Fluid Resuscitation in Head-injured Patients: Unresolved Issues


In this issue of the Journal, Shackford [1] provides a concise, authoritative review of the essential issues involved in selecting and administering resuscitation fluid in patients with combined hypovolemia and head injury. Such patients pose a particular clinical and investigative challenge because of the complex, interactive effects of head injury, shock, and rapid resuscitation on the three factors that determine cerebral blood flow (CBF), according to the equation

\[
\text{CBF} = \frac{\text{MAP} - \text{ICP}}{\text{CVR}}
\]

where MAP equals mean arterial pressure, ICP equals intracranial pressure, and CVR equals cerebral vascular resistance.

Because most clinicians measure or monitor only MAP and ICP in head-injured patients, much of the management of acute head injury has been directed at control of ICP. Few studies characterize changes in CBF, CVR, and cerebral metabolism following human head injury. Virtually no data are available to describe the changes in those variables in the acute phases of pre-hospital and early in-hospital resuscitation and stabilization. During this potentially critical interval, before ICP monitoring can be established and before neurodiagnostic information is available, clinicians must proceed empirically. Once adequate ventilation and oxygenation are insured, blood pressure and cardiac output must be promptly restored. After insuring adequate systemic oxygen delivery, attention to the adequacy of cerebral oxygen delivery might reduce the extent of subsequent neurological injury if sufficient information were available to guide cerebral resuscitative efforts.

This editorial addresses three issues of particular relevance to fluid resuscitation in head injury. These include enhanced vulnerability of the traumatized brain to secondary hypoxic or ischemic injury, impaired cerebral autoregulation following brain trauma, and impairment of the cerebral vascular response to hemodilution following shock or trauma.

Enhanced Vulnerability to Secondary Hypoxic or Ischemic Injury

Considerable clinical and animal data support the concept that acute traumatic brain injury reduces the tolerance of the brain to reductions in arterial oxygen tension (PaO₂) and cerebral perfusion pressure (MAP – ICP) that would cause no injury to normal brain. Luerssen and colleagues [2] demonstrated a prominent increase in mortality in head-injured children and adults if hypotension accompanied head injury. A systolic blood pressure less than 90 mm Hg on admission to the emergency department has also been associated with 1/13th the likelihood of subsequent favorable outcome (defined as good outcome or moderate disability) as a systolic blood pressure exceeding 90 mm Hg on admission [3].

The increase in mortality and in neurological morbidity associated with the combination of head injury and hypotension may be related to exaggerated depletion of high-energy phosphates. Ishige and colleagues [4] used in vivo phosphorus-31 magnetic resonance spectra to examine the effects of profound hypotension (MAP, 30–40 mm Hg), fluid-percussion brain injury, and the combination of hypotension and fluid-percussion injury in rats. Either insult alone resulted in a small increase in inorganic phosphate, a slight decrease in phosphocreatine, and a moderate decline in brain pH. The combination of head injury and a decrease in MAP to 30 mm Hg, however, resulted in severe depletion of high-energy phosphates, a marked increase in inorganic phosphate, and a precipitous decline in pH. Moderate hypoxemia, insufficiently severe to produce neurological injury, also worsens neurological outcome following fluid-percussion injury in rats [5]. In cats, hypoventilatory hypoxemia enhances high-energy phosphate depletion and brain acidosis following fluid-percussion injury [6].
Fig 1. In the uninjured brain, cerebral autoregulation preserves cerebral blood flow at a virtually constant level over a range of mean arterial pressure extending from 50 to approximately 150 mm Hg (solid line). A combination of clinical and experimental evidence suggest that acute head injury may alter the autoregulatory curve, as illustrated by the dotted line. The lower limit of autoregulation may be shifted upward and the upper limit may be shifted downward, thereby reducing the perfusion pressure range over which cerebral blood flow is adequate to provide metabolic substrate but not so high that cerebral blood volume and intracranial pressure are increased.

Effects of Hemorrhage on the Cerebral Vascular Response to Hemodilution

The effects of acute hemodilution on CBF have been extensively investigated in an effort to improve cerebral microcirculatory flow in ischemic stroke and in vasospasm following subarachnoid hemorrhage. In general, if hemodilution is achieved either by blood volume expansion with asanguineous fluid or by concurrent withdrawal of blood and replacement with asanguineous fluid, the resulting decrease in blood viscosity is associated with a decline in cerebral vascular resistance and an increase in CBF [8–11]. In general, the increase in CBF is less marked than the decrease in hemoglobin concentration; therefore, cerebral oxygen transport (CBF × cerebral oxygen content [CaO2]) may also decrease but to a lesser extent than the decline in hemoglobin concentration [11]. Presumably, the improvement in neurological outcome in stroke and in vasospasm following subarachnoid hemorrhage results from improved microcirculatory distribution of flow.

If an interval of hemorrhagic shock precedes acute hemodilution, however, CBF may not increase as expected (Fig 2). In anesthetized dogs subjected to a 30-minute interval of hemorrhagic shock sufficient to maintain MAP at 40 mm Hg, rapid infusion of lactated Ringer’s solution (60 ml·kg−1) increased cardiac output to 4.0 l·min−1 in comparison to a baseline of 3.5 l·min−1, while reducing hemoglobin concentration from 13.1 to 7.0 g·100ml−1 [12]. The lower hemoglobin concentra-
In the uninjured brain, isovolemic hemodilution without intervening hypovolemia produces a decrease in hemoglobin concentration that is balanced by a compensatory increase in cerebral blood flow. Cerebral oxygen transport may be modestly reduced to the extent that cerebral blood flow does not increase proportionately to the decrease in hemoglobin. Following shock or head trauma, hemodilution is not associated with a compensatory rise in cerebral blood flow, therefore, cerebral transport decreases to as great an extent as hemoglobin concentration. Open bars = pre-hemodilution; solid bars = posthemodilution.

Following resuscitation resulted in systemic oxygen transport values (cardiac output × CaO₂) approximately two-thirds of baseline. In contrast to cardiac output, CBF did not return to baseline values following resuscitation with lactated Ringer's solution and in fact did not increase above the level present during hemorrhagic shock. As a consequence, the marked reduction in hemoglobin concentration and CaO₂ was associated with a further decline in cerebral oxygen transport below that present during shock. Following asanguineous resuscitation, cerebral oxygen transport was less than half that measured at baseline. In animals with intracranial mass lesions, resuscitation after hemorrhage to a cardiac output 20% greater than pre-shock values was also associated with an additional decrease in cerebral oxygen transport after resuscitation [13]. Preliminary data suggest that experimental head trauma also prevents a compensatory increase in CBF in response to hemodilution after hemorrhage [14].

The clinical implications of these observations require continuing study. It is possible that asanguineous resuscitation may contribute to the less favorable neurological outcome and increased mortality observed in patients who have hypotension accompanying head injury. The physiological basis of abnormal, posttraumatic cerebral autoregulation is not known, and there are no data to explain the lack of appropriate vasodilation in response to hemodilution following hemorrhage or trauma. If a biochemical mechanism were identified, pharmacological interventions might be combined with fluid resuscitation to restore cerebral oxygen delivery. In addition, these experimental phenomena have yet to be confirmed in patients immediately following hemorrhage or head injury, although CBF has been extensively characterized in the later stages of acute head injury.

Pending further characterization of human cerebral circulatory responses, Shackford's review [1] provides an excellent background for the acute resuscitation of head-injured patients. Regardless of the theoretical concerns noted previously, it is essential to rapidly restore oxygenation, ventilation, blood pressure, and cardiac output in patients who have both head injury and shock. Additional research is required to improve cerebral circulatory resuscitation.

Donald S. Prough, MD
Department of Anesthesia
Bowman Gray School of Medicine
300 South Hawthorne Rd
Winston-Salem, NC 27103

References

6. Andersen BJ, Unterberg AW, Clarke GD, Marmarou A. Ef...