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TITLE: REWARMING INJURY IMPAIRS CEREBRAL BLOOD FLOW AFTER HYPOTHERMIC CPB

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Introduction: Cerebral blood flow (CBF) in dogs decreases during prolonged hypothermic cardiopulmonary bypass (CPB), but fails to improve after rewarming (1). Whether this reduction in CBF is related to the cooling-rewarming process or to prolonged CPB per se is not known.
Methods: Twenty-one anesthetized, open-chest dogs were placed on total CPB. CBF was measured by radioactive microspheres, and cerebral O_2 delivery was calculated as arterial O_2 content x CBF. Hypothermic animals (n=11) underwent 150 min hypothermic (28°C) CPB followed by rewarming, whereas normothermic animals (n=10) underwent a similar duration of CPB at 37°C. Other parameters, such as pump flow (80ml/kg/min), mean aortic blood pressure (78 ± 2 mm Hg), and hemoglobin (8 ± 1 mg%) remained constant. Measurements were taken at baseline, and after 90, 150, 210, and 270 min of CPB. In the hypothermic animals, the 270 min measurement represented rewarming.
Results: In normothermic animals, CBF remained relatively constant, with only a 9% decrease overall (Figure 1). In contrast, CBF decreased significantly with cooling in hypothermic animals (Figure 1). However, CBF failed to increase with rewarming. After rewarming, CBF remained 40% lower in the hypothermic group than the normothermic group ($p<0.05$). Consequently, cerebral O_2 delivery was 33% lower in hypothermic than in normothermic animals ($p=0.059$).
Discussion: These data indicate that a significant reduction in CBF occurs during prolonged CPB attributable to the cooling-rewarming process. After rewarming, CBF failed to increase, causing an impairment in cerebral O_2 delivery. The etiology and significance of this post-rewarming reduction in CBF (i.e., rewarming injury) are not known but may predispose to postoperative neurological dysfunction.
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References:
1. Anesthesiology 71: A554, 1989

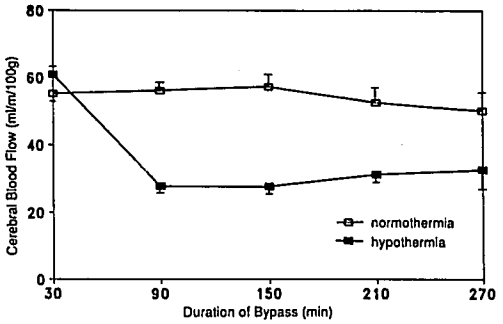

Figure 1. CBF during normothermic and hypothermic CPB. In hypothermic animals, rewarming occurred after 210 min.

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TITLE: HYPERTONIC SALINE DOES NOT REDUCE INTRACRANIAL PRESSURE OR IMPROVE CEREBRAL BLOOD FLOW AFTER EXPERIMENTAL HEAD INJURY AND HEMORRHAGE IN CATS

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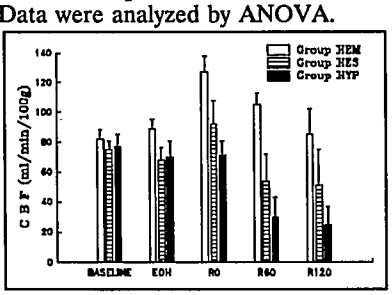
Introduction: Mild hemorrhagic hypotension following experimental traumatic brain injury (TBI) results in significant reductions in cerebral oxygen delivery.[1] Experimental evidence suggests that hypertonic saline may improve blood pressure and cerebral O_2 delivery (CDO_2) without increasing intracranial pressure.[2] Therefore, we compared the effects of resuscitation using 3.0% saline with resuscitation using 10% hydroxyethyl starch after TBI and hemorrhage in cats.
Methods: Cats were anesthetized with ketamine (25mg/kg), intubated, ventilated with 1.6% isoflurane in N_2O:O_2 (70:30) and prepared for fluid percussion TBI[1] and intracranial pressure (ICP) measurement. Isoflurane concentration was decreased to 0.8% in N_2O and cats were randomized to receive hemorrhage and resuscitation with 10% hetastarch (sham-injury, Group HEM, n=8) or trauma (2.5 atm) plus hemorrhage and resuscitation with 10% hetastarch (Group HET, n=8) or 3.0% saline (Group HYP, n=8). Cats were injured or sham-injured, hemorrhaged to 70% of pre-injury blood volume and resuscitated with an equal volume of hetastarch or saline. CBF was determined using microspheres pre-injury (BL), after hemorrhage (EOH), and 0, 60 and 120 min after resuscitation (R0, R60, R120). EEG activity was recorded and scored visually used a scale modified from Prior, et al.[3] CDO_2 was calculated as CBF x arterial O_2 content. Data were analyzed by ANOVA.
Results: Group HYP exhibited greater increases in ICP at R60 and R120 than in either Group HEM or Group HES. In the uninjured Group HEM, CBF remained above baseline at R0, R60 and R120. In contrast, CBF decreased in Groups HES and HYP at R60 and R120 (Figure). CBF, CDO_2 or EEG score were reduced at R60 and R120 in Groups HES and HYP compared to Group HEM, but were not significantly different from each at any time point.



Discussion: This data indicate that hypertonic saline is no better than hetastarch for fluid resuscitation after TBI and hemorrhage and, if the ICP increases observed here were confirmed in humans, hypertonic saline may be inappropriate for acute resuscitation of head-injured patients.
References:
1. DeWitt, et al, J Neurosurg Anesth 1:127-128, 1989
2. Prough, et al, J Neurosurg 64:627, 1986
3. Prior, et al, Br J Anaesth 50:993-1001, 1978