

# WHAT'S NEW IN MONITORING IN SEVERE HEAD INJURY

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Central nervous system (CNS) trauma is a significant cause of morbidity and mortality all over the world. In USA, each year, 500,000 patients with head injury are seen in the emergency department and of these more than 50,000 die from their injuries (1). Major cause of this menace is cerebral ischemia that ensues minutes to hours after the primary head injury. This is commonly known as secondary injury. Secondary brain lesions resulting from cerebral metabolic and hemodynamic reactions can be prevented by neurocritical care management. It must be initiated as early as possible, ideally in a prehospital setting.

In the last decade, there has been significant progress in the area of neurotrauma management. Elucidation of important pathologic mechanism leading to secondary brain lesions, a better understanding of the consequences of therapeutic agents for brain physiology, and the development of multimodality monitoring have lead to changes in standard practice. First of all, the influence of initial critical care on outcome is now clearly documented. Critical care management of severe head injuries must be regarded as a continuum that starts with initiation of critical care at the scene, uses of multimodality cerebral monitoring for further adaptation of therapeutic agents to brain hemodynamic and metabolism and ends with intense neurologic rehabilitation(2).

The two most important secondary injury processes that can be monitored, anticipated, and treated in the head injured patient are intracranial hypertension and cerebral ischemia. Recent monitoring practices as well as most of the new technologies available for monitoring patients with a traumatic brain injury address one or both of these processes.

## Routine Monitoring:

Continuous monitoring of intracranial pressure (ICP) and cerebral perfusion pressure (CPP) has become a standard in neurointensive care of severe head injured patients. In addition, head injured patients should have systemic parameters closely monitored, including ECG, heart rate, blood pressure, temperature, fluid intake and output. Routine monitoring of oxygen saturation and capnography is paramount in severely head injured patients so as to avoid unrecognized hypoxemia or changes in ventilation (3).

New devices including jugular bulb oximetry, near-infrared spectroscopy, brain tissue PO<sub>2</sub>, brain temperature, and microdialysis have significantly expanded the number of parameters that can be monitored in a patient with severe head injury.

## Monitoring Intracranial Pressure:

Intracranial pressure can not be reliably estimated by assessing the clinical features after severe head injury. Clinical manifestations of raised ICP are impossible to elicit in comatose patients. Neurologic signs, including pupillary dilation and decerebrate posturing, can occur in the absence of intracranial hypertension. Computed tomography scan signs of brain swelling, such as midline shift and compressed basal cisterns, are predictive of raised ICP. But, intracranial hypertension can manifest without these findings. It is imperative therefore, to have continuous monitoring of ICP in patients with severe head injury.

Forsyth and his colleagues had evaluated whether routine ICP monitoring in all acute cases of severe coma

reduces the risk of overall mortality or severe disability. They were not able to formulate a definitive conclusion due to lack of randomized controlled studies comparing outcome in group with ICP monitoring versus no ICP monitoring (4). Therefore, evidence that ICP monitoring alters outcome in severe head injury is lacking. However, the major advantage of ICP monitoring is, the risk is low and ICP information is useful in making therapeutic interventions.

The intraventricular ICP monitoring through ventriculostomy has been the gold standard. Nowadays, several new devices are available. An audit of the reliability of one of these new devices, Camino intracranial pressure sensor revealed failure in 10% of sensors, problems of zero drift and last but most importantly the sensitivity of the Camino devices to environmental temperature remains a problem (5).

It is common practice to immobilize the cervical spine during the early management of trauma victims.

Application of the rigid collar may increase the ICP by 4 to 5 mm of Hg. The increase in ICP was longer in patients with an initial ICP higher than 15 mm of Hg than in patients with baseline ICP lower than 15 mm of Hg (6).

Early repeated CT scanning is indicated in patients when hemorrhage is identified on the first scan and treated conservatively. One of the complications of the head injury that can result in intracranial hypertension is a delayed intracranial mass lesion due to hemorrhage. Oertel et al. demonstrated that early progressive hemorrhage occur in 49% of head injured patients who undergo computed tomography scanning within two hours of injury. Progressive hemorrhage occur most frequently in patients with cerebral contusions, and are associated with intracranial hypertension. In their logistic regression model, male sex, older age, time from injury to first CT scan and prolonged first partial thromboplastin time (PTT) were statistically significant factors related to the prediction of early progressive intracranial hemorrhage (7).

Intracranial hypertension is an important factor in determining outcome in most large series of head injury. In their retrospective review of 392 patients with severe non-penetrating injury (GCS 3-8) Clifton et al. found that GCS at admission, age, mean arterial blood pressure (>70 mm Hg), fluid balance lower than -594 ml. and ICP higher than 25 mm Hg as significant factors predicting outcome. The result suggests that dehydration should be avoided and euvolemia maintained by adequate fluid replacement after mannitol therapy, while also avoiding over hydration. The study also lends support to the guideline that CPP should be maintained at a level higher than that may not promote brain edema formation (8).

Review of literature favours the earnest recommendation pertaining to ICP monitoring in severe head injured patients. ICP monitoring is highly essential in patients with severe head injury and treatment goals should be to avoid an ICP higher than 25 mm Hg.

## Monitoring of Cerebral Ischemia:

The ideal monitor for cerebral ischaemia after traumatic brain injury is yet to be invented. This ideal monitor would have the following properties, would give regional information about cerebral blood flow (CBF), since there can be marked regional differences in CBF after trauma. Also give continuous information, since CBF evolves over time after injury. The techniques that are available fall under two general categories, those that monitor cerebral perfusion or blood flow and those that monitor cerebral blood flow adequacy.

## Cerebral Perfusion Pressure Monitoring:

The simplest measure of cerebral perfusion is the cerebral perfusion pressure (CPP) which is nothing but (MAP-ICP). Decreases in CPP can occur either through decrease in BP or increases in ICP. For equivalent levels of CPP, cerebral perfusion is impaired more by reduction in blood pressure than by increase in ICP. The most recent studies have shown that a CPP of 60 mm Hg is adequate for most patients (9). Cerebral ischaemia however, can occur despite CPP being 60 mm Hg, and therefore more specific measures of CBF may be

desirable.

Recently, there has been remarkable progress in the technologies that have made CBF measurement more feasible in critically ill neuro patients. Imaging techniques such as the stable-xenon-enhanced computed tomography (XeCT), perfusion computed tomography, perfusion magnetic resonance imaging, single photon emission computed tomography (SPECT) and positron emission tomography (PET) provide excellent regional information about CBF. These measurements of CBF, however, are a single snap shot in time. Two methods of continuously measuring local CBF are now commercially available, the 1. thermal diffusion method and the 2. laser Doppler method. But the major limitation of both these methods is, CBF was measured in a small volume of the brain, which may not reflect the whole brain or the region of importance.

A prospective controlled study was performed to compare the new thermo-dye dilution-based measurement of CBF technique with that of XeCT. Combined fiberoptic-thermistor catheters were placed in one jugular bulb and in the abdominal aorta. A 50 ml. bolus of pre cooled indocyanine green solution was injected into central vein. Determination of CBF was carried as a function of the mean transit times of coldness and dye. The thermo-dye-dilution technique was found to be reasonably reproducible technique, enabling repeated long term bed side measurement of CBF. However, the major difference in comparison to XeCT CBF measurement was the overestimation of CBF (average 45.7 ml/100G/minute) (10). Although the technique is successfully validated in patients with normal neurovascular functions, its applicability and usefulness for bedside monitoring of CBF in patients following traumatic brain injury (TBI) appears uncertain.

Traumatic brain injury (TBI) is a very heterogeneous process. Measurement of CBF through imaging techniques is of great help to the treating physicians. It provides important insights into the evolution of injury and also into the effects of treatments which may alter CBF such as hyperventilation.

von Oettingen et al. had measured CBF by using XeCT in 26 traumatic contusions and correlated regional CBF in the contusional area with the extent of tissue necrosis identified as focal atrophy on late follow up computed tomography. Mean regional CBF in those contusions was  $5.9 \pm 5.9$  ml/100G/minute. The contusions exhibited a specific regional CBF profile, presenting as a core of severe lethal ischemia surrounded by variable but gradually increasing perfusion with increasing distance from the ischemic core (11).

The effect of moderate ( $\text{PaCO}_2 = 30 \pm 2$  mm Hg.) and severe ( $\text{PaCO}_2 25 \pm 2$  mm Hg.) hyperventilation on CBF and CMRO<sub>2</sub> was studied by Diringer et al. utilizing PET. They had concluded that even profound hyperventilation is not harmful following severe head injury because the metabolism of neuronal cells are so depressed that even a reduction in CBF to less than 10 ml/100G/min. did not induce a significant change in CMRO<sub>2</sub>. Decreased delivery of oxygen was compensated by increased extraction of oxygen. The authors concluded that after TBI, brief hyperventilation produced large reductions in CBF, but didn't produce energy failure (12). Oxygen metabolism was preserved due to low baseline metabolic rate and compensatory increases in oxygen extraction function, thus, these reductions in CBF were unlikely to cause further brain injury. This study has some limitations as the experiment was carried out at a time when patients were fully resuscitated, and CPP was more than adequate. This is usually not the case when hyperventilation is being considered as an additional treatment option in a head injured patient. On the other hand the situation is totally different during the transfer of the TBI patients. Their EtCO<sub>2</sub> may be very low in 60-70% of patients due to severe hyperventilation (13).

## Monitoring Cerebral Blood Flow:

Jugular venous oxygen saturation (SjvO<sub>2</sub>) or brain tissue PO<sub>2</sub> have been used as measures of cerebral oxygenation in place of quantitative CBF measurement. Because they have been found to be very good indicator of the adequacy of CBF in relation to cerebral metabolic requirements. If the brain is hypoperfused, oxygen extraction will be increased, and SjvO<sub>2</sub> will be reduced. On the other hand, if CBF is adequate for the brain's metabolic need, then SjvO<sub>2</sub> will remain normal (14). This information is more often useful clinically than the absolute CBF values.

Cruz and his colleagues managed 45 children following severe TBI by a strict treatment protocol in which, ICP was maintained within 15 mm Hg, CPP (50-80 mm Hg) and arteriojugular oxygen saturation difference around (17-35%). They had achieved favourable outcome in 82.2% of the patients and the mortality was 4.4%. It was concluded that unfavorable outcomes were significantly related to more pronounced intracranial hypertension and more profound concomitant decreases in hemoglobin saturation by oxygen, indicating hyperoxic uncoupling between global cerebral consumption of oxygen and cerebral blood flow (15).

**Measurement of brain tissue oxygenation and metabolism** has been validated along with PET scanning by Gupta et al. to find out a novel monitoring method. The authors didn't find any correlation between end capillary oxygen tension and brain tissue oxygen pressure, but they found positive correlation between change in end capillary oxygen tension and change in brain tissue oxygen pressure. They concluded that when the sensor had been placed into a healthy brain, tissue PO<sub>2</sub> monitoring might provide a useful tool to assess the effect of therapeutic interventions in brain injury (16).

**Brain tissue oxygen pressure monitoring** along with S<sub>ij</sub>O<sub>2</sub> has been found to be very useful during hyperventilation. Imberti et al. had observed decrease in brain tissue PO<sub>2</sub> below 10 mm Hg in presence of near normal S<sub>ij</sub>O<sub>2</sub> value in 6 out of 36 patients with severe head injury during hyperventilation. The authors found regional monitoring of brain tissue oxygen pressure to be complimentary information to S<sub>ij</sub>O<sub>2</sub> values in detecting critical changes in cerebral oxygenation (17).

**Transcranial cerebral oxymetry** utilizing near - infrared technology can be monitored continuously in patients with severe head injury. It measures cerebral oxygen saturation that significantly correlates with CPP. Cerebral oxygen saturation higher than 75 suggests adequate CPP, while a value lower than 55 suggests an inadequate CPP. Cerebral oxymetry monitoring may serve as a non-invasive measurement of cerebral perfusion in patients with TBI (18).

## Monitoring of Brain Metabolism:

Brain glucose and lactate levels can be monitored with the help of imaging modalities such as magnetic resonance and PET scanning. Recently, however, the ability to measure the concentration of metabolites in the extracellular space directly and continuously, has only become feasible with the application of cerebral microdialysis (19).

Baseline microdialysis values of glucose ( $1.7 \pm 0.9$  mmol/l), lactate ( $2.9 \pm 0.9$  mmol/l), pyruvate ( $166 \pm 47$   $\mu$ mol/l), glycerol ( $82 \pm 44$   $\mu$ mol/l), glutamate ( $16 \pm 16$  mmol/l), and urea ( $4.4 \pm 1.7$  mmol/l) were measured by placing the microdialysis sensor in the frontal region of the brain in nine patients undergoing posterior fossa surgery for benign lesion(20).

Metabolic changes in brain tissue were monitored continuously by placing two microdialysis probe in worse and better position by Stahl et al. in 48 patients with severe head injury and ICP higher than 20 mm Hg. They had observed a gradual normalization of all biochemical markers from both probes independent of the location and also irrespective of the CPP. Although the results tend to support the Lund management strategies some methodological loopholes were there (21).

They had further extended their study and analyzed the results in seven patients who died due to intractable intracranial hypertension following severe head injury using two microdialysis probes. The first probe was placed near the lesion (contusion or underlying an evacuated hematoma) and the second probe was placed in the frontal region together with an intraventricular catheter (healthy area or better position). The author found that biochemical changes typical for ischemia occurred prior to elevation in ICP in the probe placed in worse position<sup>22</sup>. The same pattern was also observed in the probe in the better position, but after intractable elevation of ICP.

## Conclusion:

Advancements in technology are taking place at a rapid pace that has resulted in the availability of newer devices that make the Neuromonitoring truly multimodal. Clinical studies are needed so as to find out the best monitoring modalities that can change the management strategies of patients with severe head injury.

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