CLINICAL CONCEPTS AND COMMENTARY

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Preventing Postoperative Pulmonary Complications

The Role of the Anesthesiologist

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THE Confederate General "Stonewall" Jackson was one of the earliest known victims of a respiratory complication after surgery, dying of pneumonia 10 days after an otherwise successful ether anesthetic in 1863. Despite subsequent advances in anesthesia and surgical care, postoperative pulmonary complications (PPCs) still are a significant problem in modern practice. This commentary examines why PPCs occur and how the anesthesiologist can help prevent them.

Significance of Perioperative Pulmonary Complications

Determination of the frequency and clinical impact of PPCs in modern practice is hampered by the lack of a uniform definition of a PPC among studies. Nearly all investigators include in this definition pneumonia (definite or suspected), respiratory failure (usually defined as the need for mechanical ventilatory support), and bronchospasm. Others include unexplained fevers, excessive bronchial secretions, abnormal breath sounds, "productive" cough, atelectasis (often not defined), and hypoxemia. Even within these categories, definitions vary widely. Nonetheless, it is still clear that PPCs occur relatively frequently. In studies of noncardiac surgery, the frequency of PPCs and cardiac complications (which historically have attracted more attention from the anesthesia community) are comparable.[1] For example, in a series of adult men undergoing elective abdominal surgery, PPCs occurred significantly more frequently than cardiac complications (estimated rates of 9.6% and 5.7%, respectively) and were associated with significantly longer hospital stays.[1]

Causes of Perioperative Pulmonary Complications

A basic understanding of mechanism guides rational practice. Many PPCs, such as atelectasis and pneumonia, seem to be related to disruption of the normal activity of the respiratory muscles, disruption that begins with the induction of anesthesia and that may continue into the postoperative period. Breathing is a complex behavior requiring the coordinated activity of several muscle groups, both in the upper airway and in the chest wall. Anesthetics and many other drugs used in the perioperative period affect the central regulation of breathing, changing the neural drive to respiratory muscles such as the diaphragm. At high doses, anesthetics attenuate the activities of all respiratory muscles. However, at moderate depths of anesthesia, anesthetics may produce respiratory depression by altering the distribution and timing of neural drive to the respiratory muscles, rather than by producing a global depression of activity. For example, at 1.2 minimum alveolar concentration, halothane anesthesia depresses activity in some respiratory muscles (such as the parasternal intercostals) but actually increases activity in others (such as the transversus abdominis).[2] Thus, perioperative respiratory muscle dysfunction is, in some cases, more a matter of a lack of

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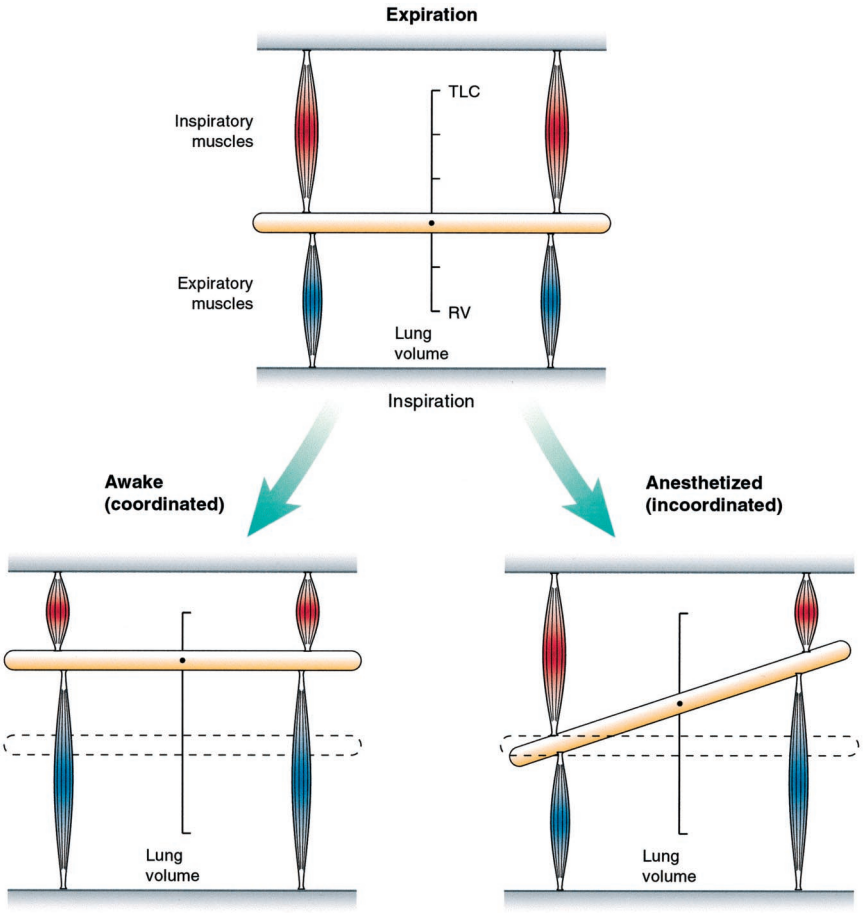

Fig. 1. Model showing how incoordination of respiratory muscles impairs lung function. The position of the midpoint of a horizontal bar, suspended between fixed surfaces by inspiratory and expiratory muscles, represents lung volume as denoted on a scale from low (residual volume, RV) to high (total lung capacity, TLC) volumes. During awake, coordinated inspiration (lower left), the bar remains horizontal (representing normal chest wall expansion), and lung volume changes efficiently. When anesthetized, muscle activity becomes incoordinated such that the bar tilts during inspiration (representing chest wall distortion), impairing lung expansion. Incoordination continues into the postoperative period after thoracic and abdominal surgery. Dashed lines in lower panels denote end expiratory position of the bar.

coordination than a lack of overall activity. As with other complex systems, this lack of coordination reduces efficiency (fig. 1), in this instance producing hypoventilation. In addition, deformation of the chest wall alters the underlying lung, decreasing the functional residual capacity and producing atelectasis in dependent lung regions. These regions of atelectasis develop in nearly all patients after a few minutes of anesthesia and may significantly impair pulmonary gas exchange.[3] Chest wall distortion and atelectasis also occur when the respiratory muscles are inactive during mechanical ventilation and persist even with positive end-expiratory pressure.

These intraoperative changes in the pattern of breathing can persist in the postoperative period as additional effects of surgical trauma come into play. Residual anesthetic effect may also contribute to postoperative changes in breathing, although the importance of this factor has not been studied. The effects of surgical trauma are most pronounced after thoracic and abdominal surgery, and they arise from at least three mechanisms (fig. 2). First, functional disruption of respiratory muscles (such as the intercostal or abdominal muscles) by incisions, even after surgical repair, may impair their effectiveness. Second, postoperative pain may cause voluntary limitation of respiratory motion. Finally, stimulation of the viscera, such as provided by mechanical traction on the gallbladder or esophageal dilation, markedly decreases phrenic motoneuron output and changes the activation of other respiratory muscles, generally acting to minimize diaphragmatic descent. These effects are only partially attenuated by vagotomy, suggesting that multiple afferent pathways mediate this reflex.[4] Thus, like anesthesia, surgical trauma can also disrupt normal coordination of respiratory muscle action, leading to persistent decreases in functional residual capacity and vital capacity, with lung atelectasis that can last for several days after surgery. The clinical impression is that this atelectasis leads to pneumonia, although this progression has not been conclusively shown. These postoperative changes in pulmonary function can be partially

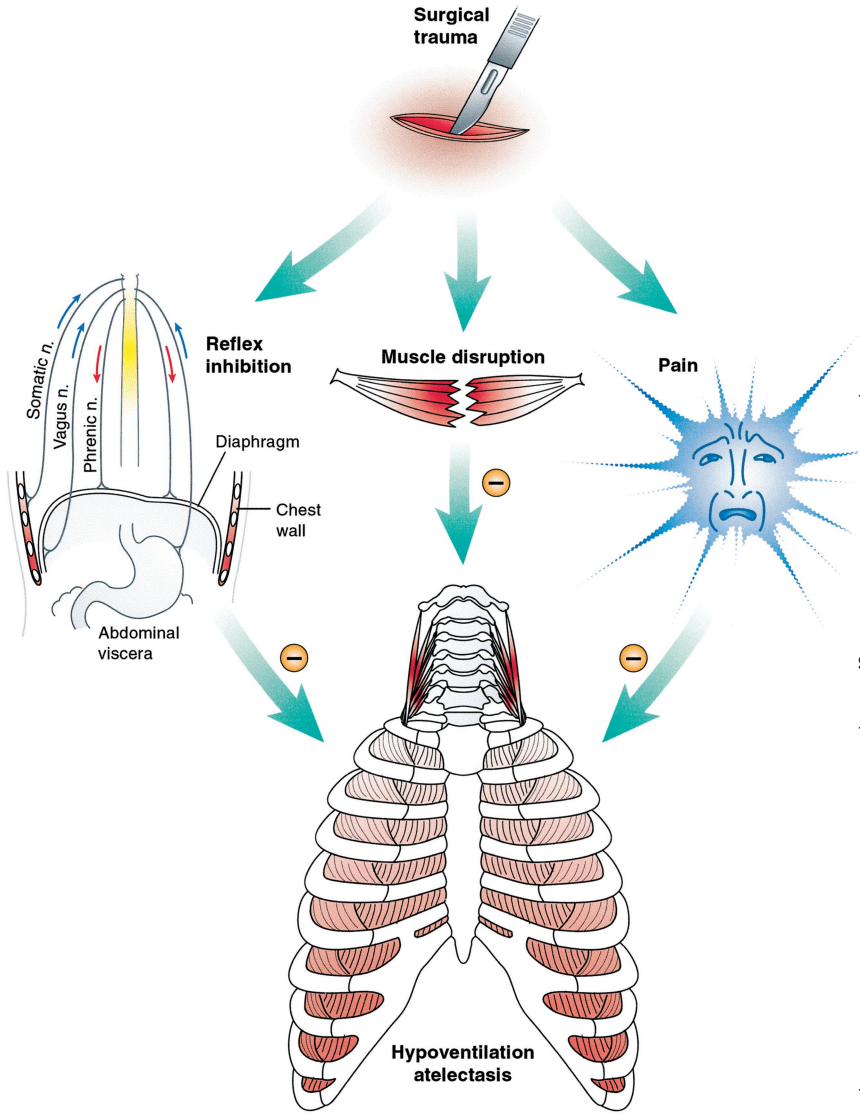

Fig. 2. Factors producing respiratory muscle dysfunction after surgical trauma. From left to right: (1) surgical trauma stimulates central nervous system (CNS) reflexes mediated by both visceral and somatic nerves that produce reflex inhibition of the phrenic and other nerves innervating respiratory muscle; (2) mechanical disruption of respiratory muscles impairs efficiency; and (3) pain produces voluntary limitation of respiratory motion. These factors all tend to reduce lung volumes and can produce hypoventilation and atelectasis.

ameliorated by using endoscopic techniques to minimize surgical trauma.[5] However, because procedures such as laparoscopic cholecystectomy still stimulate abdominal viscera (*e.g.*, through gallbladder traction), pulmonary mechanics are still affected; this is probably also true for thoracoscopy.

Other factors may also contribute to PPCs. Reflex stimulation during airway instrumentation and release of inflammatory mediators by drug administration can produce bronchoconstriction. This increased airway resistance limits especially expiratory gas flow from the lung, which, if severe, can produce hyperinflation with risk of barotrauma and gas exchange abnormalities. Anesthetic gasses and tracheal intubation may impair normal mucociliary transport. Recent studies suggest that prolonged anesthesia and surgery may impair the function of lung inflammatory cells, which could increase susceptibility to postoperative infections.[6] Finally, other respiratory outcomes are related specifically to surgical or anesthetic interventions, such as acute lung injury after cardiopulmonary bypass, pneumothorax caused by barotrauma or surgical trauma, negative pressure pulmonary edema after airway obstruction during spontaneous breathing, and aspiration pneumonitis.

Assessment of Risk

Consistent risk factors for PPCs among extant studies include surgical site (with thoracic and abdominal surgery posing the highest risk), smoking, and the presence of pulmonary disease. Although the results of pulmonary function testing, including measurements of arterial blood gasses, have proved useful in predicting pulmonary function after lung resection surgery, they do not predict PPCs.[7] For example, the degree of airway obstruction, as assessed by the forced expiratory volume in 1 s, is not a significant independent risk factor for the development of postoperative respiratory failure after abdominal surgery, even in smokers with severe lung disease.[8] Thus, pulmonary function tests should be viewed as a management tool to optimize preoperative pulmonary function as appropriate, not as a means to assess risk. For example, spirometry or peak flows may be useful to monitor the status of a patient with asthma, especially the small subset of patients who have difficulty perceiving the status of their disease. Otherwise, routine preoperative pulmonary function testing may be a waste of resources.[9]

Preparation of Patients with Lung Disease for Surgery

Schemes to prepare patients with lung disease for surgery have included prolonged preoperative hospital admission, inhaled β-adrenergic agonists, chest physiotherapy, antibiotics, intravenous aminophylline, and hydration. Proposed mechanisms of benefit include improvements in respiratory physiology and elimination of any underlying pulmonary infections. However, there have been no controlled trials demonstrating that these regimens improve outcome in patients with lung disease. Furthermore, some of their components pose risk (*e.g.*, aminophylline) or are impractical in current practice (*e.g.*, preoperative hospital admission). Although such schemes should not be routinely applied to all patients, preoperative lung function should be optimized as tailored to the needs of the individual patient. For example, symptoms should be optimally controlled in patients with asthma (usually by ensuring that airway inflammation is minimized with topical or systemic corticosteroids) because patients with more active symptoms have a higher frequency of PPCs.[10] However, there is no firm evidence that all patients with asthma should receive routine systemic corticosteroids.[10] All patients should be encouraged to quit smoking, a significant risk factor for PPCs. The optimal timing of quitting is not known and should be the focus of future investigations. Of interest, some studies find that recent cessation or reduction of smoking (within approximately 2 months before surgery) may actually increase the risk of PPCs.[11] However, these studies are not conclusive because of selection bias, with the sickest patients being more likely to reduce smoking. The long-term benefits of smoking cessation to the patient suggest that the anesthesiologist should seize the preoperative period as an opportune time to encourage quitting, recognizing that the minimum duration required to demonstrate benefit is unknown.

Beyond these measures, the single most important preoperative intervention is to educate patients regarding the proper performance of maneuvers designed to increase lung volumes, which are of proven benefit in the postoperative period (as discussed in the following section).

Prevention of Perioperative Pulmonary Complications

One goal of management is to prevent significant bronchoconstriction. The reflex irritation produced by laryngoscopy and tracheal intubation is best avoided, if possible, in patients with reactive airways, although the majority of these patients, even those with severe chronic obstructive pulmonary disease, can tolerate this if necessary.[8] A variety of measures, especially the preinduction administration of inhaled β-adrenergic agonists and muscarinic antagonists, can effectively attenuate airway reflex responses to tracheal intubation. Another approach is to avoid airway manipulation altogether by using regional anesthesia when feasible, although whether the use of these techniques actually improves respiratory outcomes is unproven. As a practical matter, some patients with severe respiratory disease use accessory muscles of breathing (such the abdominal muscles) and cannot maintain spontaneous breathing if these muscles are paralyzed by techniques such as epidural or subarachnoid block.

Another goal that has attracted much interest over recent years is to optimize postoperative pulmonary function by improving postoperative analgesia. In particular, regional analgesic techniques have the potential to improve two of the three mechanisms discussed previously (fig. 2) that produce postoperative respiratory muscle dysfunction: pain and reflex inhibition of respi-

ratory muscles, if the afferent reflex limb can indeed be targeted. The expectation is that these effects will improve respiratory muscle function, increase lung expansion, and prevent PPCs such as atelectasis and pneumonia. Is this expectation true?

There is no doubt that regional analgesic techniques, at least those using local anesthetics, can significantly alter postoperative respiratory muscle function. In general, techniques such as segmental epidural blockade with local anesthetics can increase tidal volume and vital capacity and improve indices interpreted to reflect diaphragm activity after thoracic and upper abdominal surgery.[12] These effects have been attributed to decreased pain and interruption of the afferent limb of reflex diaphragmatic inhibition. However, local anesthetic block also can paralyze other respiratory muscles such as the intercostal and abdominal muscles, which itself will change the pattern of breathing and makes it difficult to interpret various measures of diaphragmatic function.[13] Furthermore, blockade may not affect reflex inhibition mediated by afferent information carried in nerves such as the phrenic or vagus. In the only direct measurements of postoperative diaphragmatic shortening in human subjects, thoracic epidural blockade after thoracotomy did not change diaphragm electromyogram or shortening, and, in fact, produced paradoxical lengthening during inspiration in half of the patients studied, despite improving many more global indices of respiratory function such as tidal volume.[14] Thus, the effect of postoperative regional analgesia on the pattern of breathing may be complex and have unintended consequences.

Do these changes in breathing pattern and spirometric values produced by regional analgesia translate to improved clinical outcomes? This straightforward question has proved difficult to answer for a variety of reasons. As previously discussed, there is often little agreement among investigators as to what constitutes a PPC. Patients and investigators are seldom blinded for technique, perhaps introducing bias favoring regional analgesia. Patient populations, surgical technique, and analgesic regimens are heterogeneous, even within studies. For example, some studies use epidural analgesia intraoperatively, and others do not. The application of postoperative maneuvers to increase lung volume, which are of clear benefit, is often inconsistent or not reported. Finally, because major PPCs occur with relatively low frequency, studies often examine insufficient numbers of patients to make conclusions.

In an attempt to overcome the latter problem, a recent study reported meta-analyses of the effects of postoperative analgesic therapies on pulmonary outcome after a wide variety of surgical procedures, including abdominal and thoracic surgery.[15] In this study, postoperative epidural opioids significantly decreased the frequency of atelectasis, but not other pulmonary complications, when compared with systemic opioids. Epidural local anesthetics decreased the incidence of pulmonary infections and pulmonary complications overall when compared with systemic opioids. However, the individual studies examined in these analyses were often beset by the problems noted previously (and others), making interpretation of meta-analyses problematic. For example, consider the conclusion that epidural local anesthetics significantly decrease the rate of pulmonary complications compared with systemic opioids (controls). Four of the eight studies used in the meta-analysis to support this conclusion reported a very high frequency of atelectasis/pneumonia in control groups (60–70%), far greater than the other four studies (which found no benefit) and far greater than more recent series,[8] making their applicability to current practice questionable.

In one of the few studies that successfully accounts for many of these problems and that represents the largest trial to date, Jayr *et al.*[16] examined patients undergoing abdominal cancer surgery randomized to receive either continuous epidural bupivacaine and morphine or subcutaneous morphine infusion *via* a catheter that simulated epidural placement. They found that although the epidural provided excellent postoperative analgesia, superior to that afforded by the subcutaneous morphine infusion, it did not affect the frequency of PPCs as carefully defined and prospectively evaluated, either in patients with normal or abnormal lungs. Several other good studies have also failed to demonstrate significant clinical benefit of regional techniques. Thus, although regional techniques can provide excellent analgesia when properly applied, in my opinion, it is not yet clear that they consistently improve clinical respiratory outcomes.

Postoperative maneuvers to increase mean lung volumes are of proven benefit in preventing PPCs. Presumably these techniques increase lung expansile forces and discourage atelectasis. Several methods have been studied, including intermittent positive pressure breathing, deep-breathing exercises, incentive spirometry, and chest physiotherapy. Critical review, as well as a recent meta-analysis, suggests that all regimens studied are equally efficacious in reducing the frequency of PPCs (by approximately a factor of two compared with no therapy) after upper abdominal surgery.[17] Currently, incen-

tive spirometry enjoys popularity because it is simple, inexpensive, and provides objective goals for and monitoring of patient performance. Although there is little evidence that instituting these measures preoperatively benefits patient physiology, preoperative education may improve patient compliance and performance in the postoperative period. Considering that this intervention is the only one proven to reduce the frequency of PPCs, both patients and staff must be educated regarding its importance.

PPCs are an important clinical problem in modern practice, rivaling perioperative cardiac morbidity in frequency and severity in some settings. It seems that many PPCs are caused by the disruption of the normal coordination of respiratory muscle by anesthesia and surgical trauma. Although the use of postoperative regional analgesia can partially improve respiratory muscle function, there is no convincing evidence that regional techniques decrease the frequency of clinically significant PPCs. Rather, maneuvers to encourage deep breathing, such as incentive spirometry, are of proven benefit and should be the focus of preventive efforts. In an era of relentless high technology, it is perhaps comforting that such a simple technique should prove so effective.

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