

# Postoperative Respiratory Muscle Dysfunction

## Only the Strong Survive

**P**OSTOPERATIVE respiratory failure is most commonly defined as the need for mechanical ventilation for more than 48 h or unplanned postoperative reintubation. Although postoperative respiratory complications can occur through a number of different mechanisms, including infection, transfusion injury, and inflammatory lung injury from sepsis, a common factor leading to the need for prolonged mechanical ventilation is the inability of respiratory muscles to support adequate oxygenation and ventilation. The first step toward preventing postoperative pulmonary complications (PPCs) is a thorough understanding of the pathophysiology of postoperative respiratory muscle dysfunction. In this issue of *ANESTHESIOLOGY*, Sasaki *et al.*<sup>1</sup> provide just such a comprehensive review.

Postoperative respiratory failure is a major contributor to perioperative morbidity and mortality. For example, in a large cohort of surgical patients studied in the National Surgical Quality Improvement Program undergoing major vascular and general surgical operations, 3% experienced postoperative respiratory failure.<sup>2</sup> The patients who experienced postoperative respiratory failure had a 30-day mortality of 26.5% compared with a mortality rate of 1.4% in those patients who did not experience postoperative respiratory failure. In addition to morbidity and mortality, these complications are expensive. Dimick *et al.*<sup>3</sup> showed that the additional cost associated with postoperative respiratory failure was \$62,704.

Anesthesia providers play a key role in the prevention of respiratory muscle–related PPCs. Preoperative preparation, intraoperative management, and immediate postoperative care can have a major impact on the occurrence of this morbidity. For example, in a cohort of 279 patients scheduled to undergo coronary artery bypass graft surgery, preoperative respiratory muscle training consisting of 2 weeks of daily incentive spirometry against an inspiratory load resulted in statistically significant reductions in pneumonia and reduced length of stay.<sup>4</sup> The choice of anesthetic technique can impact the incidence of PPCs. Severe postoperative pain can result in shallow breathing and atelectasis, and treatment of this pain with opioids will reduce minute ventilation and

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respiratory drive. In a meta-analysis of patients undergoing thoracic and abdominal surgery, Pöpping *et al.*<sup>5</sup> showed that odds of developing postoperative pneumonia was reduced nearly 50% in patients receiving epidural *versus* intravenous analgesia. Intraoperative use of neuromuscular blockade may also

impact PPCs. In a recent prospective cohort study, Grosse-Sundrup *et al.*<sup>6</sup> demonstrated that the use of intermediate-acting neuromuscular blocking agents was associated with postoperative oxygen desaturation (<90%) and reintubation requiring unplanned intensive care unit admission. Interestingly, train-of-four monitoring and use of reversal agents such as neostigmine did not reduce the incidence of these complications.

Postoperatively, early use of continuous positive airway pressure for the prevention of hypoxemia reduced the occurrence of reintubation, pneumonia, sepsis, and intensive care unit length of stay compared with treating the postoperative hypoxemia with supplemental oxygen.<sup>7</sup> Although PPCs are most commonly associated with respiratory muscle dysfunction, it is clear that overall muscle strength and conditioning in other muscle groups have a salutary effect on respiratory function. Early mobilization of hospitalized patients can reduce a number of complications, especially those associated with mechanical ventilation. Patients in the intensive care unit frequently suffer from muscle weakness, the cause of which is often multifactorial, but which can be ameliorated with early mobilization.<sup>8</sup> Schweickert *et al.*<sup>9</sup> randomized 104 mechanically ventilated patients to either early exercise and mobilization during periods of daily sedation interruption (intervention group) or routine care during sedation interruption (control group). Patients in the intervention group had a reduced incidence of delirium and 1.5 fewer days of mechanical ventilation.

Finally, in patients subjected to mechanical ventilation, the way in which we provide ventilator support can affect outcomes. Patients requiring mechanical ventilation rapidly develop disuse atrophy of respiratory muscles. Levine *et al.*<sup>10</sup> obtained biopsy specimens from the diaphragms of 14 brain-dead organ donors before organ harvest. They showed that as early as 18 h after the initiation of mechanical ventilation, the

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biopsy specimens showed evidence of atrophy with decreased cross-sectional area of both slow- and fast-twitch muscle fibers and molecular evidence of programmed cell death. In a murine model, Mrozek *et al.*<sup>11</sup> demonstrated diaphragmatic, but not limb muscle, weakness after 6 h of mechanical ventilation. As described in this issue by Sasaki *et al.*<sup>1</sup>, respiratory muscles have primarily slow-twitch fibers, which may fatigue with overuse.<sup>3</sup> However, complete diaphragm rest will lead to atrophy. In animal models, modes that allow patient triggering and require some diaphragm activity such as assist-control ventilation seem to be able to maintain muscle integrity.<sup>12</sup>

Sasaki *et al.*<sup>1</sup> are to be congratulated for providing a clinically relevant, comprehensive review of postoperative respiratory muscle dysfunction. It is incumbent on anesthesia practitioners to be familiar with the physiology and pathophysiology of PPCs to provide optimal care for perioperative patients.

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