

Intravenous Fluids in Head Injury

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Fluid Management in the setting of head injury, especially in the presence of hypotension is a source of trepidation, since few of these therapies aimed at reduction of intracranial pressure (ICP) and maintenance of cerebral perfusion pressure (CPP) are physiologically benign. Parenteral fluid therapy, particularly in patients with brain injury may aggravate brain swelling, intracranial pressure and neurological dysfunction, resulting in increased neurological morbidity. Hypotension is documented in almost 30% of severe head injuries, and is an ominous predictor of poor outcome than hypoxia¹. Intravenous fluids are indicated for either resuscitation, where the targeted end points are hemodynamic parameters, like blood pressure, central venous pressure or pulmonary capillary wedge pressure, or for maintenance of euvolemic state, balancing urinary and insensible losses. Primary aim of fluid management in neuro-intensive care unit is to maintain vital organ perfusion and ensure substrate delivery to the injured brain while minimizing elevations in intracranial pressure, at the same time, minimizing the need for vasopressors. Most of the head injury victims with hypotension have multisystem trauma, and require rapid volume replacement. The optimal method of fluid resuscitation in the hypovolemic hypotensive brain-injured patient remains controversial. While there have been landmark advances and innovations in various aspects of head-injury management, be it diagnostic imaging, or understanding complex neurochemical events following such an injury, there has been little consensus on type of fluids (crystalloids or colloids) or their tonicity (isotonic or hypertonic) in head-injury.

Crystalloid solutions are mixtures of sodium chloride and other physiologically active solutes, of which sodium is the major component. Physiological saline and Hartmann's solution are two most commonly used crystalloids with identical volume expanding effects². They distribute throughout extracellular space. Aggravation of cerebral edema is minimal if hypo-osmolality is avoided. Commonly available Ringer's lactate is hypo-osmolar (273

mOsm/kg), while physiological saline has an osmolality of 308 mOsm/kg, and is the mainstay of fluid therapy in head-injury patients.

Solutions of 5% glucose with saline are essentially water rendered isotonic to prevent hemolysis, and at most infusion rates, insufficient glucose is infused to raise glucose levels in blood and fluid is distributed throughout total body water³. Thus, for every litre of solution infused, two-thirds will enter intracellular space and one-third will be in the extracellular space. Volume expansion is not marked, and this type of fluid may be beneficial in elderly patients with limited cardiac reserve. However, glucose infusion promotes anaerobic glycolysis and lactic acidosis in the setting of cerebral ischemia, and infusion of such fluids should be avoided in vulnerable patients.

Hypertonic saline was the first solution to be used to reduce raised intracranial pressure⁴. Its effect was found to be short lived and it gained no place in clinical practice. Of late, there has been a resurgence of interest in the use of hypertonic saline especially when combined with colloids, to expand intravascular volume^{5,6}. Rapid infusion of a small volume of hypertonic saline leads to an osmotic gradient that draws water into the intravascular compartment with osmosis of parenchymal fluid. Intracranial pressure is lowered and the effect is more marked than that with infusion of 20 percent mannitol. There is hemodilution, volume expansion and improvement in microcirculation without the need for infusion of excessive quantities of fluids. In a study of trauma victims with concomitant head injury, hypertonic saline infusion was associated with increased survival when compared that with Hartmann's solution⁷. The primary concerns with the use of hypertonic saline are pontine myelinolysis, acute renal insufficiency and hematologic abnormalities (coagulopathy and red cell lysis).

Colloidal solutions are effective in restoring blood volume by withdrawing extracellular fluid into the intravascular compartment by virtue of colloid osmotic pressure (COP). High molecular weight compounds like albumin and urea bridged gelatine are retained in the intravascular compartment and produce an oncotic gradient that tends to retain water in the capillaries. The unique structure of cerebral capillaries tends to limit the effects of

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changes in COP on water content of the brain. As compared to the intercellular pores of the non-cerebral capillaries which are about 65Å, the intercellular pores of cerebral capillaries is about 8 Å. Animal studies have confirmed that damage to blood-brain barrier increases permeability to both proteins and electrolytes so that osmotic and oncotic gradients cannot be established⁸. While resuscitation with hypertonic saline results in lower intracranial pressure, cerebral perfusion pressure is significantly high in those receiving 6% hetastarch, and neurological function was always better in the hetastarch group³. In this background, the findings a multicenter, double blind randomized study of SAFE (Saline versus Albumin Fluid Evaluation) Study⁹ can be analyzed. Out of 6997 patients with polytrauma, 492 had head trauma. While there was higher incidence of death in the albumin group, these patients were the ones who required less resuscitation fluid and had higher central venous pressure as compared to those resuscitated with saline. It is pertinent to point that, the cause of death was not defined in the study. There was no distinct benefit of using albumin. The cost of using albumin was significantly high as compared to that of saline. Thus, the SAFE study demonstrated that though albumin is a better volume expander than saline, it does not translate into improved outcome, and the results need to be interpreted with caution.

Thus, a physician resuscitating a patient with cranial trauma requiring intravenous fluid infusion really does not have a wide choice as far as the type of fluid is concerned. The issue might seem trivial, yet there are few outcome studies that address the issue. While the present data supports the use of isotonic saline, slowly there seems to be an acceptance of hypertonic saline as the initial resuscitating fluid, even in the pre-hospital phase of management of head injury¹⁰. There is however a need for caution and the problems associated with hypertonic saline must be remembered while administering such fluid. Fluids

for maintenance phase are not difficult to decide, and the daily electrolyte, glucose and creatinine levels can help in modulation of intravenous fluid therapy.

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