Choosing Tidal Volume for Ventilation

To the Editor:

Lee and associates' describe a study in which they compared two groups of patients ventilated with either high or low tidal volumes. While the data presented are interesting, there are several unanswered questions which might be clarified by the reporting of additional data.

The paper does not specify how the tidal volumes used were determined. This is important as there are often disparities between the set and delivered tidal volumes on adult volume ventilators. The most common cause of this disparity results from tidal volume not delivered to the patient because of compressible volume loss within the ventilator-circuit system. The amount of volume lost in this manner varies between makes of mechanical ventilators and between different types of ventilator tubing circuits. Unless the investigators accounted for compressible volume loss when determining the tidal volumes used in the study, the result could be that at least four groups of patients are being compared. In this paper, the majority of patients were ventilated either with a Bear 1, which does not compensate for compressible volume loss, or with a Puritan-Bennett 7200a, which does. Typical ventilator-circuit compliance for the Bear 1 is about 5 ml/cm H2O, though this may vary from 4 to 7 ml/cm H2O with individual types of tubing. In a 70 kg patient, the study design would dictate that the patient receive either 540 ml or 420 ml. Using the mean of the airway pressure changes presented in the paper (29.6 and 20.0 cm H2O for each group), one can calculate an average compressible volume loss of 148 and 100 ml for each group. These losses would result in a delivered tidal volume of 9.9 ml/kg and 4.6 ml/kg if the hypothetical 70 kg patient were ventilated by the Bear 1, compared to 12 ml/kg and 6 ml/kg if the patient were ventilated by the Puritan-Bennett 7200a. As a result, this study would still be comparing high and low tidal volume groups; however, the actual volume disparity would be greater with higher peak airway pressures as in patients with reduced lung/chest compliance or increased airway resistance. To prevent this effect from confusing the study results, data comparing the 4 groups should be presented if compressible volume loss was not taken into account with the original study design. It therefore would be valuable to see data comparing the findings of mean peak pressures and mean PEEP separated by ventilator used and by tidal volume groups to see if the differences between the groups is enlarged or narrowed if ventilator used is controlled.

It is not clear how the rate of infection was determined. For example, was incidence of infection during the time of ventilation measured, was incidence for the time period of the study measured, or was rate of infection per unit of time measured? The question is whether the increased number of infections in the high tidal volume group related to the higher tidal volume used, merely a result of the patient's being mechanically ventilated longer thus having greater exposure to nosocomial infection, or perhaps, a result of a longer time in the study for culture results to become positive? A further explanation of how infection rate was determined would be helpful.

Finally, it was not stated in the methods section whether the practitioners making the decision to extubate patients were blinded to the tidal volume group in which their patients were members. If they were not blinded then it is, of course, possible that their

References


3 Hooper RG, Kearl RA. Established ARDS treated with a sustained course of adrenocortical steroids. Chest; 1990; 97:138-43

4 Asbaugh DC, Maier RV. Idiopathic pulmonary fibrosis in adult respiratory syndrome. Arch Surg 1985; 120:530-35
knowledge of group membership could have influenced their decision as to when to extubate, making the data less reliable. Admittedly, blinding the practitioners to the tidal volumes used would be difficult to accomplish. It might have been better if a strict criteria were used (and published) to determine the time of extubation.

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REFERENCES
1 Lee PC, Helmoort CM, Cohn SM, Fink, MF. Are low tidal volumes safe? Chest 1990; 97:430-34
3 Puritan-Bennett 7800A operating manual

To the Editor:

We appreciate the opportunity to respond to Dr. Elton's letter. Tidal volume ($V_t$) was determined on the basis of delivered volume rather than the numerical setting on the ventilator. $V_t$ was readjusted by the respiratory therapist every 3 to 4 h as necessary. Thus, $V_t$ was determined by group assignment and body weight, but was not affected by the model of ventilator employed.

Rates of infection were determined for the time period that patients were in SICU. It is possible that the trend toward an increased number of infections in the high (12 ml/kg) $V_t$ group was simply due to a longer period of risk. In any event, the effect of $V_t$ assignment on the incidence of infection was not statistically significant and conclusions regarding this phenomenon must await results from a study specifically designed to address this issue.

The physicians making the decision to extubate patients were not blinded to $V_t$ group assignment. We grant that this design presented an opportunity for bias. However, blinding in a trial of this sort is virtually impossible. Using a rigid protocol to determine suitability for extubation is also impractical since many qualitative factors (eg, nature of secretions) figure into the decision.

To the Editor:

Dr. Butler's essay, "The Heart Is Not Always in Good Hands," is a worthwhile contribution to our understanding of his pioneering concept of the cardiac fossa. I wish to draw attention, however, to some erroneous material in an otherwise valuable presentation. Pulsum paradoxus is not paradoxo because of the inspiratory fall in left ventricular output when venous return is increasing. It seemed paradoxo to Kussmaul, who first described it in 1873, when he noted intermittent loss of the radial pulse despite a constant apex beat that occurred during inspiration. It is not surprising that this should be the case during increased filling of the right ventricle. Of many mechanisms contributing to pulsum paradoxus (at least during cardiac tamponade), perhaps the most important is increased inspiratory right heart filling. The right heart chambers expand, raising pericardial pressure, which further increases the external compression of the left ventricle. At the same time, the ventricular septum bulges to the left, compressing the left ventricle internally. Thus the LV is simultaneously "clamped" between internal and external compression and therefore resists filling, consequently reducing its output during inspiration. Dr. Butler also believes that "the traditional idea that the normal pericardial sac limits cardiac expansion is probably wrong," citing open-chest studies in supine animals during retraction of the cardiac fossa. That is not quite correct. The normal pericardium acutely limits cardiac expansion when the right ventricle (RV) is infarcted, the limitation being such that the pericardium becomes tightened by RV dilatation and acts just like a constricting pericardium, producing the hemodynamic pressure configuration of constrictive pericarditis. Furthermore, non-tamponading, often quite small, increases in pericardial fluid increase the coupling of the heart to the parietal pericardium and detectably exaggerate the respiratory dynamics of normal hearts.

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REFERENCES
1 Butler J. The heart is not always in good hands. Chest 1990; 97:453-60
4 Spodick DH, Paladino D, Flessia AP. Respiratory effects on systolic time intervals during pericardial effusion. Am J Cardiol 1985; 51:1033-55

To the Editor:

I appreciate Dr. Spodick's comments, which are interesting and apropos. He shows the breadth of his knowledge concerning cardiopulmonary interactions in pointing out that pulse paradoxus was called paradoxo by Kussmaul because of the loss of the radial pulse with a constant apex beat. He goes on to say that the most important mechanism of pulse paradoxus is increased inspiratory right heart filling. I mention this in my article, although I find it difficult to be sure that it is the "most important" mechanism. The analogy of clamping the left ventricle between the right ventricle and the wall of the cardiac fossa, so its output falls because the right ventricle swells during inspiration, must be a diastolic phenomenon, if true. I have always found it difficult to imagine the sturdy left ventricle discombobulated by the thin-walled right ventricle unless hypertrophic cor pulmonale is present.

Dr. Spodick is, of course, very knowledgeable about the pericardial sac and I agree with his comment that I overstated the problem with the traditional idea that the pericardial sac limits cardiac expansion. My point is that it does not normally limit cardiac expansion. It certainly prevents sudden, marked cardiac dilation, which would lead to myocardial paralysis if allowed to occur.

Finally, I am puzzled by his last sentence that non-tamponading, often quite small, increases in pericardial fluid increase the coupling of the heart to the parietal pericardium. It seems to me that the heart is fluid coupled to the parietal pericardium under normal conditions.

Again, I would like to thank Dr. Spodick for his helpful comments.

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