

Tidal Volumes during General Anesthesia

Size Does Matter!

IN this issue of ANESTHESIOLOGY, Lellouche *et al.*¹ report on the results of a prospective observational trial that investigated the effects of low, traditional, and high tidal volumes (less than 10, 10–12, and more than 12 ml/kg of predicted body weight [PBW], respectively) delivered after admission into the intensive care unit on the development of organ dysfunction after cardiac surgery. Organ dysfunction was defined as mechanical ventilation exceeding 24 h (prolonged mechanical ventilation), use of vasopressors or inotropes for more than 48 h after surgery (hemodynamic instability), or increase in creatinine levels more than 50 μ M after surgery compared with baseline values (renal failure), according to the Society of Thoracic Surgeons.[‡] Authors found that low tidal volumes, *i.e.*, less than 10 ml/kg of PBW, have been used in only about 21% of 3,434 patients included in the study, whereas the vast majority received tidal volumes more than 10 ml/kg, or even more than 12 ml/kg of PBW. Such findings are not really surprising and mirror clinical practice worldwide, where protective ventilation has been regarded as superfluous, because uninjured lungs shall be robust enough to overcome tidal volumes of that magnitude. The striking finding by Lellouche *et al.*,¹ however, is that such common practice (tidal volumes of more than 10 ml/kg of PBW) was associated with prolonged mechanical ventilation, hemodynamic instability, higher incidence of renal failure, and prolonged stay in the intensive care unit among patients who underwent cardiac surgery, compared with low tidal volumes. In addition, high tidal volumes (more than 12 ml/kg of PBW) were an independent risk factor for multiple organ failure. Organ failure was associated with increased intensive care unit stay, hospital mortality, and long-term mortality. Authors concluded that prophylactic protective ventilatory strategies should be provided in populations with an inflammatory



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different levels of stress depending on the resting end-expiratory lung volume at airway pressure of 0 cm H₂O. Thus, a tidal volume higher than 10 ml/kg may not induce harmful stress, provided the resting end-expiratory lung volume is also proportionally high. Recent studies in healthy animals showed that ventilator-associated lung injury develops only when a strain greater than 1.5–2 is reached or exceeded,³ and stress reaches the total lung capacity.⁴

Inflammation seems to modulate the response of alveolar epithelial cells to strain.⁵ Accordingly, the release of inflammatory cytokines as a response to mechanical stress is very dependent on previous activation of the inflammation signaling. In clinical practice, such phenomenon has been translated as “double hit theory,” and high tidal volumes need an “inflammatory background” or previous injury in order to be harmful. Taken together, these mechanisms suggest that there is no need for use of protective mechanical ventilation in the absence of previous lung injury.

How then to explain the findings by Lellouche *et al.*¹ in light of that body of knowledge? First, the concept of stress

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‡ www.sts.org. Accessed March 14, 2012.

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◆ This Editorial View accompanies the following article: Lellouche F, Dionne S, Simard S, Bussi eres J, Dagenais F: High tidal volumes in mechanically ventilated patients increase organ dysfunction after cardiac surgery. ANESTHESIOLOGY 2012; 116:1072–82.

and strain must take into account that displacement (tidal volume) is not uniformly distributed across the lungs. Thus, the stress resulting at the interface of zones with different elastic properties may exceed safety limits in presence of lung inhomogeneities.⁶ Accordingly, it is conceivable that nonhomogeneous ventilation and stress distribution occur in lungs that have been kept silent for hours, even if they have been partially opened by recruitment maneuvers. Second, a first hit, *i.e.*, previous inflammatory state, is commonly present in cardiac surgery. The use of extracorporeal circulation during cardiac surgery leads to a systemic inflammatory response that mimics sepsis, likely priming the lungs for a second hit, namely high tidal volumes. These findings are in line with those recently reported in a prospective randomized controlled trial showing a reduction in ventilation-free days and need for reintubation when lower *versus* higher tidal volumes were used during cardiac surgery.⁷

The limitations of the study by Lellouche *et al.*¹ are numerous. First, the authors did not perform any intervention and solely observed the consequences of the ventilatory strategies chosen. Second, lower tidal volumes have not been accompanied by increases in positive end-expiratory pressure, and high degrees of lung atelectasis may have resulted. Third, classification of tidal volumes was somewhat arbitrary, especially for low values. In the context of protective ventilation, “low” means in reality physiologic tidal volumes, which are situated around 8 ml/kg of PBW, in average. Fourth, ventilation setting during the cardiac surgery, and more specifically during the cardiopulmonary bypass, as well as records of positive end-expiratory pressure or plateau pressures in the intensive care unit, have not been reported. Fifth, cardiac surgery with extracorporeal gas exchange is associated with proportionally high systemic inflammation compared with other surgery procedures.

A recent paper showed that the cardiac surgical population is at a higher risk of postoperative pulmonary complications, compared with other surgical populations.⁸ Thus, one would conclude that protective ventilation with low tidal volumes is not required in a general population of surgical patients. In our opinion, however, such a conclusion is flawed.

There are several factors that may lead to an increased release of inflammatory cytokines during surgery. Presence of infection, manipulation of bowel, low perfusion states (ischemia/reperfusion), and aggressive blood substitution are some examples. Sometimes the anesthesiologist will be surprised by one of these complications, which are not always predictable. Also, surgery may take longer than expected, increasing the susceptibility for postoperative pulmonary complications, especially in the elderly.⁹ Thus, why not use low tidal volumes in the majority of patients, making higher tidal volumes the exception? Is the fear of development of atelectasis driving the choice for higher tidal volumes? The data from the present study¹ suggests that such fear is un-

founded, or at least that the deleterious effects of increased mechanical stress are worse than those of atelectasis. Another aspect that deserves much attention is the determination of PBW. As shown by Lellouche *et al.*,¹ anesthesiologists frequently set inappropriately high tidal volumes, particularly in female and obese patients.

Certainly, clinical trials addressing the use of protective ventilation with low tidal volumes in the general surgical population are required. However, we do believe that until such trials have been conducted, and provided there are no contraindications, tidal volumes less than 10 ml/kg set according to predicted rather than actual body weight should be the standard. For now, there is enough evidence and a strong rationale to support those recommendations. For the future, the questions to be addressed are how low tidal volumes have to be and how much positive end-expiratory pressure is needed during general anesthesia.

Paolo Pelosi, M.D.,* Marcelo Gama de Abreu, M.D., M.Sc., Ph.D., D.E.A.A.† *Department of Surgical Sciences and Integrated Diagnostics, University of Genoa, Genoa, Italy. ppelosi@hotmail.com. †Department of Anesthesiology and Intensive Care Medicine, Pulmonary Engineering Group, University Hospital Carl Gustav Carus, Dresden University of Technology, Dresden, Germany.

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