

Protecting the Brain During Severe Hemorrhagic Shock

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Some patients who have survived severe hemorrhagic shock (HS) without brain trauma seem to have some persistent neurobehavioral deficits. This report, relevant for traumatology, concerns unpublished data in monkeys and rats on studies which advance the hypothesis that cerebral vasodilation protects the brain during HS as long as mean arterial blood pressure (MAP) is maintained at or above 40 mmHg.

Monkey studies: Under normothermia throughout, lightly anesthetized cynomolgus monkeys were insulted with hemorrhage by volume-controlled blood withdrawal followed by pressure controlled maintenance of HS at MAP 30 mmHg over 2 hours (h). All-out fluid resuscitation and controlled ventilation to 20 h were followed by intensive care to 72 h with evaluation of overall performance categories (OPC 1–5) and neurologic deficit scores (NDS 0–100%). At 72 h after perfusion fixation, brain histologic damage (HD) was scored in 20 brain regions. All 72 h survivors had OPC 1 (normal), NDS 0–10% (normal), and histologically “clean” brains, with no ischemic (pink, shrunken, pyknotic) neurons.

Rat studies: Under normothermia throughout, 10 lightly anesthetized rats (in comparison with 10 sham experiments and 5 normal controls) were insulted with hemorrhage of 2 ml/100g over 10 minutes (min.), followed by further withdrawal or reinfusion of blood to maintain MAP at 40 mmHg to 60 min. without heparinization. Then, all-out fluid resuscitation to normotension and Hct of 30% were followed by MAP controlled to 1 h and observation to 10 days, for the determination of OPC, NDS, and HD scoring; and special motor and cognitive function tests. The 10 HS rats with 10 day survival had, from 72 h on, OPC 1 (normal), NDS 0–10% (normal), and under light microscopy, histologically “clean” brains, when 6 coronal sections were scored and hippocampal neurons counted (compared with shams). Motor and cognitive water-maze learning tests showed no difference between HS and sham rats. Acute (<4 h) abnormal motor function in beam balance and beam walking tests, was attributed to femoral artery ligation. An additional 10 rats were subjected to MAP 30 mmHg for 45 min. MAP of 30 mmHg frequently led to cardiac arrest before 60 min. Brain outcome results after MAP of 30 mmHg are in progress and also will be reported.

Conclusions: We conclude that clinical guidelines for hypotensive fluid resuscitation during severe HS to maintain MAP at about 40 mmHg seem reasonable to prevent even subtle brain damage.

Key Words: cerebral damage; cognitive function; hemorrhagic shock; rat outcome model

Resuscitative Moderate Hypothermia for Severe Traumatic Brain Injury

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This “mini-review” is for teaching and traumatology guidelines. Results of laboratory and clinical studies support the use of resuscitative (post-insult) hypothermia (Hth) in the prehospital and intra-hospital life support of patients in coma due to severe traumatic brain injury (TBI). Hth would be in addition to the well-characterized recent “guidelines for the management of severe head injury” promulgated by the Brain Trauma Foundation, the American Association of Neurologic Surgeons, and the Joint Section on Neurotrauma and Critical Care.

In a new *dog model* of temporary epidural balloon inflation (simulated epidural hematoma and drainage with intensive care to 96 hours (h) of injury, moderate Hth (31° C) for 5 hours, but not subsequent mild Hth (35° C) prevented the delayed rise in intracranial pressure (ICP), but could not prevent herniation during rewarming (Pomeranz et al). Moderate Hth for 48 h post-TBI also controlled ICP during Hth, but some dogs still herniated during rewarming. Moderate Hth of 48 h was associated with a hemorrhagic diathesis and pulmonary infiltration (Ebmeyer et al). In rat models of cortical contusion, moderate Hth immediately after TBI reduced inflammation and functional deficit and increased survival.

In *patients*, historical anecdotal experiences in the 1960s (by Rosomoff and Safar) included beneficial effects of mild to moderate “titrated” Hth on ICP and recovery of consciousness in cases with prolonged post-TBI coma. In a recent, randomized, clinical trial. (Marion, et al, *N Engl J Med*) patients with Glasgow Coma Scale (GCS) score of 5–7 on arrival, benefited from moderate Hth, which was initiated by surface cooling within 6 h and continued to 24 h. An increased proportion of treated patients achieved good cerebral outcome. This Hth protocol did not cause complications. TBI patients who, on admission, had GCS score of 3 or 4, had statistically similar poor outcome as did normothermic controls. Other investigators’ results support the above and also point out the deleterious effect on traumatized brain of even the mildest degree of hyperthermia.

We *recommend* that traumatologists and Disaster Medicine specialists include in prehospital and intra-hospital resuscitation protocols for severe head injury with coma, the control of temperature, shivering, and blood pressure in the field, early induction of moderate Hth, and multifaceted ICP control in the hospital.

Key Words: cerebral resuscitation; hypothermia, intracranial pressure; traumatic brain injury