Barotrauma vs Volutrauma

To the Editor:

The barotrauma vs volutrauma debate continues, but the volutrauma camp seems to have gotten the upper hand. A statement in a contemporary textbook on mechanical ventilation declared, “It is clear that microvascular lung injury and pulmonary edema during mechanical ventilation are the consequences, not of ‘barotrauma,’ but, rather, of ‘volutrauma.’” We would caution both camps that while vindication is sweet, a change in perspective may bring insight to the phenomenon of ventilation-induced injury.

The studies that brought steam to the volutrauma camp suffer from a flawed presumption. First, the researchers demonstrated that lung injury developed in animals ventilated at high pressure/high tidal volume. They simulated high pressure/low volume ventilation by thoracoabdominal strapping and found no injury in this group, concluding that high pressure per se did not cause injury. They then simulated high volume/low pressure ventilation using an iron lung to achieve lung overdistension and found injury—proving high volume to be the culprit. Unfortunately, the studies fail to consider alveolar pressure from the proper reference pressure.

Figure 1 shows the relevant pressures at end-inspiration in the experiments. In Figure 1, Top, high inspiratory pressure hyperinflates the lung, resulting in injury. In Figure 1, Center, the iron lung creates a large negative pressure at chest surface (Pcs) hyperinflating the lung while maintaining alveolar pressure (Palv) at 0. However, the distending pressure of the respiratory system (Prs=Palv–Pcs) is exactly the same as in Figure 1, Top. The distending pressure of the lung (Palv–Ppl) is also identical. In Figure 1, Bottom, the chest/abdomen is not allowed to expand. This results in very high airway opening pressure (Pao), Palv, Ppl, and Pcs, but the transpulmonary pressure (Palv–Ppl) remains low.

Furthermore, the “stretch” in the alveoli is caused by the tensile stress in the alveolar wall produced by the distending pressure. Laplace’s law states that wall stress (σ) is proportional to P (transmural pressure) times the diameter. Transmural pressure and diameter of the alveoli have equal roles in wall stress. In Figure 1, Top and Center, the transmural pressures and alveolar sizes are the same, thus the wall stresses are identical. In fact, this is how the researchers can achieve similar high tidal volumes without a change in lung compliance. Therefore, these studies have not really dissected the respective effects of pressure and volume, but they merely created an “illusion” of a low pressure situation as shown in Figure 1, Center, and high pressure as in Figure 1, Bottom.

Properly referencing one’s pressure measurements is of utmost importance, lest we arrive at misguided conclusions. For example, when a diver at a 66-foot depth is breathing at more than 2,000 cm

Figure 1. Top: High-pressure/high volume. Center: Negative-pressure/high volume (iron lung). Bottom: High-pressure/low volume.
H₂O, we'd be surprised not to find lung injury unless we realize that the ambient pressure exerted on the chest wall (PCs) is also 2 atmospheres, and transpulmonary pressure remains normal (analogous to Figure 1, Bottom). Likewise, a thin inner tube of a tire can withstand the weight of a car because the rigid outer tube limits its transmural pressure, thus keeping the wall stress low.

Undoubtedly, epithelial damage and pulmonary edema occur when alveoli are overstretched. However, the overstretching (volutrauma) results directly from distending pressure (barotrauma). There can be no strain without stress, and emerging data show that high wall stress has a pathogenic property, as expected. We propose to keep the term "barotrauma" in referring to extra-alveolar air, and to use the more accurate term "alveolar stress injury" for the phenomenon of stress/strain induced epithelial/endothelial damage. It is time that we analyze ventilator-induced lung injury using principles of physics and reconcile the barotrauma and volutrauma camps.

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REFERENCES

Propofol for ICU Sedation

To the Editor:

I read with pleasure the excellent review by Mineanda and Broyles (1995; 108:539-48) in a recent issue of CHEST regarding propofol for ICU sedation. The article was comprehensive and generally well balanced and should serve as a valuable resource to physicians involved in the ICU.

As a cardiac anesthesiologist with ICU responsibilities, I have considerable experience with propofol infusion techniques. Clearly, this drug represents a tremendous advance in anesthesia and sedation due to its pharmacokinetic profile. I believe the cost data from Mineanda and Broyles are misleading. Table 5 on page 546 (CHEST 1995; 108:539-48) describes an overstated dose and therefore cost of propofol for ICU sedation. According to their table, in a 70 kg patient, 100 to 200 mg/h is a typical low dose. This would equal 24 to 48 pg/kg/min. This is accurate. However, the authors describe a high dose of 500 to 1,000 mg/h, which equals 120 to 240 pg/kg/min. These are dosages typically used for general anesthesia in the operating room. Indeed, the 240 dose is more than I have ever used.

Based on my hospital's acquisition cost of $0.48 per mL, a 70-kg patient would use $92.16 at 20 mg/kg/min, $241.92 at 50 mg/kg/min, and $483.84 at 100 mg/kg/min, still in the middle of the midazolam range in Table 5 by Mineanda and Broyles. In most cases, if more than 100 mg/kg/min is needed, a small dose of another drug, perhaps even a neuromuscular blocker, would be more suitable as well as cost-effective.

Finally, I agree with the comments by Mineanda in his letter in Critical Care Medicine that the most practical use of propofol is to attain specific, short-term sedative goals in select patients. However, I disagree with his statement that the "cost of propofol is three to four times that of comparable doses of midazolam," and this is inconsistent with his own data.

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REFERENCE

To the Editor:

We appreciate the interest and concern of Dr. Sherman regarding propofol for ICU sedation (CHEST 1995; 108:539-48). We also welcome the opportunity to clarify the issues raised.

The concerns over dosage ranges mentioned by Dr. Sherman are obvious and noted repeatedly in the review. Table 1, in fact, thoroughly describes recommended dosage guidelines for propofol use in the ICU setting and concludes with the warning that long-term use should attempt to be limited at 50 pg/kg/min. This goal is usually not difficult to attain in the cardiac surgical ICU patient, though, unfortunately, it may need to be exceeded in medical or trauma patients, patients with relative contraindications to neuromuscular blockade, or those patients exhibiting tolerance to ongoing sedation. Though we certainly do not recommend dosages of 120 to 240 pg/kg/min for long-term sedation in any ICU patient (nor does the manufacturer), the point of including such (referenced) dosages in Table 5 is to emphasize the possibility of a number of side effects seen with such dosages; one side effect being a significant pharmacy cost. As mentioned in the review, the physician who finds himself prescribing such high doses should review the particular sedation goals in his patient and adjust infusion rates using appropriate adjuncts.

The issue of pharmacy cost of propofol is an important one, though one that is often difficult to address due to variations in acquisition cost both geographically and chronologically. In an effort to remain unbiased, Table 5, which lists typical pharmacy costs for sedation, is referenced1,2 and based on the most recent data available at the time of writing. Though the acquisition cost and methods used by these authors may be different than that used by Dr. Sherman, our own acquisition cost leads us to similar cost/24 h for midazolam as stated in Table 5 as well as the "high dosage" of propofol. As Dr. Sherman has shown, it is worthwhile for practitioners to arrive at their own data based on cost at their particular institutions.

Finally, we should emphasize that our goal in writing the review was to present the most recent data on propofol sedation in the ICU to the ICU practitioner as objectively as possible. Unfortunately, objective data on cost-benefit analysis are difficult to find. The study by Carrasco et al,3 which found that the pharmacy cost of propofol was consistently 3% to 4% at four times that of midazolam, was undertaken at one particular institution (in Spain), during a finite period of time and by certain methods. These data may or may not extrapolate to any one particular institution or practice pattern. As such, we would hope that more objective data are generated, however difficult or complex, concerning the cost-effectiveness of propofol, or other sedatives, when used in the ICU.

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