

High-frequency jet ventilation: a case report

KALPALATHA K GUNTUPALLI, ARNOLD SLADEN, MIROSLAV KLAIN

From the University of Pittsburgh School of Medicine, Montefiore Hospital, Pittsburgh, Pennsylvania

Treatment of bilateral pneumonia with pneumatoceles poses a difficult problem of management in patients requiring mechanical ventilation. The presence of a bronchopleural fistula compounds the problem. The peak inspiratory airway pressures with conventional ventilators potentially can generate an air leak, or increase the leak through an existing fistula. High-frequency jet ventilation is capable of appreciably decreasing the airway pressures during mechanical ventilation¹ and has been used in the treatment of a large airway leak.² We report the use of this method in the management of a patient with acute respiratory failure from pneumonia, pneumatoceles, and bronchopleural fistula.

Case report

A 23-year-old man was admitted after a 20-metre fall. No evidence of fracture of long bones or ribs or of pulmonary contusion was noted. The chest radiograph and arterial blood gases were within normal limits. Computed tomography showed cerebral oedema. Intracranial pressure was monitored and cerebral oedema treated with controlled hyperventilation, mannitol, and corticosteroids. Five days after admission the patient developed fever; bilateral pulmonary infiltrates were observed on the chest film. Enterococci and *Haemophilus influenzae*, both sensitive to cefamandole, were cultured from the sputum, and he was treated with appropriate doses of the antibiotic, but without improvement. Since his recovery was expected to be slow a tracheostomy was performed.

Ten days after admission pneumatoceles in the left lower lung zone and a left pleural effusion were observed on the chest film. Shortly afterwards he developed tachypnoea (respiratory rate > 45/min) and sweating, associated with a 10% pneumothorax. Mechanical ventilation provided a tidal volume of 750 ml, 10/min; positive end-expiratory pressure was 2.5 cm H₂O and fractional inspired oxygen (F_IO₂) 0.4; arterial oxygen tension (Pao₂) was 10.13 kPa (76 mm Hg), arterial carbon dioxide tension (Paco₂) 3.87 kPa (29 mm Hg), and pH 7.50. Thoracentesis was performed and a chest tube inserted, and a small air leak was noted. About 500 ml of blood-stained fluid (pH of 7.16, white cell count of $8.7 \times 10^9/l$ (97% polymorphs), glucose 1.5 mmol/l (27 mg/100 ml)) was drained. Group D streptococcus was cultured from pleural fluid and anaerobic cultures gave negative results. Tracheal aspirate at this time grew *Proteus* sp, group D streptococci and enterococci, which were sensitive to tobramycin and ampicillin, with which he was treated. At this time the patient was able to open his eyes on command. During the next 24 hours

tachypnoea persisted, with progression of bilateral pulmonary infiltrates. When the F_IO₂ was 0.4 and the positive end-expiratory pressure 7.5 cm H₂O, Pao₂ was 7.87 kPa (59 mm Hg), Paco₂ 4.27 kPa (32 mm Hg), and pH 7.48. In view of the continued respiratory distress, rapidly progressive pneumonia, pneumatoceles, and bronchopleural fistula, high-frequency jet ventilation was initiated at a rate of 100/min, driving pressure 25 lb/in² (172 kPa), inspiratory time 30%, and positive end-expiratory pressure 5 cm H₂O. The patient appeared comfortable and spontaneous respiratory rates remained less than 25 per minute. When F_IO₂ was 0.74 Pao₂ was 27.47 kPa (206 mm Hg), Paco₂ 4.13 kPa (31 mm Hg), and pH 7.50. Gradually the positive end-expiratory pressure was discontinued, and F_IO₂ decreased to 0.3; Pao₂ was then 15.73 kPa (118 mm Hg), Paco₂ 3.73 kPa (28 mm Hg), and pH 7.54. The air leak was minimal on high-frequency jet ventilation without positive end-expiratory pressure. During the succeeding six days the pneumatoceles and infiltrates resolved. High-frequency jet ventilation was discontinued and the patient was successfully decannulated. The chest tube was replaced with a Foley catheter for the drainage of empyema. The patient was responsive and able to sit in a chair.

Discussion

This patient presented a difficult problem in management because of the bilateral pneumonia, with progression to pneumatocele formation and the need to provide ventilatory support in the presence of pneumothorax and bronchopleural fistula. Mechanical ventilators, with or without positive end-expiratory pressure, can potentially cause barotrauma and generate a fistula or increase air leaks through an existing one. Several factors, such as peak respiratory airway pressure, application of positive end-expiratory pressure, and the presence of underlying lung disease, have been implicated in the causation of barotrauma. With conventional ventilators the peak inspiratory pressure changes in relation to pulmonary compliance. Varying alveolar ventilation can occur at high peak pressures, owing to greater compression volume or to the fact that the volume is vented when the pressure limit is reached. High-frequency jet ventilation is a time-cycled ventilator and uses a high-pressure source of gas to deliver jets of oxygen or air-oxygen mixtures and also entrains air or an air-oxygen mixture by Venturi principle. It has a negligible internal volume and thus an insignificant compression volume. This mode of ventilation provides adequate alveolar ventilation at lower pressures and is independent of lung compliance. The bronchopleural fistula can give rise to large inspiratory leaks resulting in decreased alveolar ventilation or expiratory leak causing an inability

Address for reprint requests: Dr KK Guntupalli, Pittsburgh School of Medicine, Montefiore Hospital, Pittsburgh, Pennsylvania, USA.

to maintain positive end-expiratory pressure and hence hypoxaemia. In our patient the air leak was not large and the aim was not to increase it. The need to provide ventilatory assistance for the management of acute respiratory failure in the presence of pneumatoceles and bronchopleural fistula contraindicated high peak airway pressures. High-frequency jet ventilation was the mode of choice in these circumstances.³

In patients breathing out of phase with the ventilators a further increase in airway pressures occurs. Narcotics or muscle relaxants may be required to control this asynchrony. High-frequency jet ventilation, by contrast, is well tolerated by the patients, can be superimposed on spontaneous breathing, and does not require the use of narcotics or muscle relaxants.⁴

In addition, high-frequency jet ventilation prevents aspiration.⁵ In neurologically compromised patients and those with loss of protective airway reflexes there is a danger of

aspiration despite adequate seal with tracheostomy cuffs. High-frequency jet ventilation is indicated in these patients quite apart from its use in acute respiratory failure.

References

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