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Spontaneous Recovery after Discontinuation of Intraoperative Cardiopulmonary Resuscitation: Case Report

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THIS case report describes the spontaneous recovery of a woman after unsuccessful cardiopulmonary resuscitation (CPR) in the intraoperative setting. Spontaneous recovery after discontinuation of CPR has been reported previously.¹⁻³ However, in contrast to previously published reports, we observed hemodynamic and electrocardiography (ECG) changes throughout the course of resuscitation, which, along with data from postoperative studies, provided insight regarding the underlying pathophysiologic events.

Case Report

A 67-yr-old woman with an enlarging aneurysm of the thoracoabdominal aorta was scheduled for endovascular implantation of a stent-graft prosthesis. Preoperative computed tomography (CT) showed an aneurysm, which extended from the distal aortic arch to the diaphragm, with a maximal diameter of 9.5 cm. Medical history was significant for hypertension and stable angina pectoris. Preoperative ECG showed sinus rhythm with a heart rate of 87 beats/min, a left anterior hemiblock, and no significant ST-T segment changes. Chest roentgenogram was negative.

Anesthesia was induced with etomidate and remifentanyl; cisatracurium was used for muscle relaxation. A 37-French double-lumen endotracheal tube was placed using fiberoptic guidance. Anesthesia was maintained using intravenous remifentanyl and sevoflurane in a 35% oxygen and nitrous oxide mixture. Monitoring included a 5-lead ECG, invasive and noninvasive blood pressure measurements, end-tidal CO₂, pulse oximetry, central venous pressure, airway pressure, flow and volume measurement, and neuromuscular function.

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The external femoral artery was cannulated, and guide wires and catheters for the endovascular insertion of the stent-graft prosthesis were placed using fluoroscopic visualization, after which an aortogram of the thoracoabdominal aneurysm was obtained. The patient remained stable during this period; systolic blood pressures ranged from 110 to 160 mmHg.

At 12:10 AM, shortly after guide wire and catheter placement, tachycardia followed by ST-segment elevation in V₅ and a rapid decrease in blood pressure were noted. Anesthetic agents were discontinued and the inspired oxygen was increased to 100%. Blood pressure was refractory to epinephrine, and heart rhythm changed to ventricular fibrillation. At 12:16 AM, the patient was defibrillated with 200, 300 and 360 J without effect. Chest compressions were initiated, and ventilation was maintained with a fractional inspired oxygen tension (F_IO₂) of 100%. Epinephrine, 1 mg, was administered in a central venous line, and the patient was again defibrillated with 300, 360 and 360 J. Patient cardiac rhythm changed to a regular, wide QRS-complex rhythm with a blood pressure of 160/105 mmHg; however, within 1 min, cardiac rhythm deteriorated to nonregular electrical activity, which was noted in several leads. Blood pressure was absent. Chest compressions were resumed while an attempt was made to rule out causes for the electromechanical dissociation, such as pneumothorax or hypovolemia. We then administered epinephrine, 5 mg, through a central line without effect. At 12:21 AM, arterial blood gas analysis showed a pH of 7.20, an oxygen tension (P_{O₂}) of 159 mmHg, a carbon dioxide tension (P_{CO₂}) of 41 mmHg, an arterial oxygen saturation (Sa_{O₂}) of 97%, a standard bicarbonate concentration of 15.5 mM, and a base excess of -11.0. We administered sodium bicarbonate, 100 mEq, and shortly afterward administered epinephrine, 10 mg. The ECG tracings continued to reveal asystole in all leads. At 12:39 AM, another series of attempted defibrillations with 360 J were performed. This had no apparent effect on cardiocirculatory function and chest compressions were resumed. At 12:51 AM, an arterial blood gas analysis showed a pH of 7.33, a P_{O₂} of 84 mmHg, a P_{CO₂} of 42 mmHg, an Sa_{O₂} of 96%, a standard bicarbonate concentration of 22.4 mM, and a base excess of -2.9. At 12:59 AM, the patient had no spontaneous circulation despite high doses of epinephrine during an extended period of time; therefore, resuscitative efforts were discontinued.

At 1:04 AM, a regular rhythm was noted on the ECG, which had not been removed. In addition, pulse was palpable and spontaneous blood pressure was 90/45 mmHg; therefore, therapy was resumed. The surgical wounds were closed, and the patient underwent emergency coronary angiography, which showed a more than 90% proximal stenosis of the left anterior descending coronary artery. The left anterior descending coronary artery was dilated and a stent was placed. During transportation to the cardiac catheterization laboratory and during the procedure, the patient continued to require an epinephrine

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infusion ranging from 50 to 300 mg/min to sustain an acceptable blood pressure; she was mechanically ventilated with minimal sedation. After the coronary angiography procedure, the patient was transported to the surgical intensive care unit, with decreasing catecholamine requirements. She showed progressive improvement in cardiocirculatory function and was extubated the next day. She was initially disoriented and somewhat combative, however, on postoperative day 3, she was alert, oriented, and without neurologic deficit.

On postoperative day 7, the patient had a massive pulmonary embolism and was administered heparin. On postoperative day 9, she had a profuse intrathoracic hemorrhage and died. Autopsy revealed a massive pulmonary embolism, evidence of a fresh intrathoracic hemorrhage, and a subacute myocardial infarction involving the posterolateral wall of the left ventricle.

Discussion

This case illustrates the spontaneous recovery of circulatory function after unsuccessful CPR in the intraoperative setting. The sequence of events allowed us to clearly demonstrate acute myocardial ischemia as the cause of the intraoperative cardiopulmonary arrest.

The cause of spontaneous return of cardiovascular function is less clear. We postulate that the intraoperative myocardial ischemia was caused by acute and complete obstruction of the stenotic left coronary artery, possibly by an endovascular plaque released at the time of guide wire manipulation within the thoracic aorta. The spontaneous return of cardiac function after CPR was probably the result of cardiac reperfusion. Complete obstruction of the left anterior descending coronary artery most likely had been reversed to some extent, but this theory is speculative.

This case report poses two questions. Was CPR discontinued too soon? An arterial blood gas obtained 20 min into resuscitation showed a pH of 7.20 with a calculated base excess of -11.0 , a P_{O_2} of 159 mmHg with the patient breathing 100% oxygen, and a P_{CO_2} of 41 mmHg.

A second blood gas, obtained after buffering with 100 mEq bicarbonate, revealed a pH of 7.33, a P_{O_2} of 84 mmHg, a P_{CO_2} of 42 mmHg, and a base excess of -2.9 . These blood gas values indicate that the patient's circulatory function was reasonably well maintained. The decision to discontinue resuscitative efforts was based on the refractoriness to extensive resuscitative efforts in an elderly patient with a major underlying pathology. Continuation of CPR at this point was considered futile. We were convinced that the patient had experienced a massive myocardial infarction, fulminant pulmonary thromboembolism, or an intrathoracic hemorrhage.

The second question is, Should CPR in the anesthetized patient with adequate ventilation and extensive monitoring in place be continued until the ineffectiveness of CPR has been clearly shown by laboratory values (*i.e.*, decreasing pH with adequate ventilation)? In one report of three patients who recovered after CPR was discontinued, the authors suggested that the patient should be carefully monitored for an additional 10 min after discontinuation of resuscitation.² Although this recommendation would help to clearly establish the inability of the patient's cardiovascular system to maintain vital organ perfusion in the absence of therapeutic interventions, it appears to be more ethical to establish this end-point before discontinuation of life support assistance.

References

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