Preventing Respiratory Depression

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LEE et al.1 evaluated closed malpractice claims related to respiratory depression. They identified 92 closed claims over 20 yr in about a third of covered anesthesiologists, which corresponds very roughly to 14 closed claims per year for all anesthesiologists nationwide among perhaps 75 million cases. Three-quarters of the patients died or were left with severe brain damage; half resulted in settlement payments, with the median being $217,000 (intraquartile range: $50,000 to $604,000). We know that only a small fraction of adverse outcomes results in malpractice claims. It is thus apparent that postoperative respiratory events resulting in death or serious injury occur at a concerning rate.

Most patients whose closed claims resulted from respiratory events were given opioids, nearly half by at least two routes—often prescribed by different physicians, and nearly half had a continuous opioid infusion at the time of the respiratory event. That said, gross overdose apparently contributed to only 16% of cases. Interestingly, only a quarter of the patients had or were at high risk of obstructive sleep apnea, which is consistent with data showing no relationship whatsoever between STOP-BANG scores (a measure of sleep apnea risk)2 and postoperative hypoxemia.3

Taken together, these data suggest that routine opioid use is a major cause of postoperative respiratory events—probably contributing more than sleep apnea. Reducing opioid use seems likely to decrease the risk of respiratory events. However, it is also obvious that opioids will remain an important analgesic strategy for years to come—and that we must thus learn to safely care for patients given opioids.

Postoperative airway obstruction or inadequate ventilatory effort should not cause catastrophic outcomes because either can be treated with opioid antagonists or mechanical ventilation. Respiratory catastrophes after routine surgery might thus reasonably be considered “never events.”

Case reviewers judged that 97% of claims probably or possibly could have been prevented by better monitoring. More intense conventional monitoring probably is not the answer though. Hypoxemia in postoperative inpatients is common, severe, and prolonged.4 Furthermore, even serious and persistent hypoxemia is unrecognized by nurses in 88% of cases (unpublished data, Department of Outcomes Research, Cleveland Clinic, December 2014). That a full quarter of the respiratory events in the series of Lee et al.1 occurred within 15 min of a nursing evaluation shows how often the current system fails.

So what can we do? Continuous monitoring is perhaps the obvious way to prevent catastrophic postoperative respiratory events. It is tempting to target continuous monitoring to high-risk patients, such as those who have a history of sleep apnea or are obese. The difficulty is that even the best prediction system will identify only a fraction—probably a small fraction—of at-risk patients. Continuous monitoring is thus probably appropriate for nearly all postoperative inpatients.

The real question is perhaps not whether to monitor continuously, but what to monitor. Surely pulse oximetry should be included. But whether to add capnography, impedance pneumography, transcutaneous partial pressure of carbon dioxide, or other monitoring systems is less obvious. (The Anesthesia Patient Safety Foundation recommends pulse oximetry for all patients given parenteral opioids and some measure of ventilation for patients using supplemental oxygen.)5 Clearly, each type of ventilation monitoring provides valuable information that supplements oxygen saturation, but each also adds complexity, cost, and false alarms. Recently developed compact battery-powered systems that communicate wirelessly reduce the burden for patients. And as interpretation algorithms become more sophisticated, the ratio of true-positive to false-positive alarms will presumably improve.

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Nonetheless, the history of medicine is littered with treatments that seemed logical and were subsequently proven unhelpful—or even harmful. There is little reason to believe that our understanding of physiology has suddenly become so good that logic alone should suffice in this case. Continuous respiratory monitoring is no different from any other clinical interventions and deserves similar formal testing including a cost-benefit analysis. The question is not simply whether to monitor; we also need to know which patients, what system(s) to use, and how to interpret the resulting data.

In summary, half the closed claims postoperative respiratory events were lethal and a quarter caused serious neurologic injury. Opioid administration probably contributed in most cases, and inadequate monitoring was the rule. It is likely that many catastrophic respiratory events could be prevented by continuous saturation and/or ventilation monitoring. However, major trials are needed to determine what should be monitored and how.

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Competing Interests

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References

5. Weigner MB, Lee LA: No patient shall be harmed by opioid-induced respiratory depression. APSF Newsletter 2011; 26:21, 26–8