After reversal of residual neuromuscular block, the patient was woken, and he made an uneventful recovery with no apparent untoward effects.

Discussion

Many techniques and lots of instruments have been designed to overcome the problem of the difficult airway and/or difficult intubation, but they are often either too complicated, too costly or unavailable, especially in poor parts of the world. In 1998, a hand-made device was produced to address this problem.

The CPT has now been used in a further 16 cases of difficult airway, and in each case a clear airway was obtained easily and rapidly at the first attempt. The device has proved to be well tolerated and is not easily displaced. Laryngoscopy, though not essential, is recommended, because it locates the epiglottis and facilitates insertion of the device.

When a difficult airway is expected, spontaneous respiration should be maintained but anaesthesia should be deep enough to allow gentle insertion and to prevent rejection of the tube after the cuff has been inflated. Many patients refuse inhalation induction with halothane, and the alternative practice in my institution is a technique based on the use of increasing amounts of analgesics (similar to the principle of pre-emptive analgesia). A benzodiazepine and small doses of thiopental and a neuromuscular blocking drug are given, while trying to maintain spontaneous respiration with halothane. The addition of small non-apnoeic doses of a neuromuscular blocking agent (e.g. atracurium 10 + 10 mg) facilitates intubation and reduces the likelihood of CPT rejection after the cuff has been inflated. The large dose of neuromuscular blocking agent used in the second patient was inappropriate, but this case is included because it demonstrates the successful use of the CPT in the presence of full muscle relaxation.

References


Use of nitric oxide for decompensated right ventricular failure and circulatory shock after cardiac arrest

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We describe a case of peri-operative cardiac arrest, severe right ventricular failure and pulmonary hypertension in a 60-year-old woman with interstitial pulmonary fibrosis. Inhaled nitric oxide therapy rapidly improved arterial oxygenation and haemodynamic variables, allowing...
Inhaled nitric oxide (iNO) is widely used in intensive care units in the UK as a powerful selective dilator of pulmonary blood vessels. It has been used to treat pulmonary hypertension in a number of situations such as after paediatric cardiac surgery, implantation of left ventricular assist devices, pulmonary embolectomy, lung transplantation and protamine-induced pulmonary hypertension. Oxygenation in acute respiratory distress syndrome may be improved by reducing ventilation/perfusion mismatch.

Inhaled nitric oxide improves right ventricular function by selective pulmonary vasodilation, which in turn improves left ventricular filling, cardiac output and systemic arterial pressure.

We report the use of iNO as a life-saving measure in a patient with pulmonary fibrosis who had a cardiac arrest after a pulmonary biopsy under general anaesthesia.

### Case history

A 60-yr-old woman was scheduled for video-assisted lung biopsy to establish a histological diagnosis of lung pathology. She had a 3.5 yr history of increasing shortness of breath, which had become acutely worse in the 10 weeks before admission. She was unable to walk more than a few yards and was oxygen dependent. She had smoked 30 cigarettes a day for 30 yr and had stopped smoking 13 yr before admission. Her past medical history included a history of thrombocytopenia, which had been treated with hydroxyurea, and this was suggested as a possible cause of the pulmonary fibrosis. Therapy on admission was salbutamol and steroid inhalers, hydroxyurea, frusenide and oxygen.

Blood gas analysis at this time showed that the pH was 7.52, \( P_AO_2 \) 8.0 kPa, \( P_ACO_2 \) 3.5 kPa, \( HCO_3^- \) 21.2 mmol litre\(^{-1}\) and \( S_AO_2 \) 93% on a fractional inspired oxygen concentration (\( FIO_2 \)) of 0.24. Pulmonary function tests showed an FEV\(_1\) of 1.31 litres and FVC of 2.59 litres. The FEV\(_1\)/FVC ratio was 0.51.

An ECG showed a sinus tachycardia with a rate of 108 beats min\(^{-1}\), right axis deviation and right atrial and ventricular enlargement. Cardiomegaly and enlarged pulmonary arteries consistent with pulmonary artery hypertension were present on a computed tomography scan of the thorax. The lung fields were abnormal, with increased interstitial markings throughout.

Pre-operative transthoracic echocardiography showed right ventricular enlargement with impaired function, pulmonary hypertension (systolic pulmonary artery pressure 40 mm Hg), moderate to severe tricuspid regurgitation and a small left ventricle with preserved function. Urea, electrolytes and full blood count were normal.

Before induction of anaesthesia, intravenous access was obtained and an arterial cannula was inserted into the left radial artery. After pre-oxygenation, propofol and remifentanil were used for induction and maintenance of anaesthesia. Arterial pressure was 95/56 mm Hg, heart rate 88 beats

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### Table 1 Cardiorespiratory and inotrope parameters against time of starting iNO. [iNO]=inhaled nitric oxide concentration. \( P_AO_2 \)=arterial partial pressure of oxygen; \( FIO_2 \)=fractional inspired concentration of oxygen; \( S_AO_2 \)=arterial haemoglobin saturation; SIMV=synchronized intermittent mandatory ventilation; BIPAP=biphasic intermittent positive airway pressure; CPAP=continuous positive airway pressure

<table>
<thead>
<tr>
<th>Time after starting nitric oxide (min)</th>
<th>0</th>
<th>15</th>
<th>120</th>
<th>240</th>
<th>480</th>
<th>600</th>
</tr>
</thead>
<tbody>
<tr>
<td>[iNO], ppm</td>
<td>0</td>
<td>4</td>
<td>4</td>
<td>10</td>
<td>9</td>
<td>0</td>
</tr>
<tr>
<td>( P_AO_2/FIO_2 ), mm Hg</td>
<td>88</td>
<td>150</td>
<td>201</td>
<td>201</td>
<td>181</td>
<td>123</td>
</tr>
<tr>
<td>Mean arterial pressure, mm Hg</td>
<td>55</td>
<td>68</td>
<td>65</td>
<td>62</td>
<td>67</td>
<td>64</td>
</tr>
<tr>
<td>Central venous pressure, mm Hg</td>
<td>25</td>
<td>12</td>
<td>11</td>
<td>15</td>
<td>8</td>
<td>12</td>
</tr>
<tr>
<td>Norepinephrine, ( \mu )g kg(^{-1}) min(^{-1})</td>
<td>0.4</td>
<td>0.4</td>
<td>0.13</td>
<td>0.13</td>
<td>0.13</td>
<td>0.13</td>
</tr>
<tr>
<td>Epinephrine, ( \mu )g kg(^{-1}) min(^{-1})</td>
<td>0.2</td>
<td>0.2</td>
<td>0.2</td>
<td>0.2</td>
<td>0.23</td>
<td>0.32</td>
</tr>
<tr>
<td>Milrinone, ( \mu )g kg(^{-1}) min(^{-1})</td>
<td>1.2</td>
<td>1.2</td>
<td>0.66</td>
<td>0.66</td>
<td>0.66</td>
<td>0.66</td>
</tr>
<tr>
<td>pH</td>
<td>7.19</td>
<td>7.23</td>
<td>7.29</td>
<td>7.37</td>
<td>7.34</td>
<td>7.40</td>
</tr>
<tr>
<td>( S_AO_2 ) (%)</td>
<td>91</td>
<td>99.3</td>
<td>98.8</td>
<td>100</td>
<td>98.5</td>
<td>94.6</td>
</tr>
<tr>
<td>Ventilatory mode</td>
<td>SIMV</td>
<td>BIPAP</td>
<td>ASB</td>
<td>ASB</td>
<td>mask CPAP</td>
<td>Face mask</td>
</tr>
</tbody>
</table>
in\(^{-1}\) and oxygen saturation (\(\text{SpO}_2\)) 99–100%. Vecuronium 10 mg was given at induction to facilitate tracheal intubation with a 37FG left-sided double lumen tube. After induction of anaesthesia, a right internal jugular venous catheter and thoracic epidural (T\(_7/8\) level) were placed uneventfully. A total of 10 ml of 0.25% bupivacaine was injected through the epidural catheter. At this time the arterial pressure was 80/50 mm Hg and heart rate 94 beats min\(^{-1}\). The epidural was not used again after this.

After induction and intra-operatively there was a slow but sustained decrease in arterial pressure to 60/25 mm Hg, the heart rate increased to 120 beats min\(^{-1}\) in sinus rhythm and central venous pressure (CVP) was increased, at 25 mm Hg. The hypotension did not respond to doses of epinephrine (25 \(\mu\)g in total) and infusion (0.1–0.2 \(\mu\)g kg\(^{-1}\) min\(^{-1}\)) or methoxamine (2 mg in total). \(\text{SpO}_2\) remained at 99% with an \(\text{FiO}_2\) of 0.5 in air. During lung biopsy the patient developed progressive hypotension and hypoxia, particularly during one-lung anaesthesia. This proved difficult to treat despite increasing the epinephrine infusion, ventilation with 100% oxygen and the use of PEEP (5 cm H\(_2\)O) and CPAP (5 cm H\(_2\)O) on the dependent and nondependent lung.

On return to two-lung ventilation the saturation improved sufficiently to consider extubation (\(\text{SpO}_2\) 97%). However following reversal of residual neuromuscular blockade, before the removal of the tracheal tube the patient suffered an electromechanical dissociation cardiac arrest.

After 20 min of advanced CPR (including the venesection of 1 litre of blood), spontaneous cardiac output returned. We then attempted to pass a pulmonary artery catheter balloon via the right subclavian vein but this could not be advanced beyond the right ventricle. At this time the arterial pressure was 75/35 mm Hg, CVP 35 mm Hg and right ventricular pressure 90/10 mm Hg.

Arterial blood gases showed severe hypoxaemia with \(P_{\text{AO}}\) 4.0 kPa, \(P_{\text{ACO}}\) 6.7 kPa, pH 7.32, with an \(\text{FiO}_2\) of 1.0 and positive pressure ventilation. The patient was already receiving an epinephrine infusion (0.2 \(\mu\)g kg\(^{-1}\) min\(^{-1}\)); she was transferred to the Cardiac Intensive Care Unit (CICU).

On arrival in the CICU, arterial pressure was 70/30 mm Hg, heart rate 100 beats min\(^{-1}\) and regular, CVP 28 mm Hg and right ventricular pressure 75/10 mm Hg. Inotropic support at this time consisted of epinephrine (0.2 \(\mu\)g kg\(^{-1}\) min\(^{-1}\), norepinephrine (0.4 \(\mu\)g kg\(^{-1}\) min\(^{-1}\)) and milrinone (1.2 \(\mu\)g kg\(^{-1}\) min\(^{-1}\)) infusions. Transthoracic echocardiography showed a grossly dilated right heart (right atrium 6.9 cm) with reduced right ventricular function, reversed septal motion and a small compromised left ventricle with preserved function. There was severe tricuspid regurgitation and an estimated pulmonary artery pressure of 65 mm Hg.

As the patient was in \textit{extremis}, a decision was made to start iNO as a life-saving measure. During the 45 min it took to set up the iNO circuit, the patient remained profoundly shocked, oxygen dependent and unresponsive to changes in inotropic support.

Inhaled nitric oxide (British Oxygen Company, Special Gases Division, Guildford, UK) at a concentration of 4 parts per million (ppm) was started using a nitric oxide delivery system (NODemo, Drager, Germany) through the ventilator (Evita 4, Drager, Germany). This machine delivers the nitric oxide into the inspiratory limb of the breathing circuit, the volume delivered being in proportion to the inspiratory limb gas flow.

Within 10 min of commencing iNO, haemodynamic and gas exchange status improved rapidly. Norepinephrine and milrinone requirements were reduced to 0.13 \(\mu\)g kg\(^{-1}\) min\(^{-1}\) and 0.66 \(\mu\)g kg\(^{-1}\) min\(^{-1}\), respectively, and the \(P_{\text{AO}}\) was decreased to 0.6. The patient’s condition stabilized and weaning from mechanical ventilation was started. The patient progressed to face mask oxygen after 10 h, extubation occurring 5 h after beginning iNO. After the tracheal tube was removed, iNO was continued, using a facial CPAP mask, through the Evita 4 ventilator for a further 4 h. After discontinuing iNO there was some deterioration in gas exchange, but it was not sufficient to warrant restarting it (Table 1).

The nitrogen dioxide concentration did not exceed 0.1 ppm and methaemoglobin concentrations were <0.5% throughout the treatment with iNO.

Haemodialfiltration was instituted on the second day after surgery because the patient developed acute tubular necrosis; it was continued until day 6 of her CICU stay. During this time her inotropic requirements decreased and all inotropes were stopped on day 5. After 7 days the patient was transferred to the High Dependency Unit and she left hospital 2 days later. Lung biopsy showed cryptogenic fibrosing alveolitis and the patient is now awaiting heart/lung transplant.

\textbf{Discussion}

We report the use of iNO in treatment of right heart failure after cardiac arrest. To our knowledge this is the first successful reported use of iNO after cardiac arrest.

Anaesthesia in patients suffering from pulmonary hypertension is difficult, particularly if the patients are to undergo procedures which require one-lung anaesthesia, when hypoxic vasoconstriction in the nondependent lung causes the pulmonary vascular resistance (PVR) to increase more than with two-lung ventilation.

We chose to use a total intravenous anaesthetic technique with a thoracic epidural for this patient. Propofol infusions minimize the ventilation/perfusion mismatch that occurs with one-lung anaesthesia.\textsuperscript{8} Thoracic epidural blockade appears to be the technique of choice for analgesia after thoracotomy.\textsuperscript{9} Although our patient was only undergoing thoracoscopy, we considered epidural analgesia essential to prevent postoperative hypoventilation and avoid ventilatory support. Although thorascopic surgery is considered less traumatic than thoracotomy, a recent study found moderate
or even severe pain scores after minor thoroscopic procedures and a decrease in FEV1 of >50%. However, the technique we used could have contributed to the systemic hypotension seen in our patient because propofol, remifentanil and epidural blockade can all have this effect.

In patients with systemic sclerosis and pulmonary hypertension, exhaled nitric oxide concentrations are lower than those in patients with systemic sclerosis who do not have pulmonary hypertension. It is likely that the same would occur in pulmonary fibrosis. This is supported by the demonstration that, in a patient with severe pulmonary fibrosis, hypoxia and pulmonary hypertension, iNO treatment reduced pulmonary vascular resistance considerably, increased cardiac output and improved oxygenation. The use of intravenous prostacyclin (PGE1) in the same patient resulted in arterial oxygen desaturation and increased dyspnoea. Yoshida and colleagues also found that iNO treatment can decrease pulmonary artery pressure in patients suffering from pulmonary fibrosis. However no improvement in arterial oxygenation was demonstrated whilst breathing room air. If, however, 1 litre min\(^{-1}\) of oxygen was then breathed during the iNO treatment, there was a further decrease in pulmonary artery pressure and a significant improvement in arterial oxygenation compared with oxygen therapy alone.

These findings suggest that iNO will benefit patients with pulmonary fibrosis who have hypoxia associated with pulmonary hypertension. Our patient had right ventricular failure because of the high pulmonary vascular resistance. The flow of blood to the left atrium and ventricle was insufficient to maintain an acceptable systemic blood pressure. All these haemodynamic problems rapidly improved when iNO reduced the pulmonary vascular resistance. iNO may be valuable in patients with pulmonary fibrosis and pulmonary hypertension who are undergoing surgery.

References
13 Yoshida M, Taguchi O, Gabazza EC et al. The effect of low-dose