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Influence of tidal volume on stroke volume variation. Does it really matter?

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Sir: We read with interest the study by Reuter et al. [1] illustrating the influence of tidal volume (V_t) on stroke volume variation (SVV) evaluated by pulse contour analysis. The SVV—evaluated either by pulse contour or arterial pressure waveform analysis—has been proposed to assess the sensitivity of the heart to changes in cardiac preload (i.e. the slope of the Frank-Starling curve) and, hence, to detect fluid responsiveness in mechanically ventilated patients [2, 3, 4].

The SVV results from the effects of mechanical insufflation-induced changes in pleural and transpulmonary pressures on cardiac preload and, hence, for a given compliance of the respiratory system, depends necessarily on V_t . In this regard, the lack of significant SVV (<10%) does not always mean that a patient is insensitive to changes in preload since it can also reflect that cardiac preload does not change significantly during mechanical insufflation. Therefore, caution should be exercised when using SVV in the decision-making process concerning volume expansion in patients ventilated with a very low V_t . However, as far as we know, such a ventilatory strategy is not recommended in patients with shock and remains questionable in patients with ARDS [5].

Moreover, we strongly believe that fluid responsiveness also depends on V_t . Indeed, increasing V_t will result in increasing the mean airway pressure and, hence, in decreasing the mean cardiac preload (leftward shift on the Frank-Starling curve). Therefore, a patient operating on the flat portion of the Frank-Starling curve (i.e. insensitive to changes in preload) may operate on the steep portion (and hence become sensitive to changes in preload) if the V_t is increased. To illustrate this phenomenon, we analyzed previously published data from 40 patients with septic shock in whom arterial pulse pressure variation (as a surrogate of SVV) and cardiac output were evaluated before and after a standardized volume load [3]. We assigned the 20 patients ventilated with a V_t of less than 670 ml (median value) to the “low” V_t group

□ Pre-infusion pulse pressure variation (%)
■ Fluid-loading induced increase in cardiac output (%)

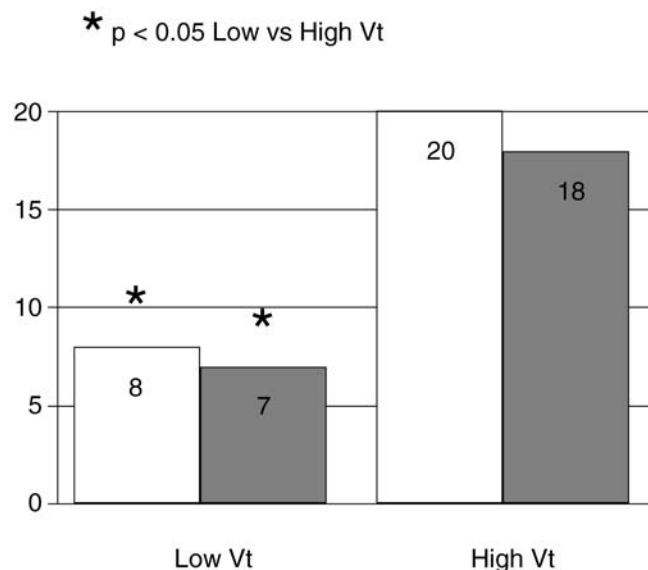


Fig. 1 Pre-infusion arterial pulse pressure variation and fluid-loading induced increase in cardiac output in the “low” and the “high” tidal volume (V_t) groups

and the 20 remaining patients to the “high” V_t group. In accordance with the findings of Reuter et al. [1], the pre-infusion pulse pressure variation was greater (20 ± 11 vs $8 \pm 5\%$, $p < 0.05$) in the “high” V_t group (Fig. 1) and correlated with V_t ($r = 0.46$, $p < 0.01$). Interestingly, the fluid loading-induced increase in cardiac output was also greater (18 ± 13 vs $7 \pm 7\%$, $p < 0.05$) in the “high” V_t group (Fig. 1) and weakly, but significantly ($r = 0.35$, $p < 0.05$), correlated with V_t .

These findings support the notion that the V_t influences not only the SVV but also the hemodynamic response to volume loading. This may explain why the SVV has been found to be a reliable predictor of fluid responsiveness in patients with a V_t ranging between 8 and 15 ml/kg [2, 3, 4]. Therefore, we believe that the influence of V_t on SVV cannot be considered as a major limitation to its use as a predictor of fluid responsiveness in most mechanically ventilated patients.

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