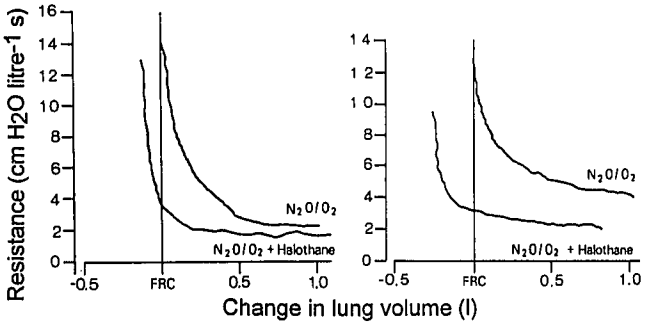

Fig. 1. To show the resistance *versus* lung volume curves in two different patients during anesthesia with nitrous oxide/oxygen/pancuronium before (N_2O/O_2) and after the addition of halothane. Despite the small increase in expiratory reserve volume (ERV) with halothane shown in the left panel, there is a large fall in resistance mainly due to the reduction in bronchomotor tone. In the patient shown on the right, the effect of change in ERV also causes a large reduction in airway resistance; again the change in shape of the curve shows a reduction in bronchomotor tone. (Redrawn.[3])

ERV causes a large reduction in airway resistance at FRC. In the examples shown in figure 1, there also is a reduction in bronchomotor tone as reflected by a change in shape of the hyperbolas from nitrous oxide/oxygen to nitrous oxide/oxygen plus halothane.[3] With neuromuscular blockade, the only explanation for such a change in lung volume with the addition of volatile agent is a shift in central blood volume. Although the effect on volume is small, the resultant decrease in airway resistance is very large and may be of considerable clinical benefit.

We are glad that Barnas *et al.* confirm our results on airway resistance in humans. It is possible that the small increase in resistance seen at higher lung volume in their studies may be due to the effect of airway lengthening. It would be of interest to see whether they find similar changes in lung volume with the addition of volatile anesthetics.

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In Reply:—We agree with Warner that one should not assume that respiratory mechanics measured in awake subjects necessarily reflect conditions in anesthetized patients. In fact, we emphasized this issue in the Discussion and mentioned several possible effects that anesthesia may have on lung mechanics, including those of inhalational anesthetics.[1] However, Warner's specific examples of studies showing why awake and anesthetized conditions differ are not relevant. He first cites three studies in which respiratory system mechanics were changed by anesthesia. This finding is not surprising, because all three studies measured mechanics of the thorax, not the lungs, and the variability in the degree of respiratory muscle contraction in the awake state may affect results. Specifically because of this possibility, we chose not to present data of mechanics on the thorax, even though they were measured. On the other hand, the data presented of lung mechanics will be independent of respiratory muscle activity. Warner also mentions a study[2] that investigated the relationship between lung volume and pulmonary resistance during halothane anesthesia. He suggests that the study proves that anesthesia affects the response to lung volume. Ironically, we cited the same study[1] to suggest the opposite. Obviously, the effects of inhalation anesthetics are controversial and, as we noted, need to be further studied. Furthermore, to imply, as Warner does, that this single study of the effects of halothane anesthesia on lung mechanics,[2] which itself is open to interpretation, proves that all studies in awake subjects are not applicable to the anesthetized state is misleading, because there are many other forms of general anesthesia whose effects on bronchomotor tone are not an issue. Indeed, as discussed, the response of lung mechanics to lung-volume changes we found in awake human subjects was qualitatively very similar to those we reported in dogs, anesthetized with barbituates and paralyzed.[3] One also must keep in mind that measurements of lung mechanics in anesthetized humans during the imposed decreases in lung volume we employed could be hazardous.

CORRESPONDENCE

Thus, the measurements we made in awake humans,[1] and which we verified in anesthetized dogs,[2] are probably the most reasonable approach.

Results from the elegant studies conducted by Lehane *et al.*[4] and Jordan *et al.*[5] agree qualitatively with our description of the relationship between lung volume and the resistive properties of the respiratory system and show, as we have noted,[1] that effects of inhalational anesthetics need to be considered. However, there are important differences between those studies[4,5] and ours[1] that make comparisons especially interesting. Those authors did not measure airway resistance directly, but instead measured total respiratory system resistance, which additionally includes the effects of lung-tissue and chest-wall tissue properties. Their measurements were made at a frequency (180/min) and tidal volume (58 ml) outside the physiologic range. As we discussed,[1] frequency and tidal volume affect airway and lung tissue properties in different ways. Moreover, we have previously shown[6,7] that chest wall tissue resistance displays significant frequency and tidal volume dependences. Although forcing waveform was kept constant in the studies cited by Lehane *et al.* and Jordan *et al.*, respiratory frequency was much higher and tidal volume was much lower than ordinarily would be used in patients. The relative contributions of airways, lung tissue, and chest wall tissue to the resistance measured and the effects of increasing lung volume did not necessarily reflect the effects of PEEP on lung properties in mechanically ventilated patients. We used forcing in a more physiologic range to facilitate comparisons to the use of PEEP, and we separated lung and chest wall properties with measurements of esophageal pressure.[1] The similarities between the results from Lehane *et al.* and Jordan *et al.* and ours may mean that frequency and tidal volume effects on airway, lung tissue, and chest wall tissue resistances are small in the ranges used for forcing in the studies. Alternately, the frequency, tidal volume, and lung volume effects on the resistances may be complex but counterbalance each other. It also must be pointed out that one cannot assume that changes caused by halothane are due to changes in airway resistance, because it has been shown that the degree of bronchoconstriction also affects lung tissue resistance.[8] Clearly, as suggested by Jones, there is still a need for further studies in this area.

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