Dead or dying? Pulseless electrical activity during trauma resuscitation

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Editor—We describe a unique case of a 67-year-old male with high-energy thoracic trauma who developed pulseless electrical activity (PEA) after tracheal intubation.

At the scene, the patient was conscious and immobilized on a vacuum mattress. He received midazolam, ketamine, phenylephrine, tranexamic acid, and ventilator support by mask for transportation to hospital. On the way, Glasgow Coma Scale (GCS) dropped to 9 (M5, E1, V3) with unresponsive pupils. By medical history, the patient was taking antiplatelet therapy. On the way, Glasgow Coma Scale (GCS) dropped to 9 (M5, E1, V3) with unresponsive pupils. By medical history, the patient was taking antiplatelet therapy.

When admitted to the resuscitation room, in-line rapid sequence intubation was performed because of rapidly increasing respiratory distress. After detection of a normal capnographic curve, positive-pressure ventilation was initiated, and a left chest tube was inserted for flail chest. Blood pressure dropped to 54/40 mm Hg with tachycardia. Despite immediate catecholamine and volume administration, PEA developed. Immediate manual ventilation was present bilaterally, and volume administration, PEA developed. Immediate manual ventilation was present bilaterally, with low end-expiratory CO₂ measurable with sufficient chest compressions. As there was an unclear cause for the PEA (exclusion of 5Hs, 5Ts), including hypothermia, hydrogen ion (acidosis), hypovolemia, hypo- and hyperkalaemia and hypoxia as well as cardiac tamponade, tension pneumothorax, coronary thrombosis and thrombosis as pulmonary embolism, toxin, a second chest tube was placed on the right to exclude missed injuries. Correct placement of both chest tubes was confirmed, and intraperitoneal and thoracic bleeding was excluded sonographically. Venous blood gas analysis showed a pH of 6.96, partial pressure of CO₂ of 11.9 kPa, haemoglobin 105 g litre⁻¹, haematocrit 0.32, and normal electrolytes. Core temperature was 35.6°C. Table I medication and medical course.

After 30 min of resuscitation using the Autopulse (AutoPulse, Sunnyvale, California), the capnographic curve deteriorated, ECG showed increasing abnormalities, and pupils were dilated with irregularities to the pupillary margins. After interdisciplinary re-evaluation, resuscitation was stopped. Four minutes later, spontaneous circulation returned, with slight thoracic movement and a palpable spontaneous pulse. Whole-body computed tomography scan showed bilateral costal fractures, contained intima rupture of the ascending aorta (Vancouver type 3), left scapular fracture with severe haematoma, and left humeral fracture. Haemoglobin decreased to 54 g litre⁻¹, and a massive transfusion protocol was initiated. During examination, onset of ventricular tachycardia with spontaneous conversion was noted.

The patient was transferred to the intensive care unit, inotropic drugs were rapidly reduced, and the humeral fracture was operatively stabilized. After 18 days, the patient was transferred to the surgical ward. Eight weeks after admission, the patient was discharged to a rehabilitation clinic. Five months after the accident according to his wife, with only seldom short memory problems.

In this instance of the Lazarus phenomenon, we hypothesize that right-heart failure caused by pulmonary hypertension attributable to hypovolaemia and hypercapnia, positive-airway ventilation, hyperinflation, and cardiopulmonary resuscitation (Autopulse) led to relative hypovolaemia. Moreover, owing to positive-pressure ventilation and end-expiratory pressure ventilation, intrathoracic pressure was increased, leading to an increase in right ventricular afterload. The combination of right-heart failure and relative hypovolaemia with little venous return led to PEA.

Reanimation in this trauma patient after intubation induced an interesting and possibly deleterious pathophysiological perturbation (relative hypovolaemia, hypoxia, and hypercapnia in combination with positive airway ventilation and Autopulse cardiopulmonary resuscitation). This is a rare but treatable cause of PEA. In retrospect, resuscitation and positive-pressure ventilation should have been stopped earlier. Differentiation between dead and dying can be subtle.

Declaration of interest
None declared.

References

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