Epinephrine during CPR significantly increases organ perfusion in a rat VF cardiac arrest model

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INTRODUCTION

Epinephrine is an established vasopressor and the primary drug administrated during cardiopulmonary resuscitation (CPR). This study evaluates the influence of epinephrine on coronary perfusion pressure (CPP), mean arterial pressure (MAP) and end-tidal CO₂ (ETCO₂) in our rat VF CPR model.

METHODS

Adult male Sprague-Dawley rats (400g) were randomized to CPR with ($n=15$) and without epinephrine ($n=10$). After 6 minutes of untreated cardiac arrest (CA) study animals were resuscitated with mechanical chest compressions, ventilations and defibrillations. In the epinephrine group, epinephrine 10 µg/kg were administered every two minutes.

Mean arterial pressure (MAP), central venous pressure (CVP) and ETCO₂ were continuously monitored and calculated for the first two minutes of CPR. Mean CPP was calculated as difference between MAP and CVP.

RESULTS

The mean CPP was significantly higher during CPR in animals resuscitated with epinephrine than without epinephrine. The epinephrine group showed also significantly higher MAP values. The mean ETCO₂ was higher in the no epinephrine group.

[mmHg]	Epinephrine	No Epinephrine
CPP	38 ± 8	23 ± 4
MAP	45 ± 8	25 ± 6
etCO₂	32 ± 6	42 ± 8

CONCLUSION

As seen in given literature epinephrine increases organ perfusion in our VF CA model. Interestingly, ETCO₂ reacted inversely and was lower in the group without epinephrine.