Postoperative extracorporeal membrane oxygenation for severe intraoperative SIRS 10 h after multiple trauma

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A 34-yr-old male suffered multiple trauma in a road traffic accident. He required right thoracotomy and laparotomy to control exanguinating haemorrhage, and received 93 u blood and blood products. Intraoperatively, he developed severe systemic inflammatory response syndrome (SIRS) with coagulopathy and respiratory failure. At the end of the procedure, the mean arterial pressure (MAP) was 40 mm Hg, arterial blood gas analysis showed a pH of 6.9, \( P_aCO_2 \) 12 kPa, and \( P_aO_2 \) 4.5 kPa, and his core temperature was 29°C. There was established disseminated intravascular coagulation. The decision was made to stabilize the patient on venovenous extracorporeal membrane oxygenation (ECMO) only 10 h after the accident, in spite of the high risk of haemorrhage. The patient was stabilized within 60 min and transferred to the intensive care unit. He was weaned off ECMO after 51 h. He had no haemorrhagic complications, spent 3 weeks in the intensive care unit, and has made a good recovery.

Keywords: acid–base equilibrium, metabolic acidosis; blood, transfusion; complications, trauma; oxygen, delivery systems, extracorporeal membrane oxygenation

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A 34-yr-old male was involved in a road traffic accident as the driver of a car. The car hit the central reservation at 70 mph, resulting in an impact with a street light on the driver’s side. The driver suffered multiple fractures together with blunt abdominal and thoracic trauma and was transferred to the nearest accident and emergency department of a hospital without thoracic surgery facilities.

He arrived with a Glasgow Coma Scale of 15, tachycardic and hypotensive, in respiratory distress (heart rate 100 beats min\(^{-1}\), arterial pressure 75/40 mm Hg, ventilatory frequency 26 bpm, oxygen saturation 90%). Clinical examination and diagnostic imaging confirmed a fractured right femur, fractured left hemipelvis, right serial rib fractures with severe lung contusion and haemothorax, a fractured right clavicle, a large haematoma spreading from the right flank down to the right buttock, and frank haematuria. At the time, there was no clinical or radiological (spiral CT) evidence of serious intra-abdominal injury.

During the 3 h the patient spent in the accident and emergency unit and the x-ray department, he required intubation and ventilation for increasing respiratory distress. He needed a total of 20 u red cell concentrates, 2 u platelets, 7 u cryoprecipitate, and 4 u fresh frozen plasma (FFP). Bilateral chest drains were inserted with an initial blood loss from the right side of 1000 ml and 400 ml in the subsequent hour. Computed tomography showed an intact thoracic aorta and no evidence of haemoperitoneum at this time. The patient was transferred to the nearby cardiothoracic centre.

On arrival, the patient had a mean arterial pressure (MAP) of 40–50 mm Hg with atrial fibrillation of 90 min\(^{-1}\), a \( \text{SpO}_2 \) of 80% on oxygen 100%, and a nasopharyngeal temperature of 35°C. The patient was reintubated with a double lumen tube and an attempt was made to stabilize his temperature by using two blood warmers (Fenwal), a humidifier in the anaesthetic circuit, and a warming blanket. The patient continued to bleed from his chest drains and was taken to theatre immediately.

Immediately before thoracotomy, the patient suffered a brief asystolic cardiac arrest on the operating table. Without delay, the surgeons performed a right thoracotomy. He was successfully resuscitated with two 50 µg boluses of epinephrine and open cardiac massage. The intraoperative
findings were several lacerations, severe contusion, and bleeding from the right lower lobe. There was a rupture of the right hemidiaphragm and a large retropleural haematoma. The right lower lobe was partially resected and haemostasis achieved. There was also significant ongoing blood loss from the airways of the right lung that only stopped after high frequency jet ventilation was applied to it. This also contributed to the control of surface bleeding from the right lung. Continuous ongoing blood loss and the presence of a diaphragmatic defect led to a laparotomy, which revealed a ruptured bladder, several liver lacerations, and 1500 ml of blood in the abdomen. The bladder and the lacerations were repaired, and the liver was packed.

In spite of hyperventilating the healthy lung with oxygen 100% and 7 cm H₂O of PEEP, jet ventilating the injured lung, and attempting to correct the metabolic acidosis with sodium bicarbonate, the blood gases deteriorated further (pH 6.9, PₐCO₂ 13–15 kPa, PₐO₂ 6–8 kPa and base excess (BE) −8.3). At this stage the airway pressures increased to 40 cm H₂O. Although four anaesthetists were actively involved in the resuscitation process, the MAP could only be maintained at 40 mm Hg with high doses of dopamine and epinephrine. Dopamine was infused at 10 μg kg⁻¹ min⁻¹ with epinephrine at 0.1 μg kg⁻¹ min⁻¹, together with intermittent epinephrine boluses of 0.5–1 mg every 5–8 min when required. Transfusion was guided by the central venous pressure, which was constantly very low (0–1 cm H₂O), the ongoing major blood loss, and visualization of the heart through the right thoracotomy. The patient was diagnosed clinically as in hypovolaemic shock.

At the end of the 6 h procedure, the nasopharyngeal temperature was 29°C and the patient’s condition continued to be critical. As a result of massive transfusion and hypothermia, and in spite of administration of clotting factors and aprotinin, the patient clinically developed disseminated intravascular coagulopathy (DIC) and was bleeding from all orifices. The DIC was confirmed by a full clotting profile. There was no urine output for the previous 3 h and attempts at active re-warming had failed. At this point the patient had received a total of 54 u blood, 12 u platelets, 10 u FFP, and 27 u cryoprecipitate.

In view of the persistence of the hypoxia, hypothermia, and acidosis, it was agreed by all the clinicians involved that the patient’s prognosis was grave, and that extracorporeal membrane oxygenation (ECMO) was the only remaining option. Heparin 50 u kg⁻¹ i.v. was given and the right internal jugular vein (drainage) and the left femoral vein (return) were cannulated percutaneously. The patient was commenced in the operating theatre on veno-venous ECMO with a flow of 4.5 litre min⁻¹ and gradually re-warmed. The ECMO circuit was as described previously, with the substitution of two Medos 7000 LT polymethyl pentene oxygenators for the usual silicone devices. During ECMO, the infusion of aprotinin was continued and an infusion of heparin was used to maintain the activated clotting time between 180 and 200 s. Within 1 h, the patient’s condition was markedly improved. His MAP had risen to 70 mm Hg and the atrial fibrillation had reverted to a sinus tachycardia. The pH was 7.4, PₐCO₂ 4.6 kPa, PₐO₂ 9.4 kPa, and BE −1.2 with a core temperature of 33°C.

While on ECMO, the patient was transferred to the intensive care unit, 6 h after the operation. The patient was anuric and continuous veno-venous haemofiltration was initiated on arrival. After 12 h, his inotrope requirements were greatly reduced and his condition was stable with a normal full blood count, normalizing coagulation, and a core temperature of 36.5°C. The double lumen tracheal tube was changed to a single lumen tube and ventilation was reduced to allow lung rest (pressure 15/5 cm H₂O, rate 10 min⁻¹, FₐO₂ 0.3). He was weaned from ECMO after only 51 h to pressure-controlled ventilation on FₐO₂ 0.4.

After 5 days the patient had a further laparotomy for removal of packs, a surgical tracheostomy, and an intramedullary nail for the left femur fracture. Continuous veno-venous haemofiltration was continued for 12 days. The patient made a good recovery with no neurological injury. He was transferred to a rehabilitation unit 6 weeks after the accident.

Discussion

We are certain that this patient would not have survived without treatment with ECMO. We were unable to maintain adequate perfusion and oxygenation, and failed to correct the coagulopathy, hypothermia, and metabolic acidosis with conventional therapy. As there was no clinical or radiological (spiral CT) evidence of intra-abdominal injury in this patient, peritoneal lavage was not indicated. In contrast, an emergency thoracotomy was indicated as there was clear evidence of serious intrathoracic injury. As soon as intra-abdominal injury was suspected because of continued hypovolaemia after control of the intrathoracic bleeding, appropriate action was taken.

The cardiac output and systemic vascular resistance were not measured in the operating theatre. This was a result of the constant demand for fluid and metabolic resuscitation during the procedure. The anaesthetists were guided by the central venous pressure which was constantly very low (0–1 cm H₂O), the major blood loss, and visualization of the heart through the right thoracotomy. The patient was diagnosed clinically as in hypovolaemic shock. A pulmonary artery catheter was not inserted in the operating room, as it would have added little to the information already available and would have consumed valuable time. In addition, a pulmonary artery catheter was not inserted on the patient’s return to the intensive care unit as it is not possible to use the thermodilution technique whilst on veno-venous ECMO with jugular venous drainage; the indicator is drained into the ECMO circuit (G. J. Peek, 1995, unpublished observations).

Transfusion-associated acute lung injury is often unrecognized. Antibodies in donor plasma react with
recipient white cells, causing capillary leak and non-cardiogenic pulmonary oedema. The combination of shock, tissue injury, and massive transfusion can culminate in the adult respiratory distress syndrome (ARDS). The diagnosis of systemic inflammatory response syndrome (SIRS) requires the presence of two or more clinical signs of a systemic inflammatory response. As some of the signs (tachypnoea, tachycardia, or hypothermia less than 36°C, etc.) are common to patients in the intensive care unit or operating theatre, SIRS can be a ubiquitous condition. The multiple trauma in this patient, together with the massive operation, prolonged hypotension, hypothermia, and blood transfusions caused multiple organ dysfunction syndrome affecting the cardiovascular, respiratory, renal, and coagulation systems. This triggered the systemic inflammatory response.

In spite of the high risk of haemorrhage, ECMO was this patient’s only chance of survival. We decided to use veno-venous ECMO and not veno-arterial ECMO or cardiopulmonary bypass for several reasons. This was a young patient with good underlying cardiac function. His main problem was impaired tissue oxygen delivery as a result of hypoxaemia (from lung contusion), hypoperfusion as a result of hypovolaemia, and high airway pressures. This led to a severe metabolic acidosis (which would have affected cardiac contractility), and respiratory distress. Hypothermia was also a significant myocardial depressant. The high dose of inotropic and vasopressor support was used to maintain an adequate perfusion pressure to vital organs. Cardiopulmonary bypass was contraindicated by virtue of the high dose of heparin required. Veno-arterial ECMO is more invasive than veno-venous ECMO and requires cannulation of either the femoral or carotid arteries. It is our experience that, when myocardial depression is a result of reversible factors (such as hypothermia, acidosis, hypoxia, and high airway pressure), it will almost always respond to veno-venous ECMO. Myocardial oxygenation is actually better on veno-venous ECMO than on veno-arterial ECMO. Veno-venous ECMO also avoids ischaemic injury to the limbs.

A similar patient was described by Plotkin and colleagues. A 25-yr-old pregnant woman presented with gunshot injuries to her chest and subsequent traumatic ARDS. This patient was commenced on ECMO 12 h after operation and weaned successfully after 41 h. Perchinsky and colleagues describe a simplified arterio-venous extracorporeal cardiopulmonary life support system. The circuit is heparin-bonded to avoid systemic anticoagulation and to reduce the risk of haemorrhage, and was used in six trauma patients in similar condition to our patient for between 5 and 23 h. Three patients survived and made a good recovery, but one suffered ischaemic injuries to the cannulated leg and required an above knee amputation.

We used heparin-bonded polymethyl pentene oxygenators. There is little advantage in terms of reduction in systemic heparin dose with heparin-bonded tubing compared with unbonded tubing. Commercially available heparin-bonded support systems based on polypropylene microporous oxygenators develop severe plasma leak from the oxygenator after 6–24 h necessitating frequent oxygenator change. Our experience is that administration of aprotinin to such patients is sufficient to control any haemorrhagic tendencies.

We believe that warming the patient too rapidly would be detrimental. Keeping his temperature at 33–34°C for the first few hours may have had a neuroprotective effect, and also played a role in the protection of his other organs. All our blood gas results were temperature corrected as we used an alpha-stat protocol. Although the slow rewarming did not help the coagulopathy, the long-term outcome of this approach was successful and he was left with no neurological damage. He was referred to the rehabilitation unit after only 6 weeks to assist him back into society.

This was a young, previously healthy patient. The reason for his renal failure was presumed to be secondary to prolonged hypotension leading to acute tubular necrosis. He suffered severe multitrauma and rhabdomyolysis most possibly contributed to his renal failure. We did not measure creatinine kinase. His renal function made a full recovery.

The significant and rapid improvement after adequate oxygenation and perfusion confirms how valuable oxygen delivery is in the severely traumatized patient. In this patient, conventional ventilation was not successful and, if continued for a longer period of time, would have contributed to further baro- and volotrauma. When lung injury is extensive, often the only successful means of adequate oxygenation is extracorporeal. In our institution, the average ECMO run for ARDS is around 2 weeks. We wean according to the patient’s ability to maintain homeostasis. A period of 51 h is therefore a short ECMO run and could only have been achieved because this patient’s metabolic status was normalized without further delay. This impressive response to veno-venous ECMO confirms its usefulness in desperate situations, where one needs short-term organ support in a patient with severe SIRS, but survivable primary injuries.

References
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An 18-yr-old man with insulin-dependent diabetes developed severe subglottic stenosis after a very brief period of intubation. Emergency tracheostomy was complicated by the development of bilateral pneumothoraces. This case highlights the importance of making an early diagnosis to minimize the risk of complications and examines postintubation subglottic stenosis in the context of poorly controlled insulin-dependent diabetes mellitus.

**Keywords**: complications, diabetes; complications, pneumothorax; complications, stenosis

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Subglottic stenosis is a late, uncommon and serious complication of intubation. Although usually associated with prolonged intubation, this is not the only factor in its development. We report a stenosis after brief intubation. Possible contributing factors include poorly controlled insulin-dependent diabetes mellitus (IDDM). A high index of suspicion for this rare but life-threatening complication is necessary.

**Case report**

An 18-yr-old man was admitted to hospital after a road traffic accident. He was a smoker with poorly controlled IDDM. Decreasing consciousness necessitated tracheal intubation after induction of anaesthesia with propofol and succinylcholine. A standard size 8.5 mm oral tracheal tube (ETT) was placed with ease, with the tip positioned 25 cm from the lips. The vocal cords and surrounding structures appeared normal. After this, sedation was provided by propofol infusion and paralysis was maintained with atracurium.

A computed tomography (CT) scan of the patient's head showed multiple small haematomas at the interface between the grey and white matter, but was otherwise unremarkable. He was admitted to the intensive care unit (ICU) and was extubated after an uneventful 36 h. He was discharged from the ICU and, some days later, home.

Two months later he re-presented complaining of progressive shortness of breath since discharge. There was no history of stridor, wheeze or other respiratory symptoms. Chest x-ray showed consolidation in the right middle lobe. Despite treatment with antibiotics, he remained dyspnoeic and developed respiratory failure. Intubation and ventilation were indicated.

Anaesthesia was induced with propofol and succinylcholine. A size 9.0 mm ETT could not be placed, and the largest tube that could be placed was size 6.0 mm. Subsequent fibreoptic examination showed a swollen epiglottis obscuring the laryngeal inlet and vocal cords. No obstruction was seen beyond the tip of the ETT. An 8 mm ETT was placed easily. Respiratory failure was attributed to epiglottic swelling, although it was unclear whether the swelling was the primary problem or the result of trauma during attempts to intubate. He was nursed with a head-up tilt of 30° and received dexamethasone 4 mg four times daily. He was weaned from ventilation on day 5. When he was examined by an ear, nose and throat (ENT) consultant using per-nasal fibreoptic laryngoscopy, a subglottic stenosis was...