Communications for this section will be published as space and priorities permit. The comments should not exceed 350 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Specific permission to publish should be cited in a covering letter or appended as a postscript.

Commenting on the ACCP Consensus Conference
Mechanical Ventilation

To the Editor:

The ACCP Consensus Conference on Mechanical Ventilation published in the December 1993 issue of Chest1 provides a useful overview of many important concepts. However, a few points merit further comment.

First, as the authors note, alveolar overdistention is increasingly recognized as the fundamental mechanism underlying ventilator-induced lung injury. Since there is no way to assess alveolar distention directly, measurements of pressure are often substituted. Among the terms used to characterize the respiratory system during mechanical ventilation are peak airway pressure (PAP), peak alveolar pressure, transalveolar pressure, transpulmonary pressure, and transthoracic pressure. These terms are not synonymous, and unfortunately are sometimes used imprecisely, causing confusion about the relationship between pressure and ventilator-induced lung injury. For example, it is stated in the discussion of adult respiratory distress syndrome that “animal studies have clearly established the damaging effects of overdistention produced by the application of high peak transthoracic pressures to normal and injured lungs.” This is incorrect: animal studies clearly show that high peak transalveolar pressures cause overdistention and lung injury, but high transthoracic pressures cause no injury when transalveolar pressure is low, as occurs when the chest wall is bound.2,4 Alveolar distention is a function of alveolar compliance and the transmural pressure applied to the alveoli. Hence, only the terms “static transpulmonary pressure” and “transalveolar pressure” reflect alveolar distention; the remaining terms bear no predictable relationship to alveolar distention.

Second, the statement that in asthma “a high PAP may also predict hyperinflation” is not quite correct. Although in asthma a high PAP may sometimes be associated with hyperinflation, it is just as likely not to correlate at all. This comes as no surprise, since PAP may or may not correlate with transalveolar pressure. In their study of patients with severe airflow obstruction undergoing mechanical ventilation, Tuxen and Lane5 found that PAP did not predict the magnitude of hyperinflation; in fact, they found PAP correlated inversely with the degree of hyperinflation.

Finally, the authors suggest the strategy of reducing Vt and increasing expiratory time in the management of patients with obstructive airways disease. In deciding whether to reduce Vt by reducing Vt or by reducing frequency, it is worth noting that in patients with severe obstructive airways disease, one gains relatively little by prolonging expiratory time beyond 3 to 4 s because of the extremely low flow rates late in expiration. As a rough example, an additional second for expiration might allow the exhalation of an additional 50 mL of gas, whereas a 100 to 200 mL reduction in Vt would have a much greater effect on minimizing hyperinflation.

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REFERENCES
5 Tuxen DV, Lane S. The effects of ventilatory pattern on hyperinflation, airway pressures, and circulation in mechanical ventilation of patients with severe air-flow obstruction. Am Rev Respir Dis 1987; 136:872-79

To the Editor:

We thank Dr. Harold Manning for his careful reading of the ACCP Consensus Conference: Mechanical Ventilation and his helpful comments. Dr. Manning brings up three points that I will comment on.

First, as stated in the Consensus document, we agree that overdistention is likely an extremely important mechanism for pulmonary barotrauma. In the experimental studies in the literature, this is usually “produced by the application of high peak transthoracic pressures . . .” as stated in the document. We do not mean to imply that high peak transthoracic pressures always produce alveolar overdistention. In the examples cited by Dr. Manning with regard to binding of the chest wall, transthoracic pressures are not increased since the relevant “outside” pressure is the pressure just outside the thoracic wall (underneath the binding), which is also elevated. We also agree that the most important pressure to measure in this regard is transalveolar pressure, but this is difficult to measure clinically, especially on a regional level.

Second, the issue of measuring the degree of hyperinflation in patients with severe airway obstruction is difficult. As stated a number of times in the document, we suggested that end-inspiratory occlusion pressure, ie, plateau pressure, is probably the best pressure to monitor with regard to the risk of overinflation. However, to the extent that the airway resistance to a specific lung unit is relatively very low, the peak pressure in that unit might reach values very close to the peak airway pressure. Hence, peak airway pressure may independently be a relatively important