

Circulation 2006;114(18):II-1195**[35] Effects of Reducing Ventilation Frequency on Brain Perfusion and Tissue Oxygen Tension in a Porcine Model of Cardiac Arrest.**

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Background: Hyperventilation during cardiopulmonary resuscitation (CPR) is harmful. We tested the hypothesis that a) 2 breaths/min (bpm) would result in higher cerebral perfusion pressures and brain tissue O₂ tension (BTO₂) vs 10 bpm and b) use of an impedance threshold device (ITD), known to increase circulation, would further enhance cerebral perfusion and BTO₂, especially with 2 bpm.

Methods and Results: Female pigs (30.4±1.3 Kg) anesthetized with propofol were subjected to 6 min of untreated ventricular fibrillation (VF) followed by 5 min of CPR (100 compressions/min, depth of 25% antero-postero diameter), and ventilated with 10 (n=11) or 2 (n=11) bpm (1.0 FiO₂, tidal volume of 12ml/kg). BTO₂ was measured with a Licox™ probe in the parietal lobe. An ITD was then used during 5 additional min of CPR. During CPR, calculated coronary and cerebral perfusion pressures (mmHg) with 10 vs 2 bpm were: 14.9±1.4 vs 13.0±1.1 (p=0.12) and 7.5±1.6 vs 6.8±1.6 (p=0.18), respectively. Carotid artery blood flow (ml/min), prospectively designated the primary endpoint, was 56.8±5.6 vs 39.2±3.2 (p<0.01) in the 10 vs 2 bpm groups and BTO₂, measured with a Licox™ probe (mmHg), was 3.9±0.6 vs 0.5±0.1, with 10 vs 2 bpm (p=0.04). After 5 min of CPR there were no significant differences in arterial pH, pO₂, and pCO₂ between groups. With CPR + the ITD, carotid blood flow and BTO₂ in the 10 vs 2 bpm groups were: 97.7 +/- 8.2 vs 38.3±3.1 (p<0.01) and 6.1±0.8 vs 0.7±0.1, (p<0.01), respectively.

Conclusions: Contrary to the initial hypothesis, during the first 5 minutes of CPR after cardiac arrest ventilation rates of 2 bpm resulted in significantly *lower* carotid blood flow and O₂ tension in brain tissue compared to 10 bpm. Subsequent addition of an ITD significantly enhanced carotid flow and brain oxygen tension values, especially in the 10 bpm group.