

Extracorporeal Membrane Oxygenation in the Management of Severe Thoracic Trauma: A Case Report

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ABSTRACT

We describe a case where extracorporeal membrane oxygenation was used for seven days to facilitate surgery and respiratory therapy in a multi-trauma patient with severe pulmonary contusions, bilateral bronchopleural fistulae with recurrent pneumothoraces. The patient made a good recovery and was discharged from hospital after three months. (**Critical Care and Resuscitation 2001; 3: 97-100**)

Key words: Artificial ventilation, ECMO, pulmonary contusions, pneumothorax, intensive care

Severe acute hypoxic lung injury is uncommon but often fatal. Standard therapy includes high inspired oxygen concentrations, mechanical ventilation and positive end expiratory pressure (PEEP). Many other interventions have been used in parallel when conventional therapy fails such as extracorporeal gas exchange,¹ prone positioning² and drugs that modify the inflammatory response.³ Indications for extracorporeal membrane oxygenation (ECMO) include acute respiratory distress syndrome (ARDS), pneumonia and pulmonary contusions.

We describe the case of a young man who suffered severe multi-trauma including pulmonary contusions, ARDS, bilateral bronchopleural fistulae and recurrent pneumothoraces for whom ECMO was used to facilitate thoracotomy and repair of pulmonary laceration and to 'rest' the lungs in the immediate post-operative period.

CASE REPORT

A 32 year old man sustained severe chest trauma following a high speed motor vehicle accident. At the scene of the accident he had a Glasgow Coma Score of 8, left flail chest, right tension pneumothorax and an unrecordable blood pressure. A right-sided needle thoracocentesis was performed and intravenous Haemaccel 1000 mL was administered which increased his systolic

blood pressure to 125 mmHg. His heart rate was 168 beats per minute, respiratory rate was 42 breaths per minute and Glasgow Coma Score increased to 12. However, on arrival at hospital he was in severe respiratory distress with gross subcutaneous emphysema to the neck, chest and abdominal wall down to the groin.



Figure 1. CXR on admission to hospital showing a right sided tension pneumothorax, extensive surgical emphysema, left sided fractured ribs and clavicle with underlying pulmonary contusions.

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The patient was intubated and had bilateral chest tubes inserted as the right-sided tension pneumothorax had re-accumulated despite the needle thoracocentesis remaining in place (figure 1). The left chest tube drained 500 mL of blood in the first hour. A diagnostic peritoneal lavage was negative. During a computerised tomography the patient became haemodynamically unstable with a further loss of 1600 mL of blood from the left chest tube, so he was taken directly to the operating room for a left-sided thoracotomy.

At operation, 2000 mL of blood was found in the left pleural space which came from bleeding intercostal vessels associated with his rib fractures. A pneumo-mediastinum was also noted. Postoperatively, the patient was transferred to the intensive care unit hypothermic (34°C), coagulopathic (activated partial thromboplastin time was 74 s and prothrombin time was 24 s) and hypoxaemic with bilateral bronchopleural fistulae. The mechanical ventilator settings on admission to intensive care were as follows: synchronised intermittent mandatory ventilation with a respiratory rate of 12 breaths per minute, tidal volume 890 mL, pressure support ventilation of 10 mmHg, PEEP 15 cmH₂O and an inspired oxygen concentration (F_IO₂) of 0.95. The arterial blood gas results revealed a pH 7.2, pO₂ 146 mmHg, pCO₂ 40 mmHg, base excess -12 mmol/L, standard bicarbonate 15 mmol/L and arterial oxygen saturation (SaO₂) 98.4%. The chest X-ray at this time revealed a re-inflated right lung and increased left sided pulmonary opacification and subcutaneous emphysema (figure 2).



Figure 2. Chest X-ray showing a re-inflated right lung and increased left sided pulmonary opacification and subcutaneous emphysema.

Within one hour of his admission to intensive care, there was increasing abdominal distension, hypotension and increasing difficulty with ventilation. He was returned to the operating room and a laparotomy was

performed which revealed 2000 mL of intraperitoneal blood and a right perinephric haematoma. A small capsular tear on the inferior surface of the right lobe of the liver was also noted and packed. The abdomen was closed temporarily with Marlex mesh.

On his return from theatre, mechanical ventilation became increasingly difficult due to increasing air leaks from his bilateral bronchopleural fistulae, causing a persistent right sided pneumothorax and increasing subcutaneous and mediastinal emphysema. A second chest tube was inserted in the right pleural space and a bronchoscopy was performed to exclude tracheal and major bronchi tears. The patient became increasingly hypoxic. The blood gases at this stage showed a pH 7.01, pO₂ 50 mmHg, pCO₂ 60 mmHg, base excess -17 mmol/L, standard bicarbonate 15 mmol/L and SaO₂ 64.9% (F_IO₂ 1.0).

A double lumen endotracheal tube was inserted in an attempt to provide differential lung ventilation (Figure 3). However, an adequate seal with the double lumen tube proved difficult to achieve so a single lumen endotracheal tube was reinserted.

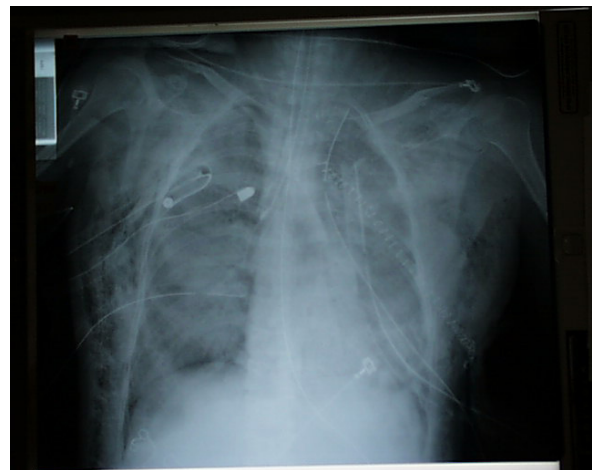


Figure 3. Chest X ray showing the patient intubated with a right bronchial double lumen endotracheal tube, three intercostal drains on the right and two on the left. Extensive bilateral pulmonary contusions and continued surgical emphysema are also seen.

After 48 hours of precarious ventilation using a pressure control mode, the packs and sponges were removed in the operating room without further haemorrhagic complications. However, the thoracic air leak remained a major problem with approximately two thirds of the tidal volume lost via the right intercostal tubes with each breath. The patient remained hypoxaemic, with a blood gas revealing a pH 7.36, pO₂ 68 mmHg, pCO₂ 37 mmHg, base excess -3 mmol/L, standard bicarbonate 21 mmol/L, SaO₂ 93.0% (F_IO₂ 0.75).

On day five he was transferred to the operating room and placed on a veno-venous extracorporeal membrane oxygenator (ECMO) to facilitate a right sided thoracotomy. At thoracotomy a right middle lobe laceration associated with one of the intercostal tubes was found and repaired. ECMO was continued postoperatively for 6 days in the intensive care unit. The vascular access from right femoral to right internal jugular vein (figure 4) allowed a blood flow of 2.8 L/minute. Oxygen was delivered at 3 L/minute. Mechanical ventilation continued at a tidal volume of 150 mL, a respiratory rate of 12 breaths/minute and PEEP 7 cmH₂O, all of which provided a SaO₂ 93%. During the period on ECMO, ventilation improved without further intervention, the air leaks diminished and the patient was weaned to pressure control ventilation and then to spontaneous ventilation with continuous positive airway pressure.

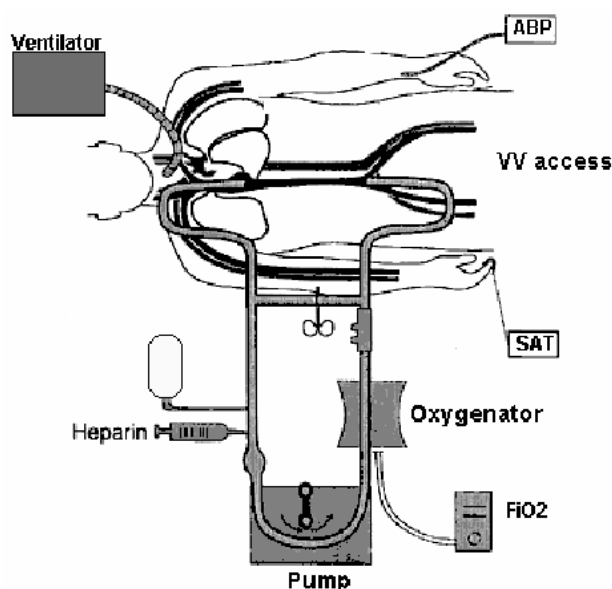


Figure 4. Schematic representation of the extracorporeal membrane oxygenation circuit used. SAT = pulse oximetry, ABP = arterial blood pressure

On day sixteen a percutaneous tracheostomy was performed and he was eventually decannulated on day 42. Recovery was slow and complicated by pneumonia. However on day 38 he underwent surgery for definitive closure of his abdomen and he was discharged to the ward the following day. He was finally discharged from hospital 70 days after the accident.

DISCUSSION

The case describes the use of ECMO to treat acute, reversible hypoxaemic respiratory failure and facilitate definitive surgery. The cause of the hypoxaemia was

multifactorial and included pulmonary contusions, ARDS, pneumothoraces and continued air leak from bilateral bronchopleural fistulae. Within eight hours of admission, all sources of internal bleeding were identified and treated but the patient was in severe respiratory failure resistant to conventional therapy.

The options for managing hypoxaemic ventilatory failure include mechanical ventilation with high inspired oxygen concentration and high PEEP, high frequency jet ventilation (HFJV), prone positioning, inhaled pulmonary vasodilators or surfactant.

These treatment modalities were unsuitable for a number of reasons. Conventional ventilatory strategies had failed and prone positioning was impractical because of cardiovascular instability and the presence of five chest drains. There is no evidence that pulmonary vasodilators and surfactant are beneficial in adults with pulmonary contusions.⁴ There have been no randomised controlled trials on the use of nitric oxide in adult acute hypoxic ventilatory failure to suggest improved outcome.⁵

Numerous studies have suggested that conventional ventilatory techniques may perpetuate or exacerbate lung injury by delaying or preventing recovery from ARDS. Ventilation with small tidal volumes to reduce end inspiratory lung volumes has been shown to improve mortality when compared with a more traditional ventilatory approach.⁶ High frequency jet ventilation may also offer an opportunity to achieve greater lung recruitment without overdistention while maintaining normal or near-normal acid-base parameters, and may also be beneficial in patients with bronchopleural fistulas. However, there have been no prospective randomised controlled studies that have demonstrated these theoretical benefits.

The ECMO process in our patient was relatively uneventful. Problems with coagulation were anticipated due to the patient's earlier coagulopathic state, however this was not borne out in practice with the blood circuit or with heparinisation. Change of the oxygenator led to a saturation of no lower than 90% and temperature control was achieved by warming the oxygenator. The patient's temperature was maintained between 36 and 37°C.

Increasing experience with prolonged bypass and ECMO have considerably improved the technique and therefore improved patient outcome.⁸ An increasingly younger population is admitted to intensive care units with severe multitrauma, surviving the initial injury yet being left with severe respiratory failure. The indications for ECMO may therefore widen and need to be reevaluated on an individual basis.

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