Title: PROLONGED REDUCTION OF COLLOID ONCOTIC PRESSURE DOES NOT INCREASE BRAIN EDEMA FOLLOWING CRYOGENIC INJURY

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Introduction. The infusion of large amounts of crystalloid solutions (e.g., lactated Ringer's, LR) can reduce colloid oncotic pressure (COP) and lead to peripheral edema. Many clinicians also believe that this can induce cerebral edema, and recommended that crystalloids be limited in neurosurgical patients. In contrast to this, recent studies have shown that an acute reduction in COP does not affect the formation of edema in either normal(1) or injured brain(2). However, these studies were relatively short , and it is possible that delayed edema formation might occur. To answer this question, we examined the influence of a prolonged (8 hours) reduction in COP on cerebral edema formation in rabbits subjected to a cryogenic brain injury.

Methods. 24 NZW rabbits were anesthetized with pentobarbital (40 mg/kg iv), paralyzed, and ventilated to normocarbia with 66% N_2O/O_2. Catheters were placed into femoral artery, abdominal aorta, and right atrium and into the right lateral cerebral ventricle, to permit blood withdrawal and recording of mean arterial, central venous and intracranial pressures respectively (MAP, CVP,ICP). When surgery was complete, brain injury was produced by pouring liquid N_2 into a funnel fixed to the exposed skull overlying the left parietal area (15 sec exposure). 30 min later, the animals were divided into 3 groups. Group 1 (Control, n=8) received only maintenance LR at a rate of 4 $ml \cdot kg^{-1} \cdot h^{-1}$. Group 2 and 3 (n=8 ea.) both underwent a 45 period of isovolemic plasmapheresis. In these groups arterial blood was withdrawn at a rate of 3-4 ml/min. Packed cells were returned to the animal and separated plasma was replaced with one of two fluids: Group 2 (Iso-COP) received 6% hetastarch in LR, while Group 3 (Hypo-COP) animals received only LR. In both cases, the volume of fluid given was adjusted to maintain MAP and CVP constant. MAP, CVP, ICP, EEG, plasma osmolality (Osm) and COP were measured repeatedly for 8 hours after beginning plasmapheresis (8.5 hours post injury), at which time the animals were killed, the brains removed and sectioned, and the water content of brain adjacent to and distant from the lesion was assessed by both microgravimetry (specific gravity [SpGr] by kerosene/bromobenzene gradient) and the wet-dry weight ($\%H_2O$). (See Fig. 1 for sampling scheme.) $\%H_2O$ of muscle and jejunum were also measured to assess peripheral edema formation.

Results. There were no intergroup differences in MAP, CVP, ICP, PaO_2, $PaCO_2$, pH, or Osm at any time during the experiment (although ICP rose in all groups). There were no COP alterations in Group 1 and 2, but as intended, LR infusion in Group 3 resulted in a stable reduction in COP from 20.6 mmHg to 10.0 mmHg. In all groups, SpGr of brain tissue was lower in the vicinity of the lesion, and hemispheric $\%H_2O$ was greater in the left than right. However, there were no intergroup differences. In contrast, $\%H_2O$ of muscle and jejunum in Group 3 (74.93 ± 0.57,and 82.18 ± 1.45) were significantly greater than in Group 1 (73.12 ±0.74, 77.58±1.85) and Group 2 (73.06 ± 0.75, 76.74±3.40).

Discussion. This study revealed that a prolonged hypo-oncotic state did not increase brain edema following cryogenic brain injury, even though it did lead measurable peripheral edema. These observations have important implications concerning the fluid management of neurological patients, and suggest that the use of iso-osmotic crystalloid solutions have no deterimental effects on the brain.

References. 1) Zornow MH, et al.: Anesthesiology 67:936,1987. 2) Zornow MH, et al.: Anesthesiology 67:A583,1987.

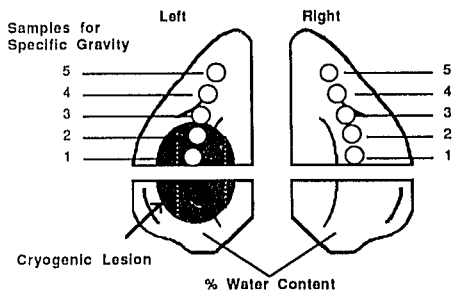

Figure 1. Brain sampling for determination of specific gravity and $\%H_2O$.

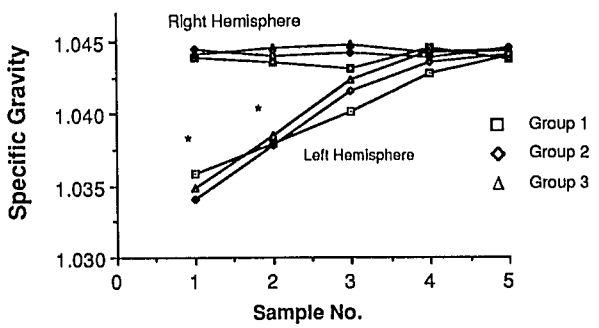

Figure 2. SpGr data. Samples from the vicinity of the lesion (*, Left, No.1,2) were lower in SpGr than other samples. ($P < 0.05$)