Communications

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Assessing the Work of Breathing

To the Editor:

In their article "Pressure Support Compensation for Inspiratory Work due to Endotracheal Tubes and Demand Continuous Positive Airway Pressure" (Chest 1998; 93:499-505), Fustro et al made several errors, calling into question their analysis and conclusions.

In the case of the mechanical model, the wrong pressures were used to calculate inspiratory work. The integral \( \int P_{\text{aw}} \, dV \) was used as in Fig 2) to estimate pump work \( (W_{\text{aw}}) \) from the area under the curve when \( P_{\text{aw}} \) was negative, ventilator work \( (W_V) \) from the area under the curve when \( P_{\text{aw}} \) was positive and their difference, "net airway work" \( (W_{\text{aw}}) \). The authors' assumption that a negative \( P_{\text{aw}} \) implied that only the pump was doing work and a positive \( P_{\text{aw}} \) implied that only the ventilator was doing work is patently incorrect since the pump "pulled," the ventilator "pushed" and both worked simultaneously throughout inspiration. \( P_{\text{aw}} \) was merely the net result of the pump "pull" and the ventilator "push." While it is true that the pump was doing more work than the ventilator when \( P_{\text{aw}} \) was negative, it was certainly not doing all the work. Actually, neither pump nor ventilator contribution to \( P_{\text{aw}} \) can be calculated from \( P_{\text{aw}} \) alone. Therefore, neither pump work, ventilator work, total work nor true net airway work can be estimated from \( \int P_{\text{aw}} \, dV \) and most of the authors' conclusions are flawed.

Since \( W_{\text{aw}} \) and \( W_V \) were determined incorrectly, the authors' "optimal pressure support" is meaningless. Even if they correctly calculated work, it is optimized by making ventilator and pump pressures equal is not clear.

In calculating "net airway work" \( (W_{\text{aw}}) \), the authors subtracted work (incorrectly) attributed to the ventilator \( (W_V) \) from work \( (\text{incorrectly)} \) attributed to the pump or respiratory system \( (W_{\text{aw}}) \). The intended significance of this is totally unclear. During most assisted breaths, this subtraction would result in negative work, since ventilator work is usually greater than inspiratory muscle work. The sum \( \int (P_{\text{aw}}-P_{\text{aw}}) \, dV \) is the total work on the system. The authors' \( \int (P_{\text{aw}}-P_{\text{aw}}) \, dV \) is totally different.

Errors were also made in the human subject studies. Inspiratory work on the lungs is \( \int (\text{transpulmonary pressure}) \, dV \) or \( \int (P_{\text{aw}}-P_{\text{aw}}) \, dV \). Unless \( P_{\text{aw}} \) is zero, as it is only during spontaneous breaths, inspiratory work cannot be calculated from \( P_{\text{aw}} \) alone. The authors assumed that it could be, while incorrectly using only \( P_{\text{aw}} \) in their calculations. The authors' graphic integration of the area under the \( P_{\text{aw}}-V \) curve therefore cannot be proportional to either inspiratory work done by the inspiratory muscles, inspiratory work done by the ventilator, or total work, their sum. (I appreciate that this error has appeared in print before.)

Nor do the authors attempt to determine how much of the subjects' inspiratory work was done by the inspiratory muscles and how much by the ventilator. Subjects are limited by what pressures and work they can generate themselves. Ventilator work is potentially limitless. Total work estimated by the authors is therefore of little significance, since most of it was likely done by the ventilator, as is usual during most assisted breaths.

Another error was the use of \( \Delta P_{\text{aw}} \) rather than \( P_{\text{aw}} \) to calculate pressure support work. Physical work is \( \int P \, dV \) and must therefore be calculated from absolute values of \( P_{\text{aw}} \). Some simple graphics will demonstrate that areas under the \( P_{\text{aw}}-V \) and \( \Delta P_{\text{aw}}-V \) curves can be grossly different.

The authors' plot of \( \Delta P_{\text{aw}} \) against "tidal volume" in Figure 7 suggests that \( \Delta P_{\text{aw}} \) was zero at both the beginning and end of a spontaneous inspiration. This is patently impossible, since endotracheal pressure must change from the beginning to the end of an inspiration, whether spontaneous or pressure-supported. Interpretation is complicated by the fact that \( \Delta P_{\text{aw}} \) was not defined.

In estimating the effect of pressure support on work, the authors ignored work on the chest wall, roughly one-half the work done under normal conditions. Adding chest wall work to lung work to obtain total work (what the body is interested in) would decrease percentage changes considerably.

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To the Editor:

We appreciate the thoughtful comments of Dr. Simmons regarding our article, and are pleased to have this opportunity to respond.

Regarding our methods and data used to calculate airway work using the mechanical model, we respond as follows: work is the product of pressure and volume. Therefore, flow-resistive work on the airway equals \( \int P_{\text{aw}} \, dV \). By this definition, areas subtended by the PV curves when \( P_{\text{aw}} \) was negative and positive clearly represent the net work done on the airway by the respiratory system (ie, mechanical pump, \( W_{\text{aw}} \)) and ventilator (\( W_V \)) respectively. In this phase of the study, we were only interested in work on the airway. Ours is an accurate method of evaluating the airway contribution to work and has precedent in the literature.1

We have defined "net airway work" \( (W_{\text{aw}}) \) as the arithmetic sum of these values \( (W_{\text{aw}} + W_V) \) for a given inspiration. When inspiration through an endotracheal tube (ETT) and ventilator circuit took place without pressure support (PS, example in Fig 2), airway pressure was negative throughout inspiration and clearly represented additional work by the breathing model (Fig 2A). As PS was added (Fig 2B, C), \( W_{\text{aw}} \) was progressively decreased but not totally eliminated, even at high levels of PS (as in standard pressure-cycled mechanical ventilation, Fig 2C). This occurs because of negative pressures needed to overcome initial demand valve resistance and time required to initiate and maintain the set level of PS. When increasing PS, \( W_{\text{aw}} \) increases as \( W_{\text{aw}} \) decreases. Though \( W_{\text{aw}} \) is clearly done in early inspiration and cannot be recovered, \( W_V \) exceeds this and assists the remainder of inspiration.

The ideal system for eliminating the added work due to the resistance of an artificial airway (Figure 2A) would be one which totally eliminates any airway pressure deflections (\( \Delta P_{\text{aw}} \)). The optimal mechanical system would have to anticipate and perfectly match patient flow demand to accomplish this. We determined that a level of PS sufficient to reduce \( W_{\text{aw}} \) and which did no more than equally
oppose it during late inspiration with \( W_i \) (ie, \( W_{iS} = W_i \) and \( W_{iaw} = 0 \)) was a reasonable approximation of this ideal system (Fig 2B).

Subsequent human subject studies were performed in part to test the mechanical model results which were based on this reasoning. It is possible that another PS system which initiates, maintains and/or terminates pressure sooner would be better suited to this function, but the PS system used was not variable in this way.

Regarding the study of human subjects, it is true that work on the lungs is \( f(P_{AW} - P_{AW}) \) dV. However, this work is a sum of work done by the subject \( f(P_{AW} - P_{AW}) \) dV and the work done by the CPAP system \( f(P_{AW} - P_{AW}) \) dV. We were interested in the work done by the subject on the lung and artificial airway and, therefore, measured \( f(P_{AW} - P_{AW}) \) dV. This rationale has been previously outlined.  

Dr. Simmons questions our use of \( \Delta P_{ES} \) rather than absolute \( P_{ES} \). If one uses absolute values of \( P_{ES} \) in computing work, one includes quantities of elastic energy transferred from the chest wall to the lungs, in addition to work done by inspiratory muscles, thereby overestimating the latter (Campbell diagram). 

We did not use the chest wall PV curve to calculate the elastic portion of inspiratory work (Weil). We assumed that, in any one subject, the PV characteristics of the chest wall were constant between studies for practical purposes. Since the FRC did not change by the design of the study (subjects were trained to maintain \( V_t \) constant), this component of elastic work was common to all conditions of measurement. Therefore, changes in work due to airway mechanical factors could be reliably compared. As an approximation, the Weil can be estimated as: \( W_{el} = \frac{V_t}{C} \) (C = lung compliance; \( V_t \) = tidal volume). This method of work estimation neglects the PV curve of the chest wall in calculating Weil and, in essence, uses \( \Delta P_{ES} \) for calculation of elastic work as we have done. Other studies, including that which we cite to reference normal work values in the discussion, have measured work in this manner.

In reference to the matter of work on the chest wall, work done by inspiratory muscles to overcome the flow-resistance of the chest wall accounts for only a small portion of the work done by the inspiratory muscles as long as \( V_t \) does not exceed 50 percent of the vital capacity and cannot be directly measured using dynamic PV curves. It is usually neglected in similar studies of work. The comments dealing with Figure 7 do require clarification. In graphing our PV relationships, pressure and volume were plotted at 0.1 sec intervals (see legend). With added airway resistance (eg, 7 mm endotracheal tube and ventilator circuit, Fig 7B), \( \Delta P_{ES} \) quickly changed from positive to negative at end-inspiration and early expiration. The absolute peak \( V_t \) and \( \Delta P_{ES} \) at the exact point of end-inspiration was, therefore, often not plotted. Such plots cannot be used to evaluate accurately dynamic compliance of the respiratory system, but have a minimal effect on work measures (areas described by the plots). In fact, in the case of added PS (Fig 7C), \( \Delta P_{ES} \) was often a negative value at end-inspiration as PS was transmitted to the pleural space.

Although Dr. Simmons takes great exception to our measurement and interpretation of work values, he fails to note another measure of patient effort, peak changes in \( \Delta P_{ES} \) during inspiration. These data (Fig 9) parallel those of work (Fig 6) with \( P_{AW} \) increasing by magnitudes similar to those of work in the presence of airway resistances. As with work, these increases are eliminated by application of appropriate levels of PS.

Finally, as can be seen from Figure 8 in our paper, the “optimal PS” values determined in our human subjects studies were consistent with relationships of \( V_t/T_t \) to “optimal PS” values derived from the mechanical model, supporting its validity.

We believe that our results support our conclusions that PS can compensate for additional work due to the resistance of artificial airways.

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The Role of Transthoracic Needle Biopsy

To the Editor:

The recent article by Levine et al (Chest 1988; 93:1152-55) on the usefulness of transthoracic needle biopsy (TTNB) in the evaluation of a solitary pulmonary nodule (SPN) presents very important data, but I believe that the statement that “TTNB is a valuable procedure in patients with a SPN in whom fiberoptic bronchoscopy is negative” is not supported. If one is faced with a patient who has a pulmonary nodule and nondiagnostic flexible bronchoscopy, the major decision to be made is whether thoracotomy is necessary for diagnostic purposes. This decision is based solely on the likelihood that the lesion is malignant. If the clinical features of the case do not indicate clearly that the lesion is benign (eg, significant central calcification) then the usefulness of the needle biopsy is solely in its ability to prove that the patient has a benign lesion and therefore does not require thoracotomy. The data presented shows that TTNB is unlikely to do this, although results might differ in a population with a higher incidence of benign lung nodules.

A patient is benefited by a needle biopsy only if a benign diagnosis is made or if an indeterminate diagnosis on needle biopsy, along with the clinical data, is sufficient to preclude the need for thoracotomy. This procedure is also useful in the occasional patient who refuses to undergo a thoracotomy without proof that he has a malignancy, or a slightly more common situation where the patient is a high medical risk and one would prefer not to send him for thoracotomy without definite evidence of malignancy. Except in these particular situations, it seems to me that the procedure adds no useful clinical information.

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To the Editor:

As Dr. Binder suggests, the “value” of a procedure cannot be assessed in black and white, but in shades of gray. We are talking about specific patients in specific parts of the country, not “cases of SPN.”

Given that view, we do regard TTNB as a valuable procedure. Many patients we see prefer to know that they have a malignancy before submitting to a costly, painful procedure (which carries more risk than TTNB) like thoracotomy. We do not feel it is inappropriate for patients to refuse thoracotomy until TTNB is done. In fact, we think their desire to know as much as they can is appropriate. As our data show, TTNB often answers the patient’s question.

Further, as Dr. Binder notes, there are patients in whom