Prolonged Artificial Ventilation
Are Tracheal Injuries the Only Problem?

To the Editor:

I wish to comment on the fine article entitled "Prevention of Tracheal Injuries in Prolonged Ventilation: Laboratory and Clinical Observations on the Use of Self-Inflating Cuffs on Ventilating Tubes" by Abouav and Finley, which appeared in the January 1977 issue (Ches 71:13-17, 1977). This timely article dealt with prevention of tracheal injuries secondary to cuff pressures on the tracheal mucosa. The studies of Abouav and Finley using the self-inflating cuff seem to obviate this problem significantly.

One of the cases of Abouav and Finley in their Table 1 (case 3) shows a patient who was maintained on an endotracheal tube for 260 hours (more than ten days), without evidence of leaks or tracheal damage. My question is "What damage is done to the vocal cords during that period of intubation, without changing to a tracheostomy within 48 to 72 hours?" Regardless of the tracheal problem, several people continue to mention to me that damage to the vocal cords can occur with prolonged nasotracheal or endotracheal intubation. I would appreciate comments from Abouav and Finley or anyone else about this potential problem, whether it really exists and at what duration of intubation one should be concerned.

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Variations in the Techniques of Pressure Breathing

To the Editor:

In the critical review entitled "The Management of Respiratory Distress Syndrome," Kleinberg indicated that application of continuous negative pressure around the chest wall of the patient "may provide a smaller risk for chronic pulmonary damage and lung rupture" and also that the "system using negative chest pressure may provide theoretic cardiovascular advantages." The references given do not support this assumption. Additional confusion is added by Kleinberg's statement that "negative-pressure ventilation promotes venous return and increases cardiac output and pulmonary blood flow." Negative-pressure ventilation does promote venous return, but it has an opposing influence to continuous negative pressure around the body, which is a form of continuous positive-pressure breathing (CPPB) in which the atmosphere supplies the positive pressure applied to the airway.

It is of clinical importance to realize that therapy with CPPB (or continuous positive airway pressure [CPAP]), the new term for CPPB) produces no better or worse physiologic effects than continuous negative pressure. Maloney and Whittenberger emphasized that the only differences between CPPB and continuous negative pressure were the slight increases in density and volume of air in the lungs and in the blood gas pressures when supra-atmospheric pressures were used; they reported the same decrease in blood pressure in a dog suffering from circulatory depression with the use of each technique.

Beck et al. reported that a similar and equal rise in venous pressure took place in normal human subjects during therapy with CPPB and continuous negative pressure. Whatever differences took place due to the use of mask or helmet-hood device were due to differences in mean applied pressure.

The work of expanding the lungs with 20 mm Hg of positive pressure (CPPB) may be expressed as the differences between 780 and 780 mm Hg. With continuous negative pressure at an intratank pressure of −20 mm Hg, the pressure of the atmosphere is positive in relation to the pressure surrounding the thorax, ie, 780 minus 740 mm Hg. There is no turbulent flow induced by CPPB or continuous negative pressure and, therefore, no effect from the change in density (20/760 or 3 percent). The increase in arterial oxygen pressure with CPPB would be approximately 0.6 percent. The increase in the functional residual capacity is approximately 3 percent.

The favorable clinical response to therapy with continuous negative pressure has been shown to be similar to that with CPPB in cases of pulmonary edema, bronchial asthma and pulmonary emphysema, andacute respiratory disturbance in infants; however, in 1972, Chernick and Vidyasagar stated that the hemodynamic influence of a pressurized hood was identical to continuous negative pressure, but in the same report, they appeared to make exceptions to their own statement. We quote: "The influence on the transpulmonary pressure is identical whether airway pressure is increased or chest wall pressure is decreased to the same extent . . . ." At the end of their report, Chernick and Vidyasagar stated: "The use of constant negative pressure around the thorax avoids placing a positive pressure on the cranium, [and] has less influence on the circulation . . . ."

Barach reported in 1958 that similar effects were produced by CPPB and continuous negative pressure; therapy with CPPB was applied by mask and the helium-oxygen pressure hood, and therapy with continuous negative pressure was applied by a tank respirator and