

Current Trends in Preliminary Cerebral Protection after Traumatic Brain Injury

Sandeep Kumar Kar

Institute of Postgraduate Medical Education and Research, Kolkata, India

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Corresponding author:
Sandeep Kumar Kar

Recently due to overwhelming growth in the automobile industry and the growing Indian economy, road Traffic accidents take a heavy toll of life. Traumatic Brain Injury being the most common cause of death and morbidity followed by injury to the spinal cord. Shearing forces due to abrupt deceleration or acceleration give a heavy thrust to the brain against the relatively immobile cranial cavity, which are termed as primary impact injuries. Primary impact injuries include macroscopic injuries like brain contusions, axial and extra-axial hematomas and microscopic insults like axonal dysfunction, ischemic cytotoxic edema, astrocyte swelling, blood-brain barrier disruption with vasogenic edema, and phasic inflammatory cell recruitment.

The area of unstable hemodynamic conditions around traumatic lesions can be described as a "traumatic penumbra". This portion of brain tissue is at risk and cerebral protection endeavors to prevent its progression to complete destruction. Similarly a 'therapeutic window' exists during which perifocal tissues may be salvaged by reperfusion or by use of pharmacological agents that support cells at risk over a critical period. Brain protection essentially attempts to salvage tissues in this "traumatic penumbra" within this therapeutic window [1].

Secondary biochemical reactions manifest themselves in three distinct ways, firstly there is disruption of calcium homeostasis as an, immediate after effect of trauma leading to raised intracellular calcium levels which activate myriad array of enzymes which cause cellular damage. Free radical mediated peroxidation and acidosis also contribute to neural tissue damage. Excitotoxicity caused by glutamates as sequelae also converge into the final pathway of increased calcium levels. Rise in the EAA (excitatory amino acids) concentration is the main cause of calcium entry via specific glutamate operated calcium channels. The immediate consequence of extracellular glutamate elevation is an enhanced stimulation of post-synaptic like receptors (NMDA, AMPA, kainate). Early initiation of monitoring after brain injury and pharmacological neuroprotection plays a pivotal role in salvaging tissues in traumatic penumbra within the window period.

Monitoring of Mean Arterial Pressure along with measurement of ICP (Intracranial Pressure) is essential for calculating and manipulating the Cerebral Perfusion Pressure (CPP=MAP-ICP) the monitoring of systemic oxygenation (by pulse oximetry, aided by arterial blood gas measurement). The need to measure core body temperature and regular blood sugar is essential as

✉ sndpkar@yahoo.co.in

Assistant Professor, Cardiac Anaesthesiology,
Institute of Postgraduate Medical Education
and Research, Kolkata, India

Tel: 9477234900

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hypothermia is neuroprotective by decreasing metabolic oxygen requirement of brain and hyperglycemia aggravates neurological damage induced by the primary impact injury and the consequent secondary biochemical neurological insult.

The therapy revolves around the concept of brain oriented life support, optimizing MAP and avoiding hypotension. Traumatic Coma Data Bank (TCDB) and from other sources which demonstrate the detrimental effects of hypotension (systolic blood pressure below 90 mm Hg) and hypoxia (PaO₂ levels below 60 mm Hg [8 kPa]) in the early and later phases of head injury on outcome [2]. Fluid therapy forms an important role in this brain oriented life support concept. Hypoosmolar and dextrose containing fluids should be avoided as they aggravate cerebral injury and edema.

Hypertonic saline raises plasma sodium and osmolality with reduction in ICP and reduction of midline shift in head injuries [3, 4]. Simma et al reported that 1.6% saline, when compared to lactated Ringer's solution as maintenance fluid in head injured children, resulted in lower ICP values, less need for barbiturate therapy, a lower incidence of acute lung injury, fewer complications and a shorter ICU stay [5]. Maintenance of oncotic pressure with albumin supplements is the strategy employed in Lund protocol. (infusion of albumin and RBC to maintain colloid osmotic pressure near normal values and capillary hydrostatic pressure is decreased by reducing systemic blood pressure by the

use of alpha 2 agonists like clonidine and dexmedetomidine), beta blockers and brain selective calcium channel blocker nimodipine.

The strategies employed to reduce ICP involve drainage of CSF and decompressive surgery (if feasible) and Hyperventilation, whose effect is limited by compensatory reductions in cerebral extracellular fluid (ECF) bicarbonate levels rapidly restore ECF pH and over time, attenuate the effect of low PaCO₂ levels on vascular tone, which can be mitigated with the use of tetra-hydro-aminomethane (THAM), which may restore ECF base levels and restore the reactivity of the cerebral circulation to carbon dioxide. In presence of hyperopia the effects of hyperventilation

can be sustained longer. Barbiturates (barbiturate narcosis from bolus doses to infusions for 24 to 72 hours or more. as post-insult injury may last for this period and cerebral edema peaks at 48 hours after an ischemic injury.)

Inhaled anesthetics like isoflurane are neuroprotective at clinically useful concentrations (<2MAC). Isoflurane is a potent inhibitor of CMR and CMRO₂ in all species studied. Isoflurane inhibits multiple voltage-gated calcium currents in hippocampal pyramidal neurons in addition to potentiating the effects of GABA. Isoflurane significantly inhibits glutamate receptor activation and ischemia induced calcium influx [6, 7] NMDA antagonists like ketamine and remecimide may also be used to combat excitotoxicity.

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