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High-frequency Positive-pressure Ventilation in Management of a Patient with Bronchopleural Fistula

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High-frequency positive-pressure ventilation (HF-PPV) has been used since 1968 for laryngeal and tracheal surgery. Extensive technical explanations are beyond the scope of this paper and we address the interested reader to an excellent reference source.[1]

High-frequency positive-pressure ventilation has also been used for long-term support in two cases of adult respiratory distress syndrome.[2] The major advantages ascribed to this form of ventilation are that airway resistances and pulmonary compliance have minimal influence on ventilation, and adequate alveolar ventilation is achieved with very low peak and mean airway pressures. We thought that these characteristics would also be valuable in solving the unique problem presented by the patient who is the subject of our report.

REPORT OF A CASE

A 42-year-old white man had an epidermoid carcinoma of the right apex of the lung. Following preoperative radiotherapy, he underwent wedge resection and internal irradiation of the chest-wall apex. Eight months later an *Aspergillus fumigatus* abscess developed in the same area. It did not respond to a course of treatment with amphotericin B. Right upper lobectomy disclosed no tumor, only abscess invading the chest wall, so an upper thoracoplasty was done as well. The postoperative course was complicated by arrhythmias, subclavian-vein thrombosis, and numerous pulmonary emboli. Despite the thoracoplasty, infection persisted, and repeated open-chest drainage of loculated abscesses was necessary.

Two months after the lobectomy, *Klebsiella pneumoniae* pneumonia developed. Tracheostomy and ventilatory support with positive end-expiratory pressure (PEEP) were necessary. After three weeks of mechanical ventilation, an episode of dyspnea, cyanosis and hyperventilation developed suddenly. Suctioning the tracheal tube yielded large amounts of frothy purulent material, similar to the fluid draining from the chest cavity. It was also found that the exhaled volume was 300–350 ml less than the delivered tidal volume; with each breath, air bubbled through the opening in the chest wall. An emergency bronchoscopy confirmed the total disruption of the right-upper-lobe bronchial stump. Surgical correction was impossible since the area was grossly infected and the patient unable to breathe spontaneously. In the next 72 hours, progressive deterioration occurred, with development of a bilateral diffuse pneumonia. Sputum cultures grew several gram-negative organisms identical to those obtained from the right-chest abscess cavity. Ventilation at high respiratory rates (as high as 30/min) and low tidal volumes (V_T) (300–500 ml) or low rates (5–10/min) and high V_T (1,000–1,400 ml) was attempted, to improve arterial oxygenation. Seventy-two hours after the sudden rupture of the bronchial stump, Pa_{O_2} was 58 torr and Pa_{CO_2} 83 torr during mechanical ventilation with 20 breaths/min, V_T 500 ml, and fractional concentration of inspired oxygen ($F_{I_{O_2}}$) .40. All other ventilatory modes had proved even less effective. Cardiac index (CI), measured with a thermodilution pulmonary-artery catheter, was 4.0 l/min/m². Pulmonary venous admixture ($\dot{Q}_V/\dot{Q}_T$) was 30 per cent of cardiac output. Pulmonary arterial mean pressure ($\bar{P}_{pa}$) was 32–35 torr; pulmonary mean capillary wedge pressure ($\bar{P}_{cw}$) was 10–12 torr. Pulmonary vascular resistance (PVR) was 750 dynes/sec/cm^{-5}/m². After consultation with all physicians involved and with the family of the patient, it was decided to attempt HFPPV. A jet ventilator, based on fluidic mechanics, was used. With this ventilator, duration of inspiration and expiration can be independently adjusted to provide any combination of ventilatory rates and inspiratory/expiratory (I/E) ratios. We selected a respiratory rate of 115/min and a I/E ratio of 1:2; this resulted in a flow of 17.2 l/min with driving pressure of 45 psig. This ventilation mode was selected because previous experiments with this particular ventilator had indicated that it guaranteed low peak airway pressure and adequate CO_2 clearance.[3] Oxygen and air, piped from the hospital's central system under a pressure of 50 psig, were

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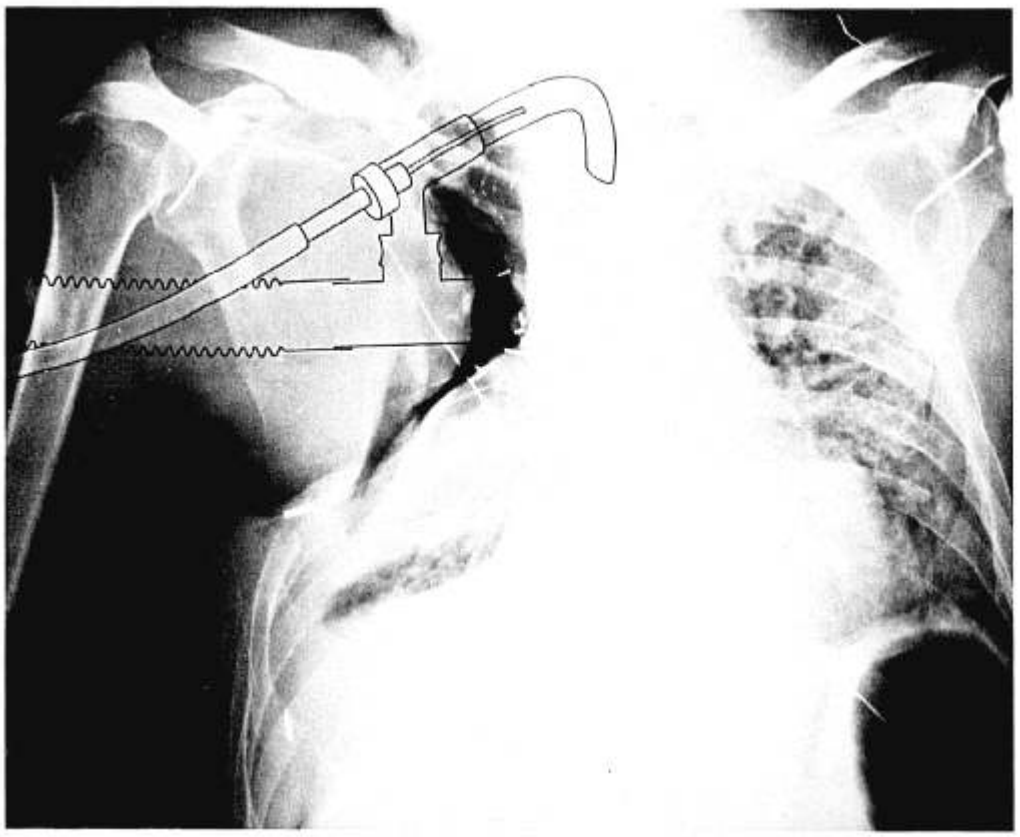

FIG. 1. Radiogram of the chest four days after initiation of jet ventilation. The superimposed drawing shows the inspiratory line of the jet and the 14-gauge Angiocath® entering the tracheotomy tube through a swivel connector. A corrugated tube, also shown in the drawing, carried warm air humidified by use of an ultrasonic nebulizer. The extent of the chest-wall fault is evident. The dilation of the gastric fundus was a recurrent problem in this patient, but always rapidly resolved without intervention.

blended to the desired proportion and supplied to the ventilator. The pressure of delivered gases was adjusted at 45 psig through the blender of the outflow regulator. The ventilator was then connected to the patient's tracheotomy tube via a 14-gauge Angiocath®, inserted into a bronchoscopic swivel connector. The tracheostomy tube was also connected via a 22-mm T-adapter to a source of air–oxygen mixture, humidified with an ultrasonic nebulizer (fig. 1). In addition to providing humidification, this system prevented entrainment of room air by the action of the jet pulse. Immediately after initiation of jet ventilation, the patient felt subjective relief from dyspnea, and his spontaneous respiration ceased.[2] Twenty minutes later, blood-gas values were Pa_{O_2} 50 torr and Pa_{CO_2} 33 torr at $F_{I_{O_2}}$ .40. The driving pressure of the jet was reduced to 35 psi; after 20 minutes more Pa_{O_2} was 114 torr and Pa_{CO_2} 47 torr at $F_{I_{O_2}}$ .40. PVR was 400 dynes/sec/cm^{-5}/m^2, CI 5.0 l/min/m^2, and $\dot{Q}_A/\dot{Q}_T$ 18 per cent of cardiac output. The flow delivered at a driving pressure of 35 psi, a rate of 115/min, and an I:E of 1:2 was 15 l/min; however, the exhaled volume of the patient, measured with an ultrasonic spirometer, was 22–25 l/min. The difference presumably resulted from gas aspirated through the open tracheotomy tube by the jet's action. The V_D/V_T, measured by collecting exhaled gases in a Douglas bag through a double-exhalation valve, was .77. Jet ventilation was continued for four days, during which time $F_{I_{O_2}}$ was lowered to .30 and driving pressure reduced to 30 psi. Mean airway pressure, measured with a saline-filled catheter advanced approximately to the carina, was 4 torr. After four days, an attempt was made to disconnect the patient from the jet ventilator, allowing him to breathe spontaneously. He was able to tolerate weaning for approximately 45 minutes before complaining of dyspnea and fatigue. Arterial blood-gas values had been Pa_{O_2} 107 torr and Pa_{CO_2} 45 torr at $F_{I_{O_2}}$ .30 during use of the jet ventilator; after 45 minutes of spontaneous breathing, they were Pa_{O_2} 48 torr and Pa_{CO_2} 53 torr at the same $F_{I_{O_2}}$. PVR had increased from 370 to 550 dynes/sec/cm^{-5}/m^2, while CI was unchanged. Weaning was continued in the following days and the patient tolerated progressively longer intervals of spontaneous respiration. Thirty-six days after initiation of jet ventilation, the patient had a Pa_{O_2} of 58 torr and Pa_{CO_2} of 51 torr after breathing humidified room air for 24 hours. He was discharged from the intensive care unit three days later and from the hospital a few weeks thereafter. A continuous regimen of parental and enteral hyperalimentation with 90 Kcal/kg/day and 2.5 g protein/kg/day had resulted in a weight gain of 8 kg of lean body mass in the last two months of hospitalization. At the time of discharge, the chest wound was half the size it had been at its maximum, and there was no air leak during spontaneous ventilation.

DISCUSSION

The predicament of this patient when the bronchial stump opened at first seemed insoluble. Surgical correction was impossible; spontaneous ventilation could not support alveolar gas exchange, and furthermore, whenever a negative intrathoracic pressure occurred, purulent fluid was aspirated into the lungs (fig. 2). Mechanical ventilation with a volume-cycled ventilator was temporarily life-saving but at the cost of maintaining the bronchial fistula widely open. Indeed, rapid deterioration of respiratory function developed despite mechanical support. The theoretical solution required a respiratory support device

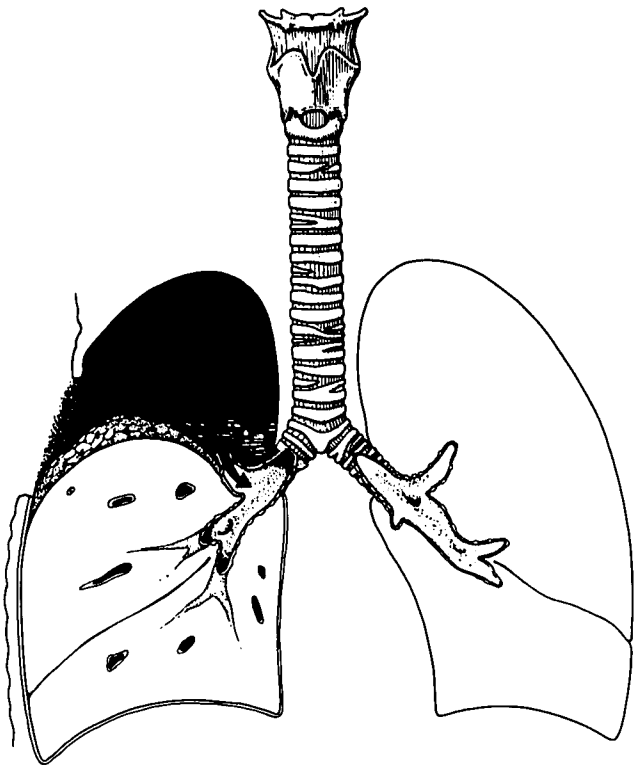

FIG. 2. Artist's representation of the lesion. The extensive loss of substance of the chest wall (approximately 35 × 15 cm) is evident, as well as the empyema cavity. The opening of the right upper lobar bronchus was in a dependent position. Careful positioning of the patient and packing of the cavity limited but did not eliminate continuous drainage of purulent material into the bronchial tree.

that could provide adequate alveolar ventilation with minimal but not negative airway pressure, even in the presence of a poorly compliant lung.

High-frequency positive-pressure ventilation had all the required characteristics. The virtual absence of internal compliance and compressible volume eliminated the influence of airway resistances and pulmonary compliance on delivered tidal volumes. At 115 breaths/min, airway pressure was minimally but constantly positive. The clinical improvement was in the patient's condition immediately evident, as indicated by decreased Pa_{CO_2} and improved arterial oxygenation.

The theoretical aspects of this form of ventilation are, however, intriguing. To begin with, V_D/V_T was unusually high, even for a patient with severe respiratory failure. Undoubtedly, a small amount of V_T was lost through the bronchial opening; this air leak, however, must have been very small, and indeed it was not clinically detectable. In any case, an air leak would have resulted only in underestimation of minute ventilation, and would probably not have affected V_D/V_T determinations, since there is no reason to suspect that the concentration of exhaled CO_2 from the right lung was markedly different than that from the left lung. Despite the very large V_D/V_T of this patient, a V_T of 180–200 ml maintained adequate alveolar ventilation. In comparison, it is inconceivable that a 60-kg patient could have been supported with a conventional volume-cycled ventilator and a V_T of 200 ml at any respiratory rate.

The principle by which HFPPV provides adequate CO_2 clearance remains obscure,[1–3] and much basic investigation remains to be done. It is worth noting that arterial blood P_{O_2} increased from 50 to 114 torr when the jet drive was decreased to provide "normal" alveolar ventilation, as indicated by the Pa_{CO_2}. It is possible that an excessively high minute ventilation resulted in eddy-flows, with redistribution of ventilation to areas of the lungs with low time-constants and consequent increases of ventilation–perfusion abnormalities. Although CI was unchanged, the decrease of PVR may be explained by better oxygenation and reduced hypoxic vasoconstriction.

We were also concerned about the effects that continuous pulses of incompletely humidified air would have on the tracheal mucosa. Repeated bronchoscopic examinations, however, failed to reveal any endobronchial damage.

In conclusion, we think that HFPPV was sufficiently indicated in this patient to justify the use of an experimental device. A number of theoretical aspects of this form of ventilation are still incompletely understood, and deserve further investigation.

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