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High-frequency oscillating ventilator use in a patient with severe pulmonary contusions

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Approximately 25% of trauma deaths can be attributed to thoracic injuries. Severe pulmonary contusions commonly result from blunt trauma to the thorax. The resulting alveolar hemorrhage and edema can make oxygenation difficult or impossible by conventional ventilation methods. High-frequency oscillating ventilation (HFOV), which delivers small tidal volumes at high respiratory rates, may provide an alternative method of treatment for these patients. Although HFOV is approved for use only in neonatal and pediatric patients, it is a potentially valuable tool for ventilating adult patients who have severe pulmonary contusions when conventional methods fail.

This article reports the successful use of HFOV as a salvage ventilation technique in the treatment of an adult-sized pediatric patient who sustained multiple injuries and severe pulmonary contusions in a motor vehicle collision. The successful outcome for this patient should stimulate further investigation into the use of HFOV in the treatment of severe pulmonary contusions in adults.

CASE REPORT

A previously healthy 16-year-old, 70-kilogram male was the unrestrained front-seat passenger in a motor vehicle collision. His car was traveling at approximately 85 mph when it hit a fire hydrant and a tree, ejecting all 4 occupants from the vehicle and killing the 2 rear-seat passengers. He was orally intubated and transported by helicopter to Baylor University Medical Center.

In the emergency department, tube thoracostomies were placed bilaterally. The patient received 4 units of packed red blood cells and 5000 cm^3 of crystalloid to improve his fluctuating blood pressure. The initial chest radiograph revealed multiple bilateral rib fractures, bilateral pneumothoraces, and bilateral upper-lobe infiltrates (*Figure 1*). Other injuries included multiple open fractures of the mandible; a closed-head injury of unknown severity; multiple transverse process fractures of the lumbar vertebrae; and fractures of the humerus, ulna, and scapula. Initial arterial blood gases (ABGs) were pH 7.02, P_{O_2} 133 torr, P_{CO_2} 55 torr, and O_2 saturation 96% at an F_{IO_2} of 1.0 delivered by manual ventilation via the endotracheal tube.

Peritoneal lavage revealed gross blood in the abdomen, and the patient was transferred directly to the operating room where splenectomy and exploration of an expanding right-flank hematoma were performed. The abdomen was packed and temporarily closed with a plastic Bogata silo bag because of continued coagulopathy and hypotension. The mouth also was explored because of continued bleeding from multiple lacerations and open fractures of the mandible. Bridle wires were placed, and the mouth was packed.

There was significant intraoperative difficulty with ventilation because of the pulmonary contusions and the presumed aspiration of blood from the oropharynx. Flexible bronchoscopy performed in the operating room showed bloody secretions without bronchial injury. Emergent head and chest computed tomography scans following the operation showed no mass lesions in the brain and severe, diffuse pulmonary infiltrates with multiple bilateral rib fractures (*Figure 2*). Throughout the computed tomography scan, the patient remained hypotensive, with progressively decreasing oxygen saturation.

Upon arrival in the surgical intensive care unit (approximately 3.5 hours after injury), the patient was profoundly coagulopathic and hypothermic, with a core temperature of 32°C (89.6°F) and a systolic blood pressure of approximately 90 mm Hg. Arterial blood gases revealed a pH of 6.96, a P_{CO_2} of 57 torr, a P_{O_2} of 50 torr, an O_2 saturation of 59%, and a base excess of –22 on pressure-control ventilation, with an F_{IO_2} of 1.0

Figure 1. Initial emergency department chest radiograph showing multiple rib fractures, bilateral pneumothoraces, and mild bilateral upper-lobe infiltrates.

Figure 2. Chest computed tomography scan revealing severe bilateral pulmonary contusions and multiple rib fractures.

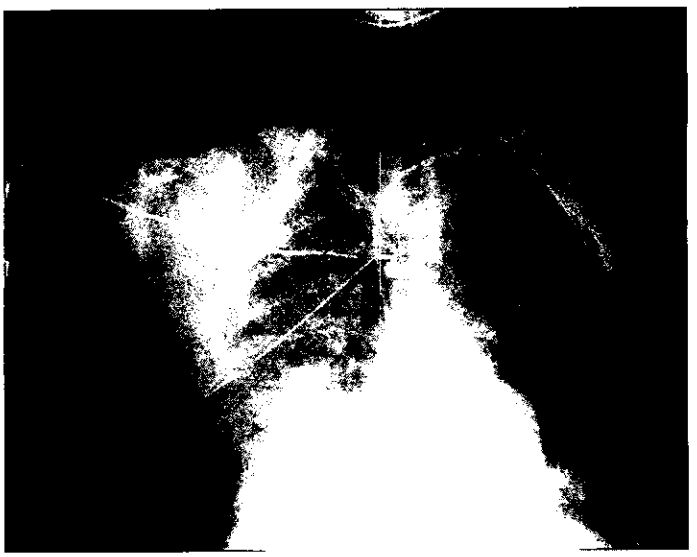

Figure 3. Chest radiograph on hospital day 1 showing almost complete consolidation of both lungs.

and positive end-expiratory pressure (PEEP) of 15 cm H_2O. The alveolar-arterial P_{O_2} gradient (A-a gradient) at this time was 592 mm Hg (normal, approximately 50 mm Hg on F_{IO_2} of 1.0), and the estimated physiologic shunt fraction (Qsp/Qt) was 78% (normal, <10%). The patient became increasingly difficult to oxygenate and ventilate and remained severely acidotic. The highest recorded mixed venous O_2 saturation was 48% via an oximetric Swan-Ganz catheter. High doses of dopamine and norepinephrine were required for blood pressure support.

The patient's respiratory status continued to deteriorate, with worsening acidosis, hypercapnia, and hypoxemia. His ventilator settings were continually adjusted, eventually reaching an inspiration to expiration ratio of 3:1, a driving pressure of 40 cm H_2O, PEEP of 15 cm H_2O, and F_{IO_2} of 1.0. The patient was placed in the prone position to maximize the relative sparing of anterior pulmonary parenchyma seen on chest computed tomography and to decrease the shunt fraction. Arterial blood gases approximately 9 hours postinjury were pH 6.99, P_{CO_2} 84 torr, P_{O_2} 26 torr, and O_2 saturation 41%; base excess was −15, and the mixed venous O_2 saturation was undetectable. In the next 4 hours, the patient had 3 separate episodes of cardiac arrest but responded to cardiopulmonary and chemical resuscitation. Arterial blood gases drawn during this 4-hour period revealed a pH of 7.01, a P_{CO_2} of 70 torr, a P_{O_2} of 22 torr, an O_2 saturation of 31%, and a base excess of −15. Chest radiographs revealed almost complete consolidation of both lungs (*Figure* 3). A decision was made to attempt high-frequency oscillating ventilation (HFOV) as a salvage attempt. The patient was placed on a SensorMedics 3100A HFOV (SensorMedics, Yorba Linda, Calif.) via a standard endotracheal tube at a rate of 300 breaths/minute and an F_{IO_2} of 1.0.

The patient's improvement was apparent within 2 hours after switching to HFOV. Five hours after initiating HFOV, the change in the patient's venous oxygen saturation (S_{VO_2}) followed an impressive curve, increasing from 48% to almost 80% (*Figure* 4). The initial ABGs on HFOV were pH 7.04, P_{CO_2} 94 torr, P_{O_2} 78 torr, O_2 saturation 92%, and S_{VO_2} 70%, and the estimated physiologic shunt fraction was 26%. Over the next several hours, the patient's acidosis continued to resolve, and the O_2 saturation stabilized at 96% to 99% (*Figure* 5). The following morning, his ABGs were pH 7.41, P_{CO_2} 34 torr, P_{O_2} 123 torr, and O_2 saturation 97%, with substantial improvement of the chest radiograph (*Figure* 6). Despite his prolonged hypoxemia and multiple episodes of cardiac arrest, the patient was able to follow simple commands on the morning of hospital day 3.

On the morning of the third hospital day, the patient returned to the operating room for a scheduled second-look laparotomy and a tracheostomy. High-frequency oscillating ventilation was used throughout the procedure. Later that day, the patient's A-a gradient (119 mm Hg), estimated physiologic shunt fraction (20%), compliance, and O_2 delivery had improved sufficiently to allow him to return to conventional pressure-controlled ventilation. The pressor agents were discontinued. By hospital day 6, the patient was transitioned to standard volume-control ventilation.

The patient required ventilatory support for 31 additional days while recovering from acute renal failure, several bouts of pneumonia, and multiple operative procedures for his injuries.

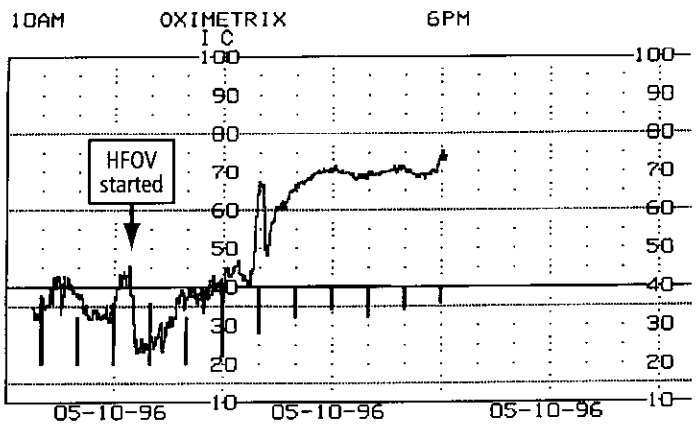

Figure 4. Mixed venous O_2 curve during initiation of HFOV (started at 12:30 PM), showing impressive improvement within 2 hours.

He was transferred to a rehabilitation hospital on hospital day 73. The patient's mental function returned to baseline, and he returned to high school. His only remaining physical difficulty is decreased range of motion and strength in his right arm that he says interferes with guitar playing.

DISCUSSION

Approximately 25% of trauma deaths ultimately can be attributed to thoracic injury (1, 2). Pulmonary injury caused by blunt trauma often is not immediately apparent and may take 48 to 72 hours to develop. Differentiation between aspiration pneumonitis, pulmonary contusion, fluid overload, intrapulmonary hemorrhage, or a combination of these can be difficult (1, 3). Pulmonary contusion commonly is caused by a forceful deceleration that leads to a sudden increase in intrathoracic pressure. The sudden increase in pressure ruptures alveoli and microvasculature, resulting in the extravasation of blood and plasma into alveoli and interstitial spaces. Alveolar hemorrhage and edema lead to a complete breakdown of the alveolar superstructure in the injured area (1–3). The extravasated blood and plasma trigger a localized inflammatory response that causes a progressive increase in pulmonary vascular resistance, leading to a decrease in blood flow to the site of the lung injury. Edema and hemorrhage in the lung also cause abnormal gas diffusion at the capillary level and subsequent localized hypoxic vasoconstriction.

The combination of increased pulmonary vascular resistance and hypoxic vasoconstriction increases shunting in a localized area, which causes a ventilation-to-perfusion mismatch and leads to systemic hypoxia and hypercapnia (2, 3). Oxygenation of contused lung segments decreases during the first 24 hours after injury, a decline that is significantly more pronounced in contused than in noncontused lung segments (1, 3).

When examining the severity of pulmonary contusion, initial chest radiographs are notoriously undependable (3). The A-a gradient and physiologic shunt fraction measurements are more reliable indicators of the physiologic significance of a pulmonary contusion (3, 4). The physiologic shunt fraction (Qsp/Qt) is a measure of the degree to which the pulmonary system contributes to hypoxia. The physiologic shunt fraction is determined

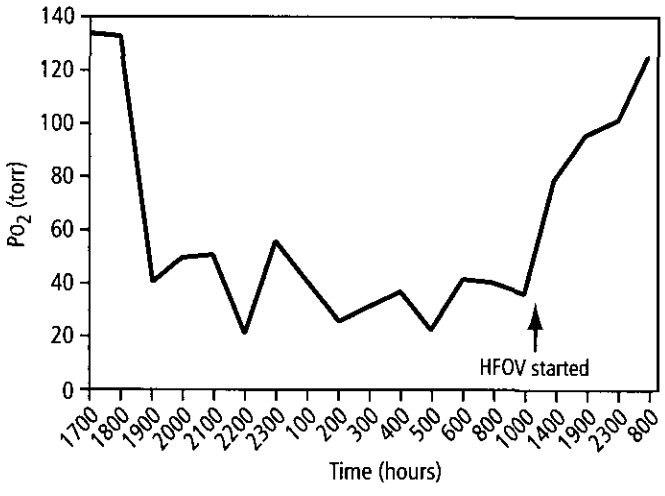

Figure 5. Graph of Po_2 over time, from initial presentation to hospital day 2.

Figure 6. Chest radiograph on hospital day 2 showing substantial improvement in pulmonary infiltrates.

using the oxygen content in arterial (CaO_2), mixed venous ($C\bar{v}O_2$), and ideal pulmonary capillary ($C\dot{c}O_2$) blood:

$$Qsp/Qt = \frac{C\dot{c}O_2 - CaO_2}{C\dot{c}O_2 - C\bar{v}O_2}$$

When 100% oxygen is breathed, the formula simplifies to:

$$Qsp/Qt = \frac{1 - \text{arterial blood saturation } (SaO_2)}{1 - \text{mixed venous blood saturation } (S\bar{v}O_2)}$$

A physiologic shunt fraction of <10% is considered normal, 10% to 19% is abnormal, 20% to 29% is significant, and >30% shunt is indicative of severe pulmonary dysfunction (5). As the shunt fraction approaches 50%, increases in FIO_2 have little effect on PaO_2 (3, 5).

High-frequency ventilation is a broad term applicable to several different types of ventilation: high-frequency positive-pressure ventilation, high-frequency jet ventilation, and HFOV. Initially described by Lunkenheimer in 1972 (6), the tidal volumes delivered usually are smaller than the anatomical dead space (1). Gas flow occurs via a "bias flow" system, and gas exchange occurs via Brownian movement; this combination allows more even gas distribution and decreases airway pressure (1, 6, 7). High-frequency oscillating ventilation delivers approximately 2 mL/kg to 5 mL/kg of gas, which is delivered at an oscillating frequency between 60 to 3000 breaths/minute via the endotracheal tube (6). High-frequency oscillating ventilation also can be delivered by an external system (e.g., Hayek Oscillator, Flexico Medical Instruments A.G., Zurich, Switzerland) using a cuirass instead of an endotracheal tube for delivery (8).

A primary benefit of high-frequency ventilation compared with conventional ventilation is a significant reduction in peak inspiratory pressure while maintaining adequate oxygenation (6, 9). Conventional ventilation often causes high peak airway pressures in diseased lungs with decreased compliance, especially when high levels of pressure control and PEEP are required for oxygenation (*Figure 7*). The large tidal volumes and high peak inspiratory pressures associated with conventional ventilation

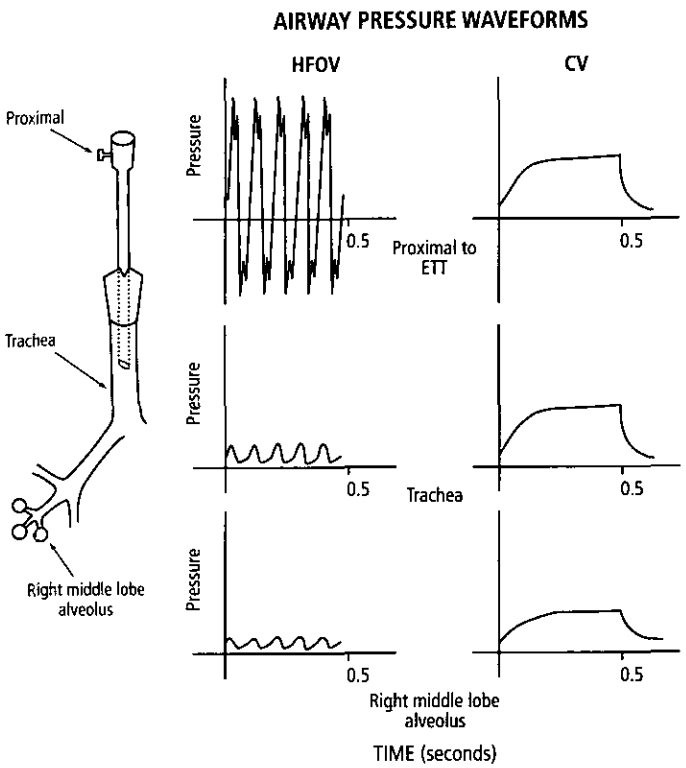

Figure 7. Differences in the pressure transmitted from the ventilator to the alveolus for high-frequency oscillatory ventilation (HFOV) and conventional mechanical ventilation (CV). These changes reflect those that occur across a 3.0 French endotracheal tube in adult rabbits with normal lungs. The figure represents data derived using a specific type of oscillator (SensorMedics 3100). ETT = endotracheal tube. (From Clark RH, Null DM: High-frequency oscillatory ventilation. In Pomerance JJ, Richardson CJ, eds: *Neonatology for the Clinician*. Stamford, Conn.: Appleton & Lange, 1993:293.)

cause cyclic overdistention of alveoli in diseased lungs that have decreased compliance and low resting lung volumes. Overdistention of alveoli, occurring when the more compliant "normal" lung areas receive the bulk of each delivered tidal volume, is the primary cause of barotrauma and decreased oxygenation (9, 10). This overdistention, or hyperinflation, leads to increased pulmonary vascular resistance in the more compliant areas of the lungs, shunting blood back to the injured areas and causing further venous admixture (3). Worsening hypoxia often leads to increased use of PEEP, which further increases pulmonary vascular resistance and creates a vicious cycle of overdistention and resulting barotrauma. Barotrauma (e.g., pneumothorax, pneumomediastinum, and permanent fibrosis) occurs in 5% to 25% of patients requiring mechanical ventilation (7, 11, 12). A benefit of HFOV is that it provides adequate oxygenation and ventilation using small tidal volumes that are delivered at rates of 60 to 3000 breaths/minute. High-frequency oscillating ventilation fluctuates pressure changes around a specific mean airway pressure (9), allowing maintenance of lung volumes slightly above the functional residual capacity (9, 10, 13). By delivering smaller tidal volumes at higher frequencies, the "normal" or more compliant areas of lung are not hyperinflated, thus reducing barotrauma to these areas and breaking the cycle of overdistention. High-frequency oscillating ventilation enhances recruitment of alveoli in damaged lung tissue, presumably because of the ability to maintain higher mean airway pressure. Eventually, the higher mean airway pressure enhances ventilation/perfusion matching, decreases dead space, and thus improves oxygenation (9). Although PEEP also can recruit alveoli from damaged areas, it does so at the cost of overdistention of normal alveoli (5).

Another potential advantage of HFOV is that adequate oxygenation can be achieved with fewer cardiovascular effects (6, 10, 11). The high levels of PEEP used with conventional ventilation can impair venous return and diastolic filling of the heart ("peeponade"), leading to decreased cardiac output and oxygenation (9). Several studies have shown that high-frequency ventilation does not cause hemodynamic compromise and may lead to a slight improvement in cardiac output (6, 10, 11, 14). This was evident in our patient, who was weaned from all pressor agents within 24 hours of placement on HFOV.

High-frequency ventilation has been used successfully in multiple studies in neonates with hyaline membrane disease (10, 15, 16), but SensorMedics 3100A HFOV is the only high-frequency ventilator currently approved for use in neonates weighing from 540 to 4600 grams. Studies using high-frequency oscillating ventilation in older children with respiratory failure have yielded promising results (9, 10). Eight months prior to our case, the Food and Drug Administration approved the SensorMedics 3100A HFOV for use in children with severe respiratory failure who are failing on conventional ventilation. There is no upper weight limit for use of this ventilator in children. In a randomized study by Arnold et al (17), 70 pediatric patients with severe diffuse alveolar disease or massive airleak syndrome, or both, received either conventional mechanical ventilation or HFOV. Arnold found that patients receiving HFOV had significantly improved oxygenation, compared with the conventional ventilation group. Furthermore, HFOV was associated with a lower incidence of barotrauma (indicated by the need for supplemental oxygen at 30 days) and improved overall outcome.

Some controversy exists as to whether high-frequency ventilation is feasible in adults. Lunkenheimer et al (13) studied HFOV in adult pig models (larger than 65 kg). These investigators concluded that adult pigs with body weights in the same range as adult humans can be ventilated effectively using high-frequency ventilation at small tidal volumes. High-frequency ventilation has been investigated clinically in adult humans in several studies (7, 9, 12, 18). Most of these studies involved the use of high-frequency ventilation in patients with bronchopleural fistulae. Several of these studies have illustrated that high-frequency ventilation may be of significant value and is not necessarily limited to salvage techniques in adult patients.

High-frequency oscillating ventilation may be a useful addition in the support of severe respiratory failure secondary to pulmonary contusion. Although these ventilators have been approved by the Food and Drug Administration for limited use in pediatric patients, they may also be beneficial in treating adult patients who have severe respiratory failure and cannot be maintained on conventional ventilators. Further studies are needed to confirm the potential benefits to adult patients. Because our

patient was 16 years old, he was considered to be a pediatric patient; however, his body weight placed him in the adult weight range. For the critically ill patient who is failing on conventional ventilation, HFOV may provide a chance of survival.

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