Small Volume Resuscitation in Hemorrhagic Shock by Hypertonic/Hyperoncotic Saline-Dextran: Effects on the Central Nervous System

S. Berger¹, L. Schürer², R. Härtl¹, C. Dautermann², R. Murr³, K. Messmer¹, and A. Baethmann¹

Introduction

Secondary ischemia of the brain is common in fatal head injury (Graham et al. 1989). Bouma et al. (1991) observed in patients a close correlation between the impairment of cerebral blood flow during the first hours after trauma and final outcome. Based on the United States traumatic coma databank study, Marmarou et al. (1991) reported that periods of elevated intracranial pressure (ICP) (>20 mmHg) or of a decreased blood pressure (<80 mmHg) were highly predictive for poor outcome from severe head injury. Since 10%–20% of head-injured patients are simultaneously affected by severe peripheral injuries causing hemorrhagic hypotension (Miller et al. 1978), primary care must reestablish and maintain an appropriate cerebral as well as systemic circulation.

Fluid resuscitation by a small volume (e.g., 4 ml/kg as rapid bolus) of hypertonic/hyperoncotic solution, such as 7.5% NaCl in 6% dextran 70 has been shown to effectively restore the cardiovascular function of polytraumatized patients in shock (Holcroft et al. 1987). This concept is currently under clinical investigation. Since up to one third of polytraumatized patients are simultaneously affected by head injury (Gennarelli et al. 1989), this form of shock treatment might also be employed in patients with an acute cerebral lesion. While hypertonic saline solutions have frequently been shown to lower ICP both clinically (Worthley et al. 1988) and experimentally (Prough et al. 1991; Zornow et al. 1990), no information is available concerning possible side effects of the mixture of hypertonic/hyperoncotic solutions on the brain. Adverse side effects exerted by hypertonic/hyperoncotic solutions, however, might evolve in the presence of a disrupted blood–brain barrier and/or during impairment of the cerebral autoregulation as in head injury.

¹ Institute for Surgical Research, Klinikum Grosshadern, Ludwig Maximilians University, Marchioninistr. 15, 81377 Munich 70, Germany
² Department of Neurosurgery, Klinikum Grosshadern, Ludwig Maximilians University, Marchioninistr. 15, 81377 Munich 70, Germany
³ Institute of Anesthesiology, Klinikum Grosshadern, Ludwig Maximilians University, Marchioninistr. 15, 81377 Munich 70, Germany

Cerebral Ischemia and Basic Mechanisms
Ed. by A. Hartmann, F. Yatsu, and W. Kuschinsky
© Springer-Verlag Berlin Heidelberg 1994
In a previous study by our group (Schürer et al. 1992), adverse effects of 7.2% NaCl in 10% dextran 60 (hypertonic/hyperoncotic saline/dextran solution, HHS) were not observed on the cerebral blood flow, cerebral tissue oxygenation, vasomotor control, or the brain water content in shock. In a second experimental series (Härtl et al., in press) we analyzed administration of HHS on the intracranial pressure acutely after a focal cerebral lesion in combination with an intracranial space-occupying process. For the present study, the influence of HHS on intracranial hypertension was studied 24 h after trauma in comparison with mannitol as standard treatment.

Methods

For the first study, six New Zealand rabbits were anesthetized using thio­pental, ventilated, and implanted with arterial and venous catheters. Anesthesia was then maintained by α-chloralose. Mean arterial pressure (MAP), central venous pressure (CVP), blood gases, body temperature, plasma electrolytes, osmolality, and colloid osmotic pressure were monitored. After a circular trephination of the skull above the left hemisphere, an epidural balloon was inserted between the skull and intact dura. A burr hole was made over the right hemisphere for manometric measurement of the ICP. A focal cold lesion of the exposed cerebral cortex was then induced by a metal rod cooled with liquid nitrogen. The skull was subsequently closed using dental cement. The ICP was slowly raised (over 30 min) by inflation of the epidural balloon until 15 mmHg was reached. Infusion of 4 ml/kg of HHS into an ear vein followed within 2 min, resulting in a decrease of the ICP. The intracranial pressure increased spontaneously thereafter due to maintenance of inflation of the epidural balloon. As soon as 15 mmHg was reached again, infusion of HHS was repeated.

For the second study, 12 New Zealand rabbits were anesthetized by i.v. ketamine/xylazine under spontaneous respiration. Upon fixation of the skull in a stereotactic frame, a circular trephination was made over the left hemisphere leaving the dura intact. A metal probe (6 mm in diameter), cooled by liquid nitrogen, was attached to the brain surface for 5 min to induce a focal lesion. An epidural balloon was then inserted directly above the lesion. The skull was closed using dental cement. Twenty-one hours later the animals were reanesthetized with thiopental, ventilated, and implanted with catheters including subdural measurement of the ICP. Anesthesia was maintained by chloralose. Physiological variables were monitored as in the first study. The ICP was raised to 17 mmHg by stepwise inflation of the balloon over 60 min. During a control period of 30 min, the ICP remained at 15–20 mmHg without changes in the balloon volume. Then, either 4 ml/kg HHS (six animals) or 9 ml/kg 20% mannitol (six animals) were infused i.v. within 2 min. The osmotic load administered in both groups was equivalent,