tumors of the central air-

ways, such as squamous cell carcinoma and small cell lung cancer. Tumors in the central airways can cause obstruction, either from intraluminal growth of tumor or extraluminal compression of the airways, and this almost always leads to dyspnea and cough (Kvale et al., 2003). Adenocarcinoma usually presents as a peripheral lesion and is, therefore, less often associated with cough; however, cough can be caused by tumor involvement of any part of the respiratory tract. Other causes of cough in patients with lung cancer include recent upper respiratory tract infection, pleural or pericardial effusion, radiation pneumonitis, vocal cord paralysis, and aspiration. Other causes of cough that are not related to cancer include asthma, postnasal drip, gastroesophageal reflux disease (GERD), chronic obstructive pulmonary disease (COPD), congestive heart failure (CHF), and medications, such as angiotensin converting enzyme (ACE) inhibitors. Cough may be dry or associated with production of sputum.

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To treat cough adequately, the cause must be determined. A thorough history and physical exam will help to determine the cause. The cough history should include onset, duration, and precipitating factors; presence or absence of sputum production and the color and odor (if any) of sputum; and current medications. The history also should include the presence or absence of any other illnesses or conditions that can cause cough (e.g., asthma, COPD, GERD), occupational or environmental exposure to irritants, and smoking history. Physical exam focuses primarily on the upper and lower respiratory tract. The oropharynx may reveal the presence of mucus, erythema, or a "cobblestone" appearance of the mucosa, which suggests postnasal drip (McDermott, 2000). Examination of the chest and lungs may reveal signs of pleural or pericardial effusion, pneumonia, or airway obstruction. When auscultating the lungs, listen for wheezing or other adventitious sounds and the presence or absence of breath sounds. The presence of stridor on inspiration suggests airway obstruction. Percussion of the chest may reveal areas of dullness, which can be seen with pneumonia or pleural effusion. Jugular venous distension, wet crackles at lung bases, and an S3 gallop suggest CHF. Observation of the respiratory rate, presence or absence of peripheral edema, and vital signs, including temperature, also are important. Diagnostic tests include evaluation of sputum, if present; radiologic tests, such as computed tomography (CT) or chest x-ray; pulmonary function testing; and bronchoscopy.

As noted earlier, treatment of cough consists of treating the underlying cause. Treatment of postnasal drip includes antihistamines, intranasal steroids, and inhalers, such as Atrovent® (3M Pharmaceuticals, St. Paul, MN). Cough from postnasal drip should subside within days to weeks with the above treatment. Cough from GERD may respond to change in lifestyle and addition of H2 antagonists. Bronchodilators and corticosteroids may be helpful in management of cough from COPD. If infection is suspected, a course of antibiotics is indicated. Cough from ACE inhibitors usually occurs within the first few weeks of treatment and resolves with discontinuation of the drug.

If cough is caused by central airway obstruction from tumor, bronchoscopic therapies using a rigid bronchoscope may provide relief. The rigid bronchoscope can be used to examine the airways, place a stent to open an obstructed airway, and facilitate laser resection of obstructing tumor (Kvale et al., 2003). In patients with lung cancer, these procedures usually are considered palliative

and may not entirely relieve cough. Cough from pleural or pericardial effusion may be helped by removal of fluid.

Pharmacotherapy and promotion of comfort is the mainstay of treatment for patients with cough from advanced bronchogenic carcinoma. In patients in whom cough is the presenting symptom from disease in the airway, treatment with chemotherapy may lead to reduction in tumor and, therefore, reduction in cough. In these patients, return of cough often signals progression of disease. Pharmacologic management includes the use of nonopioid cough suppressants, bronchodilators, corticosteroids, and opioids. The most commonly used nonopioid antitussive agent is dextromethorphan; it is available over the counter and comes in pill or liquid form. It often is available in combination with guaifenesin (e.g., Humibid DM® [Adams Laboratories, Ft. Worth, TX], Robitussin® [Wyeth Consumer Healthcare, Madison, NJ]), which is used as both an expectorant and antitussive. These agents are often of little value in patients with cough from advanced cancer. Benzonatate (Tessalon Perles® [Forest Pharmaceuticals, St. Louis, MO]) has been shown to be helpful for some (Doona & Walsh, 1998). Benzonatate is a peripherally acting drug that is available by prescription. The recommended dose is 100–200 mg, three times a day. In patients in whom bronchospasm plays a role in cough, bronchodilator therapy using inhaled ipratropium (Atrovent) has been shown to be helpful. To date, no studies have documented the role of corticosteroids in the management of cough from cancer; however, if cough is related to effects of radiation therapy (e.g., radiation pneumonitis), corticosteroids may be helpful (Kvale et al., 2003).

Opioids are currently the best available treatment for intractable cough from lung cancer. The recent American College of Chest Physicians guidelines recommend the use of opioids in the management of cough (Kvale et al., 2003). The most commonly used opioid is codeine; it is a centrally acting agent. Codeine is available in both tablet and liquid form. Low doses (10–20 mg, every 4–6 hours) are often sufficient for cough suppression (McDermott, 2000). Hydrocodone is also available in tablet or liquid form and is a good alternative to codeine or in patients who cannot tolerate codeine. The lowest effective dose of opioids should be used, and caution is recommended with use of increasing doses, as respiratory depression and hypoventilation can occur.

Other measures, such as deep breathing exercises and effective coughing, may relieve symptoms and can be taught (Ingle,

2000). Patients who smoke cigarettes should be encouraged to stop. Air humidifiers may be helpful in the management of cough in a dry environment. Warm, humidified air decreases the viscosity of secretions, which also can be helpful for cough.

Hemoptysis

Hemoptysis is the expectoration of blood and results from bleeding in the lower respiratory tract. The blood may or may not be mixed with sputum. Hemoptysis is the initial presenting symptom in an estimated 7%–35% of patients with lung cancer. It is estimated to occur in 20% of patients with lung cancer at some point during the course of the illness, and approximately 3% will die of massive hemoptysis (Beckles, Spiro, Colice, & Rudd, 2003; Kvale et al., 2003). The severity of hemoptysis is determined by the amount of blood that is coughed up. Patients who expectorate 100–600 ml of blood in 24 hours are considered to have massive hemoptysis (Kvale et al.). Hemoptysis is one of the most frightening and distressing symptoms associated with lung cancer.

The blood supply to the lungs comes from a dual supply. The pulmonary circulation system contains both arteries and veins, and it serves the function of oxygenation and elimination of carbon dioxide from the body (Guimarães, 2002). The pulmonary artery and its branches supply 95% of the blood and are a low-pressure system (Beers & Berkow, 1999). The bronchial system is a high-pressure system supplying 5% of the blood (mostly to the airways and supporting structures) and originating from the aorta (Beers & Berkow). The normal anatomy of the lung predisposes itself to the symptom of hemoptysis (Henke, 2000). This includes the close anatomic relationship of blood vessels and airways and the presence of the alveolar-capillary membrane (where the exchange of gasses between alveoli and capillaries takes place). Hemoptysis can occur from bleeding at any level in the system but most often occurs from the bronchial system (Henke).

Hemoptysis has multiple noncancerous causes, including infection (e.g., pneumonia, abscess, aspergillosis, tuberculosis), CHF, pulmonary embolism, chronic bronchitis, and pulmonary edema. In patients with lung cancer, hemoptysis can occur as a result of tumor erosion of a blood vessel. A recent retrospective analysis at a tertiary referral hospital documented the diagnosis and severity of hemoptysis in 208 patients (Hirshberg, Biran, Glazer, & Kramer, 1997). Of the 208 patients evaluated, lung cancer was the cause in 39. Bronchiectasis was the most frequent diagnosis, occurring in 41 patients.

Bronchitis and pneumonia occurred in 37 and 33 patients, respectively. The most common primary lung cancer associated with hemoptysis in this review was small cell lung cancer. The majority of patients had moderate hemoptysis caused from bronchitis and lung cancer. Moderate hemoptysis in this study was defined as less than 500 ml of blood expectorated in 24 hours.

Hemoptysis usually is easily distinguished from hematemesis. However, distinguishing hemoptysis from epistaxis (i.e., bleeding from the gums or nasopharynx) can be more difficult, necessitating a careful history (Guimarães, 2002). The history should include the amount and color of the blood; the duration of the bleeding; the presence of clots; the relationship of bleeding to rest or activity; the presence of chest pain, cough, or dyspnea; prior history of heart and lung diseases; and history of cigarette smoking. Additionally, a careful medication history should be ascertained, as aspirin, nonsteroidal anti-inflammatory drugs, and anticoagulant medicines all can cause bleeding. Diagnostic tests include laboratory exams, chest x-ray, CT scan, and bronchoscopy. Laboratory tests should include a complete blood count to determine if anemia is present and check platelet count. Coagulation tests include prothrombin time and partial thromboplastin time. Chest x-ray may reveal pulmonary infiltrates, a cavitary lesion, or atelectasis. A normal chest x-ray does not rule out pulmonary pathology as a source of bleeding (Guimarães). A CT scan or perfusion scanning also may reveal a source of bleeding, such as a pulmonary embolus or air bronchograms. Air bronchograms suggest obstruction, bronchiectasis, and chronic bronchitis, all potential causes of hemoptysis.

The most useful exam in patients who have hemoptysis is bronchoscopy. Bronchoscopy can reveal the source of bleeding and provide the physician with a means of intervention at the time of the procedure. The rigid bronchoscope is preferred, as the lumen is wide enough for suctioning of blood and debris, ventilation of the nonbleeding lung, and use of endoscopic procedures to control bleeding (Guimarães, 2002).

Treatment of hemoptysis depends on the cause of bleeding and the amount of blood that is coughed up. For most patients, hemoptysis stops spontaneously. In those with recurrent hemoptysis or those who expectorate larger amounts of blood, bronchoscopy usually is needed to identify and treat the source of bleeding. Multiple methods of "local control" to stop bleeding from endobronchial lesions are available, and all require an endoscopic procedure.

In patients in whom bronchoscopy reveals visible bleeding but no identifiable lesion, endoscopic measures, such as instillation of epinephrine solution, iced saline solution, and balloons, may be used (Kvale et al., 2003). Balloon tamponade has been shown to be effective in management of massive hemoptysis (Guimarães, 2002).

Electrocautery involves the use of electrical current to produce coagulation and vaporization of bleeding in endobronchial lesions (Prakash, 1999). Disadvantages of electrocautery include endobronchial fire, hemorrhage, and inadvertent shock to the operator or patient (Prakash). Argon plasma coagulation (APC) is a newer modality and a type of noncontact electrocautery (Morice, Ece, Ece, & Keus, 2001). A recently published study utilizing APC for control of hemoptysis and neoplastic airway obstruction showed that immediate, complete control of hemoptysis was accomplished in all patients with no recurrence of hemoptysis for a mean follow-up time of 97 +/- 91.9 days (Morice et al.). Nd:YAG laser is another older type of noncontact electrocautery; photocoagulation has been shown to be helpful in controlling bleeding in approximately 60% of patients (Hetzel & Smith, 1991). Each procedure has its advantages and disadvantages; the newer APC is associated with less risk of endobronchial fire and no risk of retinal injury, compared with Nd:YAG laser (Dumon, Shapshay, & Bourcerau, 1984; Geffin, Shapshay, & Bellack, 1980; Guimarães, 2002).

Photodynamic therapy (PDT) is another newer modality used in the palliative management of hemoptysis (Birn & Kosco, 2004). PDT also requires endoscopy; it uses lasers to activate light-sensitive pharmaceuticals to treat the lesion. PDT requires IV administration of Photofrin® (Sanofi Pharmaceuticals Inc., New York, NY), a photosensitive antineoplastic agent, 40–50 hours before the endoscopic procedure. A nonthermal laser light is used to activate the pharmaceutical agent. Approximately 24–72 hours later, another bronchoscopy is required to remove necrotic debris and perform another treatment if needed. The primary adverse effect of PDT is photosensitivity. Patients need to protect themselves from the sun or bright lights for 4–6 weeks after therapy with PDT. Outdoor activities should occur after sundown, as sunscreens do not provide adequate protection (Birn & Kosco). If an endobronchial lesion is visible and is determined not to be resectable or amenable to one of the aforementioned treatments, a course of external beam radiation therapy is recommended (Kvale et al., 2003).

Conservative management is preferred in patients with advanced cancer and small

amounts of hemoptysis (30–50 ml/day) (Henke, 2000; Ingle, 2000). Because infection is one cause of hemoptysis, a course of oral antibiotics should be started. Additionally, cough suppression with an opioid (codeine) given around the clock will help to minimize irritation. For many patients, hemoptysis can be managed successfully with the above regimen on an outpatient basis.

Although massive hemoptysis is rare, death from massive hemoptysis in patients with bronchogenic carcinoma is estimated at 59%–100% (Corey & Hla, 1987). Surgical management usually is not an option because the majority of patients have advanced disease. Intervention, if undertaken, usually begins with endotracheal intubation to maintain an adequate airway and measures to prevent asphyxiation. Some of the previously described endoscopic procedures have been used with success. Bronchial artery embolization via endoscopy also can be used (Guimarães, 2002). Other emergency and supportive care measures are used, as well. Hospitalization is recommended; the patient should lie with the bleeding lung dependent and in the Trendelenburg position to prevent aspiration of blood into the opposite lung (Guimarães). Blood transfusions, oxygen supplementation, and antitussive agents also may be used as needed. Overall, the prognosis is poor in patients with bronchogenic carcinoma and massive hemoptysis and, as noted, the majority do not survive.

Dyspnea: Prevalence

Dyspnea is a complex and distressing symptom and a difficult clinical problem to manage. It is aligned closely with a primal fear of death by suffocation, and, hence, it evokes a response that begs intervention from patients, caregivers, and health professionals. Ripamonti and Fusco (2002) reported that the prevalence of dyspnea in an advanced cancer population increases from 15%–55.5% at referral to palliative care service to 18%–79% during the last week of life. As expected, dyspnea is more common among patients with lung cancer than the cancer population in general. The prevalence of dyspnea in patients diagnosed with lung cancer ranges from 55%–87% (Dudgeon, Kristjanson, Sloan, Lertzman, & Clement, 2001; Muers & Round, 1993; Smith et al., 2001; Tanaka, Akechi, Okuyama, Nishiwaki, & Uchitomi, 2001).

In a survey of 120 outpatients with stages I–IV lung cancer that evaluated QOL, dyspnea, and the relationship between the variables, 87% of study participants experienced dyspnea. Patients with high dyspnea scores had lower QOL ($p = 0.04$) (Smith et al.,

2001). Dudgeon et al. (2001) evaluated 923 patients with cancer in the outpatient setting to assess the intensity of their dyspnea. They found that 46% of the patients had some shortness of breath. Only 4% of this study's participants had a diagnosis of lung cancer, and, for that subgroup, 84% reported dyspnea. Muers and Round (1993) evaluated the presence and severity of 12 symptoms including dyspnea in a study of 289 patients with non-small cell lung cancer. Subjects were assessed at presentation and every two months for one year or until death. Cough and breathlessness were the two most prevalent symptoms. Breathlessness of any grade was present in 216 patients (75%). Severe breathlessness was present in 8%, moderate breathlessness in 33%, and mild breathlessness in 34% of the same 216 subjects. Lastly, in a study of 157 outpatients with advanced lung cancer, Tanaka et al. (2001) found that 55% of subjects reported "clinical dyspnea," defined as dyspnea interfering with at least one of the following seven categories: work, walking, general activities, sleep (which comprise the physical domain) and mood, relationships, and enjoyment (which comprise the psychological domain). Dyspnea interfered with not only the physical domain (52%) but also with the psychological domain (23%). Although this review is not exhaustive, it indicates that dyspnea is a prevalent symptom in patients with lung cancer; it interferes with QOL and has an impact on functional and emotional status.

Dyspnea: Definition

The term dyspnea generally is applied to the sensations that individuals with unpleasant or uncomfortable respiration experience. The American Thoracic Society (ATS) (1999), in a comprehensive consensus statement, defined dyspnea as a subjective experience of breathing discomfort that consists of qualitatively distinct sensations that vary in intensity. The symptom derives from interactions among multiple physiologic, psychological, social, and environmental factors and may induce secondary physiologic and behavioral responses. This definition stresses the subjective and multifactorial nature of the dyspnea experience.

Dyspnea: Pathophysiology

A unifying theory is that dyspnea results from a disassociation or a mismatch between central respiratory motor activity and incoming afferent information from receptors in the airways, lungs, and chest wall structures. In other words, a mismatch occurs between

the motor command and the mechanical response, which produces a sensation of respiratory discomfort (ATS, 1999).

ATS (1999) classified physiologic causes of dyspnea and alternative targets for treatment as

- Heightened ventilatory demand, as demonstrated when the intensity of dyspnea increases with exertion or exercise.
- Increased impedance or resistance to ventilation as noted when the respiratory effort expended is out of proportion to the resulting level of ventilation. Asthma and COPD can narrow airways, increasing resistance to ventilation.
- Abnormalities of the respiratory muscles such as weakness or mechanical inefficiency. The pressure-generating capacity of the muscles is decreased, creating a mismatch between the central respiratory drive and achieved ventilation. Malnutrition from cancer cachexia reduces both respiratory muscle strength and maximal voluntary ventilation.
- Abnormal central perception of dyspnea caused from increased respiratory drive, as seen with blood gas abnormalities of hypoxia or hypercapnia.

Like pain, dyspnea has an affective component. The stimulus intensity of "just noticeable difference" for shortness of breath may be the same among patients with similar lung pathology; however, the affective component can vary greatly and actually modulate the intensity of the symptom (Carrieri-Kohlman, Gormley, Douglas, Paul, & Stulbarg, 1996). Hence, the threshold perception of dyspnea varies widely with individuals and is related only moderately to the degree of pulmonary dysfunction. Frequently, a discrepancy is found between severity of disease and intensity of breathing discomfort (ATS, 1999). Cognitive variables that have been shown to modify dyspnea include anxiety, depression (Dudley, Martin, & Holmes, 1964; Gift, 1991; Smith et al., 2001), personality (Chetta et al., 1998), and the meaning of the symptom for the person (Cioffi, 1991).

Dyspnea: Etiology

Dyspnea in lung cancer frequently has multiple etiologies. Possible causes of dyspnea in the general cancer population are listed in Figure 9-1. In addition to the effect of the primary lung tumor, a combination of other factors commonly contributes to dyspnea, depending on stage of disease. These include pleural effusion, anemia, cachexia, and underlying COPD. Many factors can converge to cause and contribute to the symptom of dyspnea.

Dyspnea caused directly by cancer

- Pulmonary parenchymal involvement (primary or metastatic)
- Lymphangitic carcinomatosis
- Intrinsic or extrinsic airway obstruction by tumor
- Pleural tumor
- Pleural effusion
- Pericardial effusion
- Ascites
- Hepatomegaly
- Phrenic nerve paralysis
- Multiple tumor microemboli
- Pulmonary leukostasis
- Superior vena cava syndrome

Dyspnea caused indirectly by cancer

- Cachexia
- Electrolyte abnormalities
- Anemia
- Pneumonia
- Pulmonary aspiration
- Pulmonary emboli
- Neurologic paraneoplastic syndromes

Dyspnea from cancer treatment

- Surgery
- Radiation pneumonitis or fibrosis
- Chemotherapy-induced pulmonary toxicity
- Chemotherapy-induced cardiomyopathy
- Radiation-induced pericardial disease

Dyspnea unrelated to cancer

- Chronic obstructive pulmonary disease
- Asthma
- Congestive heart failure
- Interstitial lung disease
- Pneumothorax
- Anxiety
- Chest wall deformity
- Obesity
- Neuromuscular disorders
- Pulmonary vascular disease

FIGURE 9-1. CAUSES OF DYSPNEA IN PATIENTS WITH CANCER

Note. From "Dyspnea in Cancer Patients: Prevalence and Associated Factors," by D.J. Dudgeon, L. Kristjanson, J.A. Sloan, M. Lertzman, and K. Clement, 2001, *Journal of Pain and Symptom Management*, 21, p. 100. Copyright 2001 by Elsevier. Reprinted with permission.

Dyspnea: Assessment

Assessment of dyspnea is a nursing challenge, not only because of its multiple causes but also because of its subjective nature. One of the main problems is the variable intensity of dyspnea according to activity level and time of the day (Bruera & Ripamonti, 1998). Any assessment of dyspnea should attempt to differentiate the intensity or quality of the sensation and the emotional or behavioral response to the discomfort.

Several standardized assessment tools such as the Oxygen Cost Diagram, the

Baseline Dyspnea Index, and the Borg scale exist to measure dyspnea (ATS, 1999). A simple visual analog scale, which consists of a 100-mm line with anchors at each end to indicate the extremes of “not breathless at all” to “very breathless,” can be used. Scoring is accomplished by measuring the distance from the bottom or left of the scale if horizontally oriented to the level indicated by the patient.

The most common method to assess dyspnea in the clinical setting is self-report of the level of activity at which the patient has difficulty breathing. Common activities associated with dyspnea are climbing stairs or walking uphill, walking fast on level ground, and shortness of breath with dressing or talking. Shortness of breath at rest or with no activity is most dire. One potential limitation of this assessment is that because the intensity of dyspnea depends on the rate of work performance, patients may reduce the rate of work performance and, thereby, minimize the reported intensity of the symptom. For example, a person may report the ability to climb a flight of stairs without reporting the frequent rest stops to reduce symptoms.

An evaluation of dyspnea includes a complete history of the symptom, its temporal onset (acute or chronic), descriptors, precipitating and relieving events or activities and associated symptoms, and response to medication or behavioral changes (Ripamonti & Fusco, 2002). A dyspnea-focused physical examination includes complete vital signs (blood pressure, pulse, respiratory rate, and temperature) and observation of respiratory mechanics, such as pursed lip breathing or the use of accessory muscles. Notice the presence of pallor (relative absence of oxyhemoglobin with its characteristic red color) or cyanosis at the fingertips, lips, and oral mucosa. Clubbing of the fingers and toes can be seen in patients with chronic hypoxia. Cardiac assessment includes auscultation of heart sounds, palpation of the central pulses, and observation of jugular venous distention. Lung auscultation is performed to evaluate for absent breath sounds or the presence of rales, rhonchi, wheezes, or a rub. Lung field percussion is performed to locate areas of dullness. Respiratory excursion and fremitus are assessed. Mental status signs of hypoxia include restlessness, anxiety, disorientation, and confusion (Shepherd & Geraci, 1999).

Pertinent basic diagnostic testing includes pulse oximetry at rest and with activity, complete blood count, and chemistry panel. A chest radiograph may be indicated to evaluate for infiltrates, effusions, and pneumothorax as well as heart size and position.

Pulmonary function tests that measure lung volumes and gas diffusion may be helpful to diagnose a reversible airway obstruction or hypoxemia, which can be improved with therapy. A ventilation-perfusion scan can be obtained if pulmonary embolus is suspected. The choice of appropriate diagnostic tests should be guided by the stage of disease, usefulness of the resultant information for therapeutic intervention, and the patient's wishes.

Considering the complex multidimensional nature of dyspnea, differentiating an acute and possibly reversible cause of dyspnea is important. Although dyspnea is usually a progressive complication of the lung cancer trajectory, some patients present with a sudden onset or acute exacerbation of shortness of breath. This could be considered a medical emergency depending on the presenting context and broad differential diagnosis possibilities.

Dyspnea: Treatment

The therapeutic goals in treating dyspnea are to promote patient comfort, increase exercise tolerance, and promote physical and social well-being (Carrieri & Janson-Bjerklie, 1986). Modest alterations in a number of physiologic and psychological variables, as a result of a particular treatment, can culminate in a clinically meaningful reduction in symptoms (ATS, 1999).

The optimal treatment of dyspnea is to treat reversible causes with specific therapies and to use nonspecific or symptomatic therapy to treat irreversible causes. The following therapy options are organized according to categorical causes of dyspnea: lung tumor, cancer therapy, indirect consequence of cancer diagnosis, and nonspecific palliative measures.

Dyspnea Caused by Tumor: If the lung tumor itself is causing shortness of breath, appropriate treatment with surgery, radiation, or chemotherapy will reduce symptoms. Even a minor response to oncologic therapy can improve dyspnea. Airway obstruction can be relieved with tracheobronchial stenting or laser ablation, or it can be palliated with either external beam radiotherapy or brachytherapy (Dudgeon, 2002).

Malignant pleural effusions can compromise respiration in some circumstances. Thoracentesis aimed at removing pleural fluid is beneficial in relieving dyspnea if the lung reexpands. In most instances, the pleural fluid reaccumulates shortly after thoracentesis. If relief is obtained with initial removal of fluid, pleural drainage with a chest tube and instillation of a sclerosing agent, such as talc, can be an effective

method to prevent reaccumulation of pleural fluid and associated shortness of breath (see Table 9-1).

Dyspnea Caused by Therapy: Certain chemotherapy agents or chest radiation can cause either acute or chronic pneumonitis. Corticosteroids, usually prednisone starting at 60–100 mg daily and tapered over days to weeks, are the mainstay of treatment. Occasionally, supportive oxygen and bronchodilators are required (Dudgeon, 2002). Certain chemotherapeutic agents, such as doxorubicin, can cause cardiomyopathy with risk of CHF and shortness of breath. Conventional therapy for CHF and possibly cardiology consultation are indicated.

Dyspnea as an Indirect Consequence of Cancer: Many complications of chronic illness can occur that cause or contribute to dyspnea. Some common situations encountered in lung cancer are pneumonia and anemia. Pneumonia can be treated with adequate antibiotic therapy and lead to relief of dyspnea. If appropriate to the patient's condition, anemia can be resolved with red cell transfusions or erythropoietin therapy. Short- and long-term benefits can be realized with improved function and comfort. Malnutrition, mineral and electrolyte deficiencies, and overall deconditioning also can contribute to dyspnea. Again, depending on the patient's status, attempts to correct these circumstances may improve dyspnea control.

Nonspecific Treatments of Dyspnea: Symptomatic management of dyspnea is based on three main elements: oxygen therapy, pharmacologic therapy, and general support measures and education. Usually a combination of these interventions is employed.

Oxygen Therapy: Patients who are hypoxemic on room air are quite likely to benefit from oxygen therapy (Bruera, deStoutz, Velasco-Leiva, Schoeller, & Hanson, 1993). Most authorities currently recommend oxygen for patients with hypoxic dyspnea, even in the face of increasing hypercapnia, to achieve and maintain an oxygen saturation greater than 88% (Dudgeon, 2002). However, the usefulness of oxygen for management of patients with cancer who have nonhypoxic dyspnea is questioned in the literature (Bruera & Ripamonti, 1998; Dudgeon). Oxygen may acutely reduce exertional dyspnea; however, an individual response to oxygen cannot be predicted with precision. Evidence indicates that oxygen does have beneficial symptomatic effects in COPD and probably CHF, but patients with dyspnea from cancer most frequently have restrictive pulmonary failure and might not respond in the same way (Bruera & Ripamonti).

TABLE 9-1. SYMPTOM MANAGEMENT OF LUNG CANCER

SYMPTOM	CAUSE	SIGNS AND SYMPTOMS	MANAGEMENT
Cough	Airway obstruction by tumor Gastroesophageal reflux disease Postnasal drip Smoking	Frequent and associated with distress Productive or nonproductive	Pharmacologic: Cough suppressants (opioids and nonopioids), corticosteroids, and bronchodilators Air humidifiers, fans Breathing exercises with effective cough instruction Smoking cessation
Hemoptysis	Tumor invasion of vasculature Inflammation from pneumonia, bronchitis, or bronchiectasis Bleeding diathesis Congestive heart failure	Coughed up blood in sputum • Mild: 30–50 ml per day • Moderate: 500 ml in 24 hours • Massive: more than 500 ml in 24 hours	Pharmacologic: Cough suppressants, codeine, antibiotics Bronchoscopy with laser, instillation of epinephrine solution, iced saline, balloon Intubation and palliative care
Dyspnea	Airway obstruction Pleural effusion Anemia Cachexia Underlying chronic obstructive pulmonary disease	Tachypnea Tachycardia O ₂ saturation level low Pursed lip breathing Use of accessory muscles Decreased or absent breath sounds, rales, rhonchi, wheeze, or rub Excursion or fremitus Pallor Cyanosis Clubbing of fingers or toes Activity intolerance Mental status changes	Treat the cause (cancer therapy). • Administer oxygen. • Administer medications such as corticosteroids, bronchodilators, antibiotics, or opioids. • Transfuse with blood products or treat anemia with epoetin alfa. • Drain effusions. • Assess activity tolerance and plan activities. • Teach coping strategies. • Assess and treat anxiety.
Fatigue	Disease: Dyspnea, pain, emotional distress, sleep disturbance, paraneoplastic syndromes Pain medications Treatment (radiation and chemotherapy) Anemia Cachexia and anorexia	Patient complaint Activity intolerance Anxiety, depression Cognitive dysfunction	Pharmacologic: Epoetin alfa, iron supplements, psychostimulants, antidepressants, corticosteroids Behavioral: Activity planning, psychosocial referral, restorative interventions, sleep interventions, dietary restrictions
Pain	Tumor location and nerve involvement (Pancoast tumor or brachial plexus, pleural effusion, or chest wall invasion) Metastatic involvement (back pain related to spinal cord compression, bone pain, headache, or abdominal pain) Treatment-related: Surgery or procedures (incisional), peripheral neuropathies from chemotherapy	Patient complaint Activity intolerance Anxiety, depression	Treat the cause (cancer therapy). • Assess at each visit for location, quality, intensity, relieving and aggravating factors, satisfaction with control. • Administer medications such as opioids, nonsteroidal anti-inflammatory drugs, corticosteroids, diphosphonates, adjuvants. • Manage side effects of medications. • Consider appropriate alternative interventions such as nerve block, massage, acupuncture, or distraction. • Educate patient and caretaker about use of interventions.
Esophagitis	Radiation to lungs	Dysphagia Odynophagia Reflux Epigastric pain	Pharmacologic: Oral analgesics before eating (“magic mouthwash,” Lortab® [Whitby Pharmaceutical, Richmond, VA] Rothwell’s solution), opioids, antifungals Dietary: Soft, bland, high-calorie diet; thick, soft foods and fluids; avoid alcohol and tobacco Behavioral: Use straw to drink, eat slowly, chew completely, cut foods into small pieces, take antacids before eating, maintain hydration.
Pneumonitis	Radiation to lungs	Dyspnea Cough Fever Night sweats Low O ₂ saturation	Pharmacologic: Corticosteroids, antibiotics, bronchodilators, sedatives, oxygen

Airflow over the face and nasal mucosa during oxygen administration may itself ameliorate dyspnea through poorly understood mechanisms involving modulation of afferent information by input from cutaneous nerves. In a similar way, the movement of cool air with a fan has been observed clinically to reduce dyspnea. Stimulation of mechanoreceptors on the face or a decrease in temperature of the facial skin, both mediated through the trigeminal nerve, may alter afferent feedback to the brain and modify the perception of dyspnea (ATS, 1999).

Pharmacologic Therapy: Opioids have been explored as a means to relieve dyspnea presumably because of a known respiratory depressive effect or by altering perceptual sensitivity. Opioids may alleviate dyspnea by blunting perceptual responses so that for a given stimulus, the intensity of respiratory sensation is reduced (ATS, 1999). Strong evidence from a recent meta-analysis supports the use of oral and parenteral opioids to treat dyspnea. Jennings, Davies, Higgins, Gibbs, and Broadley (2002) analyzed 18 double-blind, randomized, placebo-controlled trials of opioids for treatment of dyspnea secondary to any cause. Nine studies involved the use of oral or parenteral opioids, and nine involved nebulized opioids. This systematic review showed a statistically significant positive effect of oral and parenteral opioids on the sensation of breathlessness ($p = 0.0008$).

The intermittent use of opioids (most frequently morphine) is recommended in a population of patients who already are receiving opioids chronically. The optimal dose has not been determined. Allard, Lamontagne, Bernard, and Tremblay (1999) evaluated 25% and 50% of a four-hourly opioid dose and found them equivalent in relieving dyspnea. Dyspnea reduction was relatively greater in patients with initially low and moderate dyspnea intensity. Allard et al. concluded that 25% of the four-hourly dose of an opioid may be sufficient to reduce dyspnea. The use of sustained-released morphine and slow-release morphine has not shown benefit compared to placebo in reducing breathlessness (Dudgeon, 2002; Ripamonti & Fusco, 2002).

Although the use of nebulized opioids to reduce dyspnea may be tempting, because of local action on pulmonary receptors, a meta-analysis failed to show a positive effect of nebulized opioids on the sensation of breathlessness (Jennings et al., 2002). A synthesis of evidence about the use of nebulized opioids to treat dyspnea concluded that although scientific evidence is lacking to support the use, lower level evidence notes a positive effect in individual clinical settings in patients such as those receiving systemic opioids or experiencing dyspnea at

rest (Joyce, McSweeney, Carrieri-Kohlman, & Hawkins, 2004).

Anxiolytics have the potential to relieve dyspnea by depressing hypoxic or hypercapnic respiratory responses as well as by altering the emotional response (ATS, 1999). However, clinical trials to determine the effectiveness of anxiolytics for the treatment of breathlessness have had conflicting results. In some patients, a trial of a benzodiazepine may be reasonable, particularly in those with morbid anxiety or respiratory panic attacks (ATS), but evidence does not support the regular use of benzodiazepines in the management of dyspnea (Bruera & Ripamonti, 1998).

Because 90% of patients who develop lung cancer have a smoking history, a proportion of them may have untreated obstructive airway disease. These patients may benefit from simple bronchodilator therapy. Inhaled beta-2-adrenergic agonists (albuterol), inhaled anticholinergics, and sustained-release theophylline all have been shown to improve dyspnea in patients with stable COPD.

General Supportive Measures: The effects of cognitive, emotional, and behavioral factors on the conscious awareness of the demand to breathe can modulate the perception of dyspnea. Interventions that may assist patients to cope with their dyspnea are breathing retraining, positioning, exercise training, and education about medication use (Carrieri & Janson-Bjerklie, 1986). Pulmonary rehabilitation uses a combination of techniques to decrease energy expenditure and maximize ventilation. The benefit of pursed lip and diaphragmatic breathing retraining may be explained by the decrease in respiratory rate and the increase in tidal volume associated with their use. Positioning, such as leaning forward while sitting, may provide postural relief because of increased efficiency of the diaphragm. Patients themselves devise strategies to minimize energy expenditures. Relaxation training and similar techniques to reduce anxiety have been advocated and are clinically useful. Nurses are in a unique position to educate patients about dyspnea coping strategies. A patient's beliefs in the effectiveness of coping strategies can affect the perception of dyspnea.

Dyspnea: Conclusion

Dyspnea is a complex symptom that requires thorough assessment and attention. Effectively managing dyspnea is a clinical challenge and usually requires a combined approach of various interventions. Evidence is scant about the efficacy of many interventions primarily because of the difficulty of conduct-

ing research trials in the patient with cancer who is experiencing dyspnea. Nonetheless, dyspnea is prevalent in lung cancer and can prompt many healthcare encounters.

References

- Allard, P., Lamontagne, C., Bernard, P., & Tremblay, C. (1999). How effective are supplementary doses of opioids for dyspnea in terminally ill cancer patients? A randomized continuous sequential clinical trial. *Journal of Pain and Symptom Management*, 17, 256–265.
- American Thoracic Society. (1999). Dyspnea mechanisms, assessment and management: A consensus statement. *American Journal of Respiratory and Critical Care Medicine*, 159, 321–340.
- Beckles, M.A., Spiro, S.G., Colice, G.L., & Rudd, R.M. (2003). Initial evaluation of the patient with lung cancer. Symptoms, signs, laboratory tests, and paraneoplastic syndromes. *Chest*, 123(Suppl. 1), 97S–104S.
- Beers, M.H., & Berkow, R. (Eds.). (1999). *The Merck manual, centennial edition*. Whitehouse Station, NJ: Merck Research Laboratories.
- Birn, C.S., & Kosco, P. (2004). Flexible scopes and photodynamic therapy [Continuing education program]. *Nursing Spectrum*. Retrieved January 14, 2004, from <http://nswb.nursing-spectrum.com/ce/ce264.htm>
- Bruera, E., deStoutz, N., Velasco-Leiva, A., Schoeller, T., & Hanson, J. (1993). Effects of oxygen on dyspnea in hypoxaemic terminal-cancer patients. *Lancet*, 342(8862), 13–14.
- Bruera, E., & Ripamonti, C. (1998). Dyspnea in patients with advanced cancer. In A. Berger, R.K. Portenoy, & D.E. Weissman (Eds.), *Principles and practice of supportive oncology* (pp. 295–308). Philadelphia: Lippincott Williams & Wilkins.
- Carrieri, V.K., & Janson-Bjerklie, S. (1986). Dyspnea. In V.K. Carrieri, A. Lindsey, & C.M. West (Eds.), *Pathophysiologic phenomena in nursing human responses to illness* (pp. 191–218). Philadelphia: W.B. Saunders.
- Carrieri-Kohlman, V., Gormley, J.M., Douglas, M.K., Paul, S.M., & Stulbarg, M.S. (1996). Differentiation between dyspnea and its affective components. *Western Journal of Nursing Research*, 18, 626–642.
- Chetta, A., Gerra, G., Foresi, A., Zaimovic, A., DelDonno, M., Chittolini, B., et al. (1998). Personality profiles and breathlessness measurement in outpatients with different grading of asthma. *American Journal of Respiratory and Critical Care Medicine*, 157, 116–122.
- Cioffi, D. (1991). Beyond attentional strategies: A cognitive-perceptual model of somatic interpretation. *Psychological Bulletin*, 109, 25–41.
- Corey, R., & Hla, K.M. (1987). Major and massive hemoptysis: Reassessment of conservative management. *American Journal of Medical Science*, 294, 301–309.
- Doona, M., & Walsh, D. (1998). Benzonatate for opioids-resistant cough in advanced cancer. *Palliative Medicine*, 12, 55–58.
- Dudgeon, D.J. (2002). Managing dyspnea and cough. *Hematology/Oncology Clinics of North America*, 16, 557–577.

- Dudgeon, D.J., Kristjanson, L., Sloan, J.A., Lertzman, M., & Clement, K. (2001). Dyspnea in cancer patients: Prevalence and associated factors. *Journal of Pain and Symptom Management*, 21(2), 95–102.
- Dudley, D.L., Martin, C.J., & Holmes T.H. (1964). Psychophysiologic studies of pulmonary ventilation. *Psychosomatic Medicine*, 26, 645–659.
- Dumon, J.F., Shapshay, S., & Bourcerau, J. (1984). Principles for safety in application of neodymium-YAG laser in bronchology. *Chest*, 86, 163–168.
- Geffin, B., Shapshay, S.M., & Bellack, G.S. (1980). Flammability of endotracheal tubes during ND-YAG laser application in the airway. *Anesthesiology*, 89, 124–128.
- Gift, A. (1991). Psychologic and physiologic aspects of acute dyspnea in asthmatics. *Nursing Research*, 40, 196–199.
- Guimarães, C.A. (2002). Massive hemoptysis. In F.G. Pearson, J.D. Cooper, J. Deslauriers, R.J. Ginsberg, C.A. Hiebert, G.A. Patterson, et al. (Eds.), *Thoracic surgery* (2nd ed., pp. 717–736). New York: Churchill Livingstone.
- Henke, C.S. (2000). Hemoptysis. In D. Camp-Sorrell & R.A. Hawkins (Eds.), *Clinical manual for the oncology advanced practice nurse* (pp. 137–140). Pittsburgh, PA: Oncology Nursing Society.
- Hetzel, M.R., & Smith, S.G. (1991). Endoscopic palliation of tracheobronchial malignancies. *Thorax*, 46, 325–333.
- Hirshberg, B., Biran, I., Glazer, M., & Kramer, M. (1997). Hemoptysis: Etiology, evaluation, and outcome in a tertiary referral hospital. *Chest*, 112, 440–444.
- Hollen, P., Gralla, R., Kris, M., Eberly, S., & Potanovich, L. (1993). Quality of life assessment in individuals with lung cancer: Testing the Lung Cancer Symptom Scale (LCSS). *European Journal of Cancer*, 29A(Suppl. 1), S51–S58.
- Ingle, R.J. (2000). Lung cancers. In C.H. Yarbro, M.H. Frogge, M. Goodman, & S.L. Groenwald (Eds.), *Cancer nursing: Principles and practice* (5th ed., pp. 1298–1328). Sudbury, MA: Jones and Bartlett.
- Irwin, R.S., Boulet, L.P., Cloutier, M.M., Fuller, R., Gold, P.M., Hoffstein, V., et al. (1998). Managing cough as a defense mechanism and as a symptom. A consensus panel report of the American College of Chest Physicians. *Chest*, 114(Suppl. 2), 133S–181S.
- Jennings, A.L., Davies, A.N., Higgins, J.P., Gibbs, J.S., & Broadley, K.E. (2002). A systematic review of the use of opioids in the management of dyspnea. *Thorax*, 57, 939–944.
- Joyce, M., McSweeney, M., Carrieri-Kohlman, V.L., & Hawkins, J. (2004). Evidence synthesis: The use of nebulized opioids in the management of dyspnea. *Oncology Nursing Forum*, 31, 551–561.
- Kvale, P.A., Simoff, M., & Prakash, U.B.S. (2003). Palliative care. *Chest*, 123(Suppl. 1), 284S–311S.
- McDermott, M.K. (2000). Cough. In D. Camp-Sorrell & R.A. Hawkins (Eds.), *Clinical manual for the oncology advanced practice nurse* (pp. 127–130). Pittsburgh: Oncology Nursing Society.
- Morice, R.C., Ece, T., Ece, F., & Keus, L. (2001). Endobronchial argon plasma coagulation for treatment of hemoptysis and neoplastic airway obstruction. *Chest*, 119, 781–787.
- Muers, M.F., & Round, C.E. (1993). Palliation of symptoms in non-small lung cancer: A study by the Yorkshire Regional Cancer Organization thoracic group. *Thorax*, 48, 339–343.
- Prakash, U.B.S. (1999). Advances in bronchoscopic procedures. *Chest*, 116, 1403–1408.
- Ripamonti, C., & Fusco, F. (2002). Respiratory problems in supportive care. *Cancer*, 10, 204–216.
- Shepherd, S., & Geraci, S. (1999). The differential diagnosis of dyspnea: A pathophysiologic approach. *Clinician Reviews*, 9(4), 52–72.
- Smith, E.L., Hann, D.M., Ahles, T.A., Furstenberg, C.T., Mitchell, T.A., Meyer, L., et al. (2001). Dyspnea, anxiety, body consciousness and quality of life in patients with lung cancer. *Journal of Pain and Symptom Management*, 21, 323–329.
- Tanaka, K., Akechi, T., Okuyama, T., Nishiwaki, Y., & Uchitomi, Y. (2001). Prevalence and screening of dyspnea interfering with daily life activities in ambulatory patients with advanced lung cancer. *Journal of Pain and Symptom Management*, 23, 484–489.