Con: Low Tidal Volumes Are Indicated During One-Lung Ventilation

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The management of one-lung ventilation is aimed at prevention of arterial hypoxemia. Generous inspired oxygen concentrations and adequate tidal volumes are usually deemed the most important components. Most textbooks advise retaining the same tidal volumes of 10–12 mL/kg (1) as with normal two-lung ventilation. This choice is based on the notion that tidal ventilation less than 8 mL/kg predisposes to atelectasis, whereas larger tidal volumes (15 mL/kg) may produce alveolar over-distension, compress alveolar vessels, and increase pulmonary vascular resistance in the ventilated dependent lung. The latter may, in turn, adversely divert some blood flow to the collapsed nondependent lung.

Recent speculations about volume-related lung injury have questioned whether tidal volumes of 10 mL/kg may themselves be inappropriately large even for two-lung ventilation. Concern about large volume ventilation was originally generated by animal studies suggesting that alveolar over-inflation, and not high airway pressure, was important in the development of permeability pulmonary edema (2). Analysis of extensive trials (3) led to recommending smaller tidal volumes to prevent lung injury attributed to stretch damage of lung parenchyma with large tidal volumes. Examination of low tidal volume (5–7 mL/kg body weight [BW]) trials in patients with acute lung injury and acute respiratory distress syndrome (ARDS) actually found no consistent benefits over standard ventilator therapy with inspiratory plateau pressures of 28–32 cm H_2O (4). Regimens limiting tidal volume and over-expansion may actually be associated with development of alveolar instability and lung collapse (5). The latter may be the principal reason for the caution in extrapolating the use of low tidal volume ventilation to patients without ARDS (6). Such patients are also exemplified by anesthetized individuals, in whom reduced tidal volumes could very well promote atelectasis formation (7).

During one-lung ventilation, reduced tidal volumes (5–6 mL/kg) are aimed at avoiding the alveolar over-distension or stretching of lung parenchyma by the volumes of normal two-lung ventilation. Oddly enough, the same strategy with ventilation of two lungs often resulted in dynamic hyperinflation and intrinsic positive end-expiratory pressure (PEEPi) (7) because of the accompanying need for increased respiratory rates. The PEEPi itself is not a cause of the hyperinflation, but rather, a result of the same. The pressure actually represents the recoil pressure of the respiratory system at the elevated end-expiratory position above true functional residual capacity. The volume increase results largely from the reduced time available for expiration.

This would suggest that simply avoiding the alveolar stretch associated with periodic expansion and collapse of normal tidal volumes may not be as important as providing for alveolar recruitment and uniform alveolar distension. Both are most closely related to mean airway, or more specifically, mean alveolar, pressure. It is also important to distinguish between mean airway pressure and another important pressure, the plateau pressure. This is the pressure during an end-inspiratory pause and reflects respiratory system quasi-static compliance. The former is a composite of the time-related integration of pressure during the entire respiratory cycle, including expiration. Thus, although mean airway pressure tends to track plateau pressure, it also includes peak pressure, and any positive airway pressure during expiration (PEEP). Increased plateau

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pressures can result from excess tidal volumes, bronchospasm, or any decreases in respiratory system compliance, such as might occur with inadequate muscle relaxation. Reductions of plateau pressure can be achieved simply by addressing any of these variables.

If tidal volumes are reduced with one-lung ventilation, mean alveolar pressures are usually decreased and must be increased by the application of PEEP to preserve adequate arterial oxygenation (8). In such cases, the end-inspiratory volumes may actually approach those with standard tidal volumes because of the associated increased mean airway pressure.

If, on the other hand, tidal volumes for one-lung ventilation are not reduced from those with two-lung ventilation (10 mL/kg), both peak and plateau pressures will increase during volume ventilation with an end-inspiratory pause. The peak pressure is often an artifactual reflection of true airway pressure because of the site of its measurement proximal to the airway entrance, i.e., the endotracheal tube. In contrast, the plateau pressure during an end-inspiratory pause is a truer reflection of the momentary pressure to which the distal lung areas are exposed. In the absence of PEEP it is the most effective means of increasing mean airway pressure to provide uniform alveolar distension. A major limitation of the inspiratory pause is realized when inspiratory flow delivery rates are low, and the pause itself encroaches on expiratory time by extending the duration of inspiration. The latter may then predispose to dynamic hyperinflation (PEEPi), especially in the presence of a double-lumen tube and when obstructive lung disease is present (9). This results from the shortened expiratory time in the presence of decreased elastic recoil, i.e., increased compliance (C) and increased airway resistance (R). Both of these factors increase the time constant for passive exhalation, which is the product of $R \times C$.

The clinical efficacy of the end-inspiratory pause depends on its adequate duration as a fraction of the inspiratory time (40%–50%) to produce a mean airway pressure in the 10–12 cm H_2O range. This is achievable during volume ventilation with commonly available anesthesia machines. Any encroachment on expiratory time can be minimized by a decreased respiratory rate (8–10/min) and an increase in inspiratory flow rates. The decreased rate is the most effective means of increasing expiratory time by increasing total cycle time. For example, at a respiratory rate of 8 breaths/min the total cycle time is 7.5 s. With a I:E ratio of 1:2, inspiratory time is 2.5 s and expiratory time is a very generous 5 s.

As inspiratory flow rates are increased, the peak pressures will increase but plateau pressures during the inspiratory pause will not. Conventional wisdom has long expressed concern about using increased inspiratory flows in patients with chronic obstructive pulmonary disease because of their increased airway resistance. However, such patients actually exhibit a more normal uniform distribution of ventilation with rapid inspiratory flow rates in contrast to normal subjects (10). This suggests that differences in regional compliance are far more important than regional airway resistances in chronic obstructive pulmonary disease patients and tends to support the rationale for using an end-inspiratory pause in such patients. The pause itself functions by compensating for heterogeneity of airway time constants that result primarily from variations in regional compliances. One simply has to witness the simple manual reinflation of a collapsed lung during a thoracotomy to appreciate this concept. There is a continuous wave of segmental and lobar expansion with the continued application of sustained pressure. The more uniform distribution of ventilation results in expansion of more areas with normal time constants and thus facilitates more rapid and efficient expiration.

When deciding on appropriate volumes for any ventilation mode, it is important to consider several factors. The first is the important relation of lung volume to height as opposed to weight. This is particularly important in short obese individuals. Here one should use either ideal or predicted BW (4). The former is approximated in kilograms by the height in meters squared times 25, whereas the latter is estimated as 50 kg (males) or 45 kg (females) + 2.3 times the height in inches minus 60. Thus for example IDEAL BW = height (m^2) $\times$ 25, and PREDICTED (male) BW = 50 + 2.3 (height in inches $-$ 60).

A more important consideration is that set and delivered tidal volumes may not actually be equal. This is especially true with single-lung ventilation when total respiratory system C is by necessity decreased and lung expansion may be less than anticipated. The use of smaller volumes usually requires faster respiratory frequencies. Here the possibility of lung over-distension from air trapping or dynamic hyperinflation is more likely a result of the airway resistance imposed by the double-lumen tube and as a result of the inherent obstructive lung disease with its increased airway R and decreased lung recoil. Thus end-inspiratory as well as end-expiratory volumes may exceed intended goals.

Finally and most importantly, the use of reduced tidal volumes during one-lung ventilation will invariably require the use of PEEP to increase mean airway pressure in an attempt to provide for uniform alveolar expansion and maintain adequate oxygenation. In such cases end-inspiratory volumes may, in fact, closely resemble those associated with standard tidal volumes, as the final mean airway pressures are likely to be comparable.

For these latter two reasons and the fact that lung protection strategies for ventilation in the operating room are unproven, the author argues against any

need to reduce tidal volumes during one-lung ventilation from those with standard two-lung ventilation. The larger volumes should be maintained because they provide for more controllable and reliable conditions for optimal gas exchange.

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