Tension Pneumothorax during Apnea Testing for the Determination of Brain Death

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APNEA testing is performed routinely as an essential component of the determination of brain death. Hypoxemia, bradycardia, and hypotension are the most common and if infrequent complications of apnea testing. Preoxygenation with 100% oxygen and insufflation of oxygen into the endotracheal tube are recommended to prevent hypoxemia. This simple procedure is assumed to be relatively harmless, but it can carry major risks if performed without paying attention to certain "technical" details. We describe two cases of tension pneumothorax occurring during apnea testing and highlight the pathophysiology leading to it and the precautionary measures necessary to prevent such a catastrophic event.

Case Reports

**Case 1**

A 55-year-old hypertensive man had massive intracerebral bleeding. Three days later, examination for the determination of brain death was performed. Cardiovascular and pulmonary functions were stable before apnea testing. After preoxygenation with 100% oxygen, a 16-French suction catheter was inserted into the endotracheal tube. The catheter was advanced until it hit some resistance, which was interpreted as the carina. An oxygen flow of approximately 15 l/min was insufflated through the catheter. Two minutes later, oxygen saturation and blood pressure decreased abruptly and profound bradycardia developed. The patient was connected immediately to the ventilator but continued to deteriorate. He did not respond to intravenously administered epinephrine and asystolic cardiac arrest developed. Cardiopulmonary resuscitation was initiated. Auscultation revealed decreased air entry to the right lung. Needle thoracostomy resulted in audible escape of air and a chest tube was inserted rapidly. Despite the effective evacuation of the pneumothorax, the heart could not be restarted and "cardiac" death was determined.

**Case 2**

A 14-year-old girl sustained a severe head injury in a car accident. On the second postadmission day, she was suspected to be brain dead, and an examination for the determination of brain death was performed. During apnea testing the patient was disconnected from the ventilator and an oxygen supply tubing was placed at the connector of the endotracheal tube. Oxygen insufflation at 10 l/min was started. Approximately 1 min later, blood pressure decreased rapidly to 80/40 mmHg, pulse rate decreased to 40 beats/min, and oxygen saturation decreased to 60%. The chest looked hyperinflated. When the oxygen catheter was removed, the chest deflated and massive bloody froth welled up in the endotracheal tube. When reconnected to the ventilator the chest hardly expanded and the patient could be ventilated only by using a bag-valve. Breath sounds were diminished bilaterally and subcutaneous crepitations were palpated over the chest. Chest radiography showed bilateral massive pneumothorax. Chest tubes were inserted, a large amount of air was evacuated from both hemithoraces, and the patient's condition stabilized. An hour later, brain death was confirmed. The patient's family consented to organ donation, but the lungs and heart were damaged severely and were not harvested for transplantation.

Discussion

Hypoxemia, bradycardia, and hypotension are common complications of apnea testing in patients suspected of being brain dead. However, the major issue in the literature regarding apnea testing is the minimal level of arterial carbon dioxide tension (PaCO₂) necessary to elicit spontaneous breathing in patients with a still-active respiratory center. The issue of ensuring oxygenation is often mentioned, but the precise technique of oxygen insufflation—and especially its potential complications—have not been described sufficiently. Several key publications merely mention the flow of oxygen to be insufflated, whereas others avoid even this very basic instruction. Even the more detailed instructions are limited to a brief statement, such as "hypoxia during
disconnection should be prevented by delivering oxygen at 6 litres/min through a catheter into the trachea. The importance of each of these succinctly described "technical" details and the potential hazards of deviation are not highlighted.

Tension pneumothorax as a complication of apnea testing was never reported in the literature. Marks and Zisfein mention a case of "subcutaneous emphysema and thoracic inflation" in their series of apnea tests using apneic oxygenation. Another case occurred when a 7-French suction catheter was placed into a 3.5-mm endotracheal tube and is mentioned in passing in a letter to the editor. The absence of such reports is quite surprising because apnea testing is performed relatively frequently and despite the mentioned lack of emphasis regarding its proper performance. We posed a question regarding the occurrence of pneumothorax during apnea testing in the "PICU List" in electronic mail and were informed about three cases encountered by subscribers of this list.

The pathophysiologic mechanism leading to pneumothorax in the patients we described is most probably insufflation of a high flow of oxygen into a segment of the lung (case 1) or into both lungs (case 2), through a cannula wedged in a bronchus or in the trachea, thus obstructing air escape and leading to massive air trapping. This was thought to be the mechanism of the subcutaneous emphysema noted by Marks and Zisfein.

The two cases of tension pneumothorax we described had serious consequences. One case caused cardiac arrest (case 1), from which the heart could not be restarted and one resulted in loss of heart and lungs for transplantation (case 2).

Several techniques may be a safer alternative to oxygen insufflation through an endotracheal catheter. Oxygen delivery through a T-piece maintained high arterial oxygen tension (Pao2) during disconnection from the ventilator, and it eliminates even the remote chance of tension pneumothorax. However, others have argued that endotracheal cannula is safer than a T-piece because it better provides adequate oxygenation. We believe that a study comparing the effectiveness of oxygenation using either a T-piece or an intratracheal cannula may be warranted. An alternative, possibly safer method for apnea testing was described by Guttmann et al. It consists of preoxygenation followed by an abrupt decrease in the ventilator's delivered minute-ventilation to 1 l/min (in the adult) until arterial Pco2 rises to the level desired for apnea testing. Only then is the patient disconnected from the ventilator for a relatively brief period and observed for persistent apnea.

When oxygen insufflation through an endotracheal cannula is used, certain details should be followed strictly. First, the external diameter of the endotracheal cannula should be markedly smaller than the inner diameter of the endotracheal tube. This is especially critical in the pediatric-sized endotracheal tubes. Second, the tip of the endotracheal catheter should never be introduced beyond the tip of the endotracheal tube and should never wedge against any structure. If the catheter is thought to be "stuck", it should be withdrawn slightly. Finally, the oxygen flow should not exceed 6 l/min in adults and even less in small children. Oxygen flows of 6 l/min resulted in similar arterial Pco2 values as flows of 15 l/min. No data regarding appropriate flows in different-sized children are available. It should be remembered that the insufflated oxygen is aimed at supplanting the oxygen extracted from the lungs (oxygen consumption)—approximately 250 ml/min in the adult or 5-5 ml/kg/min in the child.

We recommend that all guidelines for the determination of brain death should include detailed instructions regarding the safe performance of apnea testing to ensure adequate oxygenation and the prevention of inadvertent pneumothorax.

References

2. Diagnosis of brain death. Lancet 1976; II:1069–70

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