

High-frequency Positive-pressure Ventilation (HFPPV):

What Role in Ventilatory Insufficiency?

RECENT DEVELOPMENTS in mechanical ventilatory support indicate that a major shift in emphasis has occurred. Although support of alveolar gas exchange is still paramount in importance, the adverse effects of positive-pressure techniques have received increasing attention during the past decade.

The major complications of ventilator therapy—pulmonary barotrauma and cardiovascular depression—were not a serious problem when diseases such as poliomyelitis were treated with negative-pressure tank ventilators. Such devices, which mimicked the natural pattern of spontaneous ventilation, were required to generate a low subambient intrapleural pressure, since pulmonary parenchymal damage was usually minimal. Shortly after the introduction of positive-pressure ventilation, Cournand and others¹ showed that blood pressure and cardiac output could be depressed significantly, particularly when a long inspiratory phase was employed. The increased duration of positive airway and intrapleural pressure subsequently was shown to decrease thoracic venous inflow, stroke volume, and cardiac output.

In 1967, positive end-expiratory pressure (PEEP) was advocated to offset the pathophysiologic changes associated with acute ventilatory insufficiency² (later called the adult respiratory distress syndrome, or ARDS). Improvement in Pa_{O_2} and decrease of intrapulmonary shunting represented beneficial effects of such therapy, but PEEP when used in conjunction with mechanical ventilation was also alleged to increase the incidence of pulmonary barotrauma and cardiovascular depression. Subsequent attempts to minimize these complications included the use of PEEP in spontaneously breathing patients³ and the development of intermittent mandatory ventilation (IMV),⁴ techniques that eliminated or decreased the adverse effects of mechanical ventilation *per se*.

Since 1967, a completely different approach, high-frequency positive-pressure ventilation (HFPPV), has been advocated by a few investigators. The techniques, apparatus, experimental studies and clinical results have been reported.⁵ Briefly, HFPPV incorporates the following characteristics: 1) ventilatory frequency approximately three times greater than that of conventional IPPV (although cycling frequencies as high as $5,000 \cdot \text{min}^{-1}$ have been reported); 2) inspiratory:expiratory ratio <0.5 ; 3) small tidal volume, usually approaching that of the calculated dead space, V_D ; 4) positive airway and subambient intrapleural pressures

throughout the entire respiratory cycle, thereby maintaining a continuously positive transpulmonary pressure.

Advantages that have been claimed for HFPPV include less cardiovascular depression as a result of lower tidal volumes and airway pressure, more efficient pulmonary gas distribution, and reflex suppression of spontaneous ventilation, thereby avoiding asynchronous patient-ventilator breathing. Most of the work thus far has emanated from Sweden, and experience in the United States has been limited. Most reports have dealt with experimental animals or patients undergoing diagnostic laryngoscopy or bronchoscopy. There has been no report of a controlled, randomized study in which the results of HFPPV were compared with results of more conventional forms of mechanical ventilatory support in patients with acute ventilatory insufficiency, although the results reported by Bland in the treatment of neonates with respiratory distress syndrome are encouraging.⁶ In this issue, however, Carlon *et al.*⁷ report a more limited application of HFPPV in the treatment of bronchopleural fistula, which is interesting, provocative, and of potentially great value. Patients in whom this complication occurs as a result of positive-pressure ventilation present a significant therapeutic challenge, which has been met with variable success.⁸⁻¹⁰ Mechanical support, which is necessary to ensure adequate alveolar ventilation, may be rendered ineffective by escape of a large portion of the tidal volume through the fistula. Attempts to improve ventilation by increasing tidal volume or inspiratory airway pressure usually compound the problem. Attempts at operative closure of the fistula usually fail, in part, at least, because of the frequent presence of infection.

Mechanical ventilatory support, which employs a low tidal volume and low peak and mean airway pressures, should decrease the tendency for volume loss through the fistula to occur and improve the distribution of gas throughout the rest of the lung. All other things being equal, improved alveolar ventilation, evidenced by a decrease in Pa_{CO_2} , might be anticipated, and indeed was found by Carlon *et al.*⁷

Many questions remain to be answered concerning HFPPV. The fact that it works in selected clinical circumstances cannot be denied, but why and how, in my estimation, is entirely unclear. Although airway pressure remains positive throughout the entire

respiratory cycle, HFPPV is not entirely analogous to conventional PEEP. Pa_{CO_2} increases in apneic patients treated with PEEP, whose lungs are not ventilated manually or mechanically, but characteristically decreases with HFPPV. Low internal compliance and circuit volume appear to be prerequisites, so that a maximum amount of the delivered tidal volume actually reaches the patient's lungs. But why is a tidal volume, which is equal only to the calculated dead space of a normal individual and probably much less than that of a patient with acute ventilatory failure, associated with a normal Pa_{CO_2} ? Speculation concerning eddy-flows and non-square-wave jet pulses down the airway, which improve alveolar mixing, really translate into the fact that we have no good idea what is happening. Classic pulmonary physiology has not prepared us to understand HFPPV! Perhaps the mechanism is similar to that which allows a panting dog to maintain normal or low Pa_{CO_2} . In the latter, the respiratory frequency is high and the tidal volume low. However, we have no knowledge whether rapid, low-tidal-volume spontaneous breathing in animals with normal lungs is analogous to the clinical setting of acute ventilatory insufficiency treated with HFPPV, in which spontaneous ventilation is deliberately suppressed.

High-frequency positive-pressure ventilation has been studied for 12 years. Thus far, most of the information is anecdotal, and the ultimate role of this support has yet to be ascertained. We need basic physiologic information and facts. Eriksson *et al.*¹¹ and Borg *et al.*¹² have demonstrated that the effects of HFPPV compared with conventional IPPV may be different under certain circumstances. In deeply anesthetized dogs total peripheral resistance was lower while cardiac output and stroke volume were higher with HFPPV than with IPPV. In contrast, when a light plane of anesthesia was employed, which allowed regulatory and compensatory cardiovascular mechanisms to remain operative, there was no significant difference in these variables with either mode of ventilation or the frequencies utilized (20–60 cycles \cdot min⁻¹). Borg *et al.*¹² suggest that the critically ill patient with ventilatory failure may simulate the deeply anesthetized dog in terms of a loss of these regulatory and compensatory mechanisms. Such an individual might indeed benefit from the cardiovascular sparing effects of HFPPV if ventilatory function can be maintained in a satisfactory manner.

It is the latter point upon which attention must be focused. In 12 years, only two cases of ARDS treated

with HFPPV have been reported.¹³ Presently, because of a lack of clinical information, HFPPV cannot be recommended as a primary form of therapy. However, it may be very useful in cases such as the one described by Carlon *et al.* We can hope that further work and prospective studies will delineate the future role of HFPPV.

ROBERT R. KIRBY, M.D.
Professor and Chairman
Department of Anesthesiology
Tulane University School of Medicine
New Orleans, Louisiana 70112

References

1. Cournard A, Motley HL, Werko L, et al: Physiological studies of the effects of intermittent positive pressure breathing on cardiac output in man. *Am J Physiol* 152:162–174, 1948
2. Ashbaugh DG, Bigelow DB, Petty TL, et al: Acute respiratory distress in adults. *Lancet* 2:319–323, 1967
3. Civetta JM, Brons R, Gabel JC: A simple and effective method of employing spontaneous positive-pressure ventilation. *J Thorac Cardiovasc Surg* 63:312–317, 1972
4. Downs JB, Klein EF, Desautels DA, et al: Intermittent mandatory ventilation: A new approach to weaning patients from mechanical ventilation. *Chests* 64:331–335, 1973
5. Sjöstrand U (editor): Experimental and clinical evaluation of high-frequency positive-pressure ventilation. *Acta Anaesthesiol Scand (suppl)* 64:1–178, 1977
6. Bland RD, Kim MH, Light MS, et al: High-frequency mechanical ventilation of low-birth-weight infants with respiratory failure from hyaline membrane disease: 92% survival. *Pediatr Res* 11:531, 1977
7. Carlon GC, Ray C Jr, Klain M, et al: High-frequency positive-pressure ventilation in management of a patient with bronchopleural fistula. *ANESTHESIOLOGY* 52:172–174, 1980
8. Downs JB, Chapman RL: Treatment of bronchopleural fistula during continuous positive pressure ventilation. *Chest* 69:363–366, 1976
9. Gallagher TJ, Smith RA, Kirby RR, et al: Intermittent inspiratory chest tube occlusion to limit bronchopleural cutaneous airleaks. *Crit Care Med* 4:328–332, 1976
10. Phillips YY, Lonigan RM, Joyner LR: A simple technique for managing a bronchopleural fistula while maintaining positive pressure ventilation. *Crit Care Med* 7:351–353, 1979
11. Eriksson I, Jonzon A, Sedin G et al: The influence of the ventilatory pattern on ventilation, circulation, and oxygen transport during continuous positive-pressure ventilation—an experimental study. *Acta Anaesthesiol Scand (suppl)* 64:149–163, 1977
12. Borg U, Eriksson I, Lyttkens R, et al: A preliminary report on experimental studies of continuous positive-pressure ventilation (CPPV) and high-frequency positive-pressure ventilation (HFPPV). *Upsala J Med Sci* 84:109–117, 1979
13. Bjerager K, Sjöstrand U, Wattwil M: Long-term treatment of two patients with respiratory insufficiency with IPPV/PEEP and HFPPV/PEEP. *Acta Anaesthesiol Scand (suppl)* 64:55–68, 1977