Management of Dyspnea in Patients With Far-Advanced Lung Disease

“One Once I Lose It, It’s Kind of Hard to Catch It . . .”

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Severe dyspnea is common among patients with end-stage lung disease but is disabling and challenging to treat. Using interviews with 2 patients with severe dyspnea, one with end-stage chronic obstructive pulmonary disease and the other with lung cancer, Drs Luce and Luce discuss approaches to helping alleviate dyspnea for patients nearing the end of life, and maximizing comfort and function for the patients’ remaining time.

The Patients’ Stories

Mrs D is a 74-year-old woman, followed up for the past 4 years for management of dyspnea secondary to emphysema. Her pulmonologist, Dr M, relates that she has had a substantial active (25 pack-years) and passive (husband smoked 2 packs per day) tobacco exposure. She is thin, with pursed-lip breathing and sternocleidomastoid contractions. Pulmonary function tests demonstrate severe airflow obstruction, air trapping, and marked reduction in diffusing capacity.

Portable oxygen allows her to continue activities outside of the home, including shopping, visiting family, and attending professional football games—her passion. On supplementary oxygen (4 L/min) her symptoms have improved, with increased exercise tolerance and resolution of pedal edema. She uses a variable regimen of inhaled ipratropium bromide, albuterol, and fluticasone propionate, in addition to oral prednisone.

Mrs I is a 65-year-old woman diagnosed with limited-stage small-cell lung cancer in 1995. She was treated with chemotherapy (cisplatin and etoposide) and radiation, with substantial decrease in the size of the tumor. She is now cared for by Dr K, a pulmonary oncologist. In December 1999, she experienced a central nervous system (CNS) recurrence of her cancer and was treated with radiation and corticosteroids. Though her neurologic symptoms improved, she became extremely fatigued and dyspneic upon exertion. Simple activities of daily living are quite difficult, in part due to her shortness of breath, and she has opted to forgo further antineoplastic therapy in favor of home hospice care.

Dyspnea is a common problem among patients with interstitial fibrosis, lung cancer, cystic fibrosis, and chronic obstructive pulmonary disease. The slow but steady progression of such diseases, often punctuated by acute exacerbations or secondary illnesses, can lead to decision-making dilemmas among patients and their caregivers, such as when to accept mechanical ventilation, when to forgo aggressive therapies, and when to make formal end-of-life care plans. Two cases, a 74-year-old woman with dyspnea secondary to emphysema and a 65-year-old woman with recurrent lung cancer and severe exertional fatigue and dyspnea, illustrate how dyspneic patients approaching the end of life can be evaluated and treated. Four management strategies for dyspnea are discussed: reducing ventilatory impedance, reducing ventilatory demand, improving respiratory muscle function, and altering central perception. Physicians should encourage end-stage lung disease patients and their families to discuss issues such as hospitalization and mechanical ventilation, to prepare advance directives, and to participate in a plan to manage their dyspnea.

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I'll depress the patient rather than relieve the anxiety. I try to make clear that the course, while highly variable, is usually inexorable, and that I'm going to be there to make sure they don't suffer.

Mrs I: It [dyspnea] absolutely wears you out because you never know when it's going to hit. You can start to shake. You can get unsteady on your feet, which drives you crazy.

Mrs I's Son: She's not a complainer . . . I think she always feels short of breath, except when she's on the oxygen tank. That relieves it to a great extent. But when she's mobile and not on oxygen, when she's able to walk, literally every 10, 15, 20 feet, she needs to stop and just take a breath.

Dr K (Mrs I's oncologist): For a while we've had some difficulty, mostly dyspnea on exertion. She is quite in tune with her own sensations, and understands how much less she is able to do because of her shortness of breath. We've talked in great detail about the fact that her progressive fatigue and loss of energy are some of the cardinal signs that things are progressing and getting closer to the end.

The word “dyspnea” is derived from the Greek roots dys, meaning difficult, and pneauma, meaning breath.1 The American Thoracic Society (ATS) defines dyspnea as “a subjective experience of breathing discomfort that consists of qualitatively distinct sensations that vary in intensity. The experience derives from interactions among multiple physiological, psychological, social, and environmental factors, and may include secondary physiological and behavioral responses.”2 Some dyspneic patients describe their experience as “difficulty getting enough air” or “shallow breathing.” Others complain of “feeling suffocated.”3

Dyspnea occurs in a variety of disorders and is common among patients with interstitial fibrosis, lung cancer, cystic fibrosis, and especially chronic obstructive pulmonary disease (COPD). Most patients with these lung diseases experience progressively severe shortness of breath as their pulmonary function declines. This downhill course often is punctuated by acute exacerbations caused either by worsening of the patients’ primary disease or by superimposed secondary illnesses such as pneumonia. An exacerbation of COPD in patients with a PaCO2 of 50 mm Hg or more on admission is associated with an in-hospital mortality rate of 11% and a 1-year mortality rate of 43%.4 Most of the patients who survive hospitalization are dyspneic for the rest of their lives, and their quality of life is often poor.5

The inexorable progression of COPD and other disorders leads to a crisis of decision-making for many patients: whether to accept mechanical ventilation or further hospitalization, when to forgo therapies that carry high risk and confer little benefit, and when to make formal plans for end-of-life care. Most patients with far-advanced lung disease prefer comfort care over aggressive interventions.6 Nevertheless, although the course of COPD has been charted,4,5 and although dyspnea in cancer patients has been shown to peak immediately before they die,6 few studies provide clear evidence that the end of life is near for patients with these and other lung diseases or give these patients and their clinicians signposts with which to make the transition from restorative to palliative care.

Despite the lack of signposts and the inherent variability among patients, it seems reasonable to suggest that the transition from restorative to palliative treatment should begin when patients with lung disease are dyspneic at rest, if not earlier. Some of these patients are homebound by this point; others may leave the home occasionally. All should be encouraged to discuss such issues as mechanical ventilation and further hospitalization with their clinicians, to prepare advance directives, and to participate in a plan to manage their dyspnea if they have not already done so.

MECHANISMS

Before dyspnea can be effectively managed, it must be understood pathophysiologically. As described by the ATS, the sensation clearly can originate from central and peripheral chemoreceptors in response to increases in PaCO2 and decreases in PaO2 and pH.2 Yet some patients may feel dyspneic despite normal arterial blood gases, whereas others do not feel dyspneic even though their blood gases show severe abnormalities.7,9 Activation of mechanical receptors in the chest wall and respiratory muscles and vagal receptors in the airways and lungs may contribute to the sensation of breathlessness caused by airflow obstruction or breathing at low lung volumes.10-13 Extrathoracic receptors, including those on the face and in the CNS, also affect the sensation of breathlessness.14-17

The sensation of dyspnea probably results from a mismatch betweenafferent information from these various receptors and respiratory motor activity.18 That is, as input from the receptors increases and the CNS perceives that the respiratory muscles cannot match the inputs and maintain adequate ventilation, dyspnea increases. According to the ATS, this increase may occur when ventilatory impedance increases, as during bronchoconstriction; when ventilatory demand increases, as during exercise; when respiratory muscle function is abnormal, as during lung hyperinflation; and when central perception of dyspnea increases, as during an anxiety attack.3

EVALUATION

Mrs D: I get a little bit short of breath more often now than I did about a year ago. As I move around a lot, walking back and forth, I start getting shortness of breath. Like walking just now to the door without oxygen, I’m real short of breath. It's gotten worse . . . the past month or two.

Dr M: At every visit I assess Mrs D according to how she's feeling, her exercise tolerance, and her ability to do activities of daily living. This past year, her symptoms have increased and her ability to do simple tasks has decreased.

Dyspnea should be evaluated in terms of its characteristics, severity—including effects on functioning—and underlying diagnosis. History-taking is essential: it clarifies what brings the sensation on, what worsens it, and what makes
it better. It also helps distinguish dyspnea from other symptoms, especially pain, fatigue, and weakness. Patients may experience pain with breathing but are not actually breathless. Others are exhausted by effort, although able to breathe adequately. Still others experience anxiety that they interpret as dyspnea. The insights of family members and friends may be useful in understanding patients’ reports of their symptoms; for example, a wife may reveal that her husband is most breathless when they argue.

Patients with COPD have been shown to reliably and reproducibly estimate the extent of physical activity that causes a given level of dyspnea. Because of this, the severity of their dyspnea can be assessed through descriptions of their ability to exercise or perform tasks of daily living. In patients with early COPD, for example, dyspnea may be so mild that it is only experienced after walking several blocks, whereas patients with far-advanced disease may become breathless with minimal activity, such as when they brush their teeth.

Pulmonary function testing may help correlate dyspnea with a physiological parameter such as the forced expiratory volume in 1 second in patients with COPD. Similarly, numerical or visual scoring systems have been used to measure the severity of dyspnea in clinical studies. Yet scoring systems are not superior to patients’ own functional assessment, and neither they nor pulmonary function testing are routinely used in patients with far-advanced disease.

The cause of dyspnea should be determined in all patients if possible. But how aggressively to work up an acute exacerbation of dyspnea in a patient whose underlying lung disease is known depends largely on the stage of the underlying disease. Other factors include the patient’s preference and the clinician’s assessment as to whether a specific finding will result in a specific treatment. For example, the finding of a pleural effusion on chest radiograph could lead to a thoracentesis; this approach might be useful in a patient with COPD who is ambulatory and whose dyspnea can be relieved by drainage of the effusion. Yet performing a chest radiograph and a thoracentesis might be ill advised in a bedridden patient for whom drainage might cause discomfort and not reduce breathlessness.

**Reducing Ventilatory Impedance**

Impedance is resistance to air movement in the lungs. It increases in patients with COPD because their airways are narrowed and their lung elastance is reduced, and in patients with interstitial fibrosis because their lung parenchyma is infiltrated. As described by the ATS, airways obstruction in patients with COPD traditionally has been treated with smoking cessation and pharmacological therapy, which typically includes inhaled β₂-agonists, inhaled anticholinergics, and inhaled and systemic corticosteroids. Inhaled corticosteroids have been shown to reduce dyspnea when given over the long term in patients with COPD. Furthermore, treatment with systemic corticosteroids results in acute improvement in clinical outcomes among patients hospitalized with COPD exacerbations. Nevertheless, neither inhaled nor systemic corticosteroids have been demonstrated to slow the rate of decline in patients’ lung function, and systemic corticosteroids in particular can cause mood changes, muscle weakness, immunosuppression, osteoporosis, and other adverse effects. In light of these effects, systemic corticosteroids should be administered on a trial basis and should not be continued if they burden patients more than they benefit them.

Most patients with COPD can clear secretions by coughing spontaneously. Patients near the end of life who are exhausted by expectorating secretions may benefit from the use of a suction catheter. Suctioning through a tracheotomy may be useful, but tracheotomies rarely should be performed solely to aid secretion clearance, and they do not reduce dyspnea per se. Anticholinergics such as atropine or scopolamine hydrobromide may reduce secretions and can reduce the intensity of a terminal “death rattle.” However, these drugs also may enhance the oral dryness that some patients experience. In such patients, oral wetting agents or aerosolized saline may be preferred.

Lung-volume reduction surgery has been demonstrated to decrease hyperinflation and improve respiratory muscle mechanics in selected patients with COPD. Few patients have been followed up long enough to determine the ideal timing and long-term effects of lung-volume reduction surgery, however, and this intervention probably should not be recommended to patients who are not ambulatory. In patients with lung cancer, interventions such as bronchial stents, brachytherapy, or photodynamic therapy may open collapsed airways and improve lung mechanics. Ventilatory impedance also may be reduced not by only draining pleural effusions but also by preventing their reaccumulation using pleurodesis. The management of malignant pleural effusions has been recently reviewed by the ATS.
but I get up at every commercial. It helps to bring the strength back in my feet and legs, and it helps the breathing a little bit.

Mrs I’s Son: My mother just takes everything at her own pace. And we realized very quickly that she needed to stop, we needed to allow time for her to get from point A to point B. It was a given, a matter-of-fact kind of thing; she just had to stop and catch her breath.

Patients with chronic lung disease have an increased minute ventilation at rest and during exercise. This increase probably results from increased carbon dioxide production, lactic acidosis, and a heightened drive to breathe. Because dyspnea increases in proportion to the need to breathe, reducing ventilatory demand may diminish the sensation of breathlessness. Exercise training using bicycle ergometers and other devices has been shown to improve aerobic capacity, decrease minute ventilation, and relieve dyspnea in some patients with COPD. Such training is most appropriate before COPD is far advanced, of course, and before patients must restrict their exercise.

Exercise training frequently is provided as part of a comprehensive pulmonary rehabilitation program that also includes education, physical and respiratory care instruction, and psychosocial support. The benefits of such programs have been demonstrated in individual studies and have been reviewed recently by the ATS. Patients who cannot attend rehabilitation programs or workshops may benefit from physical and respiratory care consultation at home.

Mechanical vibration of the chest wall inspiratory muscles during inspiration using standard physiotherapy vibrators has been demonstrated to reduce breathing discomfort in patients with far-advanced COPD subjected to experimentally increased hypercapnia. This reduction was achieved without changes in minute ventilation or lung volume, suggesting that vibration either decreases the sense of ventilatory effort or improves the match between afferent information from chest wall or muscle receptors and ventilatory commands from the CNS. Nevertheless, the overall clinical usefulness of chest wall vibration is unclear.

Oxygen

Mrs D: After a while portable oxygen is just something you get used to. It’s kind of hard at first. I just hated to put that thing on when I went outside, and I was embarrassed. But then I thought I have to do it, unless I want to be stuck. My oxygen count is 96, 95 most of the time when I’m sitting with the oxygen on. But I went to the zoo and I felt fine. The fresh air out there, I breathed wonderful.

Mrs I’s Son: My mother is weak and needs to feel like she’s getting enough oxygen, I guess. But we have the portable cart of oxygen that we tow around with us. She prefers to use it, as she’s gotten used to it.

Dr K: At first Mrs I didn’t meet the strict Medicare criteria for home oxygen. Once we initiated hospice she could get oxygen as a comfort measure. We use a prescription that meets her need for oxygen flow and at the same time is comfortable and practical. She uses a nasal cannula at a start rate of 2 to 3 L/min, and can titrate up to 6 L/min. Higher than 8 L tends to be uncomfortable, as it can dry mucosae and patients don’t like the “jet stream” effect in their nostrils.

Supplemental oxygen, used as much as possible around the clock, has been demonstrated to reduce pulmonary vascular resistance and increase survival in severely hypoxemic patients with COPD. Oxygen usually is delivered through nasal prongs but also can be administered through conserving devices such as reservoirs, demand pulse systems, and transtracheal catheters. Face masks also can be used, although they interfere with normal activity and may intensify a sense of suffocation. Oxygen may cause a marked increase in PaCO2, especially in patients with preexisting hypercarbia, either by altering ventilation-perfusion relationships in the lung or by decreasing the drive to breathe. Because of this, the effects of oxygen on arterial blood gases should be monitored at the outset of treatment in patients to whom oxygen is administered to lengthen life.

Medicare reimbursement of long-term oxygen therapy by the Health Care Financing Administration (HCFA) is based on physiological and not symptomatic guidelines (Box). Despite this fact, many patients, including those with far-advanced disease, use oxygen primarily to reduce dyspnea, which it accomplishes physiologically by decreasing ventilatory drive. Although oxygen is not universally successful in relieving breathlessness, it probably deserves a trial in all dyspneic patients who meet HCFA criteria and in dyspneic hospice patients, because their oxygen therapy is reimbursed whether or not they meet HCFA criteria. A reasonable approach is to start oxygen at 1 to 3 L/min during exercise for patients who feel breathless primarily at that time. The same starting point may be used for patients who cannot exercise, with the flow rate titrated upward for symptom relief. Arterial blood gas analysis need not be performed if such relief is the only goal.

The results of titration are difficult to predict, inasmuch as neither the flow rate nor the route of administration of oxygen has been shown to determine its effect on dyspnea in clinical trials. Furthermore, air relieves dyspnea as well as oxygen does in patients with advanced malignancies. This finding suggests that oxygen relieves dyspnea in some patients either through activation of airway receptors or through a placebo effect. In addition, cool air directed against the cheek reduces the sense of dyspnea in healthy subjects made dyspneic by breathing against resistive load, perhaps by activating receptors on the face. Whatever the explanation, breathless patients frequently are relieved by expo-
Opioids

Dr K: Mrs I had some reticence at considering the use of a narcotic. We discussed in detail that we would pick a dose that she could control, and an agent that would not be quite as sedating.

Opioids such as oral codeine and morphine have been demonstrated to acutely increase exercise tolerance and alleviate dyspnea in some patients with COPD, in part by simultaneously reducing minute ventilation and decreasing the sensation of breathlessness. Nevertheless, the long-term administration of sustained-release morphine has not been shown to be superior to placebo in reducing breathlessness in such patients, most of whom reported drowsiness, nausea, constipation, and other adverse effects. Although many patients become tolerant to these effects over time, the generally poor results of opioid administration in research studies of COPD patients suggest that these drugs probably should be used on a trial basis only for patients with recalcitrant dyspnea who do not respond to oxygen. For similar reasons, opioids should be discontinued if they are more burdensome than beneficial. However, because their analgesic properties are undisputed, opioids may be particularly useful in patients such as those with lung cancer who have pain in addition to breathlessness.

Opioids usually are initiated via the oral route, though the sublingual and transdermal routes may be used in patients who are unable to swallow or are too nauseated to reliably absorb medication. Hospitalized terminal patients may be treated with intravenous opioids by either constant infusion or patient-controlled systems. Morphine and codeine are the generally preferred oral opioids because of their cost. Morphine liquid and rapidly dissolving oxycodone hydrochloride are available for patients who cannot swallow tablets. Fentanyl citrate is the only opioid available in transdermal form, and the smallest available dose is sufficiently large that it should not be used in opioid-naive patients because of severe sedation. Clinical experience suggests that relief of dyspnea generally occurs at lower doses of opioids than are required to treat pain, but no data are available on what doses are optimal. Dosing principles and guidelines for the use of opioids may be found in the guidelines for the management of pain of the Agency for Health Care Policy and Research.

Benzodiazepines such as alprazolam and lorazepam have been shown to reduce dyspnea in some patients with COPD because they blunt ventilatory drive and the perception of breathlessness. Yet these agents may cause drowsiness, uncoordination, and dysphoria, and they probably should be used only in patients whose dyspnea is not relieved by oxygen and opioids. Phenothiazines depress ventilation, but their anticholinergic adverse effects may make them less useful than benzodiazepines. Although antidepressants have been shown to reduce dyspnea in 1 small study, their general usefulness has not been defined.

Box. Home Oxygen Therapy: Requirements for Medicare Coverage

Group I (any of the following):
1. $\text{PaO}_2 \leq 55 \text{ mm Hg or } \text{SaO}_2 \leq 88\%$ taken at rest breathing room air, while awake
2. During sleep (prescription for nocturnal oxygen use only):
   a. $\text{PaO}_2 \leq 55 \text{ mm Hg or } \text{SaO}_2 \leq 88\%$ taken during sleep for a patient whose room air $\text{PaO}_2 \geq 56 \text{ mm Hg or } \text{SaO}_2 \geq 89\%$ while awake and resting, OR
   b. Decrease in $\text{PaO}_2 > 10 \text{ mm Hg or decrease in } \text{SaO}_2 > 5\%$ associated with symptoms or signs reasonably attributed to hypoxemia (eg, impaired cognitive processes, nocturnal restlessness, insomnia)
3. During exercise (prescription for oxygen use during exercise only):
   a. $\text{PaO}_2 \leq 55 \text{ mm Hg or } \text{SaO}_2 \leq 88\%$ taken during exercise for a patient whose room air $\text{PaO}_2 \geq 56 \text{ mm Hg or } \text{SaO}_2 \geq 89\%$ while awake and resting, AND
   b. There is evidence that the use of supplemental oxygen during exercise improves the hypoxemia that was demonstrated during exercise while breathing room air

Group II: $\text{PaO}_2 = 56-59 \text{ mm Hg or } \text{SaO}_2 = 89\%$ if there is evidence of any of the following:
1. Dependent edema suggesting congestive heart failure
2. Pulmonary hypertension or cor pulmonale determined by measurement of pulmonary artery pressure, gated blood pool scan, echocardiogram, or P pulmonale on electrocardiogram (P wave > 3 mm in standard leads II, III, or aVF)
3. Hematocrit > 56

*Source: Health Care Financing Administration.
†Patients in this group must have a second oxygen test 3 months after the initial oxygen set-up.

Improving Respiratory Muscle Function

Because dyspnea probably results from an imbalance between afferent inputs and respiratory muscle output, increasing muscle strength and endurance or resting the muscles may reduce the sensation. Supplemental oral nutrition has been demonstrated to increase body weight, respiratory and other muscle strength, and to decrease breathlessness in selected patients with COPD. Supple-

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advanced lung disease who cannot consume regular food because of dyspnea. There is no evidence that cachexia can be reversed by usual dietary supplements, however. Furthermore, of the drugs commonly prescribed to increase appetite, the progestational agent megestrol acetate may enhance the sensation of breathlessness by increasing the drive to breathe.

Handheld devices that require patients to inspire through a resistance have been used to strengthen the inspiratory muscle in patients with COPD. Although some trials have suggested that inspiratory muscle training can reduce dyspnea in some patients, a meta-analysis showed little benefit overall. Inspiratory muscle training is not appropriate in patients with far-advanced COPD who may have difficulty manipulating the devices and become even more dyspneic in doing so.

Although patients with far-advanced lung disease generally are poor candidates for long-term intubation and mechanical ventilation, partial mechanical ventilatory support can help stabilize patients at home. Negative pressure ventilation administered with a body wrap is difficult to use and has been shown to be generally ineffective in treating dyspnea. Domiciliary noninvasive positive pressure ventilation, on the other hand, has been demonstrated to improve exercise tolerance and quality of life when combined with physical training in selected patients with advanced COPD. Patients who are not being ventilated may benefit from mechanical beds to support head elevation and probably should be encouraged to sit in a forward-leaning position, which improves the efficiency of the diaphragm.

**Altering Central Perception**

Many of the benefits of psychoactive drugs and pulmonary rehabilitation probably result from their effects on the perception of dyspnea in the CNS. For example, exercise training has been shown to not only increase aerobic capacity and to decrease minute ventilation but also to desensitize patients to dyspnea, perhaps by lessening their fear that the sensation itself is lethal. As noted above, rehabilitation programs frequently combine education, psychosocial support, and various behavioral approaches with exercise training. These same approaches can be used in the home, where family and group support, along with close relationships with health care providers, can help patients feel that they need not endure breathlessness on their own.

**Terminal Care**

**Dr K:** We’ve handled her switch to hospice and her family has been absolutely outstanding. When I visit, there are usually grandkids running around. As difficult a situation as it is, this maintains a certain normalcy for her, which is soothing.

**MRS I:** I’m using oxygen and I husband my energy as much as possible. I plan one thing a day. I’m more comfortable at home. I let the kids do whatever they can to make me comfortable.

During the final days and hours of life, the emphasis of care should be palliative. Patients who are dying, at home or in hospice, do not benefit from exercise training and other rehabilitative measures that may have helped earlier, and they should be encouraged to use a wheelchair and to rest. If they have not stopped smoking, they should not be pressured to stop at this time. Drugs that are intended to treat the underlying disease without relieving symptoms or are burdensome should be withdrawn. Oxygen, opioids, and perhaps benzodiazepines given to relieve dyspnea should be continued, at higher doses if necessary, assuming these therapies have been found to reduce distress. Routes of opioid and benzodiazepine administration may be shifted from oral to sublingual, rectal, transdermal, or, rarely, subcutaneous.

Opioids and benzodiazepines may depress ventilation significantly when given in high doses. Nevertheless, clinicians should be aware that administration of these drugs even to the point of terminal sedation is both ethically and legally sanctioned under the principle of double effect, provided that they are given with the intent of relieving suffering and not of hastening death. Clinicians also should remember that patients may prefer to be awake at the time of death, and that pharmacotherapy may not be an adequate substitute for the support of friends, family, and caregivers at the bedside.

**CONCLUSIONS**

Throughout the evolution of their illness and at the end of life, patients with lung disease may be comforted by the knowledge that clinicians have numerous tools with which to treat dyspnea. Indeed, patients and their families should be assured that the patients need not experience a painful and suffocating death when their lung disease is far advanced. That said, medical science knows far too little about dyspnea and how to treat this symptom. Research is sorely needed on how best to relieve breathlessness throughout the course of lung disease and especially at the end of life.

**REFERENCES**


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Resources

Agency for Healthcare Policy and Research
http://www.ahcpr.gov/clinic/cpgonline.htm
Clinical guidelines for smoking cessation, management of cancer pain, and other topics.

American Cancer Society
http://www.cancer.org
Patient-oriented pages on living with cancer, smoking cessation, and treatment.

American Thoracic Society
http://www.thoracic.org
Online journals, abstracts and meeting notices, and selected publicity of new research.

Clinical Digital Libraries Project: Respiratory Disorders Clinical Resources
http://www.slis.ua.edu/cdlp/webdcore/clinical/pulmonology/index.htm
References to clinical and research sites.

End-of-Life Physician Education Resource Center
http://www.eperc.mcw.edu
Online peer-reviewed information about instructional and evaluation materials (eg, lectures, small-group exercises, slide sets, videotapes, self-study guides, assessment tools) focused on the end of life.

National Cancer Institute
http://rex.nci.nih.gov
News and information for cancer patients, the public, and the mass media on many of NCI’s programs and resources. Site managed by the Office of Cancer Communications.

National Cancer Institute: CancerNet
http://cancernet.nci.nih.gov/index.html
Publications to order, links to other sites, and comprehensive information regarding etiology of all types of cancer; support, resources, reference materials, ongoing trials, and related news.