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TBI Audit: Audit of Monitoring, Surgery, Intensive Care and Outcome in Traumatic Brain Injuries

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Introduction

Traumatic brain injuries (TBI) represent a significant contributor to fatalities and disabilities worldwide. Those who survive moderate to severe TBIs often encounter prolonged difficulties in resuming work or education, maintaining social connections, and performing routine daily tasks.(1) Additionally, over the long term, TBI is linked to an elevated risk of deteriorating health, cognitive impairment, the onset of neurodegenerative conditions and an increased dependency for personal care throughout an individual's life. On a worldwide scale, it's approximated that there are 200 new cases of TBI per 100,000 people each year, with an associated mortality rate of 20 per 100,000 individuals annually. A single epidemiological study conducted in Bangalore by NIMHANS found that the incidence of TBIs, the mortality rate, and the case fatality rate were reported as 150 per 100,000, 20 per 100,000, and 10%, respectively.(2) Approximately 50% of trauma deaths are likely to be secondary to TBI implying one TBI related death every 3 minute in India.(3,4) Specific national-level data for TBI in India is currently unavailable as there is no national trauma registry. A lot needs to be done for prehospital care of TBI (as currently only 24% arrive within 1 hr. of injury, 30% arrive within 2-3 hrs. of injury. Only 5-8% patients in India get emergency care at scene of incident as against 90% of patients getting emergency care in Europe.(5)

Better compliance to guidelines, better infrastructure and experience of treating faculties are responsible for improved TBI outcomes in level 1 trauma centers globally. While severe TBIs are best treated in level I/II trauma centres [wherein both neurosurgeons and CT scan facility is available], many district hospitals still lack CT scanners, mechanical ventilators, rehabilitation services and even availability of Neurosurgeons.(3-6) Currently, 6 level I and over70 level II trauma centres are present in India, a greater number of level 1 trauma centres need to be added for improved care of TBI cases in India. In busy public hospitals/trauma centres, absence of beds in intensive care unit (ICU) results in severe TBI being managed in the emergency department or wards with less monitoring, physiological support, or medical attention. ICU beds are generally allocated on a first-come, first-serve basis in the absence of triage support. It is not just absence of advanced technology, but lack of availability of ICU beds, basic physiological support devices (e.g., bedside monitors, ventilators, etc.), and access to CT imaging which is a major limitation to providing care in most LMICs and India.

Understanding TBI outcomes is vital for personalized patient care, improving quality of life, efficient resource allocation, injury prevention, research, policy development and innovation in treatment and rehabilitation.

TBI Classification and Severity: The Glasgow Coma Scale (GCS) serves as a widely accepted tool for the objective evaluation of a person's level of consciousness. It offers a scale that ranges from 3 (indicating complete unresponsiveness) to 15 (indicating full responsiveness).

The Glasgow Coma Scale (GCS) was introduced forty years ago by Teasdale and Jennett as a practical method for assessing the full spectrum of disorders of consciousness, from very mild to severe. It has been broadly adopted, and is internationally utilized as an integral part of clinical practice and research. The GCS has found extensive use in classifying Traumatic Brain Injuries (TBI) into various severity levels and predicting outcomes:

- Mild TBI: Characterized by a GCS score of 13-15, with an associated low mortality rate approximating 0.1%. Term'concussion 'is often used interchangeably with mild TBI.
- Moderate TBI: Typically corresponds to a GCS score of 9-12, and it carries a mortality rate of around 10%.
- Severe TBI: Indicated by a GCS score below 9, this category has a significantly higher mortality rate, estimated at approximately 25-40%.

These distinctions help in the initial assessment, treatment decisions, and predicting outcomes for individuals who have experienced TBI, ensuring appropriate care and resources are allocated based on the severity of the injury.

The GCS aims to rate performance in three different domains of response: the eye, verbal, and motor response. For individual patients, it is recommended that in that all three

components be reported. If a GCS component is untestable due to intubation, sedation, or another confounder, the reason for this should be recorded. Although often done, a score of 1 should not be assigned because differentiation between a "true 1" and an untestable component is relevant. Graphical display of the three GCS components over time may facilitate earlier detection of changes. Assessment requires either a spontaneous response or response following application of a stimulus. At more severely disturbed levels of consciousness, the motor score has better discrimination, but in milder injuries the eye and verbal components are more relevant. Thus, each component of the scale (Eye, Verbal, Motor) provides complementary information. Strengths of the GCS are that it covers a broad spectrum of disorders of consciousness, is widely applicable, and offers an important tool for monitoring changes in the level of consciousness. Standardized approaches to both its assessment and its reporting are required in order to be able to compare evaluations over time or when communicating with other health care professionals. Spontaneous responses are first observed without stimulating the patient in any way. First, verbal stimuli are applied, such as asking a patient to obey commands and at the same time observing whether, e.g., an eye opening occurs. If a patient is not responsive, a stimulus is applied to elicit a response. The location of the stimulus (central or peripheral) should be standardized and used consistently. To describe the motor response, only the reaction of the arms should be observed, not the legs.(7-9)

Advancements in Monitoring for Traumatic Brain Injury

Traumatic Brain Injury (TBI) remains a significant public health concern, and continuous advancements in monitoring techniques are instrumental in improving patient outcomes. Monitoring patients following traumatic brain injury (TBI) via conventional examination methods might not suffice for frontline healthcare providers aiming to minimize secondary brain damage through personalized treatment.

Most interventions in TBI do not target disease, rather they remain focused on physiological targets including intracranial pressure (ICP) , cerebral perfusion pressure (CPP) and/ or multimodality monitoring (MMM).

Multimodal neurological monitoring (MMM) holds promise in early injury detection and enhancing outcomes. Through evaluating cerebral oxygenation, intracranial pressure (ICP) monitoring, Near-Infrared Spectroscopy (NIRS), pupillometry, and cerebral micro-dialysis (CMD) metabolism, clinicians can grasp the neurophysiological aspects during acute brain injury, enabling tailored therapies for individual patients at various stages of injury.

Intracranial Pressure Monitoring (ICP)

ICP is the CSF (Cerebrospinal fluid) pressure that must be exerted against a needle introduced into CSF space to prevent the escape of fluid. **Traumatic brain injury** is the most frequent indication for ICP monitoring. The presence of open fontanels and/or sutures in an infant with severe TBI does not preclude the development of intracranial hypertension or negate the utility of ICP monitoring. Elevated ICP is predictive of poor outcomes in TBI. Intracranial pressure (ICP) monitoring remains a cornerstone in TBI management [Figures 1 and 2] The gold standard for ICP measurement is via an external ventricular drain (EVD), attached to an external strain-gauge transducer. The monitor, centrally placed within the cerebral ventricles, can measure global ICP and offers the therapeutic advantage of draining CSF to reduce intracranial volume. Intraparenchymal ICP monitoring is also a reliable method but does not allow for CSF drainage. Subdural and epidural monitors have been used, but these are the least accurate methods of ICP measurement.

ICP monitoring is recommended as part of protocol driven care in patients at risk of raised ICP, to detect life threatening herniations and as integral part of MMM to allow data interpretation by these devices. Continuous ICP monitoring provides real-time data, guiding therapeutic interventions such as osmotherapy, hyperventilation, and decompressive craniectomy.(10)

Indications for ICP monitoring: If ICP monitoring is possible, all salvageable patients with severe TBI and abnormal CT scan (presence of structural brain damage) should

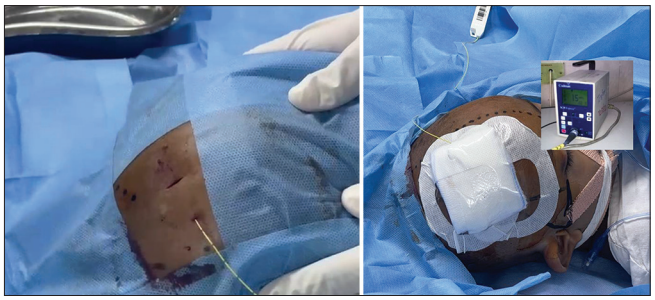

Figure 1: Left-Intraparenchymal ICP catheter inserted after Right Frontal Twist drill and tunneled out through separate incision. Right - ICP catheter with microsensor and ICP box (inset) for continuous bedside ICP monitoring

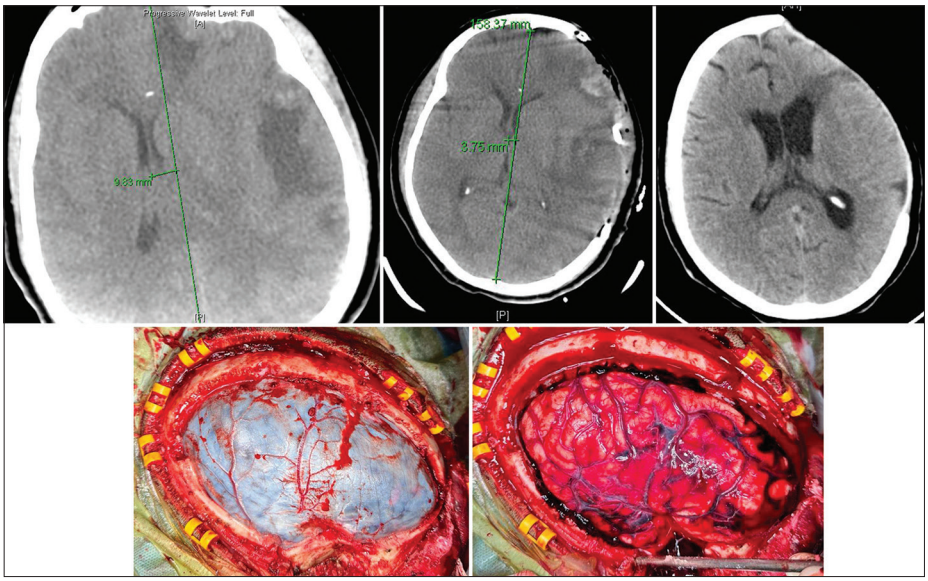

Figure 2: 40 yrs old female who was GCS 14/15 had ICP monitoring for persistent headache, significant midline shift and cisternal effacement. ICP on day 3 post injury raised to 30 mm Hg persistent. Intraop dura was tense before opening, brain was tense reflecting raised intracranial pressure. Left frontotemporal decompressive craniectomy done . Patient improved to GCS 15/15 with GOS 5, she came for cranioplasty 3 months later

undergo ICP monitoring.(4,6) In case of a normal CT scan, ICP monitoring is indicated if two of the following are present:-

1. Age > 40 years
2. Motor posturing
3. SBP < 90 mm Hg.

ICP monitoring should be considered in patients with a GCS > 8 who have structural brain damage with high risk for progression (large/multiple contusions, coagulopathy).

ICP monitoring should be considered in patients who require urgent surgery for extracranial injuries, who need mechanical ventilation because of extracranial injuries, or who evidence progression of pathology on CT imaging or clinical deterioration .

ICP monitoring is generally not indicated in comatose patients without evidence of structural brain damage or elevated ICP (compressed/absent basal cisterns) on initial CT imaging.

Patients may be observed with repeat CT imaging and forego ICP monitoring if there is no progression.

The Brain trauma foundation (BTF) guidelines (updated last in 2016) advocate for ICP monitoring in all severe TBI cases to reduce in hospital and 2 week mortality [Level IIb recommendation to use ICP data]. Also, Cerebral perfusion pressure (CPP) monitoring

is recommended to decrease 2-week mortality [Level IIb recommendation to use CPP data]. However, due to cost restraints and availability issues, intraparenchymal ICP monitoring usage varies greatly from no ICP monitoring at most centres (< 10%) to some ICP monitoring practice in few Level 1 trauma centers with advanced ICU monitoring facilities in India. BTF guidelines emphasize the need to interpret ICP values within the broader clinical context, recognizing that some patients may exhibit favorable outcomes despite persistently elevated ICP. Elevated ICP can lead to secondary brain injury, making early detection and intervention crucial. The harm associated with intracranial pressure elevation is unlikely to be uniform across patients and over time. A word of caution- ICP monitoring should not be used in isolation as a prognostic marker.

Mixed results have been noted with various trials on efficacy of ICP monitoring in TBI cases. SYNAPSE-ICU (Study on Intracranial Pressure in Intensive Care) showed better outcomes at 6 months in patients with ICP monitoring as a result of more intensive therapies.(10) Better outcomes were noted with higher compliance to BTF guidelines (which included ICP monitoring) in CHIRAG study that compared outcomes of TBI in 2 level I apex trauma centers (AIIMS, Delhi and Harborview Medical Centre, USA.(11) Nattino *et al* evaluated the association of ICP monitoring with TBI outcomes in the CREACTIVE (Collaborative Research on Acute Traumatic Brain Injury in Intensive Care Medicine in Europe) consortium database. They found no difference in mortality in patients who underwent ICP monitoring vs those who did not. Incidence of respiratory complications, infection, duration of ICU stay , mechanical ventilation and 6 month outcome was worse amongst patients who underwent ICP monitoring.(12)

The BEST TRIP randomized trial, from South American center did not show any benefit of a protocol based on intracranial pressure monitoring when compared with management based on clinical examination and serial imaging.(13) Although there was no difference in outcomes between the groups, this does not support the discontinuation of ICP monitoring in the treatment of TBI. Rather, it demonstrates the importance of aggressive treatment using ICP monitoring or frequent clinical and radiographic examination to identify intracranial hypertension. This study also challenges the currently accepted rigid ICP alert threshold of 20 mm Hg for all patients. The current accepted alert threshold is an ICP of 20 mm Hg, with a reasonable range of 20-25 mm Hg as a trigger for treatment of intracranial hypertension. Ongoing research may reveal that this threshold is dependent upon individual patient factors. An approach based on injury type and augmented by advanced neuromonitoring may lead to individualized treatment pathways.

ICP Thresholds, ICP Dose in Accordance with BTF Guidelines and CENTRE TBI Research

Over the years, various studies have employed different criteria for intracranial pressure (ICP) in patients with head injury to determine the need for surgical intervention. Although existing guidelines suggest treating a sustained intracranial pressure that is greater than 22 mm Hg and keeping cerebral perfusion pressure between 60 and 70 mm Hg, these thresholds are not absolute. In today's neurocritical care facilities worldwide, ICP threshold of 20-25 mm Hg is generally accepted. Concept of so-called ICP dose as per CENTRE TBI confirmed research(12) that integrates both the intensity and duration of an intracranial pressure event, is increasingly gaining recognition. Evolution of intracranial pressure over time needs to be considered to guide management decisions. Tolerance of intracranial pressure insults is reduced by impaired cerebral autoregulation and assessment of autoregulatory status is increasingly used to titrate cerebral perfusion pressure targets nowadays in centres where ICP, CPP monitoring facility is available. The commonly utilized ICP thresholds are summarized in Table 1 below.

ICP monitoring is important , but it is does not replace careful neurological /radiographic examination. A combination of ICP values, clinical and brain CT findings may be used to make management decisions (Level III recommendations of BTF guidelines, 2016).

Cerebral perfusion pressure (CPP), a parameter derived from ICP (Mean Arterial Pressure – ICP), is an important marker of cerebral blood flow; augmenting CPP can help to restore cerebral perfusion and oxygenation. In addition to enabling CPP measurement, ICP monitoring can provide advanced warning of impending structural brain derangements

Table 1: ICP thresholds according BTF 4th edition (2016)/BTF pediatric guidelines 3rd edition (14,15)

	Normal (mm Hg)	Threshold for treatment
Adult & older children	10-15	22 mm Hg (Level II b recommendation)
Younger children	3-7	Target <20 mm Hg
Term infants	<1.5–6	

such as contusion/hematoma progression, increased cerebral edema, and postoperative complications. The identification of ICP elevation can prompt further imaging, timely intervention, and definitive management.

ICP is a global measure that cannot distinguish among these mechanisms. Additional neuromonitoring of brain tissue oxygen tension (PbtO2), jugular venous oxygenation (SjvO2), cerebral blood flow (CBF), cerebral autoregulation, and other parameters may be helpful in identifying a more individualized approach to treatment.

Cerebral Micro-dialysis (CMD) in TBI

While it does provide the opportunity to understand cerebral metabolism, it's clinical use at the bedside is largely limited. While there is no question on its usefulness as research tool in past 3 decades, however, its value as therapeutic or prognostic tool is always being questioned.

Various forms of metabolic dysfunction (regional/dynamic) are more commonly noted than frank cerebral ischemia with advanced prehospital/ICU care these days. Cerebral micro-dialysis is an advanced monitoring technique that provides insights into the biochemical environment of the brain. CMD uses a semi-permeable catheter (20 kD,100 kD), placed in the brain parenchyma, through which a perfusate is instilled at a rate of ~0.3 uL/min into the cerebral parenchyma [Figure 3]. By sampling the interstitial fluid, one can assess markers of cerebral metabolism and inflammation. As CMD monitoring allows assessment of focal rather than global metabolism, probe location becomes an important factor for interpretation of results. Timing and duration of monitoring is also relevant, on account of the dynamic nature of the pathophysiological processes occurring in the acute phase following TBI, most monitor for 3- 5 days after injury.

CMD technique facilitates hourly sampling of small water-soluble molecules like glucose, lactate, pyruvate, glutamate or glycerol, as well as various cytokines.

Glucose is the energy substrate preferred by neurons through the process of glycolysis, although neurons can also utilize lactate as a substrate through the astrocyte neuron lactate shuttle (ANLS). The presence of cerebral hypoglycemia i.e. Glucose < 0.8 mmol/L is commonly noted in TBI patients secondary to reduced blood supply as a result of low CBF or increased demand (Seizures, hyperthermia and cerebral hypermetabolism). This highlights the need to avoid tight glycemic control that may worsen the cerebral metabolic crisis. Cerebral hyperglycemia and hypoglycemia both are associated with worse outcomes in severe TBI.(16)

CMD based abnormal metabolic states can be described following TBI, informing the etiology of physiological derangements and make tailored therapies. Patients with extradural hematomas and no mass lesions have normal LPR values, whereas patients with subdural hematomas or hemorrhagic contusions have higher LPR values . LPR directly reflects the biochemical redox state within the brain reflected by raised LPR as a metric of energy failure(17) [Figure 4]. Under anaerobic conditions/ mitochondrial dysfunction, pyruvate is converted to lactate by the enzyme lactate dehydrogenase (LDH) in the cytosol instead of entering the mitochondrial TCA cycle. This shifts the LPR, which is a measure of the metabolic redox state. Inadequate energy substrate (scarcity of glucose), impaired utilization of the available energy substrates caused by cellular damage, cerebral ischemia from raised ICP, altered cerebral autoregulation or insufficient oxygen supply can all affect metabolic processes adversely after TBI.

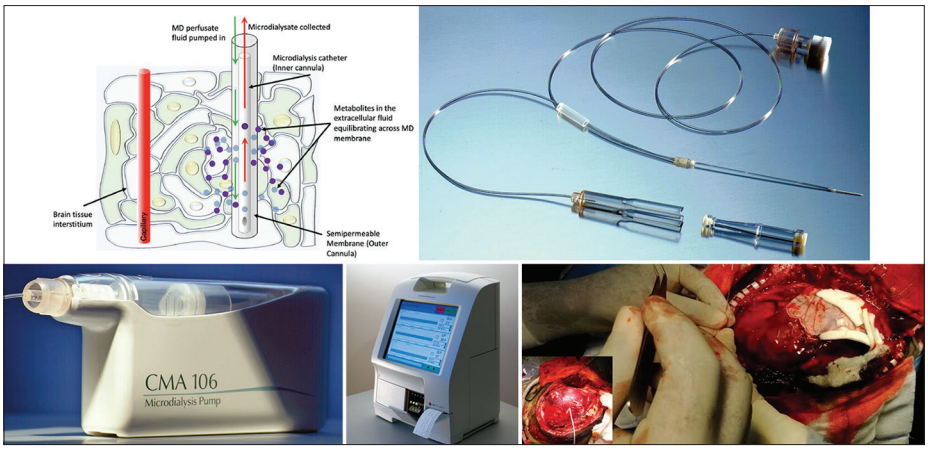

Figure 3 : Top Left – Priniciple of microdialysis (capillary action), Top right – 20 Kda Microdialysis catheter with connector and microvials. Bottom Right – Cerebral microdialysis pump for delivery of perfusion fluid at constant rate. Bottom Middle – CMD analyser for bedside monitoring of dialysate for cerebral metabolism markers (glucose, gluatamate, lactate, pyruvate, glycerol). Bottom right – Cerebral microdialysis catheter placed intraparenchymal at the end of decompressive craniectomy in pericontusional are of brain

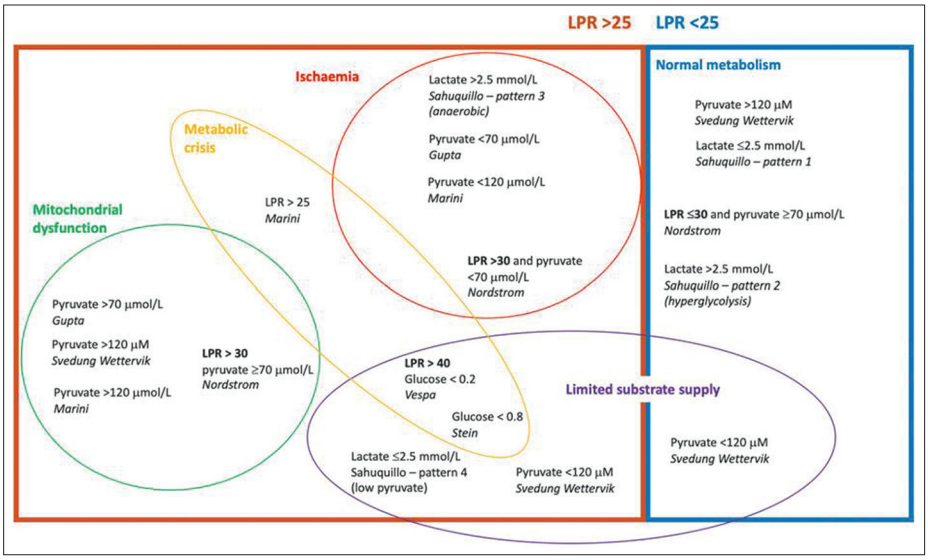

Figure 4 : Schematic representation of how different classifications of abnormal metabolic states described in the articles included in this review overlap. The included studies are by Gupta *et al*., Marini *et al*., Nordstro¨m *et al*. Sahuquillo *et al*. Stein *et al* Svedung Wettervik *et al* and Vespa *et al*

LPR (Lactate-Pyruvate ratio) is indicative of metabolic crisis. LPR elevation has been seen to occur in conditions of ischemia (with a fall in pyruvate) and also in mitochondrial dysfunction (with a near normal pyruvate level).

Mitochondrial dysfunction is an abnormal metabolic state in which there is impairment in the ability of cells to utilize energy substrates such as glucose, pyruvate and oxygen, despite availability of adequate supply. [Figure 5] Gupta *et al* noted that the unfavorable outcome group showed significantly higher episodes of both mitochondrial dysfunction and ischemia than the favourable outcome group.(18)

Variations in cerebral perfusion, oxygenation and glucose supply are associated with changes in cerebral LPR and suggest therapeutic interventions to improve cerebral metabolism. In a prospective study of 619 adult TBI patients who had ICP, CPP, CMD monitoring, LPR over the first 3-7 days post injury was elevated amongst patients with poor outcome, a nonlinear association was also observed between LPR and cerebral glucose, CPP and PRx (pressure reactivity index).(19)

L/P ratio above 25 and glycerol levels above 100 mmol/L are noted to be associated with a significantly higher risk of ICP raise within the next 3hours. This means brain energy crisis may occur before low CPP is detectable, so, L/P ratio and glycerol elevations may be early warning signals of imminent deterioration and monitoring for these markers might allow targeted therapy to be applied in a timely manner preventing further deterioration. Pathological statesfor LPR > 25 with suggested targeted interventions to correct abnormalities are [Table 3](20):

BTF guidelines/Neurotrauma society of India NTSI are silent on the usage of Cerebral microdialysis in TBI(14,21) . Cerebral microdialysis usage in TBI remains largely observational and investigative in nature. A recently held consensus meeting by the **international microdialysis forum**, led by P.J. Hutchinson, combined literature review with expert opinion to produce practical guidance for the use of cerebral microdialysis [Figure 6]. Experts recommended monitoring cerebral microdialysis in patients with or at risk of cerebral ischemia, hypoxia, energy failure, and glucose deprivation (Strong recommendation, low quality of evidence).(22)

TBI is a complex disease with substantial heterogeneity. ICP monitoring alone cannot detect all potential insults to the brain; ensuring adequate cerebral blood flow and oxygenation are important goals. Advanced neuromonitoring and assessment of cerebral autoregulation may be helpful in identifying a more individualized approach to treatment. Impaired cerebral oxygenation can occur in the face of normal ICP and CPP. Cerebrovascular pressure reactivity index (PRx) and cerebral blood flow (CBF)

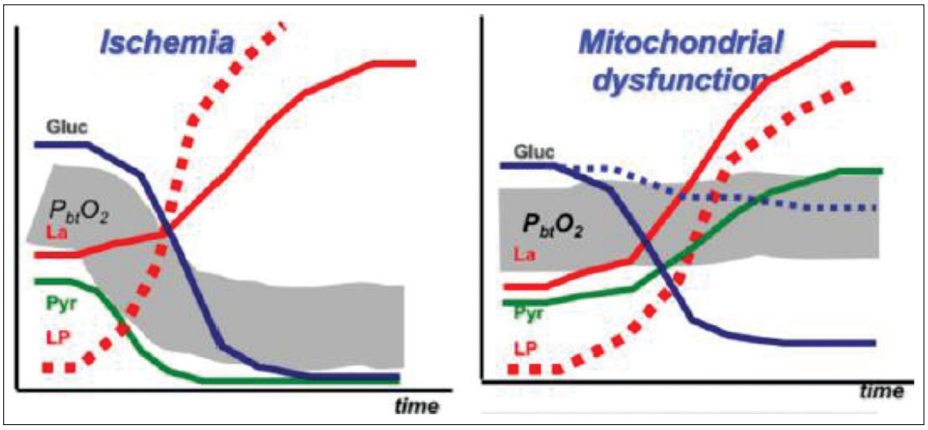

Figure 5 : Showing cerebral microdialysis based classification of ischemia and mitochondrial dysfunction in traumatic brain injury Low pyruvate levels in ischemia and high pyruvate levels in mitochondrial dysfunction patients as detected by cerebral micro dialysis. Lactate/pyruvate (LP) ratio is raised in both groups. Gluc- cerebral glucose, La Lactate, LPR Lactate/pyruvate ratio, brain tissue oxygenation, Pyr cerebral pyruvate levels

Table 2: Various Grade Level of Evidence on metabolic abnormalities detected on cerebral microdialysis are as below

Metabolic abnormality	Parameters	GRADE of evidence
Metabolic crisis	LPR >25	Moderate
Ischemia	LPR >25 and markers of anaerobic metabolism (low glucose, low pyruvate)	Low
Mitochondrial dysfunction	LPR >25 and normal substrate levels (e.g., pyruvate)	Low

Table 3: Pathological states on Cerebral Microdialysis with suggested targeted interventions

Abnormal state	ICP mmHg	PRx (autoregulation)	PbO2 mmHG	Brain Glucose mmol/L
Intracranial hypertension	>20	-	-	-
Delivery failure	<20	>0.3	-	-
Diffusion barrier	<20	<0.3	<15	-
Neuroglycopenia	<20	<0.3	>15	<1.0
Mitochondrial dysfunction	<20	<0.3	>15	<1.0

	Consensus Statement 2015	Recommendation	Evidence
Methodology of micro dialysis	20 kDa catheters for small molecules and 100 kDA for large molecules.	Flow rate of 0.3 µL/min with hourly sampling recommended.	
Reference values	**Glucose (Tier 1 intervention)**	**<0.2 mmol/L <0.8 mmol/L**	**Low glucose associated with poor outcome.**
	L/P ratio (Tier 1 intervention)	**>40 >25**	**High L/P ratio associated with poor outcome.**
	Lactate	>4 mmol/L	L/P ratio better than lactate
	Glutamate (Tier 2)	Raised in ischemia	May be useful in prognostication.
	Glycerol(Tier 3)	Limited specificity	No definitive evidence.
Management guidelines	Glucose	Raise RBS if low. Treat perfusion, if low CPP.	
	L/P ratio	If ischemia, raise CPP. If low pbtO2 , improve oxygenation.	
Catheter placement	Focal injury	a) Ipsilateral b) In normal brain	**Multiple catheters may be used.**

Figure 6 : Consensus statement on Cerebral microdialysis based treatment recommendations

monitoring can assess autoregulation status, which may help determine patient-specific CPP and ICP goals [Table 2].

Multiple studies have demonstrated an association between low brain tissue oxygen tension (PbtO2 ≤ 15 mm Hg) and episodes of jugular venous oxygen desaturation (SJvO2 ≤ 50 %) with poor outcome in TBI. Importantly, brain tissue hypoxia can occur even when ICP and CPP are normal.

Cerebral Oxygenation Monitoring

Brain tissue oxygen (pbO2) monitoring provides insights into the oxygen tension in the brain parenchyma. 4th BTF guidelines (2016) highlight the utility of pbO2 monitoring in assessing the adequacy of cerebral oxygen delivery (14). This modality is particularly beneficial in cases where traditional measures may not fully capture the complex interplay of oxygen supply and demand. Continuous pbO2 monitoring aids in identifying episodes of cerebral hypoxia, enabling clinicians to adjust therapeutic interventions promptly. The integration of pbO2 data with other monitoring parameters contributes to a more comprehensive understanding of the patient's cerebral physiology. Brain tissue oxygen tension monitoring (PbtO2) , Jugular bulb oxygen saturation and Near Infrared Spectroscopy (NIRS) techniques are used for cerebral oxygenation monitoring in advanced neuro intensive units across the globe.

A recently completed Phase II prospective randomized clinical trial investigating PbtO2-based management of severe TBI compared treatment guided by ICP alone to treatment guided by both ICP and PbtO2 (BOOST, NCT00974259). The ICP+PbtO2 management group had statistically significant decreased duration and severity of brain hypoxia along with a 10% reduction in mortality and a trend toward reduced mortality and improved neurologic outcome at 6 months. This trial supports the value of advanced multimodality monitoring in TBI patients.

Currently Brain Oxygen Optimization in Severe traumatic brain injury (BOOST-3) , a comparative effectiveness multicentric randomized study of brain tissue oxygen and intracranial monitoring versus intracranial pressure alone is ongoing wherein 1094 patients (age > 14 yrs.) with severe TBI are being enrolled, this study will assess whether the addition of brain tissue oxygenation (PbtO2) monitor will prevent more secondary injuries that will translate into improve functional outcome. The study will involve monitoring for 5 days, keeping PbO2 > 20 mm Hg and ICP < 22mg Hg.(23)

Pediatric patients (< 18 yrs of age) with severe TBI and ICP + PbO2 monitoring and treatment were noted to have better outcomes than those who undergo ICP only monitoring and treatment in a study on 39 children. Increased number of cerebral hypoxic

episodes coorelates with less favorable functional outcomes, longer hospital length of stay and longer duration of mechanical ventilator support.(24)

Brain tissue Oxygen tension monitoring (PbtO2)

PbtO2 monitors use either a Clark electrode (Licox) or an oxygen quenching method to determine the partial pressure of oxygen in brain parenchyma. The probe is usually placed in the unaffected hemisphere as this allows detection of cerebral hypoxemia that is addressable by treatment. "Normal" PbtO2 values are usually >23 +/− 7 mmHg. PbtO2 values of <15 mmHg are associated with an increasing lactate/glucose ratio and increasing glycerol levels, both of which suggest cerebral metabolic stress.(25) The commonly used cutoff is >20 mm Hg following TBI, although the BTF guidelines do not provide a specific cut off value for PbtO2. The presence of cerebral hypoxemia is detrimental to outcome with poor outcomes in patients showing longer duration, severity and non-responsiveness of cerebral hypoxemia.(26)

Low CPP and PbtO2 should be avoided in all severe TBI cases for improved outcomes . Targeting CPP to minimize PRx index (i.e. optimize autoregulation) may be beneficial in ameliorating metabolic dysfunction (noted with cerebral microdialysis), this provides a rationale for multimodality monitoring in TBI cases. Observational studies show that low PbtO2, elevated Lactate to pyruvate ration and low brain tissue glucose concentrations are associated with a poor outcome after severe TBI.

A fundamental limitation of both Cerebral microdialysis and PbtO2 monitoring is their focal nature, which only indirectly measures a heterogeneous and diffuse pathophysiology.

Jugular bulb oxygen saturation

Jugular venous oxygen saturation (SjvO2) monitoring is a technique to estimate the global balance between cerebral oxygen supply and it's requirement. Unused oxygen is detected in the internal jugular vein as it enters into the systemic circulation from the brain and this helps to measure the balance between the cerebral blood flow and the cerebral oxygen requirement. The normal value varies between 55-75%.

SjvO2 < 55% - Possible causes include hyperventilation, reduced CPP and vasospasm. Or an increased metabolic demand (seizures, injury).

SjvO2 >75% - possible causes could be due to hyperaemia, increased microvascular shunting due to TBI.

Recurrent low SjvO2 is associated with poor neurological outcome. Studies suggest that SjvO2 should be in optimal range for good outcome.(27-29)

Current BTF guidelines- SjvO2<50% may be threshold to avoid in order to reduce mortality [Level III recommendation].(14)

Near-Infrared Spectroscopy (NIRS)

Near-infrared (NIR) refers to wavelengths between 700 and 1000 nm, a range in which absorption by water molecules is low enough to allow signal transmission through tissues. NIRS measures the concentration of oxygenated Hb and deoxygenated Hb to monitor the degree of cerebral oxygenation. Early machines employed a continuous wave technology that was very sensitive to changes in extra cerebral oxygenation. However, newer techniques refine the recorded values by removing extra cranial circulation and give better idea of cerebral oxygenation using Total Hb index (THI) and Total Oxygen index (TOI).

NIRS has emerged as a valuable tool for monitoring cerebral oxygenation in TBI cases. This non-invasive technology provides real-time information about regional cerebral oxygen saturation (rSO2).

NIRS-detected episodes of cerebral hypoxia have been noted to be associated with increased mortality and poor functional outcome in some studies. There is an inverse

relationship between regional brain oxygen saturation (rSO2) and ICP and good concordance between rSO2 and brain tissue oxygen tension measured invasively based on many studies.(30)

The BTF acknowledges the potential benefits of NIRS in guiding treatment strategies and recommends its use in conjunction with other monitoring modalities.

The continuous nature of NIRS monitoring is particularly advantageous in detecting fluctuations in oxygenation levels, allowing for timely intervention to prevent secondary injury. While cerebral NIRS is a promising technique owing to its noninvasive nature, it has not yet demonstrated an ability to surpass invasive methods of brain tissue oxygenation due to concerns about its accuracy.

Pupillometry in TBI monitoring

Pupillometry, the measurement of pupil size and reactivity, is a valuable tool in TBI monitoring. Changes in pupillary response can provide early indications of neurological deterioration. Optical pupillometry is a non-invasive, objective monitoring method, measuring parameters of pupillary response and displaying a scalar value - a neurological pupil index (NPi). An impaired response on NPi has been tentatively correlated with ICP, through analysis of mean/peak NPi and ICP readings.

Studies suggest that NPi trend monitoring is a valuable complementary tool to ICP monitoring. NPi correlated well with sustained elevations of ICP>20 mm hg, variation in ICP with hyperosmolar agents was associated with normalization of NPi and the cumulative duration of abnormal NPi was significantly associated with poor outcome at 6 months.(31)

Continuous pupillometry offers an objective and quantifiable measure of neurological status, aiding healthcare professionals in identifying subtle changes that may precede more overt clinical signs. The non-invasive nature of pupillometry makes it a valuable addition to the monitoring armamentarium in TBI. The NPi appears a valuable prognostic tool, given the observed association between cumulative NPi abnormalities and 6-month neurological outcome.(31)

Integrating Monitoring Modalities (Multimodality Monitoring)

The synergy of ICP monitoring, cerebral micro-dialysis, pbO2, NIRS, pupillometry monitoring offers a multi-faceted approach to the management of traumatic brain injuries. The guidelines from the BTF underscore the importance of individualizing patient care, recognizing that a combination of monitoring modalities provides a more nuanced understanding of the dynamic nature of brain injury. By integrating real-time data from these monitoring tools, healthcare professionals can tailor interventions to the specific needs of each patient. This personalized approach enhances the precision of therapeutic strategies, minimizing the risk of secondary injuries and optimizing the chances of a favorable outcome.

Cerebral pressure autoregulation is the brain's intrinsic ability to maintain constant CBF over a range of systemic blood pressures. This mechanism protects against cerebral ischemia due to hypotension and against excessive flow that can lead to elevated ICP. Cerebral autoregulation can be assessed at the bedside in the ICU with cerebrovascular pressure reactivity index (PRx) monitoring, CBF monitoring, and Transcranial Doppler (TCD) ultrasonography monitoring. The PRx is quantified as the slope of the regression line relating MAP and ICP and can be used to establish patient-specific CPP thresholds. For patients with impaired cerebral autoregulation (PRx slope > 0.13), a lower CPP (50 – 60 mm Hg) should be considered as an option for treatment. Patients with intact autoregulation (PRx slope < 0.13) may benefit from a higher CPP (50 – 60 mm Hg). When CBF is monitored directly, autoregulation status can be assessed with a hemodynamic challenge. In patients with intact autoregulation, CBF will change minimally in response to an increase in MAP. Conversely, CBF will rise with increasing MAP in patients with impaired autoregulation. Once determined, autoregulation status can be used to set CPP goals as described above. In a similar fashion, TCD ultrasonography and hemodynamic challenge can also be used to assess autoregulation in TBI patients.(32-41)

Future Directions

The future of neurosurgical monitoring holds promise as technology advances. Trials such as CENTER TBI and ADAPT(42-44) pave the way for a more sophisticated understanding of TBI, while ongoing research may introduce Artificial Intelligence (AI) for real-time data analysis and decision support in the neurosurgical realm. Continued exploration of additional monitoring modalities and biomarkers promises to elevate the neurosurgical toolkit. The neurosurgical commitment to refining guidelines and incorporating cutting-edge monitoring technologies reflects an unwavering dedication to advancing patient care and outcomes in the complex landscape of traumatic brain injuries.

Audit of various surgical practices in TBI care

Surgical practice in TBI is not new to medical field. Trepanation which is making hole through the skull up to the dura, is one of the earliest Neurosurgical procedures. Evidence of its use is found at the beginning of Neolithic period (10,000 BC) in Mesoamerica, South America, Africa, Asia and Europe.(45) The trace of trepanations is also evident in India as early as 2350-2050 BC in Kashmir Valley region.(19) Egyptian papyri is known to be the first written evidence of brain injury about 5000 years ago, but the first written systemic approach to head injury has been attributed to Hippocrates, the great Greek physician during the 5th century BC.(46) Sushruta, who was considered as father of surgery, described cranial surgical procedures and trepanation about 100 years earlier to Hippocrates.(47) In 1896 the first scientific reference and description of an hemicraniectomy was reported by Charles Adrian Marcotte. Kocher in 1901 first reported the utilization of large decompressive craniectomy following TBI.(48)

A large traumatic hematoma should be evacuated before neurological deterioration develops, irrespective of the GCS . TBI patients presenting to the ED in coma should be taken to surgery immediately upon arrival if a large hematoma is identified as the cause of the coma. Decompressive craniectomy is effective in controlling intracranial pressure, but uncertainty exists as to its potential to improve outcome.(49,50)

Timely evacuation of an expanding traumatic intracranial haematoma in a patient with deteriorating consciousness is lifesaving. However, many patients present with a stable low-level or high-level of consciousness. Uncertainty exists, particularly in patients with an acute subdural haematoma or a traumatic intracerebral haematoma, on indications and timing of surgery, reflected in large practice variations. CENTRE- TBI data showed that where surgical equipoise exists, early evacuation of acute subdural haematoma does not lead to a better outcome compared with a strategy favoring initial conservative treatment. Conversely, conservative management in patients with mild TBI and in those with a smaller traumatic intracerebral haematoma are associated with better outcomes.

The effectiveness of a strategy preferring acute surgical evacuation with one preferring initial conservative treatment in acute subdural haematoma was recently studied in 4559 patients (31% of them had acute subdural hematomas) by CENTRE TBI investigators . It was found that a strategy preferring an aggressive approach of acute surgical evacuation over initial conservative treatment was not associated with better functional outcome. Patients with acute subdural haematoma for whom a neurosurgeon sees no clear superiority for acute surgery over conservative treatment, initial conservative treatment might be considered.(51)

Depressed skull fractures are commonly elevated if the depression is greater than the depth of the adjacent inner table, especially if located in a cosmetically important area like the forehead. Open depressed fractures are best treated surgically to prevent infection, but nonoperative management may be attempted in selected cases, limited to those without dural laceration, gross contamination or evidence of infection, or injury to the frontal sinus. In general, a depressed skull fracture over the sagittal sinus should not be treated surgically because of the high risk of uncontrollable hemorrhage.

Surgical management options for various intracranial pathologies after TBI

Surgery for TBI patients is most commonly performed to evacuate epidural hematomas (EDH), subdural hematomas (SDH), cerebral contusions, or intracerebral hematomas

(ICH) that are large enough to cause significant mass effect on the brain. Surgical evacuation of these hematomas should be performed as soon as possible.

TBI patients presenting to the ED in a coma should be taken to surgery immediately upon arrival if a large hematoma is identified as the cause of the coma.

Admitted patients who undergo neurological deterioration from delayed development or enlargement of a hematoma require prompt surgical evacuation to prevent further neurological worsening.

A formal craniotomy is necessary to perform adequate resection; there is no role for attempted burr-hole drainage of these solid clots.

Evidence-based guidelines for surgery have been compiled, but the paucity of high-quality randomized studies in this area limits the strength of recommendations.

In general, CT evidence of raised ICP, such as midline shift of ≥5 mm and/or compression of the basal cisterns is an indication for surgical evacuation of a traumatic mass lesion.

Even if a patient has a relatively high GCS score, a large traumatic hematoma should be evacuated before neurological deterioration develops from enlargement of the hematoma or swelling of the underlying brain. A lower threshold for surgical intervention may apply to posterior fossa lesions.

The spectrum of head injury may vary from concussion to large intraparenchymal hematomas and based on this its management also changes from nonsurgical to surgical options. Based on Marshall's CT grading, size of intraparenchymal hematoma >25 ml is considered to be a generalized criteria for surgical evacuation.(52) But volume is not the sole responsible factor for subjecting a patient to surgical evacuation, many other factors like neurological status of the patient, cerebral edema, basal cistern effacement, etc. are also the deciding factors.(53) Apart from intraparenchymal hematoma (ICH), depressed fracture, extradural hematoma (EDH), subdural hematoma (SDH) are also considered to be potential surgical identities.

Management of depressed skull fractures

There is uniformity is management of simple linear calvarial fracture, with most managed conservatively. Depressed fracture can be managed conservatively, if there is no evidence of dural penetration, without significant intracerebral hematoma, without (i). involving air sinuses, (ii). evidence of gross contamination and (iii). gross cosmetic deformity. Closed (simple) depressed fracture can be managed conservatively. Depressed fracture over major dural venous sinus is dangerous to elevate, especially, if patient is neurologically intact. May be best managed by conservatively.

Compound open depressed fractures are more prone to get infected and are best managed surgically, preferably within 24 hrs. of trauma . The primary aim of surgery in compound depressed fractures is to debride the infected skin margins, elevation of the bone segments, repair of dural lacerations and thorough lavage of the infected area. Antibiotics should be used for all compound depressed fracture (level III evidence).(54)

Basal skull fractures are distinct identity which leads to CSF otorrhea or rhinorrhea by dural shearing and are difficult to manage. Primarily, these types of fractures are managed conservatively in the early course, but if CSF leak does not stop spontaneously, then may be treated by surgically by plugging the defect by various skull base approaches.

Management of Extradural Hematoma (EDH)

A supratentorial EDH of volume more than 30 ml should be evacuated irrespective of GCS at presentation. If EDH volume is less than 30 ml with thickness of <15mm and midline shift of <5 mm in patients with GCS>8 without neurological deficit, can be managed by conservative methods. Patient with an acute EDH and GCS<9 with anisocoria should be treated surgically as soon as possible (Level III evidence).

Technically, hematoma evacuation and prevention of reaccumulation are the primary goals of surgery, which can be achieved by proper dealing of the bleeding sources (dura, bone margins or venous sinus) and approximation of dura with the bone margins by multiple peripheral hitches and central tenting sutures.(55,56)

Infratentorial EDH consist of approximately 5% of all EDH cases. The chances of dural sinus injury are high in this type of EDH and associated with high mortality because of mass effect to brain stem as well as obstructive hydrocephalus due to blockage of the CSF flow resulting from compression over the fourth ventricle.(57,58)

Management of Subdural Hematomas (SDH)

Acute SDH, resulting due to torn bridging veins, are usually associated with poor outcomes due to associated brain swellings and its combinations with other types of injuries (diffuse axonal injury, intraparenchymal contusions). Acute SDH with thickness of more than 10mm or midline shift (MLS) of more than 5mm should be evacuated surgically irrespective of GCS. If SDH thickness is <10mm and MLS <5mm then conservative management with or without ICP monitoring is recommended.(14,59) Decompressive Craniectomy (DC) in TBI is mostly done in cases of acute SDH.

In older times, tentorial sectioning (which released CSF from basal cisterns) was standard of care as an adjunct to decompressive craniectomy in acute TBI cases (acute SDH, traumatic brain contusions).

Conservative treatment of acute SDH may result in clinical improvement over couple of weeks in neurologically stable patients with insignificant mass lesions. Many such patients need evacuation of chronic form of hematoma (chronic SDH) by craniostomy (burr hole) when there is residual or further clinical deterioration after initial improvement. Use of twist-drill (without burr hole) and endoscopes (with burr hole) too have been used recently with satisfactory results.(60,61)

Management of intraparenchymal hematoma

There are no well accepted guidelines for intraparenchymal hematoma evacuation; however, decision of surgical evacuation is based on clinical findings, radiological grading, raised intracranial pressure (ICP) findings and surgeon preferences. Surgical procedures depend upon occurrence of cerebral edema, contusion and subarachnoid haemorrhage. Procedures may vary from DC to only hematoma evacuation by open or minimal invasive technique.(61) However, DC has changed the overall outcome of severe TBI and is one of the most commonly performed procedure nowadays, especially in situations with multiple diffuse contusions with or without acute SDH.

Decompressive Craniectomy , Decompressive Craniotomy in TBI

Decompressive Craniectomy (DC), in which a large bone flap is deliberately removed or not replaced, has witnessed a surge of popularity in recent years. DC is one of the most practiced method to counter the raised ICP following TBI. DC can be a primary procedure (combined with evacuation of a haematoma) or a secondary procedure (to decompress the brain).

Sometimes the flap is left off because massive cerebral swelling develops after evacuation of a hematoma, and at other times, the surgeon anticipates significant cerebral edema and pre-emptively leaves the bone flap off. In other cases, patients who would not normally undergo surgery may be taken to the operating room for DC if ICP begins to rise.

There is still uncertainty regarding the benefits of decompressive craniectomy, a procedure that aims to mitigate the effects of raised intracranial pressure by removing part of the skull.

A recent study casts doubt on the clinical benefit of a DC in patients with diffuse brain injury and raised ICP refractory to medical management. The randomized controlled DECRA trial demonstrated that although patients who received craniectomy achieved effective lowering of ICP, their neurologic outcomes at six months were worse than those

of patients randomized to maximal medical therapy. However, critics of this trial have highlighted unbalanced treatment groups, variability in medical treatments for the control group, high crossover rate to the surgical arm, and short-term follow-up (six months) as arguments against the conclusions of the study. The application of decompressive craniectomy for severe TBI remains a topic of lively debate.(62)

In LMICs (Low Middle Income countries) across many parts of the world, decompressive craniectomy is performed even more frequently than in HICs (High Income countries) . In LMICs, use of decompressive craniectomy appears to be often determined by resource limitations.

RESCUE-ASDH, a multicenter randomized trial comparing craniotomy versus decompressive craniectomy for patients undergoing evacuation of a traumatic acute subdural haematoma did not show benefit of craniectomy over craniotomy in the event of equipoise to replace or not to replace bone flap at the end of surgery, however a larger proportion of patients in craniotomy group had wound related complications.(63,64)

BTF update on guidelines of decompressive craniectomy

BTF - 2016 guidelines recommended DC in the management of severe traumatic brain injury [Level II A evidence]. Following reconsideration and adjudication of the evidence provided by RESCUEicp(33) as well as DECRA's(34) recently published 12-month outcome data, BTF published updated recommendation in 2020.(65)it was known that the results of the RESCUEicp (Trial of Decompressive Craniectomy for Traumatic Intracranial Hypertension BTF- 2020 retained its recommendation that a large DC not less than 12×15cm or 15cm diameter is recommended over a small fronto-temporo-parietal DC to reduce mortality and to improve neurological outcomes in patient with severe TBI. There are three new level II-A recommendations, which are based on early and late refractory ICP elevations similar to published in the reference studies.

Secondary DC performed for the late refractory ICP elevation is recommended to improve mortality and favorable outcomes, but it is not recommended for early refractory ICP elevation to improve mortality and favorable outcomes. BTF also recommends secondary DC as a treatment to control ICP, whether it is done for early or late refractory ICP elevation, but its effect on favorable outcome is uncertain.

There is level II-A for DC in severe TBI, though, it is associated with multiple issues like post-traumatic hydrocephalus, bone flap resorption, shrunken flap syndrome, etc. Floating or hinged cranioplasty appears to be a promising alternative in select cases to obviate post bone flap removal complications (level IV evidence).(66,67)

With the evolution of a para-vascular pathways which facilitates the CSF flow through the brain parenchyma, new concept of ICP control by opening of basal cisterns (cisternostomy) is suggested by few authors as an alternative to decompressive craniectomy in severe TBI cases.(68,69). While initial results are shown to be promising in few case reports, more experience and more RCT data is needed before recommending cisternostomy as standard of care to all TBI cases. In senior author personal experience and based on an ongoing prospective study, there has been no difference in outcomes noted with or without cisternostomy in severe TBI cases. There is no recommendation on cisternostomy in TBI in recently published BTF guidelines (2016) or NTSI society Indian guidelines (2017).(14, 21)

Vieira *et al*. suggested that without water-tight dural closure, DC can be performed with lesser rate of surgical complications like CSF leak, wound infection etc.(70). Rapid closure DC (as it is called) can be performed without dural closure and without compromising outcomes.(71) Procedure time is statistically reduced, but there is no statistically significant difference in CSF leak or post traumatic hydrocephalus. Exposed brain tissue is loosely covered with diverse materials (surgical, gelfoam, etc.). Shortening of duration of surgery enhances safety , might reduce blood loss and rates of infection. Emerging data suggests that RCDC is feasible, safe, time and cost sparing, and is associated with complication rate comparable/lower than water tight duraplasty [Figure 7]. Cranioplasty after RCDC is also feasible, fast and safe.

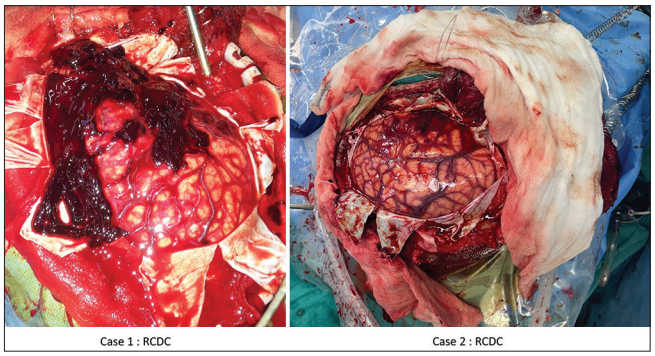

Figure 7 : Rapid closure decompressive craniectomy Case 1 and Case 2 . Both cases after hematoma evacuation, dura was left open and covered with large piece of surgical and bone flap was removed

Complications of DC among elderly patients are used to be high and associated with bad outcome, hence in selected patient's endoscopic hematoma evacuation may be a feasible option.(72-74)

Ballistic brain injuries

In the present era of war, where the spectrum of terrorism is reaching upto a great extent, military bombing injuries are not common among civilian but improvised explosive devices (homemade bombs) are occasionally use by terrorist. These explosive cause primary (due to high pressure blast waves) as well as secondary (caused by shrapnel or environmental debris) damage to victims. Depressed fracture, foreign body, diffuse cerebral edema, contusions are common after effects of blast brain injury. Damage control neurosurgery is mostly performed by neurosurgeons in these settings but even with best critical care services in these locations, the mortality is still high. The best way to improve outcome is triaging and resources management at the time of disaster.(75,76)

Recent advances in surgical care in TBI

Better imaging techniques like high resolution CT and TBI specific sequences of MRI are capable of highlighting lesions which were missed earlier. Prognostication too has improved after advancements in imaging techniques. Powered equipments like drills and bone scalpels have reduced surgical times. Localization equipments like neuronavigation, intraoperative ultrasonography (USG), mixed reality equipments and robots though not routinely used for TBI, but are available for clinical use.(77)

Advancements in information technology (IT) has enabled us to use softwares like RadiAnt or Horos DICOM viewers for virtual visualization of lesions of brain exactly like surgical fields. Interestingly, virtual surgeries too can be done using these softwares.(77,78) especially for an inexperienced neurosurgeon. We share our experience of applications of volume rendering (VR. Three-Dimensional (3-D) printing has improved to give high resolution physical model of brain or cranium and also used reconstruction of cranial defects (cranioplasty) due to TBI or after DC.

Secondary procedures in TBI patients

There are no large prospective studies defining the optimal timing of secondary extracranial surgery in patients with severe TBI. In making such decisions, close communication among the treating specialties is paramount. To avoid secondary brain injury, close monitoring during anesthesia is required to avoid hypotension, hypoxia, and hypo- or hypercarbia. While a single episode of hypotension doubles mortality, the combination of hypotension with hypoxia is associated with up to 75% mortality. If ICP is being monitored, CPP must be maintained at $\geq$ 60mm Hg.

Timing of orthopedic procedures (primarily long bone repair) does not appear to have an overall effect on outcomes in patients with severe TBI. After initial stabilization, damage control orthopedics with early external fixation is favored, with delayed definitive treatment. This minimizes the "second hit" neurological phenomenon, triggered by the inflammatory response, hypotension, hypoxia, hyper- or hypocarbia, and intracranial hypertension, all of which are common occurrences with orthopedic procedures. Timing

of spine fracture-dislocation surgery should depend on spine stability and the need for emergent spinal decompression in patients with spinal cord injury.

In patients with intractable intracranial hypertension, consideration should be given to delaying trips to the operating room for non- intracranial procedures unless life-saving procedures are required. Open laparotomy or open thoracotomy should be performed when needed, with adherence to the same general principles of avoiding secondary brain injury, as noted above. Laparoscopy should generally be avoided, especially early on, because it raises intra-abdominal pressure and also induces hypercarbia. The contribution of hypercarbia to long-term adverse neurologic outcomes is debatable, however. Routine ICU procedures, e.g., tracheostomy and percutaneous endoscopic gastrostomy may be performed once the patient's condition has stabilized.

If patients with TBI require orthopedic operations, these should ideally be delayed 24 to 48 hours for initial stabilization of intracranial hypertension. Close monitoring is required during general anesthesia to avoid high ICP, hypotension, hypoxia, and hypo- or hypercarbia. Intravenous anesthesia is preferable for severe TBI patients. Regional anesthetic techniques should be avoided in patients with intracranial hypertension.(79-85)

Audit of Intensive Care in Traumatic Brain Injuries

Current guidelines for the medical management of TBI emphasize prevention of hypoxia and hypotension. Patients with severe TBI, need optimization of cardiorespiratory physiology, control of ICP and maintenance of cerebral perfusion pressure (CPP). Initial ICU management comprises usage of sedation, mannitol/hypertonic saline , limited hyperventilation, drainage of cerebrospinal fluid, and temperature control.

Dedicated ICU care is a key element of managing TBI and suspected intracranial hypertension (SICH) in settings with limited resources as shown by BEST TRIP trial results.(86) In settings with few resources, the management more based on physician input in ICU care, more frequent clinical examinations and CT scanning are utilized . This greater physician involvement reinforces the crucial value of on-site intensivists in non-monitored TBI care in LMICs. Preliminary analysis of the findings of Consensus-Revised ICE (CREVICE) protocol showed that protocolized care is superior to non-protocolized care.(87,88) CT imaging is central to the CREVICE approach. Non-invasive methods of estimating intracranial pressure, such as measurement of optic nerve sheath diameter could help to guide decisions to initiate, escalate, and wean treatment for suspected intracranial hypertension, possibly decreasing the overall morbidity and mortality.Even if the standard in high-resource settings is monitoring intracranial pressure in severe cases, evidence is accumulating that attentive teams of intensivists and neurosurgeons can achieve a reasonably good outcome from severe TBI in low-resources settings using institutional protocols that include imaging and clinical examination alone (e.g., ICE and CREVICE). However, good post-acute care would appear essential to consolidate these benefits and reduce the inefficiencies of non-monitored management (e.g., overtreatment, increased length of stay, more decompressions, etc.).

As per CREVICE protocol, intracranial hypertension was suspected and treatment recommended in the presence of one of the following major or two of the following minor criteria:

Major criteria : Compressed cisterns , Midline shift > 5 mm, Non evacuated mass lesion.

Minor criteria : Glasgow coma sum motor score of 4 or less, Pupillary asymmetry, Abnormal pupillary reactivity, Basal cisterns patent with midline shift 0-5 mm or a high density or mixed density lesion of 25cm^3 or less, or both.

Consensus derived matrix for de-escalation of therapy in SICH based on most recent Marshall CT scan classification and clinical status exam (GCS motor score and pupillary exam) in patients who are stable after 1,2,3 or > 3 days was proposed by Chesnut *et al* which is quite handy and a useful tool for managing TBI in the absence of quantitative ICP monitoring tools, based on imaging and clinical examination alone.(87,88) One

may also consider using noninvasive tools viz Optic nerve sheath diameter for initiating/escalating/wean off treatment for SICH.

Ventilatory management and tracheostomy in ICU for TBI cases : CENTER-TBI showed substantial between-center variability in $PaCO_2$ concentrations, two thirds of patients without intracranial hypertension had $PaCO_2$ concentrations below 35 mm Hg for most of the time.(6) Hyperventilation might be indicated in patients with raised intracranial pressure due to vasodilation and hyperaemia but not in patients with raised intracranial pressure from other causes. CENTER-TBI showed that in-hospital intubation had a significant beneficial effect on outcome in patients with a GCS of 10 or lower. 20% of mechanically ventilated patients developed ventilator-associated pneumonia, which prolonged ICU stay but did not affect TBI outcome. Late tracheostomy (undertaken >7 days post injury) was associated with a longer mean ICU stay and greater odds of a worse outcome.(6)

Fluid and hemodynamic management in ICU for TBI cases : Hemodynamic management in TBI usually depends on cerebral perfusion pressure targets, and includes fluid therapy and vasoactive drugs, neither of which are addressed in current guidelines. CENTER-TBI showed that higher positive mean daily fluid balances were associated with worse clinical outcomes. Cardiac output monitoring (currently available in < 20% centres) is useful in restricted fluid administrations.

Venous thromboembolism prophylaxis in ICU for TBI cases : The use of pharmacological prophylaxis is moderately associated with more favorable outcomes.

Coagulopathy management in ICU for TBI cases: About 20% of patients with isolated TBI show abnormalities on conventional tests of hemostasis and these are associated with greater chance of mortality associated with intracranial hematomas, and worse functional outcomes, compared with patients with a normal coagulation profile. Current management remains largely based on conventional laboratory parameters, though advanced testing of platelet function, fibrinolysis, and viscoelastic testing can better characterize hemostatic defects in TBI patients.

Basic management strategies in ICU for TBI include

- Monitoring : Every 10 min (till stable) – 1 hourly (for 12 hrs.) and then 2hrly till 7 days post injury/discharge whichever is earlier
- Monitor
  - Systemic : ABC (PR, BP, Temp)
  - Neurologic : GCS, Pupils
  - Imaging monitoring : CT (at admission, 48 hrs., at discharge/ 5 days whichever is earlier)
- Limit secondary brain injury (Hypoxia, Hypotension, Temp, Glucose, Seizures)
- Optimize O2, BP, ICP and CPP. Maintain PaO2 > 100, SpO2> 95, PCo2 35-45, pH 7.35-7.45, CVP 10-12, Temp < 38 degree, MAP> 60, CPP >60, SBP >100, Glucose (80-180mg/dl), Serum Na 135-145INR < 1.4, Hb > 7g/dl, Platelet for insertion of monitors > 80,000/cc
- Manage causes of secondary brain insults (Temp, Glucose, Seizures. SCI).

Response to deterioration of TBI cases in ICU

- Report immediately
- Hyperventilate briefly, Bolus mannitol/HS
- Rapid decision (Rpt CT/ Referral/Transfer to ICU/OR)
- ICU monitoring : Ventilated (EtCo2, SaO2, ABP, CVP (PPV),ECG
- ICP monitoring : If available/ USG ONSD?/TCD?
  - GCS 3-8 , Abnormal CT, Salvageable patient,
  - Normal CT (Age> 40, M2,SBP< 90).

The recommended "3-tiered" approach to ICP management utilizes various treatments to target different mechanisms. Higher tiers reflect more intensive management that is associated with increased complications. Failure to control ICP /CPP within one tier should prompt rapid progression to the next tiers treatment option. Repeat CT imaging and neurological examination should be considered to rule out the development of

surgical lesion and guide management. Because there is often no single pathophysiological pathway of ICP elevation, management is complex. Elevated ICP can be related to a variety of mechanisms, including: edema (cellular, extracellular), cerebral venous outflow obstruction, hyperemia (loss of autoregulation, vasodilation), mass effect (expanding hematoma), and disturbances in CSF circulation. We have recommended a "tiered" approach to ICP management that utilizes various treatments to target different mechanisms. The higher tiers reflect more intensive management that is also associated with increased complications.

Treatment options for isolated ICP increase include.

Tier I (within 15 min of abnormality)

- Adjust head of bed to lower ICP (30 -45 degree) to improve cerebral venous outflow (Reverse Trendelenburg)
- Ensure temperature < 38 degree C
- Titrate analgesic/sedatives (short acting agents e.g. Propofol, fentanyl, midazolam) in intubated patients
- CSF drainage (if EVD is available) performed intermittently
- Optimise CPP to max 70mm Hg with fluid boluses or vasopressors as clinically appropriate (CPP targets to be adjusted as per cerebral autoregulation)
- Adjust ventilator for a target PaCo2 of 35-40 mm Hg (target pH of 7.35-7.45)
- Low dose mannitol (0.25-1 g/kg) given in 4-6 hourly intervals. Mannitol may be withheld in hypovolemic patients
- Low dose (Hypertonic saline HTS 3%) at 8 hourly intervals/ continuous infusion to maintain Na < 160 meq/L (250ml over 30 min). Serum Na and osmolarity should be measured every 6 hrly .
- Initiate of titrate antiepileptic drugs
- Cerebral autoregulation should be assessed (with advanced neuromonitoring) . If the patient is not autoregulating, the CPP goal should be lowered (to no less than 50mmHg) to reduce ICP – additional neuromonitoring (PbO2, SjVO2, CBF) may help determine optimal CPP.

Tier 2 (within 60 min if tier 1 therapies are ineffective) : If ICP > 20-25 mm Hg

- Repeat CT scan head/neurological examination to rule out development of a surgical mass lesion, treat surgically remediable lesions according to guidelines/institute protocols
- Adjust temperature to 35-36degree C , using active cooling measures
- Bolus neuromuscular blockade to determine effect. If effective, perfusion may be used. Weaned off rapidly upon clinical stabilisation.
- Optimise CPP : may increase CPP > 70 mm Hg with fluid boluses / vasopressors (there is potential for harm related to augmentation of CPP above 70mm Hg with vasopressors)
- Adjust ventilatory rate to target PaCo2 of 30-35 mmHg (target pH of 7.35-7.45) as long as brain hypoxia is not encountered
- High dose mannitol (1-1.5g/kg) or higher frequency of low dose mannitol (0.25-0.5g/kg) if osmolarity < 320
- High dose HTS bolus. May repeat if Na levels < 160 meq/L
- Repeat CT imaging and neurological examination should be considered to rule out development of a surgical mass lesion and to guide treatment.

Tier 3 therapies : If ICP remains > 20-25mmHg for over 60 min after Tier 2 therapies

- Adjust ventilatory rate for target PaCo2 of 30-35 mm Hg (target pH < 7.5)
- Neuromuscular paralysis via continuous infusion if there is a positive response to a bolus dose. The infusion should be titrated to maintain at least two twitches (out of a train of four) using a peripheral nerve stimulator. Adequate sedation must be utilised.
- Barbiturate or Propofol coma (as per local protocols) : initial bolus (if effective) followed by continuous infusion, rapidly weaned off upon clinical stabilisation. Decompressive craniectomy if tier 1 and tier 2 treatment are not sufficient or are limited by development of side effects of medical treatment. Hypotension is a frequent side effect of high dose therapy with these agents. Meticulous volume resuscitation should be ensured and infusion of vasopressors/inotropes may be required.

- Hypothermia (< 36C) is not currently recommended as an initial TBI treatment. It should be reserved as a rescue or salvage therapy after reasonable attempts at ICP control via the previous Tier 3 treatments have failed. Adjust temperature to 32-35degree C , using active cooling measures.

For those monitored with brain tissue oxygenation, treatment options for isolated PbtO2 < 20 mm Hg include.

Tier 1 : must begin within 15 min of abnormality
- Adjust head of bed
- Ensure temperature < 38 degree C
- Optimise hemodynamic to ensure adequate CBF and avoid diffusion gradient (correct hypovolemia, avoid hypervolemia)
- Optimise CPP upto 70 mm Hg
- PaO2 adjustments (obtain ABG first)
  - Pulmonary toilet with suctioning of secretions (no bronchoscopy)
  - Increase FiO2 to a maximum of 60%
  - Adjust PEEP by a maximum of 5cm H2O over baseline
- Adjust minute ventilation to achieve a PaCO2 of 38-42 mm Hg (target pH of 7.35-7.45). PaCo2 should be increased if pH < 7.35, PaCo2 shouldn't be lowered if pH > 7.45
- Initiate or titrate antiepileptic medications.

Tier 2 therapies for low PbO2 : initiate within 60 min if tier 1 therapies are ineffective.
- Increase sedation
- Decrease ICP < 15 mm Hg
- CSF drainage (if available)
- Neuromuscular blockade
- Optimise CPP
- PaO2 adjustment (obtain ABG first, increasing PaO2 > 150 mm Hg implies overtreatment) : Perform bronchoscopy for pulmonary toilet, increase FiO2 to a maximum of 100% (wean rapidly when clinically stable, decrease FiO2 by 5% every 30 min), adjust PEEP to increments of 3-5 cm H2O
- Adjust minute ventilation to increase PaCO2 to 40-45 mm HG (target Ph 7.35-7.45)
- Transfusion of red blood cells.

Tier 3 therapies for low PbO2 (< 20mmHg)
- Adjust minute ventilation to increase PaCo2 > 45 mm Hg
- Increase cardiac output with inotropes (e.g. dobutamine)
- Assess for vasospasm with Transcranial doppler
- Hyperventilate.

Pediatric patients with TBI : Transferring children with TBI to a pediatric trauma center leads to decreased morbidity and mortality. If this is not possible, they should be transported to an adult trauma center capable of treating pediatric patients. Pediatric TBI protocols should incorporate age appropriate physiologic parameters. Because children of different ages have differing blood pressure and ventilation parameters, it is important to maintain meticulous adherence to age appropriate parameters. Data from well-designed, controlled studies on acute management of TBI in the pediatric population are limited. With the exception of age appropriate parameters for blood pressure, the tiered approach for management of intracranial hypertension and operative management outlined in previous sections also apply to children.[89]

Elderly patients with TBI: Neurologic evaluation of the elderly patient with TBI can be complicated by pre-existing dementia, cognitive decline, or hearing/vision deficits; careful determination of pre-injury neurological baseline via family and caregivers is important. Anticoagulants and anti-platelets medications can exacerbate the sequelae of TBI; reversal of these medications, if feasible, is an important early management goal.

Older age is associated with higher mortality and worse functional outcomes following TBI. However, age, in isolation, should not be considered a valid reason for treatment limiting decisions. History obtained from the patient or family can be very helpful, as

comorbid conditions can profoundly affect the impact of a TBI on an elderly patient. In addition, medications that are frequently utilized in the elderly can exacerbate TBI (anticoagulants/anti-platelets) or confound evaluation. With the increasing use of Novel Oral Anticoagulants (NOAC) (for example, rivaroxaban/apixaban, dabigatran) the approaches to reversal are evolving. Case series have reported success with tranexamic acid and Factor Eight Inhibitor Bypass Activity (FEIBA)/Four factor PCC(Prothrombin citrate complex) concentrates to reverse dabigatran (with or without hemodialysis) and Prothrombin Complex Concentrate (PCC) for rivaroxaban/apixaban. It is suggested that each center develop its own protocol for rapid reversal of anticoagulants using local expertise.

Neurologic evaluation of the elderly patient with TBI can often be complicated by pre-existing dementia, cognitive decline, or hearing/vision deficits. Family and caregivers can be invaluable sources of information when trying to determine a neurologic "baseline." Determining the appropriate level of diagnostic evaluation is important. One study found that in elderly patients with mild head injury, 14% of patients had evidence of traumatic lesion on head CT, with 20% of those lesions requiring neurosurgical intervention. Therefore, the American College of Emergency Physicians recommends that a head CT be obtained in any patient age ≥ 65 years who presents with mild head injury.

There is a paucity of information related to acute management of intracranial hypertension resulting from TBI in the elderly. Age-related changes in intracranial space are known to lower ICP significantly, with a concomitant rise in CPP. Further, cerebral autoregulation and pressure reactivity indices are known to decrease over time. These changes can be complicated by comorbid conditions and medications that are more common in the elderly patient sustaining TBI. Well-studied recommendations for optimal CPP thresholds in the elderly are lacking.

It is clear that as age advances, the risks of mortality and poor functional outcome from TBI increase. This is true for all types of brain injury, but most striking with a GCS < 9. Despite this grim prognosis, 30% of elderly TBI patients with severe TBI can survive to leave the hospital. There is tremendous variability in the aggressiveness of medical care following traumatic brain injury. This likely is due to local, regional, and cultural differences in how care is provided. Many of those deaths occur early after brain injury and likely reflect early decisions to withdraw life-sustaining therapy. At this time, due to the lack of sufficient prognostic tools, it is difficult to determine which patients may go on to have a meaningful recovery. Arbitrary age thresholds for limitations of care should be avoided. Rather, a detailed discussion with the family and decision-makers should center around the severity of injury, comorbid conditions, and respect for a patient's previously expressed wishes.(90-97)

Outcome measures tools in TBI: Around 50% of patients with mild TBI presenting to hospital do not recover to pre-TBI levels of health and wellbeing by 6 months after injury. Outcome in women after TBI is poorer compared with men. There is little understanding of individual differences in recovery after TBI. Age-specific norms are absent for many outcome instruments. Post-traumatic stress disorder is noted in over 20% of patients after TBI.(6)

Long-Term Outcomes: Bio-psycho-socio-ecological model capture individual differences that can substantially affect the outcome trajectory after a traumatic brain injury, in some cases leading to a better outcome and in others leading to a worse than expected outcome. [B=biological (e.g., brain injury severity, host response, and genetics); P=psychological (e.g., coping skills and mental health); S=social (e.g., social support and employment); and E=ecological (e.g., health-care systems). BPSE=bio-psycho-socio-ecological](6).

Age, preinjury mental health, coping skills, and the influence of other life course events, such as litigation and access to health care, all these including acute injury characteristics, the type and extent of TBI pathology, concomitant polytrauma, affect TBI outcomes in the long run. Recovery is often slow or incomplete after mild TBI, hence timely multidimensional outcome evaluation and therapy is needed for TBI survivors.

When selecting an outcome measure, it is essential to begin by defining what needs to be measured: whether it's activity (limitation), impairment, or participation. The second

consideration is whether to assess capacity or actual performance, and this choice guides the type of assessment, such as laboratory testing, observation, or patient/proxy reports. It is also crucial to review each instrument for content, syllabus, and the necessary instructions for administration and scoring.(98)

There are various tools to assess different aspects of TBI outcomes, including impairment, functional limitations, activity levels, participation, and quality of life, enabling a comprehensive evaluation of the impact of TBI on individuals.

Traumatic brain injuries (TBI) cause both immediate and ongoing effects that result in lasting disabilities, increased long-term mortality, and a reduced lifespan. A single TBI or repeated injuries can lead to additional health issues such as permanent weakness of limbs, sensory symptoms , language problems, visual symptoms, seizures, sleep disorders, neurodegenerative diseases, hormonal imbalances, and psychiatric disorders.(99) A significant number of psychiatric conditions that frequently emerge following traumatic brain injury are linked to neuropsychological challenges. These conditions include behavior, mood, and cognitive functions, with memory, attention, and executive function often being affected like depression, anxiety disorders, adjustment disorders, and post-traumatic stress disorder.(100-102)

Memory plays a crucial role in our daily lives, and cognitive problems arising from traumatic brain injury (TBI) have severe consequences for those affected. Changes in the neural circuits related to memory function are a significant factor contributing to memory issues resulting from TBI.(103)

Few prognostic models have been developed for mild TBI. The best known are the CRASH (for GCS ≤14) and the UPFRONT models. Published models contain different predictors to those on moderate to severe TBI and include, besides age, GCS, extra-cranial injuries, alcohol intoxication, sex and gender, education, and pre-injury mental health. Injury severity, however, remained one of the strongest predictors of GOSE, showing that what injury does to the patient (e.g., injury severity) is also relevant for global outcome after mild TBI. However, validation of existing models for predicting the GOSE in CENTER-TBI data showed poor performance.

Outcome after TBI is multidimensional. Despite the value of the GOSE for describing functional outcome, it lacks detail in characterizing the heterogeneous nature of impairments, particularly those resulting from mild TBI. Global functional outcome measures should be complemented by domain-specific instruments, providing more comprehensive assessment. New models for predicting persisting post-concussion symptoms were developed in CENTER-TBI in a cohort of 1605 patients with complete outcome assessment at 6 months. The Core model explained only 4% of variance; extending this model with other variables available at admission increased the explanation of variance to 9%–12%, and adding information obtained at 2–3 weeks in a cohort of 476 patients, for whom this information was available, increased the explained variance from 6% to 37%.

Despite the broad acceptance of prognostic models in TBI research, they are not often used in clinical practice. This discrepancy might be partly due to the low precision of prediction in individual patients. Prognostic models have been developed and extensively validated for moderate and severe TBI. No well-validated models exist for mild TBI, nor do models exist that are applicable across all ranges of TBI severity. Robust prognostic models exist for moderate to severe TBI, but these only account for 35% of the variance in outcome. Performance of various prognostic models in mild TBI is enhanced by including information obtained at 2-3 weeks after injury. Incomplete recovery in mild TBI (GOSE <8) represents a prognostic endpoint with clinical, research, and societal relevance. Models for predicting post-concussion symptoms after mild TBI have used different approaches in defining post-concussion symptoms. Quality indicators developed for TBI are restricted to the ICU setting and are not yet ready for translation into clinical practice.

Impairment/Functional Limitation Masures

- Glasgow Coma Scale (GCS).

Activity/Activity Limitation Measures

- Barthel Index
- Functional Independence Measure.

Participation/Participation Restriction Measures

- Craig Handicap Assessment and Reporting Technique (CHART)
- Community Integration Questionnaire (CIQ).

Measures that Cross International Classification of Functioning (ICF) Domains

- Rancho Los Amigos Level of Cognitive Functioning Scale (Rancho or LCFS)
- Disability Rating Scale (DRS)
- Functional Status Examination (FSE)
- Glasgow Outcome Scale (GOS)
- Neuropsychological Assessment
- Quality of Life after Brain Injury (QOLIBRI).

In a study by Ruet's *et al*. only 15% of patients evaluated, did not suffer somatic or neurological disability. Most reported various complaints, including balance, motor weakness and headaches. Cognitive and behavioral problems were prevalent and often more disabling than somatic issues in the long-term. The likelihood of post-traumatic epilepsy increased with TBI severity, affecting about 10% of the sample. Nearly all stabilized after an 8-year follow-up.(104) Approximately 25% of the patients experienced clinically significant anxiety or depression, with the management of emotional issues being a common need. Assessing global outcomes using the GOSE score, around 28% of subjects had moderate to severe disability, requiring assistance for daily activities. Another 37% had an upper moderate disability, and the remaining 33% showed good recovery, with 16% in the upper range.

McCrea *et al*.'s prospective study found that at 2 weeks post-injury, 94% of severe TBI patients and 79% of moderate TBI patients had moderate to severe disability. About 80% needed assistance in daily life. By 12 months, half of the severe TBI group and three-quarters of the moderate TBI group could function independently for at least 8 hours per day. Many individuals with moderate TBI showed significant improvement between 2 weeks and 12 months, regaining independence. However, it's essential to note that a substantial proportion still experienced disabilities.(105)

In a retrospective study of 209 patients conducted by Rosyidi *et al*, they examined the outcomes of traumatic brain injury (TBI) patients based on CT scan results categorized according to the Marshall classification.

In the mild brain injury group, 32 patients (15.31%) achieved a good outcome. For the moderate brain injury group, 50 patients (23.92%) had good outcomes, while 40 patients (19.14%) experienced poor outcomes. In the severe brain injury group, 18 patients (8.61%) had good outcomes, but a significant 69 patients (33.01%) had poor outcomes.

Assessing these patients using the Glasgow Outcome Scale (GOS) revealed that 33 (15.79%) patients had fatal outcomes, 2 (0.96%) were in a vegetative state, 18 (8.61%) had severe disabilities, 23 (11%) exhibited moderate disabilities, and 133 (63.64%) achieved good recovery or had no disability.

Moreover, in addition to the GOS, a modified Rankin Scale (mRS) was used to assess TBI patient outcomes. The study included 100 patients with favorable outcomes and 109 patients with poor outcomes, based on the Marshall CT classification.

In Forslund *et al*.'s longitudinal study, most participants had stable Glasgow Outcome Scale Extended (GOSE) scores across different time intervals (1-2 years, 2-5 years, and 5-10 years). Approximately 57-67% showed no change. Between 1-2 years and 2-5 years,

21-22% saw improvement by one GOSE category, dropping to 7% in the 5-10 year period. Conversely, 9-13% had a one-category decrease between 1-2 years and 2-5 years, while this increased to 30% from 5-10 years.(106)

Overall, among 77 participants with GOSE data at both 1-year and 10-year follow-up, 77% experienced changes in their GOSE scores across all time points. Among those with consistent GOSE scores at 1 and 10 years, over a third showed changes. Specifically, in the 1-10 year follow-up, 26% increased by one to three GOSE categories, 36% remained unchanged, and 38% decreased by one to two categories.

Over a 15-year study, Corrigan *et al*. found that traumatic brain injuries (TBIs) and other brain injuries are not static but continue to change over time. Some individuals may experience a decline, influenced by various factors including aging, neurodegenerative processes, and lifestyle choices. To address this, a clinical approach is needed, treating brain injuries as chronic conditions. This approach involves identifying risk factors, early detection protocols, evidence-based treatments, and self-management training.(107)

Anderson *et al*.'s prospective study showed that, survivors of severe traumatic brain injury (sTBI) patients had increased long-term mortality compared to a matched population. Among severe TBI patients, those with a poor outcome at 1 year had even higher mortality rates. Older age was linked to worse outcomes, while higher Glasgow Outcome Scale (GOS) scores at 1 year correlated with better long-term results. Mental fatigue symptoms were a common lasting issue.(108)

Rehabilitation and Treatment: Individuals who undergo traumatic brain injury (TBI) may navigate various care paths and receive diverse interventions to aid in their recovery from the physical, cognitive, emotional, and behavioral consequences of the injury. Care options encompass inpatient and/or outpatient rehabilitation, nursing home care, and community-based services. While some may require lifelong care, others might benefit from periodic follow-ups to proactively address factors that could lead to deterioration.

Research supports both the effectiveness and cost-efficiency of TBI rehabilitation.(109,110) Despite the potential benefits of comprehensive interdisciplinary inpatient rehabilitation for many moderate to severe TBI patients, most are discharged to home or skilled nursing facilities, which may not offer intensive, specialized therapy or robust reassessment opportunities.

For individuals with less severe TBI, the recovery process can be impeded by limited access to TBI-informed outpatient physical and mental health services, as well as a lack of awareness or resources to support their return to work and school. It is important to recognize that TBI has repercussions not only for the injured person but also for their families and caregivers. Families require culturally sensitive information about their loved one's injury, expected recovery trajectory, and strategies to alleviate commonly reported burdens. In TBI rehabilitation interventions, a patient-centered approach is vital, considering not just the individual with the injury but also their family or caregivers [Table 4].

Summary of TBI audit: Robust trauma care networks, physician assisted retrieval to decrease secondary insults is urgently needed in lines with CENTRE TBI research results. Adherence to guidelines (Current Brain trauma foundation living guidelines or tailored national/center specific guidelines) improves outcomes – some guideline adherence is always better than no guidelines as most TBI guidelines (national/ global) are more or less similar. CHIRAG study showed that physicians in LMICs can achieve outcomes after TBI (with strict guideline adherence) similar to their counterparts in HICs.

Future of monitoring in ICU with ICP, CPP, Cerebral microdialysis and brain oxygenation tools is promising and cannot be ignored. It is recommended to use monitoring tools wherever available. For developing nations, less expensive ICP monitoring tools/ noninvasive tools (Optic nerve sheath diameter by USG) need to be researched .

However, is not a must to have MMM tools for all TBI cases especially in centres with limited resources. Currently available published/ongoing studies have shown comparable outcomes with treatment based on Imaging/ clinical examination treatment based on ICP/MMM monitoring.

Table 4: Summarizing the key findings from the mentioned studies

Study	Key Findings
1) Ruet's *et al*.	15% of patients evaluated didn't suffer somatic or neurological disability. - Various complaints reported, including balance, motor weakness, and headaches. - Cognitive and behavioral problems were more disabling than somatic issues. - Likelihood of post-traumatic epilepsy increased with TBI severity, affecting about 10%, with stabilization after an 8-year follow-up. - About 25% of patients experienced clinically significant anxiety or depression. - 28% had moderate to severe disability, 37% had upper moderate disability, and 33% showed good recovery, with 16% in the upper range using the GOSE score.
2) McCrea *et al*.	At 2 weeks post-injury, 94% of severe TBI patients and 79% of moderate TBI patients had moderate to severe disability. - About 80% needed assistance in daily life. - By 12 months, half of the severe TBI group and three-quarters of the moderate TBI group could function independently for at least 8 hours per day. - Significant improvement was observed in many individuals with moderate TBI between 2 weeks and 12 months.
3) Rosvidi *et al*.	Outcomes of TBI patients based on CT scan results categorized according to the Marshall classification. - Varying outcomes in different TBI severity groups. - Glasgow Outcome Scale (GOS) and modified Rankin Scale (mRS) used to assess TBI patient outcomes.
4) Forslund *et al*.	Participants generally had stable Glasgow Outcome Scale Extended (GOS) scores across different time intervals. - Some showed improvement in GOSE scores over time, while others had declines. - In the 1-10 year follow-up, 26% increased by one to three GOSE categories, 36% remained unchanged, and 38% decreased by one to two categories.
5) Corrigan *et al*.	Traumatic brain injuries (TBIs) and other brain injuries continue to change over time. - Some individuals may experience a decline influenced by various factors, including aging, neurodegenerative processes, and lifestyle choices. - A clinical approach is needed, treating brain injuries as chronic conditions, involving identifying risk factors, early detection protocols, evidence-based treatments, and self-management training.
6) Anderson *et al*.	Severe traumatic brain injury (sTBI) patients had increased long-term mortality compared to a matched population. - Poor outcome at 1 year was associated with higher mortality. - Older age correlated with worse outcomes, while higher Glasgow Outcome Scale (GOS) scores at 1 year correlated with better long-term results. - Mental fatigue symptoms were common in the long term.

Major reforms in policy decisions on prehospital care services are needed to reduce delay in time to surgery and improve outcomes. As level I trauma centres have better outcomes compared to level 2 trauma centres for TBI care, it is suggested to have more level 1 trauma centres for developing nations to improve outcomes.

Decompressive craniectomy is still used as an option in resource constrained ICU set ups/ centres. When a decision is taken to do DC in TBI, it is better to do it early and do it Big (size minimum 15x10 cm). Novel approaches to decompressive craniectomy need to be validated still.

Traumatic brain injuries (TBI) have long-term consequences, including cognitive impairments, behavioral issues, and increased health risks. Classifying TBI severity with tools like the Glasgow Coma Scale helps in initial assessment and treatment decisions. Various outcome measures assess different aspects of TBI impact. TBI outcomes is crucial for personalized care and resource allocation. Long-term outcomes vary, with some experiencing improvements in the first year but many facing lasting challenges. Rehabilitation is crucial, but access to comprehensive care can be limited. Recognizing the impact on families and caregivers is important, and a patient-centered approach is essential. High quality acute and post-acute care can achieve good outcomes in patients with severe TBI, infrastructure development is urgently needed for saving life and improving outcomes in most developing nations across the globe.

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Conflicts of interest

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