Femoral vessel occlusion increases cardiac and cerebral perfusion in a pilot cardiac arrest porcine model

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Introduction: Closed chest compressions manually circulate blood to the vital organs during cardiac arrest; however, cardiac arrest cases remain 90% fatal. The use of adjuncts to prioritize vital organ perfusion, such as the Resuscitative endovascular balloon of the aorta (REBOA), have shown clinical promise, but lack translatability to community cardiac arrest cases. The purpose of this study was to evaluate if external femoral vessel occlusion can increase cardiac and cerebral perfusion in an established porcine model of cardiac arrest.

Methods: Eight adult domestic pigs were enrolled in this pilot study. The animals were anesthetized and ventilated while arterial monitoring lines were placed and baseline hemodynamic measures were collected. External femoral vessel occlusion (FVO) was compared with surgical ligation during native heart function. Next, cardiac arrest was electrically-induced, fluorescent microspheres were injected into circulation, and tissue perfusion was measured following CPR or CPR with FVO. At the conclusion of the experiment, heart, brain, and skeletal muscle were collected for analysis.

Results: During native heart function, FVO demonstrated a statistically significant increase in carotid systolic blood pressure (11mmHg, n=8, $p<0.05$), representing a 16% increase in mean arterial pressure. External FVO was non-inferior in its effects to surgical ligation. During cardiac arrest, CPR with FVO raised carotid systolic blood pressure by a mean of 67.7mmHg, a 51.2% increase over CPR alone (35.2mmHg) (CPR: n=2, CPR + FVO: n=2). CPR with FVO demonstrated a 10-fold increase in coronary blood flow by spectrofluorimetric analysis (CPR: 0.012mL/min/g, n=1, CPR + FVO: 0.122mL/min/g, n=2) and a 50-fold increase in cerebral blood flow (CPR: 0.001mL/min/g, n=1, CPR + FVO: 0.071mL/min/g, n=2) **(Fig. 1).** Sartorius muscle demonstrated no evidence of muscle damage, inflammation, or edema on histologic examination and CPR with FVO tissue was indifferentiable from control animal tissue.

Conclusion: CPR with FVO for thirty minutes during cardiac arrest increased mean arterial pressure and increased cardiac and cerebral perfusion over CPR alone. These findings suggest FVO may be an underutilized adjunctive strategy during resuscitation and may present a therapeutic opportunity to enhance perfusion during cardiac arrest to reduce morbidity and mortality.

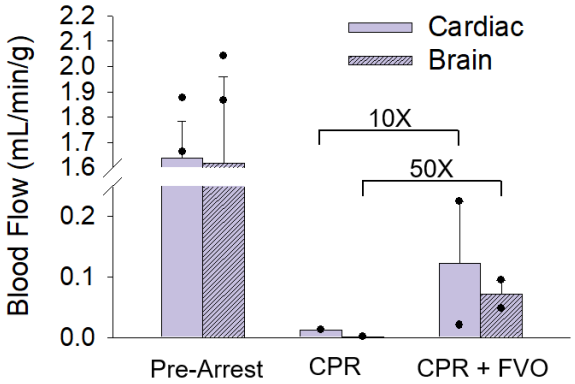

Fig. 1) Cardiac and cerebral blood flow determined by fluorescent microspheres. There is a 10-fold increase in blood flow to the heart and a 50-fold increase in blood flow to the brain with FVO (Pre-Arrest: n=3, CPR: n=1, CPR+FVO: n=2).