Barotrauma during apnoea testing for brain death determination in a five-year-old boy

The apnoea test is an essential part of brain death confirmation. The procedure most frequently employed to perform apnoeic oxygenation consists of introducing a catheter into the endotracheal tube, advancing it to the trachea while oxygen at a flow of 6 l/min is administered. Although apnoeic oxygenation has traditionally considered a safe technique, cardiovascular and iatrogenic complications are reported. Patients are at risk of hypoxaemia, bradycardia and hypotension and, when performed in the above manner, barotrauma is a potential complication.

A five-year-old boy with a past history of Angelman syndrome with intellectual and developmental delay and seizures under treatment with clobazam and sodium valproate was transferred to our hospital after three days of fever of respiratory origin with increasing dysnoea and apnoeic pauses. Out-of-hospital CPR was performed by an emergency team for 20 minutes because of apnoea and pulselessness. After initial resuscitation, he suffered three further episodes of asystole while he was being transferred to the hospital. On admission at the emergency ward he was in coma with a Glasgow Coma Score of 3, bilateral unreactive pupils, absent brainstem reflexes, profoundly hypotensive and with mild hypothermia. The patient was admitted to the intensive care unit where his neurological state remained unchanged.

We examined the child to confirm brain death when polyuria and hypernatraemia appeared, nearly 24 hours after the first cardiac arrest. Core temperature was 36.6 °C, Glasgow Coma Score 3 without motor response in all extremities, pupils were dilated without response to bright light, absent oculocephalic reflex and caloric responses to irrigation in each ear with 50 ml of cold water, absence of facial motor response and corneal, jaw, pharyngeal and tracheal reflexes. Blood gases performed after 10 minutes’ preoxygenation showed a pH 7.51, P\textsubscript{O\textsubscript{2}} 200 mmHg and P\textsubscript{CO\textsubscript{2}} 41 mmHg. After ventilator disconnection, an oxygen catheter of 4 mm inner diameter was inserted inside an endotracheal tube 26 cm in length and an inner diameter of 6 mm and advanced to the carina. An oxygen flow of 6 l/min was insufflated through the catheter. One minute later his chest, neck and face became massively hyperinflated and cyanosed. Blood pressure decreased rapidly to 76/45 mmHg and oxygen saturation decreased to 85%, pulse rate increased from 110 to 130 beats/min. When the oxygen catheter was removed and the patient was reconnected to the ventilator the chest and face deflated and blood pressure and oxygen saturation rose to normal values. Palpation over the face, neck, chest and abdominal wall demonstrated subcutaneous emphysema. Air entry to the left lung was decreased. A chest X-ray showed massive barotrauma with subcutaneous emphysema over the face, neck, chest and abdominal wall, left-sided pneumothorax and pneumoretroperitoneum. Given the chronic disability associated with his genetic disorder, the irreversibility of the brain insult and no potential for organ donation, therapy was limited and a chest drain not inserted. The patient died six hours later.

Demonstration of absent spontaneous respiratory efforts by apnoea testing is an essential part of the determination of brain death. It is commonplace to avoid concomitant hypoxaemia and adverse cardiovascular responses by means of a tracheal catheter with oxygen at a flow of 6 l/min.

There are several reports in medical literature of pulmonary barotrauma associated with apnoea testing\textsuperscript{2-5}. The first description was cited as “subcutaneous emphysema and thoracic inflation\textsuperscript{6}” but most of the cases developed a tension pneumothorax, sometimes bilateral\textsuperscript{6}. Other forms of extra-alveolar air like interstitial gas, pneumomediastinum\textsuperscript{7} and pneumoperitoneum\textsuperscript{8} are also described. The proposed pathophysiologic mechanism leading to barotrauma associated with this technique is insufflation a high flow of oxygen through a catheter wedged in a bronchus or trachea or, mainly in children, a tracheal catheter wedged in a small bore endotracheal tube with insufficient room for gas escape leading to massive air trapping. This complication could have potential legal implications if brain death had not been established or could jeopardise organ procurement when the patient is a candidate for organ donation.

To avoid this complication, some authors advocate the use of other systems to perform apnoeic oxygenation, such as the T-piece and the CPAP systems, bulk diffusion and setting the ventilator rate to zero providing a continuous flow of oxygen via the endotracheal tube. When oxygen insufflation is performed through a tracheal catheter some precautions must be taken: first, assuring a small external diameter of this catheter, significantly smaller than the inner diameter of the endotracheal...

tube is mandatory, especially in children: second, the catheter should not be introduced beyond the tip of the endotracheal tube and if resistance is noted it must be withdrawn slightly. Third, the oxygen flow should not exceed 6 l/min in adults and probably less in small children. Finally, some authors have discouraged the use of tracheal catheters because of the technical difficulties reported.

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References

Remifentanil for supraventricular tachycardia

We report the inadvertent use of high dose remifentanil proving beneficial for rate control in a patient with supraventricular tachycardia (SVT) scheduled for DC cardioversion. A 76-year-old male weighing 82 kg presented with acute shortness of breath and palpitations. On examination, he was dyspnoeic and in visible distress. His pulse was regular but rapid at 180 /min, blood pressure was 154/78 mmHg and SpO₂ 94% on air. Auscultation of the chest revealed occasional crackles. Blood investigations were unremarkable except for a low serum sodium (125 mmol/l). ECG revealed narrow complex tachycardia at rate of 183 /min (Figure 1). He had a past medical history of hypertension, paroxysmal atrial fibrillation, asthma, bronchiectasis, hyperthyroidism and rheumatoid arthritis. His regular medications included flecainide, salmeterol, frusemide, carbimazole, tramadol, paracetamol and aspirin. He was referred to a cardiologist after vagal manoeuvres failed to control the heart rate. The cardiology team started him on digoxin, and switched flecainide to disopyramide. He was scheduled for DC cardioversion 48 hours after admission when pharmacological therapy failed to control the rate and symptoms.

FIGURE 1: Pre remifentanil showing SVT.

According to U.K. Resuscitation Council guidelines, the management of stable and regular narrow complex tachycardia involves use of vagal manoeuvres, adenosine and beta blockers. Our patient did not respond to carotid sinus massage and asthma precluded the use of adenosine and beta blockers. Although digoxin has prominent vagotonic effects, it is not very effective in decreasing the heart rate in the presence of enhanced sympathetic drive. Disopyramide is a Class 1a antiarrhythmic and acts by blocking the cardiac sodium channels. Its role in the presence of hyponatraemia is questionable.

FIGURE 2: Post remifentanil showing restoration of normal sinus rhythm.
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