is offset by an increase in mean systemic pressure, which restores venous return to previous levels.[2] Until intrathoracic pressure rises to a point beyond which compensatory mechanisms fail, there will be no change in cardiac output. A number of other factors (*e.g.*, changes in pulmonary vascular resistance) have been shown to have potential effects on cardiovascular function during positive-pressure breathing. However, changes in intrathoracic pressure consistently have been shown to be the most important in hemodynamic function.

In our experience with similar dog preparations,[3] circulatory hemodynamics during positive-pressure ventilation were unchanged from those measured during spontaneous ventilation, even at tidal volumes and airway pressures higher than those used by Muneyuki *et al.* Although the current study did not have a spontaneously ventilating control group, the reported hemodynamic measurements are similar to those we have observed. If, in fact, the increase in airway pressure with either mode of ventilation in the present study was not great enough to cause a change in hemodynamic status from that found during spontaneous ventilation, then it is not surprising that differences in airway pressures between alternating lung and synchronous ventilation were not associated with differences in hemodynamic status.

This point should be clarified, since it detracts from an obviously well-designed and well-executed study. If *mean* airway pressure during alternating lung ventilation is significantly lower than *mean* airway pressure during synchronous ventilation, there may be a difference in the hemodynamic effects of these two modes of ventilation under circumstances in which increased airway pressure does cause a decrease in cardiac output (*e.g.*, hypovolemia). Perhaps the authors will pursue their studies further and investigate this possibility.

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(Accepted for publication June 21, 1983)

Anesthesiology
60:75, 1984

In reply: In our study, mean airway pressure decreased significantly as well as peak airway pressure when the ventilation mode was changed from synchronous to alternating. Cardiac output, however, did not change significantly. This may be a result of the compensatory mechanisms of the cardiovascular system. It is also conceivable that the advantageous effects of lowered airway pressure during alternating ventilation were offset by unfavorable effects on the cardiovascular system. We agree that it is not surprising that differences in airway pressure between the two ventilation modes used in our study were not associated with significant changes in cardiac output, considering the responses observed by Dr. Otto and his colleagues. Lastly, it deserves emphasis that the purpose of our study was not to clarify the differences in cardiovascular functions between spontaneous and artificial ventilation modes but rather to observe any differences between synchronous and alternating ventilation modes under the conditions of artificial ventilation with complete muscle relaxation.

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(Accepted for publication June 21, 1983.)