Intensive Care Med (1984) 10:197 – 200

Intensive Care Medicine

Methods and devices

Combined use of HFPPV with low-rate ventilation in traumatic respiratory insufficiency

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Accepted: 21 February 1984

Abstract. Two patients with chest injuries, flail chest and respiratory failure were mechanically ventilated by a system composed of 2 Bennett respirators and an independent source of gas. This system provides high-frequency positive pressure ventilation (HFPPV), low-frequency conventional mechanical ventilation (LFCMV) and high inspiratory flow of fresh gas (HIF), through the independent source. This system made use of the advantages of HFPPV and also solved the problem of possible CO_2 retention. Using this system we could ventilate the patients while they were fully conscious and cooperative, thus eliminating the need for sedatives and muscle relaxants. Time of mechanical ventilation was shortened since the internal pneumatic fixation was very good and made it possible for the fractured ribs to unite rapidly. Restoration of spontaneous breathing was immediate after disconnection from the ventilator. We suggest this method as another mode of ventilation for patients with flail chest and respiratory failure.

Key words: Lung contusion – Flail chest – Internal pneumatic fixation – HFPPV – Low rate mechanical ventilation

Thoracic injuries continue to be a major cause of morbidity and mortality in war and civilian casualties [8]. The definition and treatment approach of flail chests are still controversial [14]. Most authors agree that the respiratory distress accompanying a flail chest is the consequence of direct trauma to the lung with no relationship to the portion of chest in paradoxical movement [11]. There is a clinical trend to classify flail chest injuries according to the severity of their effect on pulmonary physiology in order to select optimum therapy for each case. Tradionally there have been two approaches to management, surgical fixation of the thorax [7] and "internal pneumatic fixation" [3].

The clinical picture often includes coexisting hemothorax and/or pneumothorax which prevent optimal pulmonary function and must be drained as soon as possible, whether the management is going to be surgical or conservative [8 – 11]. Some patients do not require ventilatory and/or surgical treatment since their respiratory function is good, others will need ventilatory support due to deterioration of respiratory function [10].

The best mode of ventilation will provide good oxygenation and alveolar ventilation and minimize the complications of mechanical ventilation. Various modes have been tried including, PEEP combined with IMV [9], CPAP with IMV and CPAP through a face mask [12]. The duration of ventilatory support with these modes averaged 3 – 10 days and there was little change in mortality [3 – 9].

We prefer to use HFPPV for the treatment of flail chest and lung contusion. This preference is based upon proven experimental advantages of this mode of ventilation, namely: low peak airway pressure, less cardiovascular interference and successful oxygen and CO_2 exchange and distribution accomplished in a patient who is conscious, cooperative and under less stress [13, 1].

We present here our experience in the management of two patients with flail chests and with probable fat embolism in one, and contused lung in the other, using our ventilatory method which combines HFPPV and low rate conventional mechanical ventilation with high inspiratory flow and free release of over-flow gas.

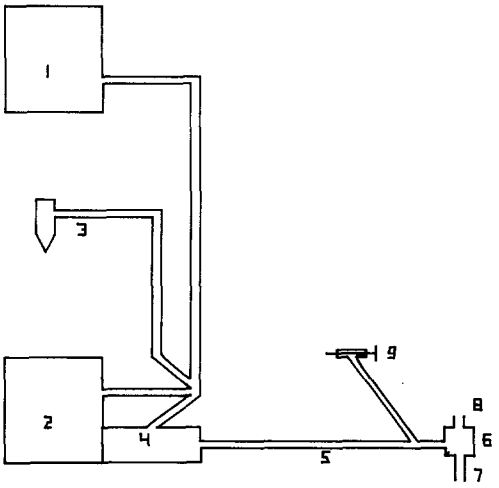

Fig. 1. 1. Bennett Ma-2B; 2. Bennett MA-1B; 3. mixer High Flow; 4. humidifier; 5. inspiratory flow; 6. swivel connector; 7. tube to patient; 8. free release; 9. expiratory valve

Methods

HFPPV/low frequency conventional mechanical ventilation with high inspiratory flow and free release (HFPPV/LFCMV and HIF/FR) was performed using two conventional ventilators, Bennett MA-2B and Bennett MAI-B (International Corp., USA).

High frequency was achieved by turning both the sensitivity and flow rate buttons of the MA-2B ventilator to maximal capacity and shutting off the tidal volume button. This procedure provides ventilatory rates of 140 – 160 and tidal volumes of 50 – 70 ml.

The outflow of this ventilator was connected through a Y connector to the inflow of the Humidifier (Cascade) of the other ventilator (Bennett MAI-B) which provided independently a tidal volume of 10 to 12 ml/kg, with rate of breaths 2 – 6 per min, according to the patient's ventilatory status. We also added an independent high inspiratory flow of 15 l/min of fresh gas, passed also through the same cascade of the Bennett MAI-B. Therefore, the patients received ventilation through one cascade only. Both Bennett respirators and the free high flow of gas were set to give the same oxygen concentration in the inspired air. The patients were nasotracheally intubaded with soft cuffed tubes, connected to the ventilation system through a Swivel connector which had a leak pore, 1 to 3 mm diameter, in order to provide free release of the overflow (Fig. 1).

Intravascular catheters were inserted for monitoring femoral artery pressure (SAP), right atrial pressure (CVP), pulmonary artery pressure (PAP) and pulmonary capillary wedge pressure (PcWP). Vascular pressures were measured with Ailtech MS 20 pressure transducer (Eaton Corp City of Industry, CA 91748, USA). Pulmonary vascular (PVR) and total peripheral (TPR) resistances were calculated from the cardiac index (CI) and the difference between mean pulmonary artery and pulmonary wedge pressure for PVR, and between mean arterial and right atrial pressures for TPR. Cardiac output was obtained by the thermodilution technique (Cardiac output computer 924/C, Mennen-Greatbach, Israel) using the average of three serial determinations. pH, arterial and mixed venous blood gases were determined by ABL3 blood gas analyzer (Radiometer, Denmark); results were corrected according to the patient's temperature. All calculations were performed on a desk top computer system (CASIO-Fx-702 P, Japan).

Patients and results

Case reports

Patient 1. A 61-year-old woman was admitted to the orthopedic department with multiple trauma due to a road accident. Her injuries included fractures of right tibia, left femur and sternum. Forty-eight hours later she began to show symptoms of respiratory distress and stupor. At that time she was noticed to have paradoxical movement of the chest, petechiae over the abdomen, thrombocytopenia (75000/ml), respiratory acidosis (pH: 7.28, PCO_2: 49 mmHg) and severe hypoxemia (PaO_2: 48 mmHg, PaO_2/FIO_2 = 60) (probable fat embolism). Chest X-ray revealed diffuse pulmonary infiltrates and fractures of right ribs 2 – 3 and sternum.

The patient was transferred to the GICU. On admission she was stuporous, cyanotic and had respiratory distress. Blood pressure was 120/65 mmHg and pulse rate 135/min. Nasotracheal intubation was performed immediately and mechanical ventilation was initiated using the combination of HFPPV, (rate 140/min, V_T : 50 ml), and low frequency conventional ventilation (rate : 6/min, V_T : 700 ml), with the addition of high inspiratory flow rate of gas (15 l/min) and free release of the over flow gas, through a 2-mm pore. Peak airway pressure for HFPPV was 8 cm H_2O and PEEP 6 cm H_2O, for the low rate ventilation peak airway pressure was 34 cm H_2O and PEEP 8 cm H_2O (intratracheal measurements). Arterial and pulmonary artery catheters were inserted and the following results obtained within 1 h of starting combined mode HFPPV/LFCMV: CVP: 12 mm/Hg, PAP: 34 mmHg, PcWP: 9 mmHg, CI: 3.47 l/min per m^2, SAP: 120/75, PVR: 278 dyne/cm^{-5} per second, TPR: 1060 dyne/cm^{-5} per second, Qsp/Qt: 18.3%. Blood gas analysis revealed normoventilation (PCO_2: 34 – 38 mmHg) and good oxygenation (PaO_2:

84 – 136 mmHg) with FIO_2 0.6. In the following hours it was possible to reduce the FIO_2 to 0.3.

The patient was ventilated for 3 days and the hemodynamic status remained stable. She did not require sedatives and was fully conscious and cooperative. The internal pneumatic fixation was very good. On the second day of ventilation the low frequency rate was lowered to 4 breaths/min and on the third day to 2 breaths/min. On the fourth day the mechanical ventilation was stopped and the patient immediately began breathing spontaneously, maintaining normal blood gases; 12 h later the patient was extubated. The chest X-ray improved.

On the day of discharge from the GICU, she had minimal paradoxical movement of the chest and normal blood gases. One week later she had no flail chest at all.

Patient 2. A 68-year-old man was admitted to the Emergency Room after falling from a height of 5 m. On admission he was restless, and physical and X-ray examinations revealed cyanosis, fractures of the anterior ribs, 2 – 7 right and 4 – 5 left, with flail chest and bilateral pneumo-hemothoraces with diffuse infiltrates in both lungs, fracture of left tibia, left Colles fracture and compression fracture of L1 vertebra without neurological deficit. Intercostal drainage was performed bilaterally and the patient was transferred to the GICU.

Initial tests showed a respiratory acidosis (pH: 7.23, PCO_2: 46 mmHg) and hypoxemia (PaO_2: 40 mmHg, PaO_2/FIO_2: 50). The patient was intubated (naso-tracheally) and mechanical ventilation was initiated immediately using the combination of HFPPV (rate 146, V_T: 70, low frequency conventional (rate: 4/min, V_T: 850 ml) and high inspiratory flow rate of 15 l/min, and free release pore of 2 mm diameter. Peak airway pressure for HFPPV was 10 cm H_2O and for the low rate ventilation peak airway pressure was 38 cm H_2O and PEEP was 10 cm H_2O. Hemodynamic measurements taken 45 min after starting ventilation revealed CVP: 10 mmHg, PAP: 43 mmHg, PcwP: 14 mmHg, CI: 4.37 l/min/m^2, SAP: 150/100, PVR: 267 dyne/cm^{-5} per second, TPR 1097 dyne/cm^{-5} per second and Qsp/Qt: 21%. These remained stable except for the pulmonary shunt which fell to 12% in the first 4 h.

Immediately after starting ventilation there was great improvement in blood gas status (pH: 7.38, PCO_2: 30 – 39 mmHg, PaO_2: 80 – 105 mmHg, FIO_2: 0.5 – 0.3). The rate of low frequency ventilation was gradually tailed. During mechanical ventilation the patient needed no sedatives and was fully awake and cooperative. The internal pneumatic fixation was very good. After 4 days ventilation was stopped and the patient immediately began to breathe spontaneously through a CPAP system. Eighteen hours later the patient was extubated, the drains were taken out and he was discharged from the GICU. A week later there was no flail chest and pulmonary functions were normal.

Since the patients were stuporous, informed consent was obtained from the next of kin.

Discussion

Direct serious trauma of the chest can cause fractures of ribs and sternum, rupture of the pleura and contusion of the lung parenchyma, resulting in flail chest, hemo and/or pneumothorax, rupture of lung tissue and airways, and bleeding in the lung tissue [10]. Most authors agree that the main problem is damage to the lung tissue per se and not the paradoxical movement of the chest wall [15]. Efforts must therefore be made to ameliorate the lung tissue damage. Since the description by Avery et al. of internal pneumatic fixation of the flail chest [2], surgical management of flail chest is no longer in use. Most patients with thoracic trauma die because of extra thoracic injuries. The influence of the lung injury itself on the mortality increases when the patient needs ventilatory assistance, because of the deleterious effects of positive pressure ventilation on the damaged lung tissue [9]. Many attempts have been made to find the best method of ventilation for these patients who already have damaged lung tissue. The latest method is IMV with PEEP [3], but results with this method showed the average time of ventilation to be 10 days with little improvement in survival. The combination of HFPPV and high oscillatory frequency has already been described by El-Baz et al. [4]. HFPPV has many advantages over normal frequency positive pressure ventilation (NFPPV) as a result of the lower airway pressure. These advantages include less depression of cardiac output, better oxygenation due to more uniform distribution of the inspired air and better diffusion of the air in the terminal airways [1, 6, 13], but in some patients we noticed difficulties in CO_2 removal when we used frequencies higher than 2 Hz. Another problem of HFPPV was the difficulty of humidification of the inspired air.

We attempted to solve these problems by the addition of a few conventional respirations and addition of a high flow of water vapour saturated gas. This high flow of gas also abolishes the high compressible volume and compliance of the respirator and corrugated tubes. (E. Barzilay, A. Lev, 1982, unpublished data). PEEP was achieved by two means: (a) it is an unseparable part of HFPPV, and (b) by the high flow of gas which lowers the compliance of the tubes and

adds some permanent internal pressure to the tubing system.

Using this system of combined HFPPV/LRCMV and HIF/FR we achieved normo ventilation and good oxygenation while keeping a low peak airway pressure, very good internal pneumatic fixation and a shortening of the duration of mechanical ventilation, compared to results obtained with other methods of ventilation [3]. We must emphasize that the patients were fully conscious and cooperative during ventilation and required only analgesics but no sedatives or muscle relaxants, had no spontaneous respiration and could restore their spontaneous breathing immediately after being disconnected from the respirators. To our knowledge this is the first description of such a system. Although the number of patients is small, we would like to suggest this method as another mode of ventilation for patients with contused lung, with respiratory failure and flail chest, who need internal pneumatic fixation.

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