IMPROVING POSTOPERATIVE RESPIRATORY OUTCOME 5RC1

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Postoperative pulmonary complications (PPCs) such as atelectasis and pneumonia are still a significant source of morbidity in modern practice, rivaling cardiovascular complications in frequency and severity in some settings [1]. What can the anesthesiologist do to improve postoperative respiratory outcomes?

WHAT CAUSES POSTOPERATIVE PULMONARY COMPLICATIONS?

Many factors responsible for PPCs are 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 [2]. 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. Thus, perioperative respiratory muscle dysfunction is in many cases more a matter of a lack of coordination than a lack of overall activity. As with other complex systems, this lack of coordination reduces efficiency, 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 and other effects produce the ventilation-perfusion inequalities characteristic of anesthesia.

These effects of anesthesia can persist into the postoperative period, though via different mechanisms, as the effects of surgical trauma come into play. These are most pronounced following thoracic and abdominal surgery, and arise from at least three mechanisms. First, functional disruption of respiratory 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 motor neurone output and changes the activation of other respiratory muscles, in general acting to minimize diaphragmatic descent. These changes may be particularly marked in the obese. Postoperative changes in pulmonary function can be partially ameliorated by utilizing endoscopic techniques to minimize surgical trauma, although pulmonary mechanics are still affected.

Other factors that may contribute to PPCs include 1) reflex stimulation during airway instrumentation and release of inflammatory mediators by drug administration, increasing airway resistance and limiting expiratory gas flow from the lung; if severe this can produce hyperinflation with risk of barotrauma and gas exchange abnormalities; 2) impairment of normal mucociliary transport by anesthetic gasses and endotracheal intubation which may delay clearance of pathogens and promote retained secretions; 3) impairment of lung inflammatory cell's function by prolonged anesthesia and surgery, which could increase susceptibility to postoperative infections; 4) impaired upper airway reflexes postoperatively, with may increase the risk of aspiration, and; 5) incomplete reversal of neuromuscular blockade.

HOW CAN RISK BE ASSESSED?

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 have proved useful in predicting postoperative pulmonary function following lung resection surgery, they do not predict PPCs [3]. 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.

CAN REGIONAL ANALGESIA PREVENT POSTOPERATIVE PULMONARY COMPLICATIONS?

Epidural analgesia has the potential to improve two of the three mechanisms that produce postoperative respiratory muscle dysfunction: pain and reflex inhibition of respiratory muscles. This should improve respiratory muscle function, increase lung expansion, and prevent postoperative pulmonary complications. However, there is still considerable doubt regarding whether regional analgesia significantly improves postoperative respiratory outcomes [2].

There is no doubt that epidural analgesia, at least if local anesthetics are used, can significantly alter postoperative respiratory function [4]. In general, techniques such as segmental epidural blockade with local anesthetics increase tidal volume and vital capacity, and improve indices interpreted to reflect diaphragm activity after thoracic and upper abdominal surgery [5]. 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 [6]. Further, 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 the diaphragm electromyogram or shortening of the muscle, 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 [7]. Thus, the effect of postoperative epidural analgesia on the pattern of breathing may be complex and have unintended consequences. Such complexities may also apply to effects to other organ systems, such as gastrointestinal motility.

Do these changes in breathing pattern and spirometric values produced by epidural analgesia translate to improved clinical outcomes? This straightforward question has proved difficult to answer for a variety of reasons [2]. A recent study reported meta-analyses of the effects of postoperative analgesic therapies on pulmonary outcome following a wide variety of surgical procedures, including abdominal and thoracic surgery [8]. In this analysis, 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 above (and others), making the interpretation of meta-analyses problematic. For example, four of the eight studies used in the meta-analysis to support this conclusion reported an inordinately 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, making their applicability to current practice questionable.

Although detailed review of all trials is not possible here, the most useful recent trials include two large unmasked multicenter trials with relatively less standardization of care, and two smaller, double-masked single center trials employing strict protocols for care (the term “masking” is used in preference to “blinding” to recognize that true blinding may be difficult to achieve in these studies).

Each of the former [9-12] enrolled approximately 1000 patients, studying epidural analgesia as the treatment (opioids with or without local anesthetics) beginning before incision, and allowing substantial variations in practice among patients. Overall, they found few differences in outcome between those receiving and not receiving epidural analgesia, with the exceptions that 1) respiratory failure was less frequent (for some types of surgery), and 2) postoperative pain control was improved by epidural analgesia.

In the first masked trial, Jayr et al [13] examined 153 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 postoperative pulmonary complications, as had been carefully defined and prospectively evaluated, either in patients with normal or abnormal lungs (few of these subjects were obese). Norris et al [14] randomized 168 patients undergoing abdominal aortic aneurysm repair to receive either thoracic epidural anesthesia combined with general anesthesia or general anesthesia alone intraoperatively with either intravenous or epidural analgesia postoperatively (4 treatment groups). A combination of epidural opioids and local anesthetics was used. They found no differences in outcomes, other than a shortened time to extubation in the epidural group (all patients received some period of mechanical ventilation postoperatively). Pain scores were equivalent in all groups, perhaps reflecting the relatively high dose of intraoperative opioids permitted by not planning for immediate extubation. Hospital length of stay, their primary outcome, was not different (median of 7 days).

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 when applied in isolation.

How can we help prevent pulmonary complications in patients at risk?

Table 1. Management of Patients at Risk for Pulmonary Complications

PREOPERATIVE	
Evaluation and assesment	• Assess general physical status (pulmonary, cardiac, neurologic disease) and treat any reversible signs/symptoms
	• Spirometry used only as guide for treatment.
	• CXR only when necessary to evaluate symptoms
	• Arterial blood gases as needed to evaluate signs/symptoms
	• Cessation of cigarette smoking (as long before surgery as possible)
	• Treat any reversible component (antibiotics, bronchodilators, corticosteroids)
	• Consider postponing elective surgery if improvement of pulmonary function is possible
	• Education regarding postoperative deep breathing/incentive spirometry
	• Enforce strategies for postoperative early ambulation (pain management, etc)
Anesthetic/Surgical Planning	• Consider regional anesthesia for excellent pain control, not necessarily to reduce pulmonary risk
	• Limit duration of surgery as possible
	• Consider use of laparoscopic techniques
INTRAOPERATIVE	• Consider laryngeal mask airway or similar, especially in those with bronchospasm
	• Careful and vigilant use of long acting muscle relaxant (to avoid postoperative muscle weakness)
	• Consider use of local anesthetics for operative field infiltration
	• Maintain adequate hydration to allow mobilization of airway secretions
	• Mechanical ventilation: consider recruitment maneuvers (lung expansion followed by PEEP) to achieve and maintain a dequate hemoglobin saturation.
	• Consider use of lower FIO2 to reduce resorption atelectasis
	• Consider “permissive hypercapnia”, and avoid high pressure/ large tidal volume ventilation which may cause barotrauma or volutrauma
POSTOPERATIVE	• Maintain tracheal intubation until full reversal of neuromuscular drugs is achieved
	• Use multimodal therapy such as non-steroidal antiinflammatory agents, dexmedetomidine, nerve blocks, etc. to maximize analgesia and minimize the use of opioids that may depress ventilation
	• Early postoperative respiratory therapy (deep breathing, incentive spirometry)
	• Encourage early ambulation
	• Continue adequate hydration to allow mobilization of secretions

There have been no controlled trials demonstrating that a specific preparation scheme improves outcome in patients with lung disease. Nevertheless, preoperative optimization of lung function would seem reasonable as tailored to the needs of the individual patient (Table 1). The clinician should use whatever diagnostic tools (such as pulmonary function tests) necessary to ensure optimization, recognizing that absolute values of these tests have little predictive ability. As smoking is a significant risk factor for PPCs, all patients should be encouraged to quit. Although it may take at least a month for clinical benefit to be realized, the assertion that more brief abstinence is actually detrimental is not supported by existing data, and patients should not be discouraged from quitting at any time on this basis [15]. Beyond optimization of lung function, the single most important preoperative measure is to educate the patient regarding the proper performance of maneuvers designed to increase lung volumes, which are of proven benefit in the postoperative period [16]. Education regarding strategies for early postoperative ambulation may also be helpful.

In the planning for anesthesia and surgery, regional anesthesia should be considered as appropriate, recognizing that analgesia per se may not affect outcome. Laparoscopic techniques may minimize perioperative changes in pulmonary mechanics, although it is unclear whether this translates into improved clinical outcomes.

Intraoperatively, minimizing airway irritation through use of devices such as the laryngeal mask airway would seem appropriate. Recruitment maneuvers and the use of lower inspired oxygen fractions will decrease intraoperative atelectasis, an effect that may persist into the postoperative period. Whether this improves clinical outcomes is not yet known. For patients requiring intraoperative mechanical ventilation, standard ventilator settings (tidal volume of 10-12 ml/kg, rate of 10-12 min^{-1}) produce hypocapnia. Considering recent data concerning ventilator-induced lung injury in the critically ill, it may be prudent to employ smaller tidal volumes, although again there is no outcome data to support any particular method of intraoperative mechanical ventilatory support.

Postoperatively, maneuvers to increase mean lung volumes are of 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 [16]. Currently, incentive spirometry enjoys popularity because it is simple, inexpensive, and provides objective goals for and monitoring of patient performance; anesthesiologists should ensure that it is consistently performed. Rapid postoperative mobilization using techniques such as “multimodal” therapy may improve several measures of postoperative outcome [17]; trials will be necessary to determine if this benefit includes PPCs.

References

1. Lawrence VA, Hilsenbeck SG, Mulrow CD et al. Incidence and hospital stay for cardiac and pulmonary complications after abdominal surgery. Journal of General Internal Medicine 1995;10:671-8.
2. Warner DO. Preventing postoperative pulmonary complications: the role of the anesthesiologist. Anesthesiology 2000;92:1467-72.
3. Lawrence VA, Page CP, Harris GD. Preoperative spirometry before abdominal operations: A critical appraisal of its predictive value. Archive of Internal Medicine 1989;149:280-5.
4. Warner DO, Warner MA, Ritman EL. Human chest wall function during epidural anesthesia. Anesthesiology 1996;85:761-73.
5. Pansard JL, Mankikian B, Bertrand M et al. Effects of thoracic extradural block on diaphragmatic electrical activity and contractility after upper abdominal surgery. Anesthesiology 1993;78:63-71.
6. Duggan J, Drummond GB. Activity of lower intercostal and abdominal muscle after upper abdominal surgery. Anesthesia & Analgesia 1987;66:852-5.
7. Fratacci MD, Kimball WR, Wain JC et al. Diaphragmatic shortening after thoracic surgery in humans. Effects of mechanical ventilation and thoracic epidural anesthesia. Anesthesiology 1993;79:654-65.
8. Ballantyne JC, Carr DB, deFerranti S et al. The comparative effects of postoperative analgesic therapies on pulmonary outcome: cumulative meta-analyses of randomized, controlled trials. Anesthesia & Analgesia 1998;86:598-612.
9. Peyton PJ, Myles PS, Silbert BS et al. Perioperative epidural analgesia and outcome after major abdominal surgery in high-risk patients.[comment]. Anesthesia & Analgesia 2003;96:548.
10. Rigg JR, Jamrozik K, Myles PS et al. Epidural anaesthesia and analgesia and outcome of major surgery: a randomised trial. Lancet 2002;359:1276-82.
11. Rigg JR, Jamrozik K, Myles PS et al. Design of the multicenter Australian study of epidural anesthesia and analgesia in major surgery: the MASTER trial. Controlled Clinical Trials 2000;21:244-56.
12. Park WY, Thompson JS, Lee KK. Effect of epidural anesthesia and analgesia on perioperative outcome: a randomized, controlled Veterans Affairs cooperative study. Annals of Surgery 2001;234:560-9.
13. Jayr C, Thomas H, Rey A et al. Postoperative pulmonary complications. Epidural analgesia using bupivacaine and opioids versus parenteral opioids.[comment]. Anesthesiology 1993;78:666-76.
14. Norris EJ, Beattie C, Perler BA et al. Double-masked randomized trial comparing alternate combinations of intraoperative anesthesia and postoperative analgesia in abdominal aortic surgery. Anesthesiology 2001;95:1054-67.
15. Warner MA, Offord KP, Warner ME et al. Role of preoperative cessation of smoking and other factors in postoperative pulmonary complications: a blinded prospective study of coronary artery bypass patients. Mayo Clinic Proceedings 1989;64:609-16.
16. Thomas JA, McIntosh JM. Are incentive spirometry, intermittent positive pressure breathing, and deep breathing exercises effective in the prevention of postoperative pulmonary complications after upper abdominal surgery? A systematic overview and meta-analysis. Physical Therapy 1994;74:3-10.
17. Kehlet H. Multimodal approach to control postoperative pathophysiology and rehabilitation. British Journal of Anaesthesia 1997;78:606-17.