Management of Dyspnea at the End of Life: Relief for Patients and Surgeons

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I first met John, a 72-year-old retired professor, in the trauma room where he was brought after a motor vehicle crash while driving to his physician's office. He was in severe respiratory distress with obvious chest trauma, multiple fractures, and an oxygen saturation of 86% on oxygen mask. He was intubated and placed on mechanical ventilation with minimal improvement in his oxygen saturation. His workup revealed multiple rib fractures, pneumothorax, pulmonary contusion, and traumatic brain injury. I spoke with his family during his resuscitation when they told me of his severe chronic obstructive pulmonary disease. He had been intermittently on steroids and home oxygen during the last 6 months. I was dismayed to hear the news, because I could already foresee a long complicated intensive care course for John if he survived, with a high likelihood that he would become ventilator dependent.

John's family mentioned an advance directive, which stated his wishes that he would not want to be ventilator dependent at the end of his life. During the first few days of his hospitalization I hoped that some of his pulmonary injury would be treatable and reversible, allowing him to be weaned from the ventilator. But after 10 days there was little progress or improvement. He became hypoxemic and tachypneic when the ventilator was weaned. I discussed John's condition with his family again, and they brought the advance directive; John's wishes were clear, he would not want a prolonged course on mechanical life support. His primary care physician concurred that he and John had several discussions about mechanical ventilation for his pulmonary disease and that he would not want to be ventilator dependent.

After a family conference in which consensus was reached unanimously, John's ventilatory support was withdrawn after intravenous administration of lorazepam and morphine. To everyone's surprise, John did not die right away. He lingered in the intensive care unit for 2 days in a comatose state with labored, spontaneous breathing. While all those concerned recognized John's wishes were appropriately honored by withdrawal of the ventilatory support, they became increasingly distraught. "Doctor, is he suffocating off the respirator? I can't watch him suffer like that. Can't you do something?" I was conflicted. It was difficult to tell whether he was suffering from suffocation. He did have fast labored breathing, but he was also comatose and therefore unaware of discomfort. Should I treat him with high-dose morphine just in case, and make the respiratory signs go away, even if it may hasten his death? If that happens, will I have performed euthanasia?

As surgeons, many of us have felt uncomfortable in this clinical situation. Many physicians who care for the dying believe that dyspnea is the most distressing symptom at the end of life. Dyspnea is one of the most terrifying symptoms of advanced illness for patients and families. The sensation of breathlessness is not only frightening in its own right but also conjures up images of suffocation, drowning, or smothering. To compound this fear, many physicians misunderstand the symptom of breathlessness and its management or have their own fears that its treatment will hasten death or deterioration of function. But dyspnea is treatable, as are other symptoms at the end of life and physician expertise in its management can go a long way to provide comfort and allay patient and family fears. What is the mechanism of breathlessness and how does one assess the symptom? What is the appropriate management of dyspnea not only for the pa-
Dyspnea is a subjective sensation of inability to catch one's breath or an uncomfortable awareness of breathing. It is a common symptom at the end of life with reported prevalence of 21% to 70%, depending on the series.\textsuperscript{2,3} Clearly, patients with COPD, congestive heart failure, and lung cancer are most likely to experience dyspnea, but in the National Hospice Study patients without underlying pulmonary disease accounted for 25% of those experiencing breathlessness.\textsuperscript{4} Dyspnea may accelerate or worsen as death approaches, particularly in patients without pulmonary disease.

Dyspnea is a symptom perceived by the patient; there are no physiologic or physical signs that indicate the level of dyspnea. Distress from breathlessness is not well correlated with the degree of hypoxemia or hypercarbia on arterial blood gases, airway obstruction on pulmonary function tests, nor the objective measures on physical exam such as cyanosis and tachypnea usually used to assess respiratory distress. Multiple studies have shown that what onlookers or caregivers perceive as distressing, ie, labored rapid breathing may not be distressing to the patient. The corollary is also true: patients without physical signs of shortness of breath or blood gases with hypoxemia may experience severe subjective dyspnea.

The pathophysiology of dyspnea is complex and not well understood, and together with its subjective nature makes its successful treatment hard to assess. Psychological and social factors interact with physiologic ones to generate a sensation of breathlessness; the relative contribution of each of these factors is difficult to discern in any individual. Anxiety may be a more important cause of dyspnea in some people than hypoxemia.

Dyspnea is caused by a cortical perception of stimulation of the respiratory center in the brain. Central and peripheral chemoreceptors detect hypoxia or hypercarbia, which, in turn, stimulate ventilation. But because many patients who are tachypneic from increased ventilatory demand do not experience dyspnea, this may not be the primary mechanism. Probably the mechanoreceptors in the lung and chest wall are more important in the pathophysiology. These detect changes in lung volume, stretch, and ratio of pressure generated by respiratory muscles to its maximum potential pressure.\textsuperscript{5} This may explain why dying patients experience dyspnea in the absence of hypoxemia or lung disease, because progressive muscle weakness (from cachexia, malnutrition, inanition) requires a proportionally greater workload of breathing to maintain homeostasis. There are also receptors on the face from the fifth cranial nerve that can generate a sensation of dyspnea. So the major pathophysiologic components of dyspnea can be divided into three main paradigms. A perceived increase in respiratory effort or work of breathing to overcome a pulmonary load may be seen in patients with airway obstructive disease or a large pleural effusion. An increase in the proportion of chest wall strength and respiratory muscles required to maintain homeostasis is the mechanism found in patients with neuromuscular diseases and cancer cachexia. Last, an increase in ventilatory requirements, minute volume due to exercise, sepsis, anemia, acidosis, or hypoxemia can cause dyspnea.\textsuperscript{6}

Assessment

Many patients may report significant dyspnea in the absence of physical signs of distress or pulmonary function abnormalities; conversely, patients with profound physiologic derangements by objective measures may deny a sense of dyspnea. This, coupled with a complex and mysterious pathophysiology as described above, make the assessment of patients with dyspnea difficult. Because it is a subjective symptom, its assessment is best based on patient report or visual analogue scales, similar to pain scales. In patients who are unconscious, no subjective information is available and assessment becomes more problematic based on physical signs such as tachypnea, tachycardia, grimacing, and agitation, which can be poor proxies for dyspnea. Several authors have reported qualitative differences in the “language of dyspnea”\textsuperscript{5,6} as described by patients, depending on the underlying cause for distress. Asthmatics tend to describe dyspnea as “chest tightness,” and those with obstructive pulmonary disease report an inability to “get a deep breath.”

There are many scales for dyspnea assessment but few have been validated across different patient groups with different diagnoses. The majority have been developed for those with COPD or asthma, and cannot necessarily
be extended to terminally ill patients with cancer or multiple organ failure, for example. Functional scales that describe the occurrence of dyspnea based on activity are useful: Level 1 is no dyspnea; Level 2 is dyspnea on vigorous exertion such as climbing stairs; Level 3 is dyspnea with routine ambulation, and so on. Visual analogue scales are also widely used.7

Assessment should be made first for the intensity, duration, and incidence of dyspnea. Dyspnea may occur only with exercise, and the patient may limit exertion to avoid any dyspnea, to the extent that he or she does virtually no activity but does not report dyspnea as distressing. This can be circumvented by assessing the patient first at rest and then with some programmed activity, such as walking or repetitive arm motion if the patient is bedridden. This same maneuver can then be used to assess the therapeutic effect of any medications on dyspnea, with the patient as his or her own control for this “trial.” Because dyspnea is subjective and varies spontaneously for each patient this “N of 1” trial is very useful to titrate opioids for therapy.8 A search for a treatable underlying cause should be done, even if the patient is terminally ill or in the last weeks of life, because several conditions such as pleural effusion, pericardial effusion, and bronchial obstruction may be amenable to specific surgical therapy with minimal burdens and good symptomatic improvement. A study of dyspnea in cancer patients found that the most common treatable causes of dyspnea were bronchospasm, hypoxia, and anemia.9 Many patients may have several underlying causes for their dyspnea, and their relative importance to symptoms may change over time, requiring reassessment. Diagnostic tests directed at treatable causes are effective. Chest radiographs, CT scans, and ultrasonography cause minimal discomfort and may yield new effusions, pneumonia, bronchial collapse. On the other hand, repeated pulmonary function tests, saturation monitoring, and arterial blood gases are not correlated with the degree of dyspnea nor do they provide information on underlying causes or diagnoses, and should be avoided.

The role of cultural and psychological factors should not be underestimated in assessing dyspnea in any patient. Fear and anxiety of choking, of not being able to catch one’s breath, of dying alone by suffocation, or of abandonment may be the salient features of dyspnea for some. Some may severely limit their activity, even going to the bathroom at night for fear of uncontrolled breathlessness and no obvious source of help. Loss of air may be significant for the family either culturally or historically. One only has to hear families describe their harrowing experience watching one parent die of lung cancer or pulmonary disease to see that their greatest fear may be for the other parent to succumb to the same. This sort of anxiety can contribute to the patient’s dyspnea.

Management: Treatment of underlying causes
Management of dyspnea should first be directed at treatable underlying causes. Although the majority of patients will not have such a cause, certainly, at the initial assessment or if there is a significant change in symptoms, they should be investigated. Surgeons are often called to perform procedures for palliation of dyspnea; the syndromes most amenable to surgical therapy are pleural effusion, pericardial tamponade, and endobronchial obstruction. The judicious use of surgery in these situations can improve symptoms greatly. The most common of these is pleural effusion. Because dyspnea may be determined to a large extent by stretch of mechanoreceptors in the pleural cavity and chest wall, the presence of pleural effusion is a very important cause of dyspnea. This sense of breathlessness is a direct result of the fluid in the pleural cavity, not of the associated physiologic abnormalities in gas exchange. These, in fact, may not be apparent; significant dyspnea can be present in the absence of serious hypoxemia or hypercarbia. For this reason, the existence of a pleural effusion should always be sought when a change in dyspnea occurs. Pleural effusion can be treated by simple thoracentesis and aspiration of the fluid. Thoracentesis is relatively painless, can be performed in virtually any setting, and can be repeated, but it carries a small risk of pneumothorax and lung puncture. Chronic indwelling thoracentesis catheters for drainage that can be used in hospice or home settings have been described, although they increase the risk of infection.10 Reaccumulation of fluid is usually a concern in the majority of patients, and because many patients with malignant effusions have a life expectancy of 6 to 9 months, a more definitive procedure may provide better palliation. Options include tube thoracostomy with or without pleurodesis, video-assisted thoracoscopy with pleurodesis, and pleuroperitoneal shunts. Tube thoracostomy can be painful, may limit activity, and patients generally must remain in a hospital inpatient setting. Video-assisted thoracostomy is well de-
scribed for pleurodesis. It allows for a more complete pleurodesis with lower recurrent effusion rates, excellent palliation of dyspnea, and a less than 3% mortality and morbidity rate. It must be performed under anesthesia. All these factors should be weighed along with the goals of care for each patient when recommending these procedures.

Pericardial tamponade is a less common cause of dyspnea but should be considered in the assessment of dyspnea in patients with a history of lung, breast cancer, and chronic renal failure. The reduction of cardiac output leads to a sensation of breathlessness, in the absence of hypoxemia. Relief of tamponade will generally yield an immediate improvement in symptoms unless other causes of dyspnea are present. Pericardial aspiration of fluid is helpful but fluid generally reaccumulates. Catheters percutaneously placed under ultrasonographic guidance are described, with some relief of symptoms, but infection is a risk and longterm palliation is unusual. Consideration for a pericardial window should be made, but selection of patients should be based on the likelihood of symptomatic improvement versus the risk and burden of anesthesia and surgery. For many the latter outweighs the former.

Bronchial obstruction in patients with Stage III lung cancer can cause significant symptoms of dyspnea, hemoptysis, and cough. Survival may be calculated in months, so effective palliation can provide important comfort for this terminal period. Multiple modalities are available for relief of obstruction: endobronchial brachytherapy, ablation with laser, cautery, cryotherapy or photodynamics, and insertion of endobronchial stents. Endobronchial brachytherapy with or without external beam radiation provides excellent palliation. Palliation rates of 80% for dyspnea and 95% for hemoptysis are reported in several series. Surgical ablative therapies for endobronchial lesions using laser, cautery, etc, report palliation of dyspnea in 70% of patients. All these modalities must be performed by rigid bronchoscopy, usually with general anesthesia.

Bronchial obstruction from extrinsic tumor can be treated by endobronchial stents. A variety of stents are available, including silicone, self-expanding wire stents, and covered wall stents. Again bronchoscopy is required for insertion. Although palliation rates are high initially, complications of stent migration, obstruction, and bleeding occur over long periods.

Management: Symptomatic therapy
Despite the above syndromes, the majority of dyspnea is not amenable to specific therapies directed at underlying cause. The therapeutic goal is aimed at ameliorating the sensation of breathlessness; for this symptomatic relief, oxygen, opioids, and benzodiazepines are the mainstays of therapy. The EPEC curriculum (Education for Physicians on End of Life Care) describes this multimodality approach with both pharmacologic and nonpharmacologic therapies.

Oxygen
Patients who are dyspneic with demonstrated hypoxemia may benefit from oxygen, but the majority of patients with dyspnea do not have low oxygen saturation. Oxygen is a powerful symbol of medical care that is probably more important than its actual therapeutic value in the relief of dyspnea. Many patients with dyspnea feel better with oxygen therapy even when hypoxemia is not present, or when no demonstrable improvement in oxygen saturation can be documented. Again, levels of hypoxemia do not correlate with dyspnea so monitoring of oxygen saturation or arterial blood gases is not useful in assessing whether oxygen works.

Some clinical series in patients with dyspnea imply that oxygen likely has a strong placebo effect. Interestingly, a similar benefit can be had from a fan blowing cool air across the face; this is probably related to fifth cranial nerve receptors in the face that feed back to the cerebral cortex. Multiple randomized trials have shown that oxygen therapy relieves dyspnea in COPD patients, particularly dyspnea with exercise. Its role in dyspnea at rest is less clear. Because the goal is to relieve the symptom of dyspnea, regardless of hypoxemia, a trial of oxygen therapy should be used for each patient. It is easy to use, causes minimal discomfort, and its symbolic role can go a long way in providing comfort in intangible ways. To assess its efficacy, the method of trial with “N of 1” as described by Bruera and colleagues is very useful. The patient is his or her own control during alternating trials of oxygen versus room air, while subjective reports of dyspnea are obtained. This can be done at rest or on exertion, as oxygen may only be needed with exercise. If the benefit of oxygen over room air is unclear, the use of fans, cool air, open window, and relaxation techniques should be explored. Oxygen, while easy to use, is expensive and may be burdensome at home for some.
**Opioids**

Opioids are the primary pharmacologic therapy for dyspnea. Just as the pathophysiology of dyspnea is complex and poorly understood, so is the mechanism of opioid relief of dyspnea. The most important mechanism is likely the suppression of the central ventilatory drive in response to rising CO₂ levels, or the suppression of the cortical sensation of rising CO₂. However, studies have shown that arterial partial pressure of CO₂ does not necessarily change significantly in patients receiving morphine, and it is now clear that breathlessness is not solely based on central perception of hypercapnia. Opioids are known to bind to receptors in the airways themselves, but there is little clinical evidence that this mediates their relief of dyspnea. Opioids clearly have important central effects of analgesia and euphoria that palliate dyspnea. They have other indirect effects on cardiopulmonary physiology that are beneficial, such as increased venous capacitance, decreased venous return and cardiac congestion, increased pulmonary vasodilation, decreased pulmonary hypertension, and decreased metabolic rate and oxygen consumption. Many of these mechanisms interact in the dyspneic patient to alleviate breathlessness.

Regardless of the mechanism of action of opioids in treating dyspnea, multiple randomized clinical trials have shown their efficacy for this symptom. The majority of these were conducted in patients with chronic end-stage lung disease, although several reports in advanced cancer confirm its therapeutic role. Opioids can be prescribed through any administration route: cutaneous patch, subcutaneous, parenteral, oral, inhaled, or per rectum. The dosing schedule should be directed at the occurrence of dyspnea, which is most often intermittent. Intensity of breathlessness can change spontaneously unrelated to apparent factors, or increase related to exertion. Contrary to pain management, where long-acting regular medication dosing works best, dyspnea should be treated with intermittent dosing. Opioids can be administered 30 minutes before anticipated activity or dyspnea for the best effect. Continuous morphine for dyspnea is effective, but in studies on cancer patients, led to increases in sedation that were not desirable, as well as to increases in CO₂ levels. Studies on intermittent dosing of morphine found fewer problems with sedation and high CO₂, but adequate relief of dyspnea. But some patients have chronic, constant breathlessness and for them constant infusions or around the clock opioids are more effective. In patients who are comatose, where sedating effects are not an issue, continuous infusions are also recommended.

Selection of an opioid for dyspnea is based primarily on the dosing route preferred. The majority of studies in chronic dyspnea have been in COPD patients; here oral formulations of codeine, hydromorphone, or hydrocodone have been described as efficacious. For patients who are opiate naïve hydrocodone 5 mg q 4 hours, or codeine 30 mg q 4 hours is useful for mild dyspnea. A breakthrough dose can be repeated every 2 hours. For severe dyspnea morphine 5 to 15 mg liquid or tablets, oxycodone 5 to 10 mg q 4 hours is used. For patients already receiving opioids for pain on a fixed-dose schedule, additional medication equal to 50% of the basal dose can be added every hour. A short-acting opioid such as morphine is preferred rather than long-acting slow-release formula, so that rapid titration can be done based on hourly assessments. For critically ill patients or those who have withdrawal of life support, parenteral or subcutaneous bolus followed by infusions of morphine or fentanyl are recommended.

**Benzodiazepines**

Because fear and anxiety can be distressing components of the sense of breathlessness, judicious use of benzodiazepines can alleviate suffering. Unfortunately, several randomized trials looking at the symptom relief of benzodiazepines have yielded contrary results. These trials were primarily in patients with obstructive pulmonary disease who reported little improvement in dyspnea after benzodiazepines versus placebo. Nevertheless many palliative care practitioners recommend an anxiolytic. These should be started at very low doses with frequent repeat administration to titrate to dyspnea reduction. Once an effective dose is reached it should be given every 4 to 6 hours. Again, a variety of dosing routes are appropriate such as oral, sublingual, or parenteral, depending on the patient and setting.

**Nonpharmacologic therapy**

There are many nonpharmacologic modalities that are useful for the treatment of dyspnea. Because anxiety can be a large component of dyspnea, efforts to minimize anxiety-producing factors in the environment can go a long way in providing comfort. Fans, open windows, and avoidance of clutter with machines or beeping of monitoring devices help to decrease the sense of breathlessness. Anxiety of family members and caregivers...
around suffocation and choking can exacerbate the sense of dyspnea for the patient. Time taken to educate and support the family regarding their reactions around the patient’s tachypnea and labored breathing is time well spent, as often these observed signs are not distressing to the patient unless they become anxious because others appear so. Some have recommended relaxation techniques, hypnosis, massage, and acupuncture as therapy for dyspnea. The patient’s report of dyspnea offers an opportunity to explore fears related to the illness or other aspects of the patient’s life. This kind of discussion can not only be therapeutic for dyspnea but also preparation for other future developments.

Dyspnea after withdrawal of the ventilator: Palliation and the “double effect”

Management of dyspnea after the withdrawal of the ventilator deserves special consideration. Although this therapy involves the same modalities of oxygen, opioids, and benzodiazepines, this setting is usually highly charged both for the patient, family, and physicians because of the perceived conflict of palliation and euthanasia.

Withdrawal of life support, especially withdrawal of ventilatory support, has become common practice in the terminal care of the critically ill patients. In intensive care units, the majority of the patients who die do not receive cardiopulmonary resuscitation, but instead undergo a process of limited life support as death becomes likely. In lieu of continued suffering from futile medical intervention, the patient’s proxy consents to withdrawal of treatment, anticipating a peaceful end to what has often been a tumultuous clinical course. As in the case presented here, few bedside scenes evoke greater consternation than the sight of a dying patient in apparent respiratory distress trying unsuccessfully to catch adequate breath after the ventilator is removed. Such a scenario is brief in the passing of a patient whose death is imminent, but it can present a major challenge in a patient who has even the slightest degree of consciousness and lingers much longer than anticipated. The proxy or the family become increasingly frustrated with each passing hour—or days or weeks. As the surgeon feels the mounting pressure from the patient’s proxy or family to “do something” short of euthanasia, the apparent respiratory distress after withdrawal of life support becomes a paramount issue. Many surgeons are concerned that adequate treatment of symptoms with opioids and benzodiazepines may hasten death and may question the ethical basis for such treatment. From a practical standpoint, when titrated to symptom relief, administration of high-dose opioid should not lead to accelerated death after withdrawal of life support. In a prospective study evaluating why and how sedatives and analgesics were administered during withdrawal of life support, there was little evidence that the medication actually hastened death. The surgeon should find comfort in the knowledge that when opioid dose is titrated to the patient’s respiratory signs, acute and direct respiratory demise is rare. In a study of cancer patients treated for pain, no respiratory demise occurred despite the average maintenance dose of 20 mg/hour, up to as high as 360 mg/hour of intravenous morphine. Several excellent protocols detailing procedures, dosing recommendations for symptom relief after withdrawal of the ventilator can be found in Education for Physicians on End-of-Life Care (EPEC) and End of Life Physician Education Resource Center (EPERC).

First the goal of care should be clarified. If the patient is conscious and able to report the symptom of dyspnea, this should be used to assess therapy for comfort. Complete sedation may not be desirable for this patient, and control of dyspnea should be balanced with the goal of alertness and ability to communicate. Nonpharmacologic modalities such as a fan, cool air, open window, and massage may be very useful here to minimize sedating medication requirements. More often in this situation the patient is comatose and the therapeutic goal is to relieve observed distress such as tachypnea and labored breathing as witnessed by the family and staff. A respiratory rate less than 30 and an absence of agitation, restlessness, and grimacing should be sought.

The starting dose and maintenance dose should be determined with the patient’s opioid history in mind. A relatively opioid-naïve patient may be started on a much lower dose than a patient with longterm opioid intake with high level of tolerance. Boluses of morphine should be administered in incremental doses every 10 minutes until the respiratory rate is slowed. Then the total required dose to achieve satisfactory clinical effect should be converted into an hourly rate as the initial maintenance dose. Thereafter, the dose should be titrated up or down to the patient’s respiratory signs. If the patient lives on for a long period after withdrawal of ventilatory sup-
port, as in the case presented here, development of tolerance should be anticipated and the maintenance dose should be adjusted frequently.

Because of tolerance, it is not uncommon for patients undergoing withdrawal of life support to exhibit signs of respiratory discomfort and agitation despite surprisingly high doses of opioid.31 When the maintenance dose is increased, the adjustment should be made in sufficient increments. For example, in the setting of continued physical signs of respiratory distress, it would be subtherapeutic to increase the intravenous morphine dose from 4 mg/hour to 5 mg/hour, or from 8 mg/hour to 10 mg/hour. These represent only a 25% increase. Instead, the dose increase should be in increments of 50% to 100%, for example, from 4 mg/hour to 8 mg/hour, or from 8 mg/hour to 12 or 16 mg/hour, repeatedly until the desired clinical effect is reached.

As in other settings and perhaps more often after ventilator withdrawal, anxiety and agitation commonly occur with dyspnea and should be treated with benzodiazepines. Usually, 1 to 2 mg of lorazepam is administered intravenously as a bolus, and a maintenance dose started based on the initial response, with titration to patient comfort without bradypnea. A hypnotic agent such as propofol can also be used for shortterm additive effects but is impractical for longterm use. Neuromuscular blockers should never be used for control of dyspnea because they provide no palliation and prevent assessment of comfort.

Sometimes the tracheobronchial secretions can accumulate from inaccessible airway and inability to suction, leading to discomfort and rattling noises during inspiration and expiration. This “death rattle” causes a great deal of anxiety on the part of onlookers, but can be minimized by timely administration of anticholinergic agents before the onset of profuse pulmonary secretion. Once present, secretions should be controlled by frequent suctioning of the posterior oropharynx and discreet use of nasopharyngeal and nasotracheal suction if absolutely necessary. Other nonpharmacologic techniques to reduce breathlessness also include semiupright positioning, oxygen, or cool-misted air administration. It is essential for the proxy or family to receive explanation and reassurance about the respiratory signs and their significance, and the rationale for specific therapy as the patient approaches death. It is also crucial that the family be counseled that the patient may survive “terminal weaning” (not a desirable term) and live days, weeks, or even longer. Unexpected survival can provoke as much a crisis as expected demise!

In cases of extubation of patients who are not likely to survive the absence of ventilatory support or when dyspnea is occurring when demise is imminent, spiritual preparation for the patient, the patient’s family, and the caregivers is equally important as the pharmacologic preparation outlined above. The services of a chaplain or another individual entrusted with spiritual care identified by the patient or family should be offered.

With the above armamentarium, dyspnea after withdrawal of the ventilator can be successfully treated. But many surgeons fear directly causing or hastening death from sedation and analgesia and this can be a barrier to adequate therapy and palliation. Although this is a theoretical concern, there is very little clinical likelihood of opioid administration causing death based on the studies cited above.30,31 In addition, the ethical and legal precedents all suggest that palliation is morally sound and distinct from euthanasia based on the principle of “double effect.” Euthanasia is defined as “the act or practice of killing or permitting the death of hopelessly sick or injured individuals . . . in a relatively painless way of reasons of mercy.”32 Palliation, in the context of the present discussion, may be defined analogously as the act or practice of alleviating the suffering of hopelessly sick or injured individuals for reasons of mercy, with or without the double effect of hastened death.

Double effect, a concept derived originally from moral theology, is defined as follows: “An act with primary intention of doing good which produces a secondary effect that is harmful may be considered to have double effect.”33 Four conditions must be satisfied to qualify as a double effect.33,34

1) The harm (hastened death) occurs as a side effect to the achievement (palliation) of the primary act (high-dose opioid administration), which is directly aimed at or intended.
2) The primary act (high-dose opioid administration) directly aimed at is itself morally good (palliation), or at least morally neutral.
3) The good effect (palliation) is not achieved by way of the bad (hastened death). That is to say, hastened death must not be the primary means to palliation of suffering. This is where, at least theoretically, palliation is distinct and sep-
arate from euthanasia, in which the palliation of suffering occurs as an intended result of hastened death.

4) The bad consequence (hastened death) must not be so serious as to outweigh the good effect (palliation).

Application of double effect to end-of-life therapeutic decisions is not without controversy.35 Validity of the moral principle depends heavily on the moral agent’s “intent.” In reality, determination of the physician’s “intent” to palliate suffering vis-a-vis to hasten death is a complex if not impossible task. Recent US Supreme Court decisions over *Washington v. Glucksberg* (1997) and *Vacco v. Quill* (1997) provide clarification, if not directive, for the practicing surgeons. As Chief Justice William H Rehnquist affirmed in his majority opinion, the State may “permit palliative care related to (refusal of unwanted lifesaving treatment) . . . which may have the foreseen but unintended “double effect” of hastening the patient’s death.”36 And Justice Sandra Day O’Connor concurred that a patient in terminal illness has “no legal barriers” to receive medications to alleviate suffering, “even to the point of causing unconsciousness and hastening death. . . .”36 In the *New England Journal of Medicine*, a legal expert has interpreted that these opinions form a “basis for concluding that a Court majority has found that states must not impose barriers on the availability of palliative care for terminally ill patients.”36

The patient’s right to comfortable death after withdrawal of life support is further elucidated in *State of Georgia vs. McAfee* (1989).37 In this case, a quadriplegic patient who was incapable of spontaneous breathing asked for not only withdrawal of life support but also administration of a sedative agent in order to be spared of the consequent suffering. He had previously attempted to become disconnected from the ventilator but had been unable to do so because of severe discomfort experienced when the ventilator was disconnected. The Supreme Court of Georgia not only granted him the right of patient autonomy to refuse ventilatory support, but unanimously affirmed that his “right to be free from pain at time ventilator was disconnected was inseparable from right to refuse medical treatment.”37

Clarification of the above legal and ethical precedents support the use of medications for palliation of dyspnea even if the unintended consequence is a hastened death. Inadequate treatment of dyspnea because of misunderstanding of these precedents can result in unnecessary suffering for the patients and families.

### Summary

I returned to John’s bedside. We started a morphine infusion, and intermittent lorazepam, titrating them to his respiratory rate. His grimacing and noisy breathing abated and he appeared more comfortable. I spent a few minutes with his family to explain what to expect in the next hours. One day later he died peacefully, with his family at the bedside.

More and more surgeons will have the opportunity to ensure a peaceful death for their patients, such as John and his family experienced. Surgeons must recognize that withdrawal of life support is the beginning of heightened, intensive palliative care, when symptoms of dyspnea should be treated aggressively. The moral reasoning and legal precedents of palliation versus euthanasia provide a foundation for this therapy, and there should be no ethical barrier to treating dyspnea adequately. Once this is understood, much of the physician’s discomfort around treating dyspnea is removed, and attention can be paid to alleviation of the patient’s distress. Surgeons who care for dying patients must understand both the pathophysiology and the subjective experience of dyspnea, for its mechanism is complex and ill-defined and has implications for therapy. Whenever possible, surgical or interventional procedures should be offered to ameliorate the underlying reversible conditions such as pleural effusion, pericardial tamponade, or bronchial obstruction. The main therapy is pharmacologic, such as opioids and anxiolytics, supplemented by oxygen and other nonpharmacologic maneuvers. Management of dyspnea after withdrawal of ventilatory life support deserves heightened attention to patient and family discomfort and anxiety.

### Appendix

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