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Adrenalin significantly increases intracranial pressure in hypovolemic cardiac arrest model in male piglets

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Introduction

Resuscitation from hemorrhagic shock and subsequent cardiac arrest (CA) is a major clinical challenge in the care of trauma patients [1]. We hypothesized that adrenalin given during cardiopulmonary resuscitation (CPR) will improve cerebral perfusion response in hypovolemic cardiac arrest.

Methods

Twenty-one anesthetized male piglet (with a weight 24.3 \pm 1.3 kg) were bled 35% via femoral artery to a mean arterial blood pressure of 25 mm Hg during 15 min. Later piglets were subjected to 8 mins untreated ventricular fibrillation followed by 15 min open-chest CPR. At 9 min of CA piglets received amiodarone 1.0 mg/kg and hypertonic-hyperoncotic solution 4 ml/kg infusion for 20 minutes. At the same time vasopressin 0.4 U/kg was given intravenously to vasopressin group (VAS, n = 9), while adrenalin group received adrenalin 20 μ g/kg (ADR, n = 12). Internal defibrillation was attempted from 11 min of CA to achieve restoration of spontaneous circulation (ROSC). Experiment was terminated at 3 hours after resuscitation.

Results

No significant differences were observed in resuscitability/ survival between the groups. There were no significant differences in cerebral cortical blood flow or protein S-100 β levels after ROSC between the groups. Intracranial pressure was significantly higher in ADR group compared with

VAS group in the post resuscitation phase. Jugular bulb pH and base excess was higher in the ADR group 15 (p = 0.0003 and p = 0.0002) and 30 min after ROSC in comparison with the VAS group (p = 0.02 and p = 0.001). Besides, oxygen extraction in jugular bulb samples was greater in the VAS group compared with the ADR group at the same time points (p = 0.03 and p = 0.003, respectively).

Conclusion

Resuscitation with adrenalin significantly increased intracranial pressure though improvement of cerebral perfusion was reflected by higher jugular bulb pH, base excess and lower oxygen extraction ratio with no significant changes in cerebral cortical blood flow.

References

1. Lockey D, Crewdson K, Davies G: Ann Emerg Med 2006, 48(3):240-4.